

Recombinant Human VCAM-1/CD106 protein (GST Tag)

Catalog Number: PDEH100841

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

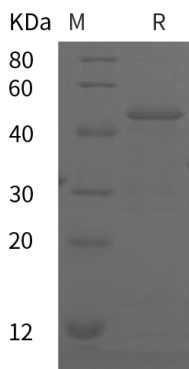
Description

Species	Human
Source	E.coli-derived Human VCAM-1 protein Gln109-Ser318, with an N-terminal GST
Calculated MW	48.0 kDa
Observed MW	45 kDa
Accession	P19320
Bio-activity	Not validated for activity

Properties

Purity	> 95% as determined by reducing SDS-PAGE.
Endotoxin	< 10 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



> 95 % as determined by reducing SDS-PAGE.

Background

Vascular cell adhesion molecule 1 (VCAM-1), also known as CD106, is a cell surface sialoglycoprotein belonging to the immunoglobulin superfamily. Two forms of VCAM-1 with either six or seven extracellular Ig-like domains are generated by alternative splicing, with the longer form predominant. VCAM-1 is an endothelial ligand for very late antigen-4 (VLA-4) and $\alpha_4\beta_1$ 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.