

Recombinant Human VCAM-1/CD106 Protein (His Tag)

Catalog Number: PDMH100409

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

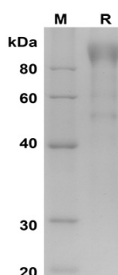
Description

Species	Human
Source	Mammalian-derived Human VCAM-1 protein Phe25-Glu698, with an C-terminal His
Calculated MW	76.7 kDa
Observed MW	100 kDa
Accession	P19320
Bio-activity	Not validated for activity

Properties

Purity	> 90% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU/mg 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 a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Human VCAM-1/CD106 proteins, 2 µg/lane of Recombinant Human VCAM-1/CD106 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 100 kDa.

Background

For Research Use Only

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VCAM-1 is a single-pass type I membrane protein, contains 7 Ig-like C2-type domains. It is an endothelial ligand for very late antigen-4 (VLA-4) and $\alpha 4\beta 7$ integrin expressed on leukocytes, and thus mediates leukocyte-endothelial cell adhesion and signal transduction. VCAM-1 expression is induced on endothelial cells during inflammatory bowel disease, atherosclerosis, allograft rejection, infection, and asthmatic responses. During these responses, VCAM-1 forms a scaffold for leukocyte migration. VCAM-1 also activates signals within endothelial cells resulting in the opening of an "endothelial cell gate" through which leukocytes migrate. VCAM-1 has been identified as a potential anti-inflammatory therapeutic target, the hypothesis being that reduced expression of VCAM-1 will slow the development of atherosclerosis. In addition, VCAM-1-activated signals in endothelial cells are regulated by cytokines indicating that it is important to consider both endothelial cell adhesion molecule expression and function during inflammatory processes.

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