Recombinant Human ECM1

Catalog Number: 3937-EC

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Ala20-Glu540, with a C-terminal 6-His tag Accession # AAH23505
N-terminal Sequence Analysis	Ala20
Predicted Molecular Mass	59.7 kDa

SPECIFICATIONS

SDS-PAGE	77-87 kDa, reducing conditions
Activity	Measured by the ability of the immobilized protein to support the adhesion of B16-F1 mouse melanoma cells. When 5 x 10^4 cells/well are added to Recombinant Human ECM-1 coated plates (10 µg/mL with 100 µL/well), >30% will adhere after 30 minutes at 37 °C. **Optimal concentration depends on cell type as well as the application or research objective.**
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

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 $CC(X_{7-10})C$ 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).

References:

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