Spinal muscular atrophy (SMA) is a common human autosomal recessive neurodegenerative disease that leads to the death of spinal cord motor neurons. SMA occurs with a frequency of 1 in 10,000 individuals and is the most common cause of infant mortality. SMA arises from deletion of small nuclear ribonucleoproteins (snRNPs) and spliceosomes (6, 7). In this study, we report the first evidence that SMN protein interacts with the transcription corepressor mSin3. Our deletion analysis reveals that the mSin3-interacting domain is encoded by exon 6 of SMN. In mammalian cells, SMN is expressed as the highly related mSin3A and mSin3B proteins (8). mSin3 associates with histone deacetylases (HDACs), methyltransferases and other factors to regulate the accessibility of chromatin (9–12). In light of findings from our group and others, it is possible that a subset of SMN protein may be involved in the repression of genes critical to motor neuron survival.

MATERIALS AND METHODS

Cell Culture—Human embryonic kidney 293 cells were cultured in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal bovine serum (Invitrogen), 1% L-glutamine, and 1% penicillin/streptomycin (Invitrogen) at 37 °C under 5% CO2.

Plasmids—Myc-epitope-tagged expression plasmid pCS2-MT-SMN was generated by cloning full-length SMN cDNA in-frame into the EcoRI-XbaI sites of the pCS2-MT vector. SMN deletion mutants lacking exons 1, 5, 6, or 7 were generated by polymerase chain reaction and cloned in-frame into the same EcoRI-XbaI sites of pCS2-MT vector. Point mutations S262I, Y272C, T274I, G275S, and G279V were introduced into full-length SMN cDNA by the Gene Editor™ site-directed mutagenesis kit (Promega). For generation of pCS2-Gal4-MT-SMN, the Gal4 DNA-binding domain was cloned in-frame into the unique Clai site within the pCS2-MT-SMN vector.

Transfection and Luciferase Assay—Two duplicate wells of 65% confluent 293 cells in 6-well plates were transfected with 500 ng of firefly luciferase reporter pG5-SP1-Luc (9), pG5-Elb-Luc, (13) or pG4–14D-Luc (14), 100 ng of pCS2-Gal4-MT-SMN, and 10 ng of pCMV-β-gal (30 ng of pRL-SV40), plus 30 μl of DOTAP transfection reagent (Roche Applied Science). 48 h later, cells were assayed for firefly luciferase activity on a TD-20e luminometer (Turner Designs, Sunnyvale, CA). To assess inhibition of histone deacetylases, cells were treated with trichostatin A (TSA) at different concentrations 1 day before the luciferase assay. Transfection was repeated at least three times; the luciferase activity was normalized to the internal β-galactosidase or Renilla luciferase control and then compared with the luciferase activity from cells transfected with the pCS2-Gal4-MT empty vector.

Antibodies—The rabbit polyclonal anti-mSin3A (K-20), anti-mSin3B (AK-12), anti-HDAC2 (C-19), and an unrelated rabbit polyclonal anti-hemagglutinin (HA) epitope (Y-11) were purchased from Santa Cruz Biotechnology (Santa Cruz, CA). The mouse monoclonal anti-SMN anti-
The 9E10 mouse monoclonal anti-Myc antibody was purchased from Sigma.

Immunoprecipitation and Western Blotting—1/100 293 cells were
lysed in 0.4 ml buffer A (10 mM Tris, pH 7.4, 100 mM NaCl, 2.5 mM
MgCl2, 0.5% Triton X-100, 10 mM dithiothreitol) supplemented with
protease inhibitor and phosphatase inhibitor mixture (Sigma). 25/100
ml of the respective antibodies were incubated with 40/100 ml of protein A/G
agarose (Santa Cruz Biotechnology) for 30 min at 4°C in 0.2 ml of
buffer A. The antibody-protein A/G-agarose complex was then incu-
badated with 0.25 ml of fresh cell lysate for 6 h at 4°C on a rotating
wheel. After one 10-min wash with buffer A and two washes with
RIPA buffer (50 mM Tris, pH 7.4, 150 mM NaCl, 1% Nonidet P-40,
0.5% sodium deoxycholate, 0.1% SDS), 40 µl of 1× SDS sample buffer
was added to the agarose beads and denatured at 96°C for 5 min. 10
µl of the sample was loaded onto an SDS-10% polyacrylamide gel.
After separation and transfer to a polyvinylidene difluoride PVDF
membrane, the protein samples were blotted with anti-SMN, anti-
mSin3A, or anti-HDAC2 antibodies. Protein bands were
visualized using the ECL Western blotting analysis system
(Amersham Biosciences).

