

Supplementary Materials for

Non-invasive electrical stimulation restores corneal nerve density in diabetic neuropathy by promoting axon growth in trigeminal ganglia neurons

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Figs. S1 to S7

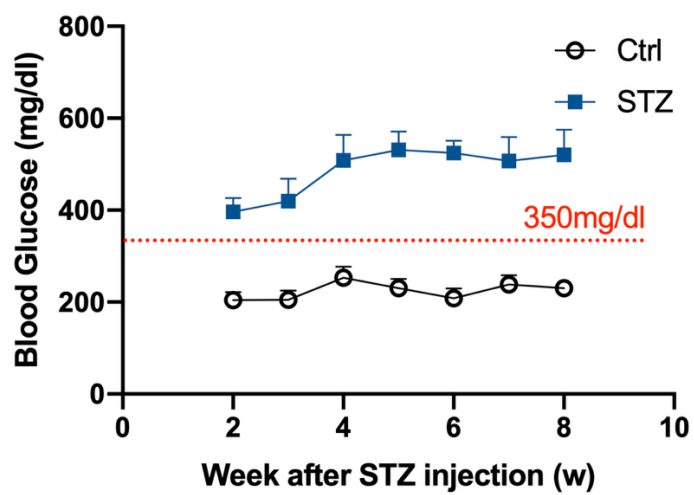
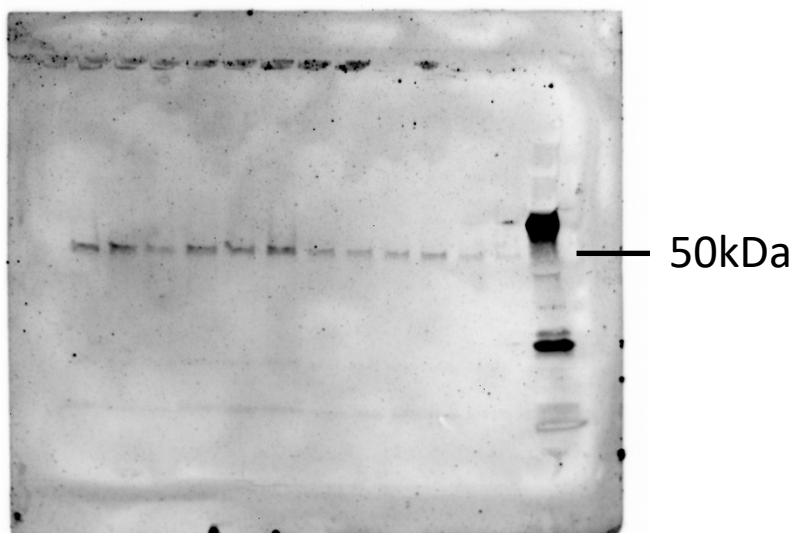


Fig. S1.

Blood glucose measurement of STZ model. Random blood glucose > 350mg/dl is regarded as diabetic.

GAP43 Raw Image



GAPDH Raw Image

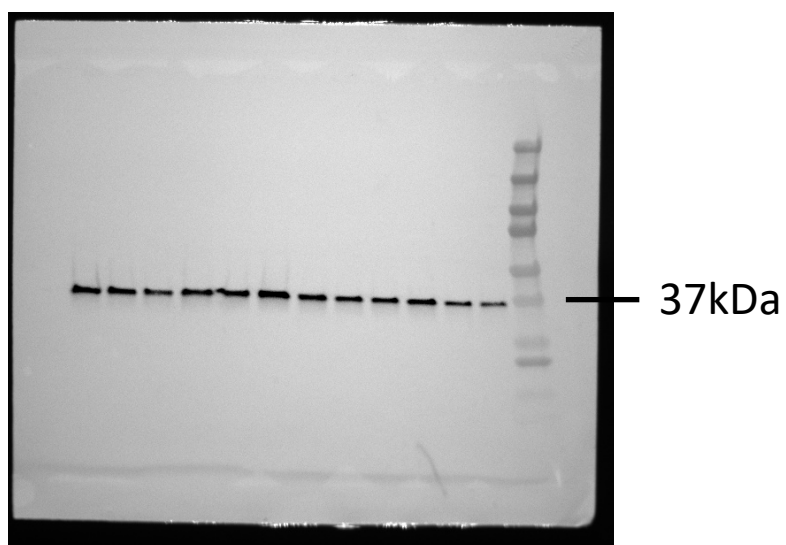


Fig. S2.

