Gemin2 (4): sc-135918



The Power to Question

BACKGROUND

Spinal muscular atrophy (SMA) is an autosomal recessive neurodegenerative disease characterized by loss of motor neurons in the spinal cord. SMA is caused by deletion or loss-of-function mutations in the SMN (survival of motor neuron) gene. Gemin2 (formerly known as SIP1 for SMN interacting protein) associates directly with SMN and is a part of the SMN complex containing Gemin3 (a DEAD-box RNA helicase), Gemin4, Gemin5 and Gemin6, as well as several spliceosomal snRNP proteins. The SMN complex plays an essential role in splicesomal snRNP assembly in the cytoplasm and is required for pre-mRNA splicing of the nucleus. It is found in both the cytoplasm and the nucleus. The nuclear form is concentrated in subnuclear bodies called gems (Gemini of the coiled bodies). The SMN-Gemin2 complex is associated with spliceosomal snRNAs U1 and U5. Gemin2 is expressed in spinal cord. It can be induced by TGFβ treatment and expression is high in several E-cadherin negative human carcinoma cell lines. SMN is expressed in a wide variety of tissues including brain, kidney, liver and spinal cord, and moderately in skeletal and cardiac muscle.

REFERENCES

- 1. Fischer, U., et al. 1997. The SMN-SIP1 complex has an essential role in spliceosomal snRNP biogenesis. Cell 90: 1023-1029.
- 2. Coovert, D., et al. 1997. The survival motor neuron protein in spinal muscular atrophy. Hum. Mol. Genet. 6: 1205-1214.
- Monani, U., et al. 1999. A single nucleotide difference that alters splicing patterns distinguishes the SMA gene SMN1 from the copy gene SMN2. Hum. Mol. Genet. 8: 1177-1183.
- Meister, G., et al. 2000. Characterization of a nuclear 20S complex containing the survival of motor neurons (SMN) protein and a specific subset of spliceosomal Sm proteins. Hum. Mol. Genet. 9: 1977-1986.
- Mourelatos, Z., et al. 2001. SMN interacts with a novel family of hnRNP and spliceosomal proteins. EMBO J. 20: 5443-5452.
- Comijn, J., et al. 2001. The two-handed E box binding zinc finger protein SIP1 downregulates E-cadherin and induces invasion. Mol. Cell 7: 1267-1278.
- 7. Online Mendelian Inheritance in Man, OMIM™. 2001. Johns Hopkins University, Baltimore, MD. MIM Number: 602595. World Wide Web URL: http://www.ncbi.nlm. nih.gov/omim/
- 8. LocusLink Report (LocusID: 8487). http://www.ncbi.nlm.nih.gov/LocusLink/

CHROMOSOMAL LOCATION

Genetic locus: SIP1 (human) mapping to 14q21.1.

SOURCE

Gemin2 (4) is a mouse monoclonal antibody raised against amino acids 36-156 of Gemin2 of human origin.

PRODUCT

Each vial contains $50 \mu g lgG_1$ in 0.5 ml of PBS with < 0.1% sodium azide, 0.1% gelatin, 20% glycerol and 0.04% stabilizer protein.

APPLICATIONS

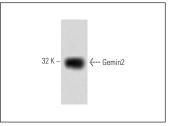
Gemin2 (4) is recommended for detection of Gemin2 of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)] and immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

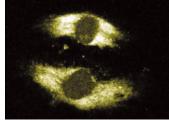
Suitable for use as control antibody for Gemin2 siRNA (h): sc-42129, Gemin2 shRNA Plasmid (h): sc-42129-SH and Gemin2 shRNA (h) Lentiviral Particles: sc-42129-V.

Molecular Weight of Gemin2: 32-34 kDa.

Positive Controls: Hep G2 cell lysate: sc-2227, K-562 whole cell lysate: sc-2203 or HeLa whole cell lysate: sc-2200.

DATA





Gemin2 (4): sc-135918. Western blot analysis of Gemin2 expression in K-562 whole cell lysate.

Gemin2 (4): sc-135918. Immunofluorescence staining of HISM cells showing cytoplasmic staining.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures. Not for resale.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.