# Resource Summary Report

Generated by RRID on Aug 3, 2026

## Mouse Anti-SMN Monoclonal Antibody, Unconjugated, Clone 8

RRID:AB_397973
Type: Antibody

### Proper Citation

BD Biosciences Cat# 610646, RRID:AB_397973

### Antibody Information

**URL:** http://antibodyregistry.org/AB_397973

**Proper Citation:** BD Biosciences Cat# 610646, 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:** 610646

**Record Creation Time:** 20250820T010336+0000

**Record Last Update:** 20250820T095531+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 RRID .


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.

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.

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

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.

Vivacqua G, et al. (2020) Motor Neurons Pathology After Chronic Exposure to MPTP in Mice. Neurotoxicity research, 37(2), 298.