Raw Western blot image of Fig 2.

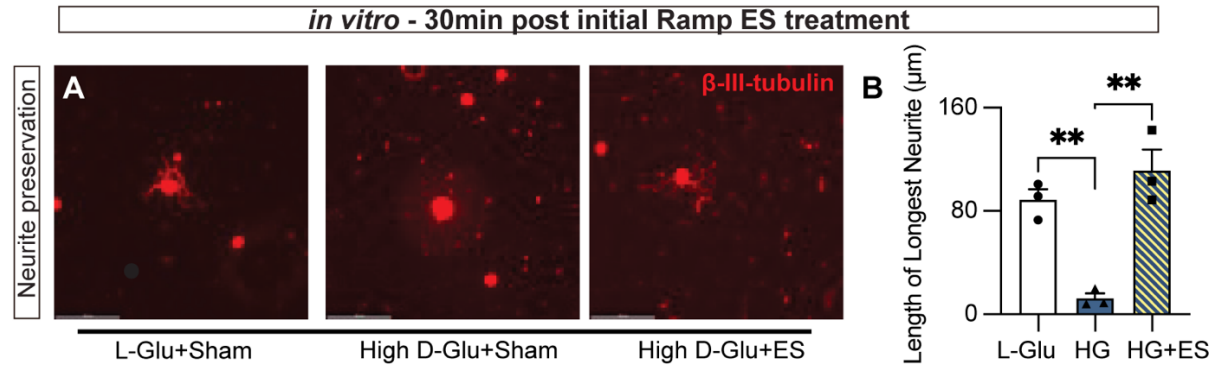


Fig. S3.

In vitro ES treatment in high glucose (100mM) condition. L-glucose 100mM served as osmolarity control (A). The length of longest neurite of quantified in (B). n=3. **, $p<0.01$.

