

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human BTLA in direct ELISAs.
Source	Recombinant Monoclonal Rabbit IgG Clone # 2161A
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Mouse myeloma cell line, NS0-derived human BTLA Ile25-Ser150 Accession # AAP44003
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 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	Human PBMC

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

B- and T-lymphocyte attenuator (BTLA; CD272) is a 35 kDa type I transmembrane glycoprotein in the CD28 family of T cell co-stimulatory molecules (1-3). Mature human BTLA contains a 127 amino acid (aa) extracellular domain (ECD), a 21 aa transmembrane sequence, and a 111 aa cytoplasmic domain. The two ITIM motifs and three Tyr phosphorylation sites in the cytoplasmic tail transmit inhibitory signaling (4-5). The ECD of human BTLA shares 42% and 44% aa identity with that of mouse and rat BTLA, respectively. A splice variant lacking the transmembrane domain has been reported (6). Unlike other CD28 family members, the BTLA Ig domain in the ECD is of the I-type rather than V-type, and BTLA does not form homodimers (7). BTLA is also unusual in its interaction with the TNF superfamily member HVEM rather than with B7 family ligands (8). BTLA is expressed on T cells, B cells, macrophages, dendritic cells, and NK cells (9). Its expression is low in naïve T cells and increases during antigen-specific induction of anergy. In B cells, BTLA expression is highest in mature naïve cells (9). BTLA apparently limits T cell numbers, since its deletion results in overproduction of T cells, especially CD8⁺ memory T cells that are hyper-responsive to TCR cross-linking (10). Under the control of RORγt and IL-7, BTLA regulates the homeostasis and inflammatory responses of γδT cells (11). The binding of BTLA and HVEM does not preclude the concurrent binding of other HVEM ligands such as LIGHT or Lymphotoxin-α (12).

References:

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