

DESCRIPTION

Source Human embryonic kidney cell, HEK293-derived
Cys34-Val1116, with a C-terminal 6-His tag
Accession # NP_003053

N-terminal Sequence Analysis Cys34

Predicted Molecular Mass 121 kDa

SPECIFICATIONS

SDS-PAGE 119-149 kDa, reducing conditions

Activity Measured by its ability to enhance neurite outgrowth of E16-E18 rat embryonic cortical neurons. Recombinant Human Slit3, immobilized at 5 µg/mL on a 96-well plate, is able to significantly enhance neurite outgrowth.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in MOPS, NaCl and Ethylene Glycol with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 500 µg/mL in PBS.

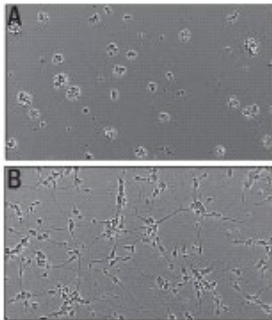
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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



Recombinant Human Slit3 (Catalog # 9255-SL) Induces Cortical Neurite Outgrowth. A) Untreated E16-18 embryonic rat cortical neurons. B) Neurite outgrowth in E16-18 embryonic rat cortical neurons treated with 5 µg/mL of Recombinant Human Slit3.

BACKGROUND

Slit3 is an approximately 200 kDa member of the Slit family of large secreted axon guidance molecules that are ligands for ROBO receptors (1, 2). It contains a mitochondrial targeting sequence and may also be retained intracellularly (3). Mature human Slit3 consists of 4 cassettes of leucine-rich repeats (LRR) flanked by LRR N-terminal and C-terminal domains, followed by multiple EGF-like domains, a Laminin G-like domain, and a C-terminal cysteine-rich domain. Alternative splicing generates additional isoforms with a substituted cysteine-rich domain or a deletion in the last EGF-like domain and Laminin G-like domain. During development, Slit3 is expressed in the ventral neural tube, developing sensory organs, limb buds, and developing areas of the limbs in patterns that overlap with but are discrete from Slit1 and Slit2 (1, 2, 4). Axons will not be allowed to recross the floor plate unless all three Slit genes are disrupted, suggesting some overlap in Slit function (5). Slit3 is also expressed in the lung, kidney, skeletal muscle, and heart, both during development and postnatally (6-8). Mice with genetically disrupted Slit3 show abnormalities in diaphragm and kidney development (7, 8). Slit3 is additionally expressed by vascular endothelial cells and smooth muscle cells (9). It binds to both ROBO1 and ROBO4, but it is the ROBO4 interaction that mediates Slit3-induced angiogenesis (9). It also can enhance the chemokine-induced migration of monocytes (10). The related Slit2 protein is cleaved *in vivo* (at a site conserved in Slit3), and the resulting C-terminal fragment binds Plexin A1 and retains the ability to repel axon migration (4, 11). The corresponding C-terminal fragment of Slit3 is able to bind heparin and neutralize its anti-coagulant activity (12). Within this fragment (aa 1120-1523), human Slit3 shares 93% amino acid identity with mouse and rat Slit3.

References:

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