**Supplementary Figures and Table**

**Figure S1. Two distinct *smn-1* RNAi feeding bacterial strains reduce the expression of an SMN-1::GFP fusion protein and cause similar changes in *tos-1* splicing.**

(A) Transgenic animals expressing either GFP or an SMN-1::GFP fusion protein in the body-wall muscles were treated with two distinct *smn-1* RNAi feeding bacterial strains and the expression of GFP was observed under a fluorescent microscope. Both RNAi clones strongly reduced the expression of SMN-1::GFP but not that of GFP. Scale bar: 0.2 mm. (B) Western blot showing that the expression of the SMN-1::GFP fusion protein was significantly reduced by the RNAi clones. (C) Both RNAi clones similarly altered the splicing of *tos-1*. W and O: *smn-1* RNAi clones obtained from a whole-genome RNAi library [38](#_ENREF_38) and an ORFeome RNAi library,[39](#_ENREF_39) respectively.

**Figure S2. *smn-1(ok355**)* enhances the increased *tos-1* intron 1 retention caused by *uaf-1(n4588)*.**

(A) RT-PCR experiments examining *tos-1* splicing in different mutants.Genotypes are labeled on top. (B) RT-PCR experiments examining the effects of different mutations on the recognition of the cryptic 3’ splice site of *tos-1* intron 1.

**Figure S3. Body length analysis of mutant animals.**

(A) Pictures of animals with the indicated genotypes on day 3 and day 5 post the L1 larval stage. Scale bar: 1 mm. (B) Body length quantification of the mutants. 15 animals were measured for each genotype. Statistics: difference between the two compared datasets. \*\*: p<0.01 (Student’s TTEST). Bars: standard deviations.

**Figure S4. Independent survival trials indicate that *uaf-1(mut)* extends the lifespans of *smn-1(ok355**)* mutants.**

Figure 3A of the main text is the average of A, B and C, Figure 3B is the average of D and E, Figure 3C is the average of F, G and H and Figure 3D is the average of I and J. Statistics: difference between two lifespan curves. p<0.001 (logrank test) between *smn-1(ok355**)* mutants and *smn-1(ok355**); uaf-1(mut)* double mutants for each individual trial.

**Figure S5. Reducing the expression of *uaf-1* by RNAi does not rescue the locomotion or lifespan defects of *smn-1(ok355**)* mutants.**

(A) P0 animals at the young adult stage were fed HT115 *E. coli* expressing either control RNAi or dsRNA targeting *uaf-1* [31](#_ENREF_31), respectively. On day 6 and day 7, bodybends of F1 progeny were scored. At least 20 animals were scored for each genotype. Statistics: difference between the two compared datasets. N.S.: no significant difference (Student’s TTEST). Bars: standard errors. (B) The survival of F1 progeny was monitored starting on day 6 post the RNAi feeding. *uaf-1(RNAi)* did not obviously increase the lifespan of *smn-1(ok355**)* mutants. 50 animals were scored for each genotype.

**Figure S6. RT-PCR analysis of U6 snRNA expression.**

To verify the dramatic changes of U6 snRNA expression as detected by qRT-PCR (Fig. 6), we amplified U6 cDNAs at two different PCR cycles using the same aliquots of total cDNA templates and examined the amplified DNA on 2% agarose gels. The results indicate an obvious increase of U6 in *smn-1(ok355**)* and *smn-1(ok355**); uaf-1(n4588)* mutants on (A) day 3 and (B) day 5 post the L1 larval stage. Two biological replicates of each genotype were examined.

**Figure S7. Gonad development of mutant animals.**

To examine whether *smn-1(ok355**); uaf-1(mut)* double mutants had an apparent developmental delay, we identified gonads of the animals on day 2.5 post the L1 larval stage (dotted red lines). *smn-1(ok355**)* single mutants and *smn-1(ok355**); uaf-1(n4588 n5127)* double mutants had similar gonadal development and body size, suggesting a similar postembryonic development. The smaller gonad in *smn-1(ok355**); uaf-1(n4588)* double mutants was probably caused by the *uaf-1(n4588)* mutation, which by itself causes severe gonad developmental defects (X. Gao and L. Ma, unpublished observations) [31](#_ENREF_31). These results suggest that the longer lifespan and improved motor functions of *smn-1(ok355**); uaf-1(mut)* double mutants are likely not caused by delayed or arrested development of these animals. Scale bar: 0.1 mm.

**Table S1. List of PCR primers.**

|  |  |  |
| --- | --- | --- |
| Gene |  | Primer sequences |
|  |  |  |
| 18S *rRNA* | Forward | TAGTGAGACGCCCAACAGC |
|  | Reverse | TGGCATCGTTTACGGTCAG |
| U1 *snRNA* | Forward | CGGAATCCCCATGGTGAG |
|  | Reverse | CGATACGCAAAAATTAAGCTG |
| U2 *snRNA* | Forward | CGCTTCTTCGGCTTATTAG |
|  | Reverse | GGACGACCCTTGGGAAGT |
| U4 *snRNA* | Forward | GCGATAACGTGACCAATGAG |
|  | Reverse | GTATGCCACCCATGTTTCAG |
| U5 *snRNA* | Forward | GTTCCTCTGCATTTAACCGTG |
|  | Reverse | CAAGGGGACTCCAAAAATTGTAT |
| U6 *snRNA* | Forward | TTCCGAGAACATATACTAAAATTGG |
|  | Reverse | AAAATTTGGAACGCTTCACG |
| *plst-1* | Forward | TGTATGCTGATCTTCAAAATGGTGTC |
|  | Reverse | AGCTTATGGAATGTCCGTACAACTC |
| *bus-19* | Forward | ATGACACGACTCTTCATACTCCCTG |
|  | Reverse | TTATACAATTTCCATCTTTTTGACTTTCTCG |
| *Y71A12C.2* | Forward | ATGCTGCGAATTTGCGGCTTG |
|  | Reverse | CTAGGAAACTTTAACTCCATGCAGCTCATC |
| *tag-175* | Forward | ATGGAAACAAAATCATCCCAAACTTC |
|  | Reverse | TTAATCCGATTTGAGCTTCTTCTTGAG |
| *act-1* (loading control for *stasimon* homologs) | Forward | CAATCCAAGAGAGGTATCCTTACC |
|  | Reverse | AGCGGTGGTGGTGAAAGAGT |