Recombinant Mouse Semaphorin 3E Fc Chimera

Catalog Number: 3238-S3

DESCRIPTION

Source

Spodoptera frugiperda, *Sf* 21 (baculovirus)-derived

N-terminal Sequence Analysis	Asn27
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	112 kDa (monomer)

SPECIFICATIONS

SDS-PAGE	110-120 kDa, reducing conditions
Activity	Measured by its ability to cause collapse of chick embryonic dorsal root ganglia (DRG) neuron growth cones. 2.5-5.0 µg/mL of Recombinant Mouse Semaphorin 3E Fc Chimera causes >50% growth cone collapse in the presence of 10 ng/mL of Recombinant Human β-NGF (Catalog # 256-GF).
Endotoxin Level	<1.0 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 Tris, NaCl and Tween® 20. 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 3E (Sema3E), previously known as SemaH, is one of six Class 3 (secreted) semaphorins which function in axon guidance and/or vascular tip cell guidance during development (1). Sema3E contains a seven-blade β-propeller sema domain, a cysteine-knot PSI domain, an Ig-like domain, and a basic region. Dimerization and cleavage within the basic region are required for the repulsing activity of class 3 semaphorins (2). Sema3E can also be cleaved at a furin consensus sequence C-terminal to the sema domain, resulting in a 61 kDa form that does not dimerize and is highly expressed in tumor cell lines with metastatic potential (3, 4). Mature mouse Sema3E shares 90% and 98% aa sequence identity with human and rat Sema3E, respectively. Sema3E signaling is transduced by Plexin D1 which may also be associated with Neuropilin 1 and/or VEGF R2 (2, 5, 6). Its interaction with Plexin D1 inhibits axon migration in the neocortex and forebrain (6, 7), although it can attract axons that express both Plexin D1 and Neuropilin 1 (6). Sema3E promotes axonal growth (5), the development of glutamatergic synaptic specificity (8, 9), and the development of GnRH producing neurons (10). Genetic disruption of either Sema3E or Plexin D1 in mouse causes excessive and disorganized vascular growth and branching, indicating the importance of this ligand-receptor pair for vascular guidance (11, 12). In addition, Sema3E is up-regulated by inflammatory macrophages and damaged hepatocytes (13-15). It inhibits smooth muscle cell proliferation and migration in the asthmatic airway (16), promotes hepatic stellate cell activation and wound healing (15), and regulates the migration of developing thymocytes (17).

References:

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