

DESCRIPTION

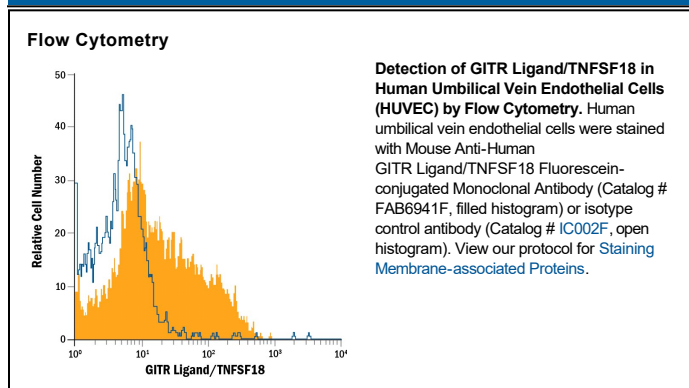
Species Reactivity	Human
Specificity	Detects human GITR Ligand/TNFSF18.
Source	Monoclonal Mouse IgG ₁ Clone # 109101
Purification	Protein A or G purified from ascites
Immunogen	<i>S. frugiperda</i> insect ovarian cell line Sf 21-derived recombinant human GITR Ligand/TNFSF18 Glu52-Ser177 Accession # Q9UNG2
Conjugate	Fluorescein Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm (FITC)
Formulation	Supplied 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	10 µL/10 ⁶ cells	See Below

DATA



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

GITR (glucocorticoid-induced TNF receptor superfamily-related protein, also named AITR, activation-inducible TNF receptor superfamily-related protein) and GITR ligand (GITRL) are novel members of the TNF receptor (TNFR) and TNF superfamilies (SF) that have been designated TNFRSF18 and TNFSF18, respectively. Human GITRL cDNA encodes a 177 amino acid residues type II membrane protein. The carboxy-terminal extracellular domain shows sequence identity to TNF/TNFSF2 (21%), Fas ligand/TNFSF6 (21%), TRAIL/TNFSF10 (18%), and lymphotoxin α/TNFSF1 (18%). GITRL is constitutively expressed in human umbilical vein endothelial cells but is not expressed in resting or stimulated T cell lines, B cell lines or peripheral blood mononuclear cells. GITR, the receptor for GITRL, is expressed at low levels in peripheral blood T cells, bone marrow, thymus, spleen and lymph nodes. In contrast to mouse GITR, expression of human GITR is not induced by treatment with dexamethasone, but is up-regulated by antigen-receptor stimulation or by treatment with soluble anti-CD3 plus anti-CD28 or PMA plus ionomycin. Ligation of GITR has been found to induce nuclear factor (NF)-κB activation via TNF receptor-associated factor 2 and protect cells from TCR activation-induced cell death. It has been proposed that GITRL and GITR may modulate T lymphocyte functions in peripheral tissues.

References:

1. Nocentini, G. *et al.* (1997) *Proc. Natl. Acad. Sci. USA* **94**:6216.
2. Kwon, B. *et al.* (1999) *J. Biol. Chem.* **274**:6056.
3. Gurney, A.L. *et al.* (1999) *Current Biology* **9**:215.
4. Kwon, B. *et al.* (1999) *Current Opinion in Immunology* **11**:340.