Gel Filtration—9.4 ml of frozen lysate from 293 cells expressing
wild-type or mutant SMN protein was thawed, loaded onto a Superose
6 10/300 GL high performance column and size-fractionated with the
eletion buffer (10 mM Tris, pH 7.4, 100 mM NaCl, 2.5 mM MgCl2) using
a fast protein liquid chromatography system (Amersham Biosciences). The
cluent was collected at 0.5 ml/fraction, separated by SDS-PAGE,
and blotted with an anti-SMN or anti-Myc antibody to detect fractions
containing endogenous and Myc-epitope-tagged SMN proteins. The
molecular sizes of protein complexes in the collection were calculated
according to a chromatogram of standard markers provided by the
manufacturer.

RESULTS

Endogenous SMN Interacts with mSin3/HDAC—A schematic
representation of human SMN protein. Localizations of
SMA-associated missense mutations are indicated. SMN is
a ubiquitously expressed protein (15) and is abun-
dant in 293 cells (Fig. 1B, lane 1, upper panel). To
determine whether SMN protein is associated with mSin3
in vivo, 293 whole-cell lysate was immunoprecipitated with the rabbit
K-20 anti-mSin3A antibody (Fig. 1B, lane 2) or with the rabbit
AK-12 anti-mSin3B antibody (Fig. 1B, lane 3). When
the immunoprecipitates were blotted with a mouse mono-
clonal anti-SMN antibody, it was found that both anti-
mSin3A and anti-mSin3B antibodies were able to coimmuno-
precipitate endogenous SMN protein. To ensure that this
SMN-mSin3 interaction is specific, an unrelated rabbit poly-
clonal Y-11 anti-HA antibody was used in the same experi-
ment and failed to coimmunoprecipitate SMN (Fig. 1B,
lane 4).

Because mSin3 is known to complex with other histone mod-
ification enzymes (9, 14, 16), we carried out experiments to
address whether SMN can also associate with mSin3-interact-
ing proteins such as HDACs. Lysate from 293 cells (Fig. 1C,
lane 1) was immunoprecipitated with a goat polyclonal anti-
HDAC2 (Fig. 1C, lane 2) or a goat IgG as a negative control (lane 3),
then blotted with anti-SMN (top panel), anti-mSin3A (middle panel), and
anti-HDAC2 (bottom panel). Western blotting revealed
that the anti-HDAC2 antibody coimmunoprecipitated both mSin3A and SMN proteins, whereas the control goat IgG did not bring down either of them in the experiments. These findings suggest that a subset of SMN forms stable intracellular complexes with mSin3/HDAC.

