

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived human SPARC-like 1/SPARCL1 protein
Ile17-Phe664, with a C-terminal 10-His tag
Accession # Q8N4S1

N-terminal Sequence Analysis Ile17

Predicted Molecular Mass 74.9 kDa

SPECIFICATIONS

SDS-PAGE 118-144 kDa, reducing conditions

Activity Measured by its ability to inhibit the cell growth of Mv1Lu mink lung epithelial cells. Schiemann, B.J. *et al.* (2003) Mol. Biol. Cell. **14**:3977. The ED₅₀ for this effect is 0.5-2.0 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 PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in sterile 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.

BACKGROUND

SPARCL1 (Secreted Protein, Acidic and Rich in Cysteines-like 1), also known as hevin, SC1 or MAST9, is a member of the SPARC family of extracellular glycoproteins (1, 2). SPARCL1 is an anti-adhesive protein that is widely expressed in tissues such as brain, heart, lung, muscle and kidney, but not liver (3, 4). Human SPARCL1 contains a 16 amino acid (aa) signal sequence and a 648 aa mature region with four domains: a 416 aa N-terminal acidic region, a 23 aa follistatin-like domain, a 55 aa kazal-like segment and a 48 aa EF-hand/calcium-binding domain (3, 4). SPARCL1 is predicted at 75 kDa, but migrates at ~130 kDa, which has been explained either by disulfide-linked homodimerization or by glycosylation and high acidity (3-5). Some truncated forms have been reported. In mouse, a 55 kDa C-terminal fragment is the only form in kidney and represents a portion of SPARCL1 in other tissues (6). In humans, a 25 kDa form is increased in liver tumors that are encapsulated, while the full-length form is down-regulated in many epithelial cell-derived tumors (7, 8). SPARCL1 inhibits adhesion and spreading on a variety of substrates (5, 9). It is thought to cause antiadhesive signaling that terminates neuronal migration, consistent with production by glial and neuronal cells during development or in response to trauma (10). In tonsillar high endothelial venules (HEV), SPARCL1 may induce endothelial cell dissociation, promoting extravasation (3). SPARCL1 binds collagen; in mice, deletion causes dermal collagen fibrils that are smaller in diameter and deficient in decorin (6, 11). Human mature SPARCL1 shares 67%, 69%, 78%, 76%, 72% and 72% aa identity with mouse, rat, equine, canine, porcine and bovine SPARCL1, respectively. The follistatin-like, kazal-like and calcium-binding domains of SPARCL1 show 61% aa identity with corresponding regions of SPARC.

References:

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