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

Catalog Number: PKSH032380

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

#### Description

**Species** Human

Source HEK293 Cells-derived Human ESAM protein Gln30-Ala247, with an C-terminal Fc

Calculated MW 50.8 kDa
Observed MW 60-80 kDa
Accession Q96AP7

**Bio-activity** 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

°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 of PBS, pH 7.4.

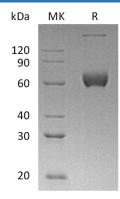
Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants

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**

# Elabscience®

## Elabscience Biotechnology Co., Ltd.

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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 6 9% 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.