**SMN-mSin3A Interaction Requires the Exon 6 Region of SMN Protein**

In the next experiment, we sought to map the SMN subdomain(s) responsible for interacting with mSin3. Full-length SMN and SMN deletion mutants lacking exons 1, 5, 6, or 7 were cloned into the mammalian expression vector, pCS2-MT. A schematic representation of SMN deletion mutants is shown (Fig. 2A). After transfection of 293 cells with these SMN deletion constructs, the resultant lysates were incubated with K-20 antibody which can coimmunoprecipitate endogenous mSin3A and endogenous SMN (Fig. 1B). Myc-tagged full-length SMN and its deletion mutants were expressed in 293 cells, as confirmed by Western blotting (Fig. 2B, lanes 2–6, upper panel). As expected, Myc-tagged full-length SMN was shown to associate with mSin3A (Fig. 2B, lane 2, middle panel). Removal of the N-terminal region encoded by exon 1 did not have a significant effect on the interaction between SMN and mSin3, as demonstrated by the presence of Myc-SMN-Δ1 in the anti-mSin3A immunoprecipitate (Fig. 2B, lane 3, middle panel). Although internal deletion of exon 5 did not affect mSin3A association with Myc-SMN-Δ5 (Fig. 2B, lane 4, middle panel), internal deletion of exon 6 abolished SMN-mSin3A interaction, as demonstrated by the absence of Myc-SMN-Δ6 from the anti-mSin3A immunoprecipitate (Fig. 2B, lane 5, middle panel). Myc-SMN-Δ7 was still capable of binding to mSin3A at a level comparable with full-length Myc-SMN (Fig. 2B, lane 6, middle panel). Thus, it seems that SMN region of amino acids 241–278 encoded by exon 6 is the SMN subdomain that mediates association with mSin3A. To show the presence of endogenous mSin3A, the immunoprecipitates were blotted with the K-20 anti-mSin3A antibody (Fig. 2B, lanes 1–6, lower panel).

**Wild-type SMN Represses Transcription in an HDAC-dependent Manner**

As a well documented transcription corepressor, mSin3A is known to exert its repressive effects by means of association with histone deacetylase, histone methyltransferase, as well as other chromatin remodeling factors (9–12). Because our data showed that SMN protein could interact with mSin3/HDAC, we tested whether directing SMN to a particular promoter would inhibit transcription through recruitment of mSin3A and its associated HDAC activity. Because SMN has not been reported to bind to DNA in a sequence-specific manner, we constructed the expression plasmid pCS2-Gal4-MT-SMN in which the Gal4 DNA-binding domain is fused to the Myc-SMN protein. 293 cells were then transfected with pCS2-Gal4-MT-SMN plus a luciferase reporter containing five consensus Gal4-binding sites and an SP1 site in the promoter region (Fig 3A). When compared with the pCS2-Gal4-MT vector, expression of Gal4-Myc-SMN resulted in repression of the reporter gene activity by more than 70% (Fig. 3B, compare columns 1 and 2). Furthermore, this repressive function of Gal4-Myc-SMN seems to require HDAC activity because the repression was significantly relieved by treatment of the cotransfected cells with TSA, a well known in-
The firefly luciferase activities were measured and normalized to untreated or were treated with TSA at the designated concentrations. The transfected cells were left untreated or were treated with TSA at the designated concentrations. The luciferase reporter gene are indicated. pG5-SP1-Luc reporter gene construction was cotransfected into 293 cells either with pCS2-Gal4-MT empty vector or pCS2-Gal4-MT-SMN. The transfected cells were left untreated or were treated with TSA at the designated concentrations. The firefly luciferase activities were measured and normalized to Replin II luciferase controls, then compared with the empty vector (bottom panel). Results of five experiments are presented as mean ± S.D.

To determine the minimal region(s) required for transcriptional repression, the same Gal4 DNA-binding domain was fused in-frame to SMN deletion mutants and then cotransfected with the pG5-SP1-Luc reporter construct. SMN mutants lacking exons 1, 5, or 7 repressed transcription from the reporter gene as strongly as the wild-type SMN. In contrast, SMN mutant lacking exon 6 no longer possessed such repressive activity (Fig. 4, top panel). To rule out the possibility that the observed differences were due to inconsistency in transfection and/or protein expression, 293 lysates expressing these Gal4-Myc-SMN mutants were separated by SDS-PAGE for protein analysis. Western blotting showed that the Gal4-fused SMN proteins were expressed at comparable levels in all these samples (Fig. 4, bottom panel), thus repression of the reporter gene by Gal4-fused SMN is unlikely to be an artifact. Instead, these results suggest that targeting of SMN to a promoter can result in recruitment of mSin3 by means of the exon 6 subdomain of SMN, and that subsequent repression of the reporter gene is mediated, at least in part, through TSA-sensitive histone deacetylases.

