

Recombinant Human CD111/Nectin-1 Protein (Fc Tag)



DIA-AN[®]
by Elabscience

Catalog Number: PKSH031066

Note: Centrifuge before opening to ensure complete recovery of vial contents.

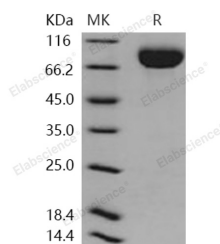
Description

Synonyms	Poliovirus Receptor-Related Protein 1; Herpes Virus Entry Mediator C; Herpesvirus Entry Mediator C; HveC; Herpesvirus Ig-Like Receptor; HIgR; Nectin-1; CD111; PVR L1; HVEC; PRR1; ED4; HIgR; HV1S; HVEC; nectin-1; OFC7; PRR; PVRR; PVRR1; SK-12
Species	Human
Expression Host	HEK293 Cells
Sequence	Met 1-Thr 334
Accession	NP_002846.3
Calculated Molecular Weight	61.0 kDa
Observed molecular weight	75-85 kDa
Tag	C-hFc
Bioactivity	Immobilized rh PVRL3 at 1 µg/ml (100 µl/well) can bind recombinant human Nectin-1 with a linear range of 0.156-5 ng/ml.

Properties

Purity	> 98 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage	Generally, 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 Normally 5 % - 8 % trehalose, mannitol and 0.01 % Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 98 % as determined by reducing SDS-PAGE.

Background

Poliovirus receptor-related 1 (herpesvirus entry mediator C; nectin-1; CD111), also known as PVRL1 is a cell adhesion molecule belonging to the immunoglobulin superfamily that can bind to virion glycoprotein D (gD) to mediate entry of herpes simplex viruses (HSV) and pseudorabies virus (PRV). CD111/Nectin-1/PVRL1 colocalizes with E-cadherin at

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adherens junctions in epithelial cells. The disruption of cell junctions can result in the redistribution of nectin-1. To determine whether disruption of junctions by calcium depletion influenced the susceptibility of epithelial cells to viral entry, Madin-Darby canine kidney cells expressing endogenous nectin-1 or transfected human nectin-1 were tested for the ability to bind soluble forms of viral gD and to be infected by HSV and PRV, before and after calcium depletion. It has been revealed that binding of HSV and PRV gD was localized to adherens junctions in cells maintained in normal medium but was distributed, along with nectin-1, over the entire cell surface after calcium depletion. Both the binding of gD and the fraction of cells that could be infected by HSV-1 and PRV were enhanced by calcium depletion. Taken together, CD111/Nectin-1/PVRL1 confined to adherens junctions in epithelial cells is not very accessible to virus, whereas dissociation of cell junctions releases nectin-1 to serve more efficiently as an entry receptor.

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