Recombinant Human ESAM Protein (aa 30-247, Fc Tag)

Catalog No. PKSH032380

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

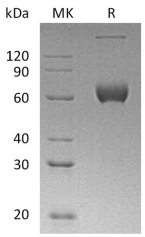
Description

Synonyms	Endothelial Cell-Selective Adhesion Molecule;ESAM
Species	Human
Expression Host	HEK293 Cells
Sequence	Gln30-Ala247
Accession	Q96AP7
Calculated Molecular Weight	50.8 kDa
Observed molecular weight	60-80 kDa
Tag	C-Fc
Bioactivity	Not validated for activity

Properties

Purity	> 95 % 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℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ 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 of 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



> 95 % as determined by reducing SDS-PAGE.

Background

Endothelial Cell Adhesion Molecule (ESAM) is a 55 kDa type I transmembrane glycoprotein member of the JAM family of immunoglobulin superfamily molecules. The 390 amino acid Human ESAM contains a 216 amino acid extracellular

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domain (ECD) with a V-type and a C2-type immunoglobulin (Ig) domain. The ECD of human and mouse ESAM share 69% amino acid identity. ESAM is specifically expressed at endothelial tight junctions and on activated platelets and performs homophilic adhesion activity. The adaptor protein membrane-associated guanylate kinase MAGI-1 has been identified as an intracellular binding partner of ESAM. In addition; ESAM at endothelial tight junctions participates in the migration of neutrophils through the vessel wall; possibly by influencing endothelial cell contacts. ESAM-deficient mice were described with lowered angiogenic potential; and accordingly; overexpression of ESAM is closely associated with certain tumor growth and metastasis.

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