SMA-associated Mutation Y272C May Affect SMN-mSin3A Interaction—It is known that homozygous deletion of the telomeric SMN is responsible for SMA in 94% of cases. About 4% of patients with SMA retain at least one allele of the telomeric SMN (1). In these cases, intragenic mutations are thought to be the cause of SMA. At the present time, several SMA-related missense mutations are known to be within or immediately adjacent to the exon 6 region. These mutations include S262I, Y272C, T274I, G275S, and G279V (Fig. 1A). Among these mutations, Y272C is the most frequently occurring one that may be associated with type I SMA (19).

To investigate the binding of these mutants to mSin3A, we performed site-directed mutagenesis to introduce these point mutations into wild-type SMN. 293 cells were then transfection with the resultant missense mutation constructs, and the lysates were subjected to immunoprecipitation with the K-20 anti-mSin3A antibody. Western blotting showed that the expression levels of Myc-SMN missense mutants were comparable for all constructs (Fig. 5, lane 1-7, upper panel). The amount of Myc-SMN-Y272C that was immunoprecipitated with K-20 anti-mSin3A antibody was lower than that of Myc-SMN protein (Fig. 5A, compare lanes 1 and 3, middle panel). However, the decrease of Myc-SMN-Y272C in the immunoprecipitate was not as complete as Myc-SMN-Δ6 (Fig. 5A, compare lanes 3 and 7, middle panel). There was no significant change in binding of other mutant proteins to mSin3A (Fig. 5, lanes 2, 4, 5, 6, middle panel). Taken together, these data suggest that the SMA-associated Y272C missense mutation may have an adverse effect on SMN-mSin3A interaction, whereas other SMN missense mutations have no appreciable effects on the binding under our experimental conditions.

To investigate whether SMN missense mutations also affect repression of the luciferase reporter gene, we constructed expression plasmids by fusing Gal4 DNA-binding domain to these SMN missense mutants. Compared with the wild-type protein, these SMN missense mutations showed minimal effects on luciferase activity and, therefore, did not seem to cause derepression of the reporter gene (Fig. 5B). To examine whether potential derepression by the Y272C missense mutation could be obtained with a different promoter construct, we tested
another luciferase reporter, pG4–14D-Luc, which contains four consensus Gal4-binding sites upstream of the 14D promoter (14). When pG4–14D-Luc was co-transfected into 293 cells, 10-fold repression of the luciferase activity by Gal4-Myc-SMN was observed (Fig. 5C, compare the first two columns). However, none of the five SMN missense mutations had any effect.

**Fig. 5. Analysis of mSin3A interaction with SMN missense mutations.** A, 293 cells were transfected with pCS2-MT-SMN missense mutants, and the lysates (top panel) were immunoprecipitated with the anti-mSin3A antibody. The immunoprecipitates were blotted with the anti-Myc antibody to detect the Myc-SMN missense mutants (middle panel) or blotted with the K-20 antibody to show the existence of mSin3A (bottom panel). B, cells were transfected with pG5-SP1-Luc reporter construct and pCS2-Gal4-MT-SMN missense mutants. After 40 h, the firefly luciferase activities were measured and normalized to the β-galactosidase controls (top panel). The same lysates were also blotted with the anti-Myc antibody to show expression of the Gal4-Myc-SMN missense mutants (bottom panel). C, cells were transfected with pG4–14D-Luc reporter (top panel) and pCS2-Gal4-MT-SMN missense mutants. Firefly luciferase activities were measured and normalized to the Renilla luciferase controls (bottom panel). D, cells were transfected with pG5-E1b-Luc reporter (top panel) and pCS2-Gal4-MT-SMN missense mutants. Firefly luciferase activities were measured and normalized to the Renilla luciferase controls (bottom panel). All transfection experiments were repeated at least three times; results are shown as mean ± S.D.
on derepression (Fig. 5C, columns 2-7).

