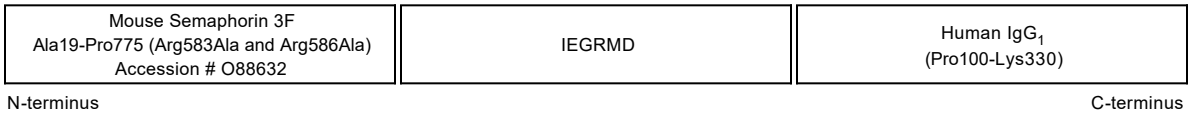
Recombinant Mouse Semaphorin 3F Fc Chimera, Truncated

Catalog Number: 3237-S3

DESCRIPTION

Source

Mouse myeloma cell line, NS0-derived



N-terminal Sequence Analysis	Ala19
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	111.6 kDa (monomer)

SPECIFICATIONS

SDS-PAGE	110-122 kDa, reducing conditions
Activity	Measured by its ability to inhibit proliferation of the A549 human lung carcinoma cells. The ED_{50} for this effect is 40-120 ng/mL.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in Citric Acid and NaCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile 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.

BACKGROUND

Semaphorin 3F (Sema 3F; previously Sema IV) is one of six Class 3 (secreted) semaphorins which in the mouse share 40 - 50% amino acid (aa) identity. Class 3 semaphorins are potent chemorepellents that function in axon guidance and/or vascular tip cell guidance during development (1). Sema 3F is expressed in the developing nervous system, especially in the dorsal spinal cord (2, 3). In adults, Sema 3F is expressed in the lung and most other tissues (2). Crystal structures of semaphorins reveal that the 500 aa N-terminal Sema domain forms a seven-blade β-propeller similar to that found in integrin molecules. Fourteen conserved cysteine residues and one or more N-glycosylation sites are thought to be critical for forming the secondary structure (4). Isoform A is missing aa 153 - 183 within the Sema domain relative to the long form (isoform B) but appears to have similar activity. C-terminal to the Sema domain, Sema 3F has a basic domain, a cysteine-knot plexin/semaphorin/integrin (PSI) domain, an Ig-like domain, a cysteine for dimerization and another basic domain at the C-terminus. Dimerization and cleavage at the C-terminus are required for repulsing activity of class 3 semaphorins (5). Mouse Sema 3F shares 96%, 99%, 92%, 97% and 82% aa identity with human, rat, bovine, canine and chick Sema 3F, respectively. Type 3 semaphorins transduce signals through transmembrane plexins, either directly or by binding associated neuropilin receptors. Sema 3F signaling is transduced by type-A plexins, especially Plexin-A3, via interaction with neuropilin-2 (3, 6). Genetic disruption of either Sema 3F or neuropilin-2 alters motor axon trajectory to the ventral forelimb (3). Sema 3F is deleted or downregulated in many metastatic tumors. Restoration of Sema 3F decreases tumorigenicity, vascularization and adhesiveness, most likely through repulsive interactions, VEGF antagonism and downstream integrin regulation (7).

References:

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