

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human ECM-1 in direct ELISAs.
Source	Monoclonal Mouse IgG ₁ Clone # 966248
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human ECM-1 Ala20-Glu540 Accession # Q16610
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% 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.

ELISA Optimal dilution of this antibody should be experimentally determined.

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. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Extracellular matrix protein-1 (ECM-1) is an 85 kDa, secreted glycoprotein important in connective tissue organization (1-3). Of three identified splice variants the 540 amino acid (aa) form, ECM-1a, is the most widely expressed, with the highest expression in the placenta and heart (2). ECM-1b (415 aa) is found only in tonsil and associated with suprabasal keratinocytes (2, 4). Since ECM-1b expression is differentiation-dependent, a role in terminal keratinocyte differentiation has been suggested (4). ECM-1c (559 aa) accounts for approximately 15% of skin ECM-1 (5). Human ECM-1a contains a 19 aa signal peptide and a 521 aa secreted portion that includes an N-terminal proline-rich, cysteine-free region, two tandem repeat domains, and a C-terminal domain. There are six repeats of a CC(X₇₋₁₀)C motif (x = any aa) within the tandem repeat and C-terminal domains. These motifs are involved in ligand binding to members of the albumin family, and are expected to form two (in ECM-1b) or three (in ECM-1a) "double loop" structures (2). Mature human ECM-1a shows 69%, 71%, 72%, and 76% aa identity with corresponding isoforms of mouse, rat, canine, and bovine ECM-1, respectively. ECM-1 is over-expressed in many malignant epithelial tumors and has demonstrated angiogenic activity (6, 7). A variety of ECM-1 mutations, mainly within the first tandem repeat, are considered causative of lipoid proteinosis, a condition showing thickened and irregular extracellular matrix within connective tissue (8). In the autoimmune condition lichen sclerosis, auto-antibodies mainly recognize the second tandem repeat or the C-terminus of ECM-1 (9). These domains also bind the extracellular matrix molecules fibulin-1 and perlecan (5, 10). The phenotypes of lipoid proteinosis and lichen sclerosis support a role for ECM-1 as a "biological glue" in the dermis (1).

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