To investigate whether repression by Gal4-Myc-SMN can work on a TATA-containing promoter, the pG5-E1b-Luc reporter construct (13) was used in co-transfection with pCS2-Gal4-MT-SMN. This reporter construct contained five consensus Gal4-binding sites upstream of the E1b promoter. Sequence analysis revealed the presence of a TATA box ~30 bp from the transcription initiation site. Although Gal4-Myc-SMN was able to repress expression of the luciferase reporter gene driven by this TATA-containing promoter, the five SMN missense mutations did not show appreciable derepression as compared with the wild-type SMN (Fig. 5D).

**Exon 6 Region of SMN Mediates Higher Order Complex Formation**—These findings suggest that either SMN mutants do not derepress gene expression or our luciferase reporter approach is unable to assess the impairment of SMN missense mutations on potential gene repression. Previously, a cell viability and proliferation assay also failed to differentiate SMA-related missense mutants from wild-type SMN (20). If these SMA-related missense mutations indeed contribute to the pathological process of motor neuron death, they would be expected to lack at least some of the functions of wild-type SMN protein.

Interestingly, a previous in vitro study reported that the exon 6 of SMN encodes a self-oligomerization region, and we showed in this study that the same exon 6 region is important for interaction with mSin3 in vivo. These findings suggest that the exon 6 region of SMN may be involved in the formation of higher order protein complexes inside cells, and SMA-related missense mutations may be deficient in such a function. To test this hypothesis, lysate from 293 cells expressing Myc-epitope-tagged SMN, SMN-Δ6, or SMN-Δ7 was size-fractionated through a Superose column; all of the fractions were then analyzed by Western blotting. In 293 cells, endogenous SMN protein existed as multi-protein complexes exceeding 40,000 kDa. However, there are more abundant smaller Y272C-containing complexes, which peaked around 700 kDa (Fig. 6B). In comparison with the wild-type SMN, we suspect that protein supra-complexes containing the Y272C missense mutation may be less stable and easier to dissociate into smaller protein complexes under our experimental conditions.

**DISCUSSION**

Based on results from this and other studies, it seems that SMN can be a component of large protein complexes that are found both in cytoplasmic and nuclear compartments (6). Cytoplasmic SMN plays an essential role in spliceosomal snRNP biogenesis and is required for the transport of the snRNP complex into the nucleus (21). In the nucleus, SMN protein is required for regenerating an active splicing complex (22). SMN protein has been reported to associate with the nuclear transcription activator E2 of papillomavirus (23), the DEAD box protein dp103 (also called GEMIN 3) (24, 25), the SIP-1 (SMN-interacting protein-1) (26), the small nuclear RNA-associated protein fibrillarin (27), GAR1 (28), the zinc-finger protein ZPR1 (29), the Ewing’s sarcoma protein EWS (30), and RNA polymerase II (31). Finally, there is evidence that SMN may also be involved in pro- and anti-apoptotic pathways (32, 33).

**SMN** is an essential gene in diverse organisms. A reduction of the SMN protein because of deletion and/or mutation in the telomeric SMN causes SMA. Studies on SMA have suggested that motor neuron cells, for reasons still not clear, are more...
sensitive than other cell types to an insufficient amount of SMN protein. It is not known which function(s) carried out by SMN is especially critical to the survival of motor neurons.

In this work, we have studied protein interactions between SMN and mSin3, a transcription corepressor involved in histone deacetylation and chromatin remodeling. We have found that interaction of SMN with mSin3 is dependent upon an intact exon 6 region of SMN. Interestingly, an earlier study noted that a previous study (20) also encountered a similar difficulty in differentiating SMN missense mutants from the wild-type protein in a proliferation assay of an SMN-depleted chicken pre-B cell line. In the same study, the effect of exon 6 deletion was not demonstrated, this may reflect the difficulty in correlating the SMA-associated missense mutations with aberrant cellular function using non-motor neuron cells. It should be noted that a previous study (20) also encountered a similar difficulty in differentiating SMN missense mutants from the wild-type protein in a proliferation assay of an SMN-depleted chicken pre-B cell line. In the same study, the effect of exon 6 deletion was dramatically different, as the SMN-Δ6 mutant was no longer able to support the survival of the SMN-depleted cells.

