SANTA CRUZ BIOTECHNOLOGY, INC.

SMN (N-19): sc-7804

SANTA CRUZ BIOTECHNOLOGY

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 of SMN (survival of motor neuron) gene. SMN, also known as Gemin1, SMN1, SMNT and BCD541, exists as four isoforms produced by alternative splicing. SMN is oligomeric and forms a complex with Gemin2 (formerly SIP1), 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. The SMN complex is found in both the cytoplasm and the nucleus. The nuclear form is concentrated in subnuclear bodies called gems (for Gemini of the coiled bodies). Cytoplasmic SMN interacts with spliceosomal Sm proteins and facilitates their assembly onto U snRNAs and nuclear SMN mediates recycling of pre-mRNA splicing factors. Nearly identical telomeric and centromeric forms of SMN encode the same protein, however, only mutations in the telomeric form are associated with the disease state SMA. SMN is expresed in a wide variety of tissues including brain, kidney, liver, spinal cord and moderately in skeletal and cardiac muscle.

CHROMOSOMAL LOCATION

Genetic locus: SMN1/SMN2 (human) mapping to 5q13.2; Smn1 (mouse) mapping to 13 D1.

SOURCE

SMN (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of SMN of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-7804 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

SMN (N-19) is recommended for detection of SMN (also designated Gemin1) of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

SMN (N-19) is also recommended for detection of SMN (also designated Gemin1) in additional species, including canine, bovine and porcine.

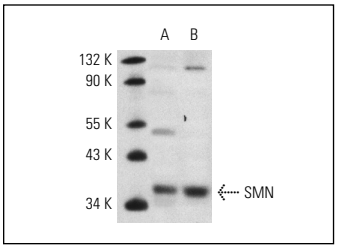
Suitable for use as control antibody for SMN siRNA (h): sc-36510, SMN siRNA (m): sc-36511, SMN shRNA Plasmid (h): sc-36510-SH, SMN shRNA Plasmid (m): sc-36511-SH, SMN shRNA (h) Lentiviral Particles: sc-36510-V and SMN shRNA (m) Lentiviral Particles: sc-36511-V.

Molecular Weight of SMN: 39 kDa.

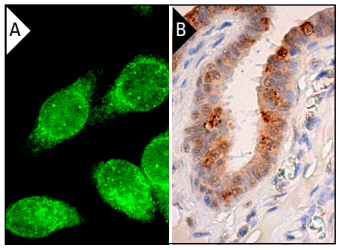
STORAGE

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

DATA



SMN (N-19): sc-7804. Western blot analysis of SMN expression in HeLa (**A**) and Hep G2 (**B**) whole cell lysates.



SMN (N-19): sc-7804. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic and nuclear staining (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human fallopian tube tissue showing cytoplasmic and nuclear staining of glandular cells (**B**).

SELECT PRODUCT CITATIONS

1. Charron, F., et al. 2001. Tissue-specific GATA factors are transcriptional effectors of the small GTPase Rho A. Genes Dev. 15: 2702-2719.
2. Hebert, M.D., et al. 2001. Coilin forms the bridge between Cajal bodies and SMN, the spinal muscular atrophy protein. Genes Dev. 15: 2720-2729.
3. Li, L., et al. 2006. Dynamic nature of cleavage bodies and their spatial relationship to DDX1 bodies, Cajal bodies, and gems. Mol. Biol. Cell 17: 1126-1140.
4. Tsai, L.K., et al. 2006. Establishing a standardized therapeutic testing protocol for spinal muscular atrophy. Neurobiol. Dis. 24: 286-295.
5. Tsai, M.S., et al. 2006. Abolishing Trp53-dependent apoptosis does not benefit spinal muscular atrophy model mice. Eur. J. Hum. Genet. 14: 372-375.

RESEARCH USE

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

PROTOCOLS

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