

DESCRIPTION

Source Human embryonic kidney cell, HEK293-derived
Lys31-Leu150, with a C-terminal 6-His tag
Accession # Q7Z6A9-2

N-terminal Sequence Analysis Lys31

Predicted Molecular Mass 15 kDa

SPECIFICATIONS

SDS-PAGE 23-33 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When Recombinant Mouse HVEM/TNFRSF14 Fc Chimera (Catalog # 2516-HV) is immobilized at 0.5 µg/mL, 100 µL/well, the concentration of Recombinant Human BTLA that produces 50% of the optimal binding response is approximately 0.1-0.5 µg/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

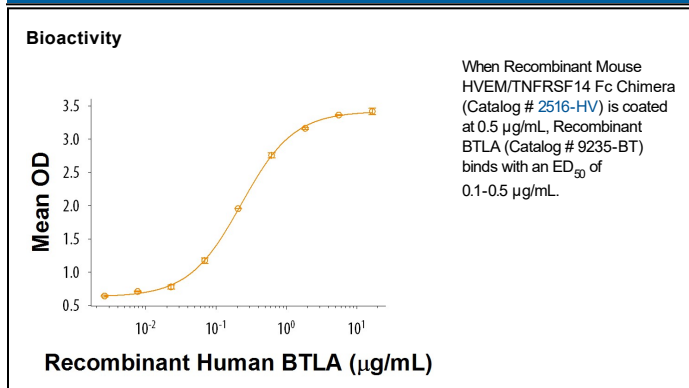
Reconstitution Reconstitute at 500 µg/mL in PBS.

Shipping The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA



BACKGROUND

B- and T-lymphocyte attenuator (BTLA; CD272) is a 35 kDa type I transmembrane glycoprotein in the CD28 family of T cell costimulatory 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 crosslinking (10). Under the control of ROR γ t and IL-7, BTLA regulates the homeostasis and inflammatory responses of $\gamma\delta$ T cells (11). The binding of BTLA and HVEM does not preclude the concurrent binding of other HVEM ligands such as LIGHT or Lymphotoxin- α (4).

References:

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