The telomeric SMN gene differs from the centromeric SMN gene by a single nucleotide, which results in expression of centromeric SMN transcripts that typically lack exon 7. The exon 7-deficient protein product of the centromeric SMN gene is unstable and rapidly degraded (1, 4). In our transient transfection studies, we successfully over-expressed SMN mutant that harbors deletion of the exon 7. It is likely that this is because of the stabilizing effect of the Myc-epitope tag on the SMN moiety.

Recent studies have demonstrated that the transcription coactivator CBP is trapped by polyglutamine repeats and that the resultant CBP (a histone acetyltransferase)-depletion may be linked to the neurodegenerative phenotype of Huntington's disease (35). On the other hand, our results suggest that SMN protein may functionally interact with the transcription corepressor mSin3 and its associated histone deacetylases; SMN-Δ6 deletion completely abolishes this interaction, whereas the Y272C missense mutation may impair it. In addition to 293 cells, we have carried out the same experiments in other types of cells and obtained similar results (data not shown). Although further experiments are needed, we postulate that SMN may be involved in transcriptional repression of critical genes in motor neuron cells of the spinal cord, possibly by means of mSin3-associated histone modification enzymes.

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REFERENCES

1. Lefebvre, S., Burglen, L., Reboulet, S., Clermont, O., Burlet, P., Viollet, L., Benichou, B., Cruaud, C., Millasseau, P., and Zeviani, M. (1995) Cell 80, 155–165
2. Nicole, S., Diaz, C. C., Frager, T., and Melki, J. (2002) Muscle Nerve 26, 4–13
3. Rudnik-Schoneborn, S., Forkert, R., Hahnne, E., Wirth, B., and Zierz, K. (1996) Neuropediatrics 27, 8–15
4. Lorson, C. L., Hahnen, E., Androphy, E. J., and Wirth, B. (1999) Proc. Natl. Acad. Sci. U. S. A. 96, 6307–6311
5. Monani, U. R., Lorson, C. L., Parsons, D. W., Prior, T. W., Androphy, E. J., Burghes, A. H., and McPherson, J. D. (1999) Hum. Mol. Genet. 8, 1177–1183
6. Liu, Q., Fischer, U., Wang, F., and Dreyfuss, G. (1997) Cell 90, 1013–1021
7. Fischer, U., Liu, Q., and Dreyfuss, G. (1997) Cell 90, 1025–1029
8. Ayer, D. E., Lawrence, Q. A., and Eisenman, R. N. (1995) Cell 90, 767–776
9. Yang, L., Mei, Q., Zielieksa-Kwiatowska, A., Matsui, Y., Blackburn, M. L., Benedetti, D., Krumm, A. A., Taborsky, G. J., Jr., and Chansky, H. A. (2003) Biochem. J. 369, 651–657
10. Wysocka, J., Myers, M. P., Laherty, C. D., Eisenman, R. N., and Herr, W. (2003) Genes Dev. 17, 896–911
11. Shi, S., Saurin, A. J., Imbalzano, A. N., and Kingston, R. E. (2001) Genes Dev. 15, 603–618
12. Zhang, Y., and Dufau, M. L. (2002) J. Biol. Chem. 277, 33431–33438
13. Zhang, Y., Woodford, N., Xia, X., and Hambarger, A. W. (2003) Nucleic Acids Res. 31, 2168–2177
