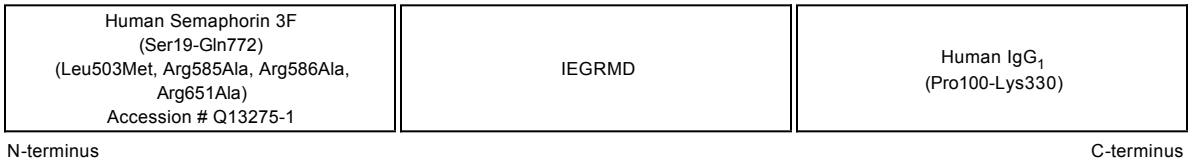
Recombinant Human Semaphorin 3F Fc Chimera

Catalog Number: 9878-S3

DESCRIPTION

Source

Mouse myeloma cell line, NS0-derived human Semaphorin 3F protein



N-terminal Sequence Analysis	Ser19
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	111 kDa

SPECIFICATIONS

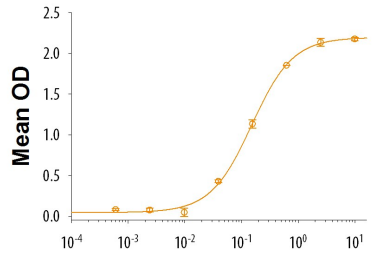
SDS-PAGE	91-125 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human Neuropilin-2 Fc Chimera (Catalog # 2215-N2) is immobilized at 2 µg/mL, 100 µL/well, it binds Recombinant Human Semaphorin 3F Fc Chimera. The concentration of Recombinant Human Semaphorin 3F Fc Chimera that produces 50% of the optimal binding response is 0.1-0.6 µg/mL.
Endotoxin Level	<0.10 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 Tris and NaCl with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in water.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	• 12 months from date of receipt, ≤ -20 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 3 months, ≤ -20 °C under sterile conditions after reconstitution.

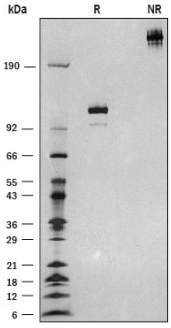
DATA

Binding Activity



When Recombinant Human Neuropilin-2 Fc Chimera (Catalog # 2215-N2) is immobilized at 2 µg/mL, 100 µL/well, Recombinant Human Semaphorin 3F Fc Chimera (Catalog # 9878-S3) binds with an ED_{50} of 0.1-0.6 µg/mL.

SDS-PAGE



1 µg/lane of Recombinant Human Semaphorin 3F was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by silver staining, showing bands at 91 - 125 kDa and 200 - 250 kDa, respectively.

bio-techne®
Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449

Recombinant Human Semaphorin 3F Fc Chimera

Catalog Number: 9878-S3

BACKGROUND

Semaphorin 3F (Sema 3F; previously Sema IV) is one of six Class 3 (secreted) semaphorins. 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 amino acid (aa) N-terminal Sema domain forms a seven-blade beta-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 2 is missing aa 153-183 within the Sema domain relative to the long form (Isoform 1) 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). Human Sema 3F shares 96% aa identity with mouse and rat Sema 3F. Sema 3F signaling is transduced by type-A plexins, especially Plexin-A3, via interaction with neuropilin-2 (3, 6). Sema 3F-Npn-2-Plexin-A3 signaling regulates AMPA-type glutamate receptor (AMPAR) homeostatic downscaling in response to increased neuronal activity of cortical neurons (7). Genetic disruption of either Sema 3F or neuropilin-2 alters motor axon trajectory to the ventral forelimb (3). Sema 3F is deleted or down-regulated in many metastatic tumors such as colorectal cancer, oral squamous carcinoma, lung cancer, and many others (8-10). Restoration of Sema 3F decreases tumorigenicity, vascularization and adhesiveness, most likely through repulsive interactions, VEGF antagonism and downstream integrin regulation (11).

References:

1. Kruger, R.P. *et al.* (2005) Nature Rev. Mol. Cell Biol. **6**:789.
2. Eckhardt, F. and A. Meyerhans (1998) Neuroreport **9**:3975.
3. Huber, A.B *et al.* (2005) Neuron **48**:949.
4. Gherardi, E. *et al.* (2004) Curr. Opin. Struct. Biol. **14**:669.
5. Adams, R.H. *et al.* (1997) EMBO J. **16**:6077.
6. Yaron, A. *et al.* (2005) Neuron **45**:513.
7. Wang Q. *et al.* (2017) Neuron. **96(5)**:1084.
8. Liu, Y. *et al.* (2017) Cell Mol Biol Lett. Dec 28; **22**:32.
9. Gao, X. *et al.* (2015) Int J Clin Exp Pathol. **8(10)**:12766.
10. Potiron, V.A. *et al.* (2007) Cancer Res. **67(18)**:8708.
11. Chedotal, A. *et al.* (2005) Cell Death Differ. **12**:1044.

bio-techne®
Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 **Canada** TEL 855 668 8722 **China** TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449