

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human LAG-3 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2A} Clone # 1009614
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human LAG-3 Met1-Leu450 Accession # P18627
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	Supplied 0.2 mg/mL 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	0.25-1 µg/10 ⁶ cells	Human PBMC treated with PHA and HEK293 Human Cell Line Transfected with Human 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), also known as CD223, is a member of the immunoglobulin superfamily (IgSF). The mature LAG-3 protein is a 496 amino acid (aa) membrane protein with a 421 aa extracellular region which contains four IgSF domains, a 21 aa transmembrane region and a 54 aa cytoplasmic region. LAG-3 and CD4 molecules share < 20% aa sequence homology but have a similar structure (1, 2). Both molecules bind to MHC class II. LAG-3 binds to MHC class II with higher affinity compared to CD4. Both LAG-3 and CD4 genes are located on the distal part of the short arm of chromosome 12.

LAG-3 is an activation-induced molecule, expressed on activated T cells and NK cells, but not on resting T cells. Studies using LAG-3^{-/-} mice have shown significant delay of T cell apoptosis following antigen stimulation and increased size of memory T cells pool following infection (3, 4). It also has been reported that anti-LAG-3 antibodies up-regulate T cell activation by blocking interaction of LAG-3 and MHC class II. The study has demonstrated that LAG-3 is selectively expressed on activated CD4⁺CD25⁺T_{Reg} cells and plays a role in their suppressive activity (5). This evidence indicated, unlike the interaction of CD4 with MHC class II that plays a positive role in T cell activation, LAG-3 binds to MHC class II and negatively regulates T cell activation through LAG-3 signaling. On the other hand, studies have shown that binding of LAG-3 to MHC class II molecules on antigen presenting cells induce maturation of dendritic cells and cytokine secretion by monocytes through MHC class II signal transduction (6). Taken together, LAG-3 may have two major functions, it negatively regulates T cells activation through LAG-3 signaling and stimulates antigen presenting cells which express MHC class II.

References:

1. Triebel, F. *et al.* (1990) J. Exp. Med. **171**:1393.
2. Baixeras, E. *et al.* (1992) J. Exp. Med. **176**:327.
3. Workman, C.J. and D.A. Vignali (2003) Eur. J. Immunol. **33**:970.
4. Workman, C.J. *et al.* (2004) J. Immunol. **172**:5450.
5. Huang, C.T. *et al.* (2004) Immunity **21**:503.
6. Andrae, S. *et al.* (2003) Blood **102**:2130.

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