

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Fas Ligand in direct ELISAs and Western blots. In Western blots, this antibody shows approximately 50% cross-reactivity with recombinant mouse (rm) Fas Ligand and less than 5% cross-reactivity with rhAPRIL, rhBAFF, rhEDA-A2, rhGITR Ligand, rhLIGHT, rhOX40 Ligand, rhTRAIL, rhTNF- α , rhTRANCE, rhTWEAK, or rhVEGI.
Source	Monoclonal Mouse IgG ₁ Clone # 154922
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human Fas Ligand Pro134-Leu281 Accession # P48023
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 μ m filtered solution in PBS.

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
Western Blot	1 μ g/mL	Recombinant Human Fas Ligand/TNFSF6 (Catalog # 126-FL)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Fas Ligand (FasL), also known as CD178, CD95L, or TNFSF6, is a 40 kDa type II transmembrane member of the TNF superfamily of proteins. Its ability to induce apoptosis in target cells plays an important role in the development, homeostasis, and function of the immune system (1). Mature human Fas Ligand consists of a 179 amino acid (aa) extracellular domain (ECD), a 22 aa transmembrane segment, and a 80 aa cytoplasmic domain (2). Within the ECD, human Fas Ligand shares 81% and 78% aa sequence identity with mouse and rat Fas Ligand, respectively. Both mouse and human Fas Ligand are active on mouse and human cells (2, 3). Fas Ligand is expressed on the cell surface as a nondisulfide-linked homotrimer on activated CD4⁺ Th1 cells, CD8⁺ cytotoxic T cells, and NK cells (1). Fas Ligand binding to Fas/CD95 on an adjacent cell triggers apoptosis in the Fas-expressing cell (2, 4). Fas Ligand also binds DcR3 which is a soluble decoy receptor that interferes with Fas Ligand-induced apoptosis (5). Fas Ligand can be released from the cell surface by metalloproteinases as a 26 kDa soluble molecule which remains trimeric (6, 7). Shed Fas Ligand retains the ability to bind Fas, although its ability to trigger apoptosis is dramatically reduced (6, 7). In the absence of TGF- β , however, Fas Ligand/Fas interactions instead promote neutrophil-mediated inflammatory responses (3, 8). Fas Ligand itself transmits reverse signals that costimulate the proliferation of freshly antigen-stimulated T cells (9). Fas Ligand-induced apoptosis plays a central role in the development of immune tolerance and the maintenance of immune privileged sites (10). This function is exploited by tumor cells which evade immune surveillance by upregulating Fas Ligand to kill tumor infiltrating lymphocytes (8, 11). In gld mice, a Fas Ligand point mutation is the cause of severe lymphoproliferation and systemic autoimmunity (12, 13).

References:

1. Lettau, M. *et al.* (2008) *Curr. Med. Chem.* **15**:1684.
2. Takahashi, T. *et al.* (1994) *Int. Immunol.* **6**:1567.
3. Seino, K-I. *et al.* (1998) *J. Immunol.* **161**:4484.
4. Suda, T. *et al.* (1993) *Cell* **75**:1169.
5. Pitti, R.M. *et al.* (1998) *Nature* **396**:699.
6. Schneider, P. *et al.* (1998) *J. Exp. Med.* **187**:1205.
7. Tanaka, M. *et al.* (1998) *Nature Med.* **4**:31.
8. Chen, J-J. *et al.* (1998) *Science* **282**:1714.
9. Suzuki, I. and P.J. Fink (2000) *Proc. Natl. Acad. Sci.* **97**:1707.
10. Ferguson, T.A. and T.S. Griffith (2006) *Immunol. Rev.* **213**:228.
11. Ryan, A.E. *et al.* (2005) *Cancer Res.* **65**:9817.
12. Takahashi, T. *et al.* (1994) *Cell* **76**:969.
13. Lynch, D.H. *et al.* (1994) *Immunity* **1**:131.