14. Laherty, C. D., Yang, W. M., Sun, J. M., Davie, J. R., Seto, E., and Eisenman, R. N. (1997) Cell 89, 349–358
15. Coovert, D. D., Le, T. T., McAndrew, P. E., Strasswimmer, J., Crawford, T. O., Mellott, J., Brown, J. N., Langer, K. D., and Shiekhattar, R. (1998) Cell 93, 517–526
16. Zhang, Y., Iratni, R., Erdjument-Bromage, H., Tempst, P., and Reinberg, D. (1997) Cell 90, 357–364
17. Marks, P. A., Rifkind, R. A., Richon, V. M., and Breslow, R. (2001) Clin. Cancer Res. 7, 759–760
18. Zhang, Y., Xie, X., and Hambarger, A. W. (2003) Biochemistry 42, 15152–15160
19. Wirth, B. (2000) Hum. Mol. Genet. 15, 2263–2276
20. Wang, G., and Dreyfuss, G. (2001) J. Biol. Chem. 276, 45387–45393
21. Meister, G., Buhler, D., Lageragger, B., Zobawa, M., Lottspeich, F., and Fischer, U. (2000) Hum. Mol. Genet. 9, 1977–1986
22. Pellizzoni, L., Kataoka, N., Charroux, B., and Dreyfuss, G. (1998) Cell 95, 615–624
23. Strasswimmer, J., Lorson, C. L., Breiding, D. E., Chen, J. J., Le, T., Burghes, A. H., and Androphy, E. J. (1999) Hum. Mol. Genet. 8, 1219–1226
24. Campbell, L., Hunter, K. M., Mogahpeghi, P., Tinsley, J. M., Brasch, M. A., and Davies, K. E. (2000) Hum. Mol. Genet. 9, 1093–1100
25. Charroux, B., Pellizzoni, L., Perkinson, R. A., Shevchenko, A., Mann, M., and Dreyfuss, G. (1999) J. Cell Biol. 147, 1181–1194
26. Young, P. J., Man, N. T., Lorson, C. L., Le, T. T., Androphy, E. J., Burghes, A. H., and Morris, G. E. (2000) Hum. Mol. Genet. 9, 2669–2677
27. Jones, K. W., Gorzynski, K., Hales, C. M., Fischer, U., Badbanchi, F., Terns, M. P. (2002) J. Biol. Chem. 277, 38645–38651
28. Whitehead, B. E., Jones, K. W., Zhang, X., Cheng, X., Terns, M. P., and Terns, M. P. (2002) J. Biol. Chem. 277, 48087–48093
29. Gangwani, L., Mikrut, M., Theroux, S., Sharma, M., and Davis, R. J. (2001) Nat. Cell. Biol. 3, 376–383
30. Young, P. J., Francis, J. W., Lince, D., Coon, K., Androphy, E. J., and Lorson, C. L. (2003) Brain Res. Mol. Brain Res. 119, 37–49
31. Pellizzoni, L., Charroux, B., Rappaport, J., Mann, M., and Dreyfuss, G. (2001) Cell Biol. 152, 75–85
32. Iwahashi, H., Kguchi, Y., Yasuhara, N., Hanafusas, T., Matsuwasu, Y., and Tsujimoto, Y. (1997) Nature 390, 413–417
33. Kerr, D. A., Nery, N. P., Truscott, R. J., Chau, B. N., and Hardwick, J. M. (2000) Proc. Natl. Acad. Sci. U. S. A. 97, 13312–13317
34. Lorson, C. L., Strasswimmer, J., Yao, J. M., Baleja, J. D., Hahnne, E., Wirth, B., Le, T., Burghes, A. H., and Androphy, E. J. (1998) Nat. Genet. 19, 63–66
35. Steffen, J. S., Bodai, L., Pallos, J., Poelman, M., McCambell, A., Apostol, B. L., Kazantsev, A., Schmidt, E., Zhu, Y. Z., Greenwald, M., Kurokawa, R., Housman, D. E., Jackson, O. R., Marsh, J. L., and Thompson, L. M. (2001) Nature 413, 730–743
Survival Motor Neuron (SMN) Protein Interacts with Transcription Corepressor mSin3A
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