Recombinant Human MIA

Catalog Number: 9250-IA

DESCRIPTION

Source	*E. coli*-derived Gly25-Gln131, with an N-terminal Met Accession # Q16674
N-terminal Sequence Analysis	Met, Gly25
Predicted Molecular Mass	12 kDa

SPECIFICATIONS

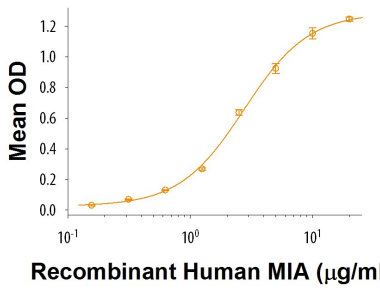
SDS-PAGE	10 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human Fibronectin Fragment 4 (Catalog # 3624-FN) is immubilized at 1 µg/mL, 100 µL/well, Recombinant Human MIA binds with a typical ED_{50} of 1-6 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE with silver staining, under reducing conditions.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS and Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 400 µg/mL in PBS.
Shipping	The product is shipped with polar packs. 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.

DATA

Bioactivity



When Recombinant Human Fibronectin Fragment 4 (Catalog # 3624-FN) is coated at 1 µg/mL, Recombinant Human MIA (Catalog # 9250-IA) binds with a typical ED_{50} of 1-6 µg/mL.

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BACKGROUND

Melanoma Inhibiting Activity (MIA), also known as cartilage-derived retinoic acid-sensitive protein (CD-RAP), is an approximately 11-15 kDa protein that is secreted as a noncovalent homodimer and is structurally related to OTOR/Otoraplin and MIA-2 (1). Mature human MIA contains a SH3 domain and shares 90% and 92% amino acid sequence identity with mouse and rat MIA, respectively (2). Alternative splicing generates a short isoform that lacks the SH3 domain (3). MIA is widely expressed in developing and regenerating cartilage and in the endothelium and parenchyma of developing lungs (4). MIA disrupts cellular interactions with the extracellular matrix by binding to Integrins α4β1 and α5β1 (5). It competes with Fibronectin fragments for Integrin binding and interferes with Integrin signaling (5). It also functions as a chemoattractant for mesenchymal stem cells and enhances their BMP-2 and TGF-β3 induced differentiation into chondrocytes [tscheud]. MIA-deficient mice exhibit delayed chondrocyte differentiation but enhanced chondrocyte proliferation and cartilage repair (7). MIA is up-regulated in several cancers including malignant melanoma, lung adenoma, metastatic oral squamous cell carcinoma, neurofibromatosis type 1 (NF-1)-related tumors, and pancreatic cancer (2, 4, 8-10). It is selectively secreted and internalized from the trailing pole of migrating cells (11, 12). This polarization reduces cellular attachment to the matrix at the trailing pole and contributes to directional tumor cell migration (2, 10, 13, 14).

References:

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