Recombinant Human Slit3 (aa 1120-1523)

Catalog Number: 9067-SL

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived Thr1120-Ser1523, with a C-terminal 6-His tag Accession # O75094
N-terminal Sequence Analysis	Thr1120
Predicted Molecular Mass	45 kDa

SPECIFICATIONS

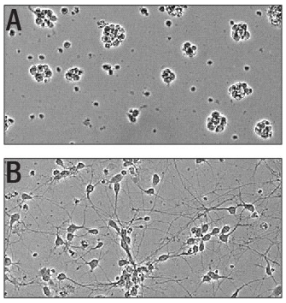
SDS-PAGE	56-63 kDa, reducing conditions
Activity	Measured by its ability to enhance neurite outgrowth of E16-E18 rat embryonic cortical neurons. Recombinant Human Slit3, immobilized at 1.25-2.5 µg/mL on a 96-well plate, is able to significantly enhance neurite outgrowth.
Endotoxin Level	<0.10 EU per 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 and NaCl. 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 # 9067-SL) Induces Cortical Neurite Outgrowth. A) Untreated E16-E18 embryonic rat cortical neurons. B) Neurite outgrowth in E16-E18 embryonic rat cortical neurons treated with 1.25 µg/ml of Recombinant Human Slit3.

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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 is secreted to the extracellular space and also localizes in mitochondria via a mitochondria localization sequence (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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