

Resource Summary Report

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Mouse Anti-SMN Monoclonal Antibody, Unconjugated, Clone 8

RRID:AB_397973

Type: Antibody

Proper Citation

BD Biosciences Cat# 610647, RRID:AB_397973

Antibody Information

URL: http://antibodyregistry.org/AB_397973

Proper Citation: BD Biosciences Cat# 610647, RRID:AB_397973

Target Antigen: SMN

Host Organism: mouse

Clonality: monoclonal

Comments: Immunofluorescence

Antibody Name: Mouse Anti-SMN Monoclonal Antibody, Unconjugated, Clone 8

Description: This monoclonal targets SMN

Target Organism: rat, canine, mouse, dog, human

Defining Citation: [PMID:16786553](#)

Antibody ID: AB_397973

Vendor: BD Biosciences

Catalog Number: 610647

Record Creation Time: 20250820T032120+0000

Record Last Update: 20250820T160154+0000

Ratings and Alerts

No rating or validation information has been found for Mouse Anti-SMN Monoclonal Antibody, Unconjugated, Clone 8.

No alerts have been found for Mouse Anti-SMN Monoclonal Antibody, Unconjugated, Clone 8.

Data and Source Information

Source: [Antibody Registry](#)

Usage and Citation Metrics

We found 24 mentions in open access literature.

Listed below are recent publications. The full list is available at [NIF](#).

Huang WP, et al. (2025) M6A-dependent RNA condensation underlies FUS autoregulation and can be harnessed for ALS therapy development. *Science advances*, 11(30), eadx1357.

Pagiazitis JG, et al. (2025) Catecholaminergic dysfunction drives postural and locomotor deficits in a mouse model of spinal muscular atrophy. *Cell reports*, 44(1), 115147.

Xiang T, et al. (2025) Loss of SMN Impairs Osteoblast-Osteoclast Coupling via IGF1-Akt-OPG Axis in Spinal Muscular Atrophy. *FASEB journal : official publication of the Federation of American Societies for Experimental Biology*, 39(20), e71095.

Hodgson RE, et al. (2024) C9orf72 poly-PR forms anisotropic condensates causative of nuclear TDP-43 pathology. *iScience*, 27(10), 110937.

Wang L, et al. (2024) Arginine methylation-enabled FUS phase separation with SMN contributes to neuronal granule formation. *Cell reports*, 43(8), 114537.

Hann SH, et al. (2024) Depletion of SMN protein in mesenchymal progenitors impairs the development of bone and neuromuscular junction in spinal muscular atrophy. *eLife*, 12.

Muiños-Bühl A, et al. (2023) Long-Term SMN- and Ncald-ASO Combinatorial Therapy in SMA Mice and NCALD-ASO Treatment in hiPSC-Derived Motor Neurons Show Protective Effects. *International journal of molecular sciences*, 24(4).

Oiwa K, et al. (2023) Monomerization of TDP-43 is a key determinant for inducing TDP-43 pathology in amyotrophic lateral sclerosis. *Science advances*, 9(31), eadf6895.

Kim JK, et al. (2023) A spinal muscular atrophy modifier implicates the SMN protein in SNARE complex assembly at neuromuscular synapses. *Neuron*, 111(9), 1423.

Ikenaka A, et al. (2023) SMN promotes mitochondrial metabolic maturation during myogenesis by regulating the MYOD-miRNA axis. *Life science alliance*, 6(3).

Tisdale S, et al. (2022) SMN controls neuromuscular junction integrity through U7 snRNP. *Cell reports*, 40(12), 111393.

Ravel-Chapuis A, et al. (2022) A novel CARM1-HuR axis involved in muscle differentiation and plasticity misregulated in spinal muscular atrophy. *Human molecular genetics*, 31(9), 1453.

Marasco LE, et al. (2022) Counteracting chromatin effects of a splicing-correcting antisense oligonucleotide improves its therapeutic efficacy in spinal muscular atrophy. *Cell*, 185(12), 2057.

Du LL, et al. (2022) NOVA1 promotes SMN2 exon 7 splicing by binding the UCAC motif and increases SMN protein expression. *Neural regeneration research*, 17(11), 2530.

Muinos-Bühl A, et al. (2022) Combinatorial ASO-mediated therapy with low dose SMN and the protective modifier Chp1 is not sufficient to ameliorate SMA pathology hallmarks. *Neurobiology of disease*, 171, 105795.

Quinodoz SA, et al. (2021) RNA promotes the formation of spatial compartments in the nucleus. *Cell*, 184(23), 5775.

McCormack NM, et al. (2021) A high-throughput genome-wide RNAi screen identifies modifiers of survival motor neuron protein. *Cell reports*, 35(6), 109125.

Buettner JM, et al. (2021) Central synaptopathy is the most conserved feature of motor circuit pathology across spinal muscular atrophy mouse models. *iScience*, 24(11), 103376.

Pagliarini V, et al. (2020) Combined treatment with the histone deacetylase inhibitor LBH589 and a splice-switch antisense oligonucleotide enhances SMN2 splicing and SMN expression in Spinal Muscular Atrophy cells. *Journal of neurochemistry*, 153(2), 264.

Sapaly D, et al. (2020) The Small-Molecule Flunarizine in Spinal Muscular Atrophy Patient Fibroblasts Impacts on the Gemin Components of the SMN Complex and TDP43, an RNA-Binding Protein Relevant to Motor Neuron Diseases. *Frontiers in molecular biosciences*, 7, 55.