

Product Data:**Product SKU:** RPES3690**Size:** 50µg**Species:** Mouse**Expression host:** HEK293 Cells**Uniprot:** PP86176**Protein Information:****Molecular Mass:** 39.8 kDa**AP Molecular Mass:** 52 kDa**Tag:** C-Fc**Bio-activity:** Immobilized mouse PVR-His at 10 µg/ml (100 µl/well) can bind mouse TIGIT-Fc, The EC50 of mouse TIGIT-Fc is 0.25-0.55 µg/ml.**Purity:** > 90 % as determined by SDS-PAGE**Endotoxin:** < 1.0 EU per µg of the protein as determined by the LAL method.**Storage:** Lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.**Shipping:** This product is provided as lyophilized powder which is shipped with ice packs.**Formulation:** Lyophilized from sterile PBS, pH 7.4**Reconstitution:** Please refer to the printed manual for detailed information.**Application:** Functional ELISA**Synonyms:** T-cell immunoreceptor with Ig and ITIM domains;TIGIT;V-set and transmembrane domain-containing protein 3; VSTM3

Immunogen Information:

Sequence: Met1-Gly141

Background:

TIGIT, also known as V-set and transmembrane domain-containing protein 3 (VSTM3) or V-set and immunoglobulin domain-containing protein 9 (VSIG9) is a new surface protein containing an immunoglobulin variable domain, a transmembrane domain and an immunoreceptor tyrosine-based inhibitory motif (ITIM). TIGIT is expressed on regulatory, memory, activated T cells and NK cells. It binds PVR with high affinity, and PVRL2 with lower affinity, but not PVRL3. Knockdown of TIGIT with siRNA in human memory T cells did not affect T cell responses, however, TIGIT inhibits NK cytotoxicity directly through its ITIM. TIGIT suppresses T cell activation by promoting the generation of mature immunoregulatory dendritic cells. The binding of PVR to TIGIT on human dendritic cells enhanced the production of IL10 and diminished the production of IL2p40. In addition, TIGIT counter inhibits the NK-mediated killing of tumor cells and protects normal cells from NK-mediated cytotoxicity thus providing an 'alternative self' mechanism for MHC class I inhibition.