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Product datasheet for TP726700

Pvr Mouse Recombinant Protein

Product data:

Product Type:	Recombinant Proteins
Description:	Recombinant Mouse PVR (C-mIgG2AFc)
Species:	Mouse
Expression cDNA Clone or AA Sequence:	Asp29-Leu348
Tag:	C-mIgG2AFc
Buffer:	Lyophilized from a 0.2 um filtered solution of PBS, pH7.4.
Note:	Recombinant Mouse Poliovirus Receptor is produced by our Mammalian expression system and the target gene encoding Asp29-Leu348 is expressed with a mIgG2AFc tag at the C-terminus.
Storage:	Lyophilized protein should be stored at < -20°C, though stable at room temperature for 3 weeks. Reconstituted protein solution can be stored at 4-7°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Stability:	12 months from date of despatch
RefSeq:	NP_081790
Locus ID:	52118
UniProt ID:	Q8K094
Synonyms:	Poliovirus receptor; CD155 antigen; Nectin-like protein 5; Nectin-2; Tage4 receptor; Pvr; PVR; Necl5; CD155

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Summary: Mouse poliovirus receptor (PVR, CD155) is a type I transmembrane (TM) glycoprotein that is a member of the nectin-related family of adhesion proteins within the immunoglobulin superfamily. It binds other molecules including vitronectin, Nectin3, DNAM1, CD96, and TIGIT, but does not bind homotypically. CD155 includes a 28 aa signal sequence, a 318 aa extracellular domain (ECD) with one N-terminal V-type and two C2-type Ig-like domains, a 24 aa TM segment and a 38 aa cytoplasmic tail. Epithelial, endothelial, and many immune cells show low CD155 expression. It is up-regulated on endothelia by IFNÎ³, and is highly expressed on immature thymocytes, lymph node dendritic cells, and tumor cells of epithelial and neuronal origin. On migrating cells, it is concentrated at the leading edge, where it binds basement membrane vitronectin, recruits Nectin-3-expressing cells, and cooperates with PDGF and integrin Î±vÎ²3 to promote cell migration. Binding of monocyte DNAM-1 to endothelial cell CD155 promotes transendothelial migration. Enhanced CD155 expression in tumor cells contributes to loss of contact inhibition and increased migration, but also allows tumor cell recognition and killing by DNAM-1or CD96 expressing NK cells.