

Differential induction of muscle atrophy pathways in two mouse models of spinal muscular atrophy

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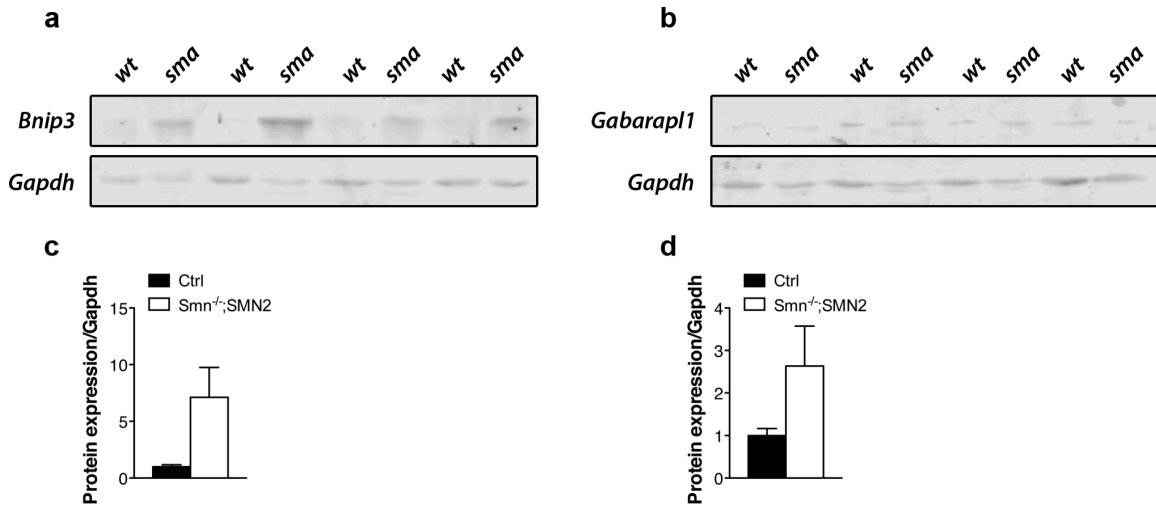
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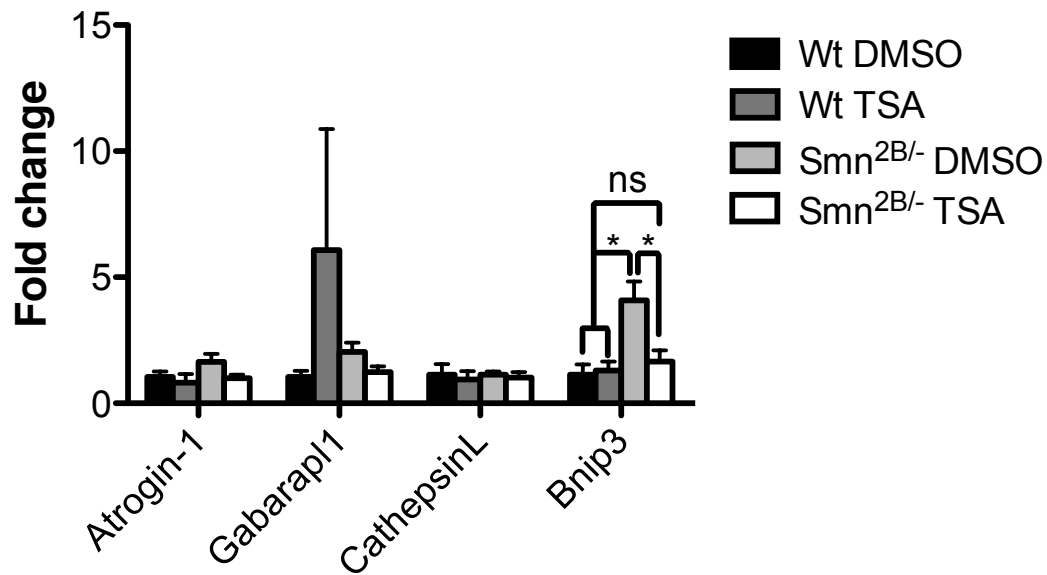
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Supplementary Table S1: List of primers used in this study and their respective annealing temperatures.

Gene target	Forward primer	Reverse primer	Ta
Atrogin-1	CGTCTCACTTTCCCCTCAAG	GACTCCCAGCCATCCAATTAG	57
MuRF1	AGTGTCCATGTCTGGAGGTCGTTT	ACTGGAGCACTCCTGCTTGTAGAT	60
GabarapL1	CATCGTGGAGAAGGCTCCTA	ATACAGCTGGCCCATGGTAG	62
Bnip3	TTCCACTAGCACCTTCTGATGA	GAACACCGCATTTACAGAACAA	60
CathepsinL	GTGGACTGTTCTCACGCTCAAG	TCCGTCCTTCGCTTCATAGG	60
FoxO1	CAAAGTACACATACGGCCAATCC	CGTAACTTGATTTGCTGTCCTGAA	60
FoxO3	CCTCATCTCAAAGCTGGGTAC	GGTTTTCTCTGTAGGTCTTCCG	63
FoxO4	AAATGCCAGCCTCGGCCAGC	GGCATTCTCCAGTAACAG	60
Gapdh	TCGGTGTGAACGGATTTG	GGTCTCGCTCCTGGAAGA	62



Supplementary Figure S1. *Smn*^{-/-};*SMN2* hearts show a trend toward increased **Bnip3** and **Gabarap11** protein levels at P5. (a) Western blot of Bnip3. (b) Western blot of Gabarap11. (c) Quantification of Bnip3 protein expression showing a trend toward elevated protein levels compared to control. (d) Quantification of Gabarap11 protein expression showing a trend toward elevated protein levels compared to control.



Supplementary Figure S2. **TSA administration to *Smn*^{2B/-} mice restores *Bnip3* expression in hearts to normal levels.** Atrogin-1, Gararapl1 and CathepsinL were unchanged as observed above. TSA treatment did not alter their expression. The increased expression of *Bnip3* was effectively brought back to wild type control levels. (N=3 for all experiments; $p \leq 0.05$ for *)