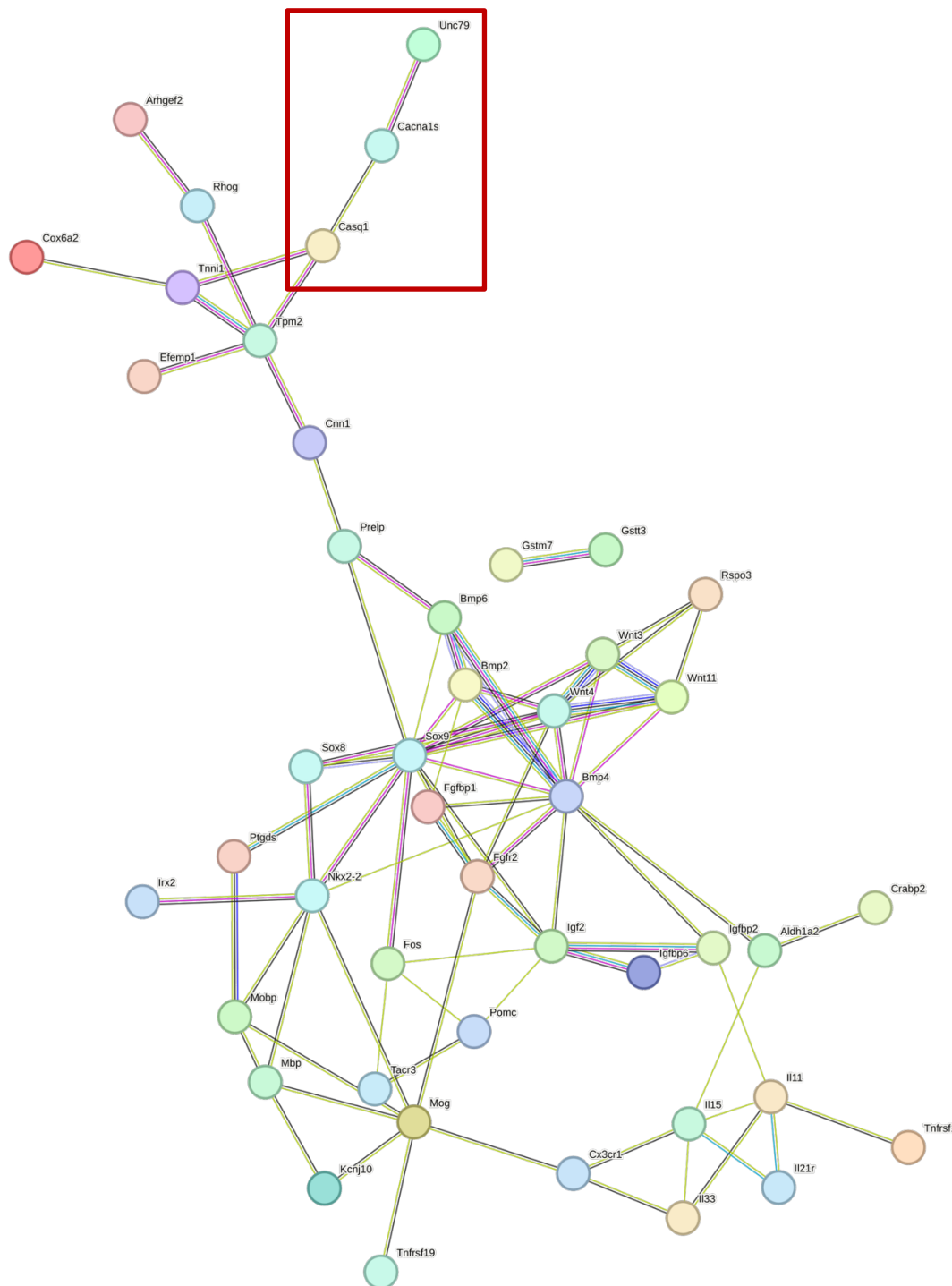


Fig. S4.

Gene network analysis of DEGs identified between the TGs from sham-treated and ES-treated keratectomy injury. Genes related to membrane potential and Ca^{2+} signaling are highlighted in the red box.

