Recombinant Mouse Serpin F1/PEDF

Catalog Number: 8295-SF

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Gln20-Thr417, with a C-terminal 6-His tag Accession # BAA31978.1
N-terminal Sequence Analysis	No results obtained: Gln20 inferred from enzymatic pyroglutamate treatment revealing Asn21.
Predicted Molecular Mass	45 kDa

SPECIFICATIONS

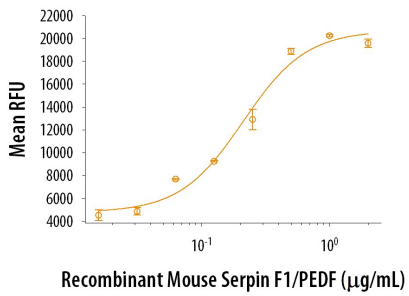
SDS-PAGE	50-56 kDa, reducing conditions
Activity	Measured by its ability to enhance the adhesion of Saos-2 human osteosarcoma cells to bovine Collagen I coated plate. Eth, E.K. *et al.* (2007) Cancer Gene Therapy. **14**:616. The ED_{50} for this effect is typically 0.1-0.6 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE with silver staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS and NaCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 µg/mL in steril 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.

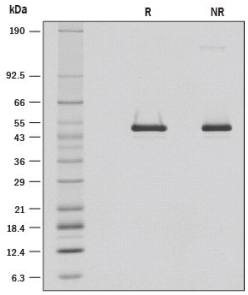
DATA

Bioactivity



Serpin F1/PEDF-induced Adhesion. Recombinant Mouse Serpin F1/PEDF enhances the adhesion of Saos-2 human osteosarcoma cells to a bovine Collagen I coated plate. The ED_{50} for this effect is typically 0.1-0.6 µg/mL.

SDS-PAGE



1 µg/lane of Recombinant Mouse Serpin F1/PEDF was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by silver staining, showing R bands at 48.8, 42.5 kDa and NR bands at 140.8, 47.9, 42.1 kDa.

bio-techne
bio-techne.com
Global info@bio-techne.com techsupport@bio-techne.com
North America TEL 800 343 7475 Europe | Middle East | Africa TEL +44 (0)1235 529449
China info.cn@bio-techne.com TEL +86 (21) 52380373
Rest of World bio-techne.com/find-us/distributors TEL +1 612 379 2956

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BACKGROUND

Serpin Peptidase Inhibitor, clade F (Serpin F1), also called Pigment Epithelium-Derived Factor (PEDF), EPC-1, and PIG35, is a member of the Serpin superfamily of serine protease inhibitors (1-5). This superfamily is comprised of two protein groups with dissimilar functions. One group demonstrates protease inhibition while proteins in the other group display no protease inhibition, but rather, perform diverse functions, such as molecular chaperones, circulating transporters, and tumor suppressors (2). Serpin F1 is part of this latter group. It is a 50 kDa, monomeric phosphoglycoprotein that is comprised of three β-sheets, 8-10 α-helices, and a C-terminal reactive center loop (RCL), a structure common to all Serpins (2-5). However, unlike Serpins that exhibit protease inhibiting activity, the RCL of Serpin F1 does not undergo a conformational change, a prerequisite for anti-protease activity (3, 5). Such cleavage does, however, generate a 46 kDa fragment that possesses nonprotease-associated bioactivity (6). Mouse Serpin F1 displays 88% and 93% amino acid sequence identity with the human and rat orthologs, respectively.

Serpin F1 is a multifunctional protein that is synthesized by multiple cell types and is expressed in many tissues including the retinal pigment epithelium, liver, bone, connective, heart, and adipose tissues (1, 3, 5, 7, 8). It has been shown to bind to several different cell surface molecules including the PEDF Receptor, Laminin Receptor, LRP-6, and the F_1 ATP Synthase (5, 9-13). It also has binding affinity for several extracellular matrix components, such as Heparin, Heparin Sulphate, Hyaluronan, and Collagens (5, 3). It is believed that the multiple and varied biological activities attributed to Serpin F1 are due to its interactions with these different cell surface molecules. Serpin F1 has been shown to be involved in neurogenesis, neuronal cell survival, angiogenesis, tumorgenesis, stem cell survival and multipotency, and inflammation (2-5, 10, 12-15). In humans, Serpin F1 has been suggested to play a role in choroidal neovascularization, obesity and insulin resistance, cardiovascular disease, osteogenesis imperfecta, and cancer (3-5, 15-19).

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Global info@bio-techne.com techsupport@bio-techne.com
North America TEL 800 343 7475 Europe | Middle East | Africa TEL +44 (0)1235 529449
China info.cn@bio-techne.com TEL +86 (21) 52380373
Rest of World bio-techne.com/find-us/distributors TEL +1 612 379 2956