

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human ESAM in direct ELISAs.
Source	Monoclonal Mouse IgG _{2B} Clone # 1021723
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human ESAM Gln30-Ala247 Accession # Q96AP7
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide

*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

	Recommended Concentration	Sample
Flow Cytometry	0.25-1 µg/10 ⁶ cells	HUVEC human umbilical vein endothelial cells

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. <ul style="list-style-type: none"> 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

Endothelial cell-selective adhesion molecule (ESAM) is a 55 kDa type I transmembrane glycoprotein that belongs to the JAM family of immunoglobulin superfamily molecules (1, 2). Human ESAM is synthesized as a 390 amino acid (aa) protein composed of a 29 aa signal peptide, a 216 aa extracellular region, a putative 26 aa transmembrane segment, and a 119 aa cytoplasmic domain. The extracellular region contains one V-type and one C2-type Ig domain and is involved in homophilic adhesion (1). In the cytoplasmic domain, there is a docking site for the multifunctional adaptor protein MAGI-1 (3). The extracellular region of human ESAM shows 90%, 74%, 69%, and 67% aa identity with monkey, canine, mouse, and rat extracellular ESAM, respectively. ESAM is expressed on endothelial cells, activated platelets, and megakaryocytes and can be found associated with cell-to-cell junctions. Whether ESAM is restricted to a particular junctional type is not clear (1, 2). ESAM deficient mice have no defect in vascularization but do have reduced angiogenic potential. This may be due to a decreased migratory response to FGF-2 (4).

References:

- Hirata, K-I. *et al.* (2001) *J. Biol. Chem.* **276**:16223.
- Nasdala, I. *et al.* (2002) *J. Biol. Chem.* **277**:16294.
- Wegmann, F. *et al.* (2004) *Exp. Cell Res.* **300**:121.
- Ishida, T. *et al.* (2003) *J. Biol. Chem.* **278**:34598.

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