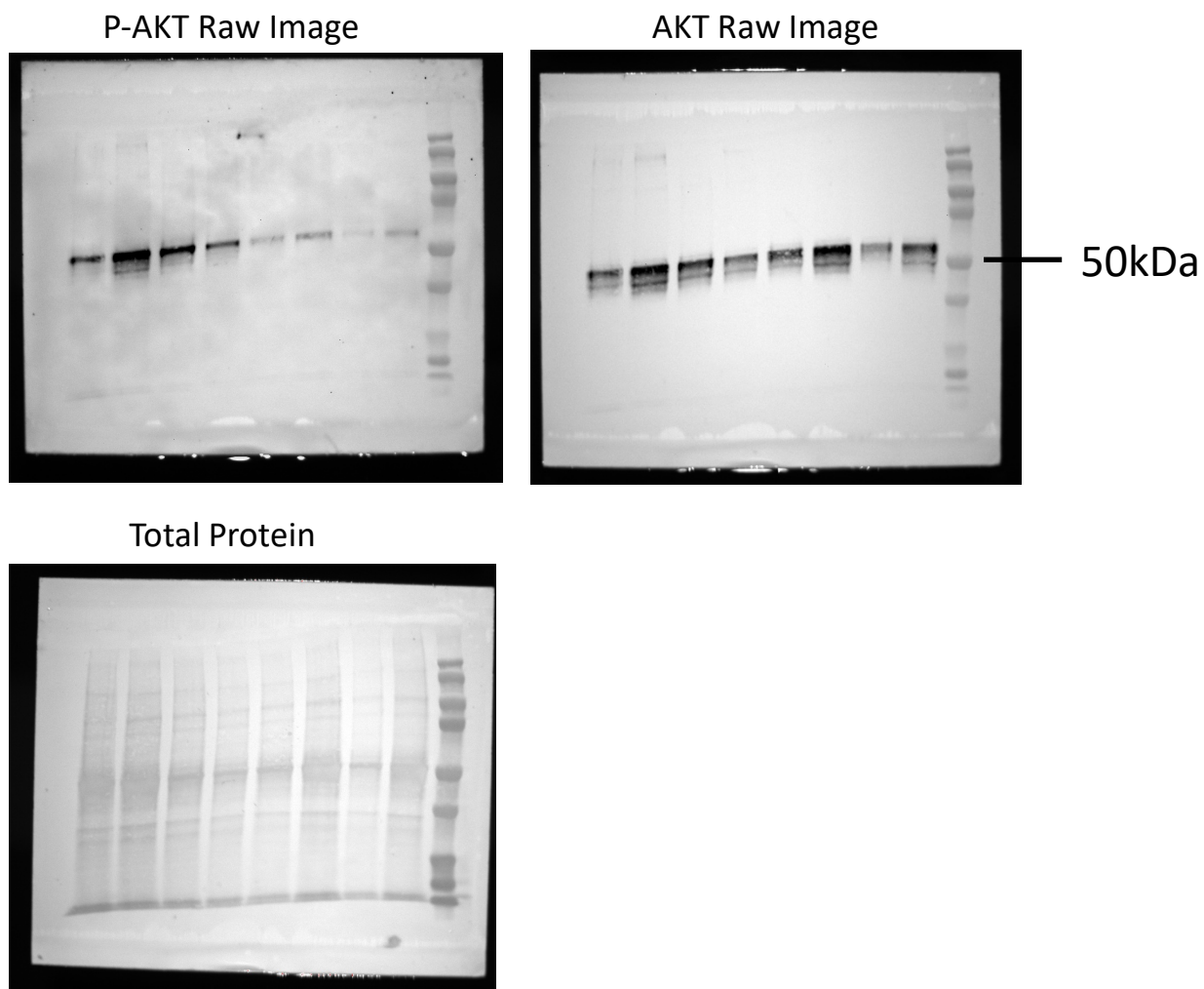


Fig. S5.
Raw Western blot image of Fig 3.

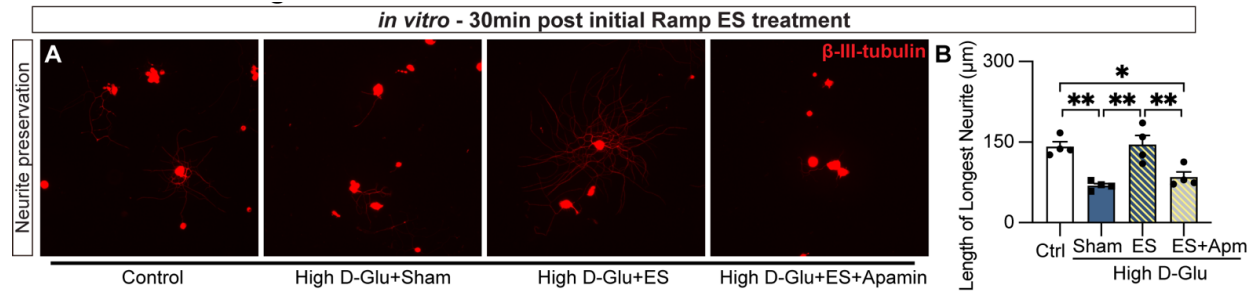


Fig. S6.

In vitro ES treatment in normal glucose control and high glucose (100mM) condition, with sham, ES, ES combined with Apamin treatments (A). The length of longest neurite of quantified in (B). n=4. *, $p<0.05$; **, $p<0.01$.

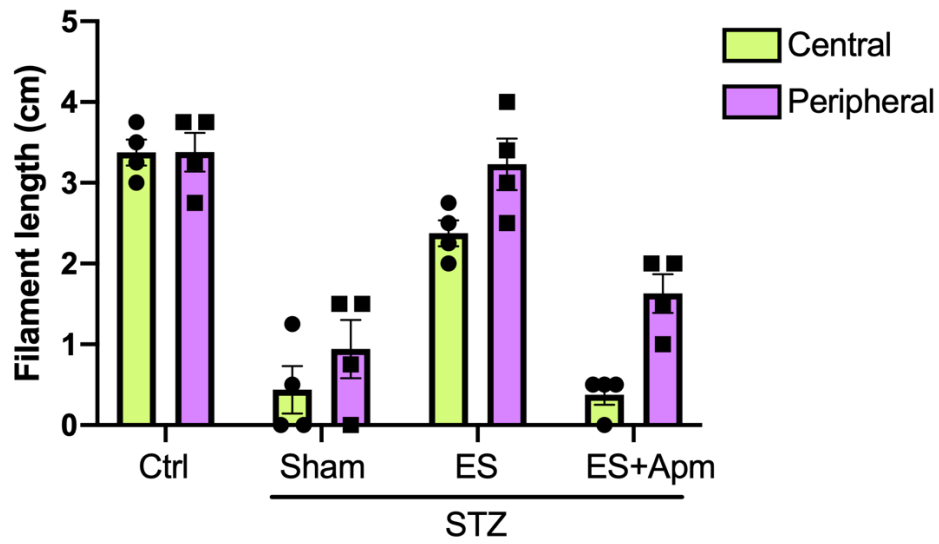


Fig. S7.

Cochet-Bonnet esthesiometer result from non-DM control, STZ-induced T1MD mice with sham treatment, ES treatment, and ES combined with Apamin, in central and peripheral cornea.