

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



Catalog Number:PKSH032380

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

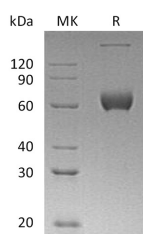
## Description

<b>Synonyms</b>	Endothelial Cell-Selective Adhesion Molecule;ESAM
<b>Species</b>	Human
<b>Expression Host</b>	HEK293 Cells
<b>Sequence</b>	Gln30-Ala247
<b>Accession</b>	Q96AP7
<b>Calculated Molecular Weight</b>	50.8 kDa
<b>Observed molecular weight</b>	60-80 kDa
<b>Tag</b>	C-Fc

## Properties

<b>Purity</b>	> 95 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	< 1.0 EU per µg of the protein as determined by the LAL method.
<b>Storage</b>	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.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	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.
<b>Reconstitution</b>	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 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

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certain tumor growth and metastasis.

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