Supplemental figure 1. PRL dose optimization for SMN induction in SMA mice **model**. SMA Δ 7 mice were treated daily with PRL (0.5 and 2.5 mg/kg) from P1 for 6 days, then sacrificed at P7. Brain and spinal cord tissues were harvested Western blot analysis. (a) Representative Western blot showing effect of PRL on SMN protein in brain samples of SMA Δ 7 mice treated with Saline (control, lane 1) or PRL (0.5 & 2.5 mg/kg; lane 2 & 3 respectively) (each lane represents individual animal). (b) Representative Western blot showing effect of PRL on SMN protein in spinal cord samples of SMA Δ 7 mice treated with Saline (control, lane 1) or PRL (0.5 & 2.5 mg/kg; lane 2 & 3 respectively) (each lane represents individual animal). Supplemental figure 2. PRL treatment does not affect SMN protein level in heart tissues of SMA Δ 7 mice until time of death. SMA Δ 7 mice were treated

daily with PRL (2.5 mg/kg) from P1 onward. Heart tissues were harvested upon death for Western blot analysis. (a) Representative Western blot showing the effect of PRL on SMN protein in heart samples of SMA Δ 7 mice treated with saline (control, lane 1,2 & 3) or PRL (lane 4, 5 & 6).

Supplemental figure 3. Genotyping of transgenic mice using two different primer sets.

Supplemental figure 4. Comparison of SMN induction in SMA mice model (*mSmn-/-;hSMN2+/+, hSMNA7+/+*) after PRL treatment with carrier treated heterozygous transgenic mice (*mSmn+/-;hSMN2+/+, hSMNA7+/+*). SMAA7 and heterozygous mice were treated daily with saline or PRL (2.5 mg/kg; SMAA7 mice only) from P1 for 6 days, then sacrificed at P7. Brain and spinal cord tissues were harvested Western blot analysis. (a) Representative Western blot showing effect of PRL on SMN protein in brain samples of SMAA7 and heterozygous mice treated with Saline (control, lane 1 & 3 respectively) or PRL (2.5 mg/kg; lane 2) (each lane represents individual animal). (a) Representative Western blot showing effect of PRL on SMN protein in spinal cord samples of SMAA7 and heterozygous mice treated with Saline (control, lane 1 & 3 respectively) or PRL (2.5 mg/kg; lane 2) (each lane represents individual animal). (a) Representative Western Supplementary figure 5. Comparison of SMN induction in motor neurons in SMA mice model ($mSmn-/-;hSMN2+/+, hSMN\Delta7+/+$) after PRL treatment with carrier treated heterozygous transgenic mice (mSmn+/-;hSMN2+/+,

hSMN Δ 7+/+). SMA Δ 7 and heterozygous mice were treated daily with saline or PRL (2.5 mg/kg; SMA Δ 7 mice only) from P1 for 6 days, then sacrificed at P7. Brain tissues were harvested for Immunohistochemistry analysis. Representative merged Confocal images [SMN/alexa488 (green) + HB9/alexa 568 (red; motor neuron marker) + Hoechst (blue)] for different tissues are shown. Representative Confocal images showing effect of PRL on SMN protein expression in brain stem motor neurons samples of SMA Δ 7 and heterozygous mice treated with Saline (A & C respectively) or PRL (B). Scale bars: 10 μ M.

Supplemental videos. PRL treatment ameliorates on disease phenotype in SMA mice model. (a) P13 SMA mice (control; saline treated). (b) P21 SMA mice (PRL-treated). (c) P13 untreated heterozygous mice.









a-SMA **b-HET or WT** c-HET or WT d-HET or WT e-SMA f-HET or WT g-HET or WT h-HET or WT i-HET or WT

a-SMA **b-HET** c-WT d-HET

e-SMA

g-HET

h-HET i-HET

f-WT





