

Supplementary Materials for
4E-BP1–dependent translation in nociceptors controls mechanical hypersensitivity via TRIM32/type I interferon signaling

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The PDF file includes:

Figs. S1 to S3
Legend for data S1

Other Supplementary Material for this manuscript includes the following:

Data S1

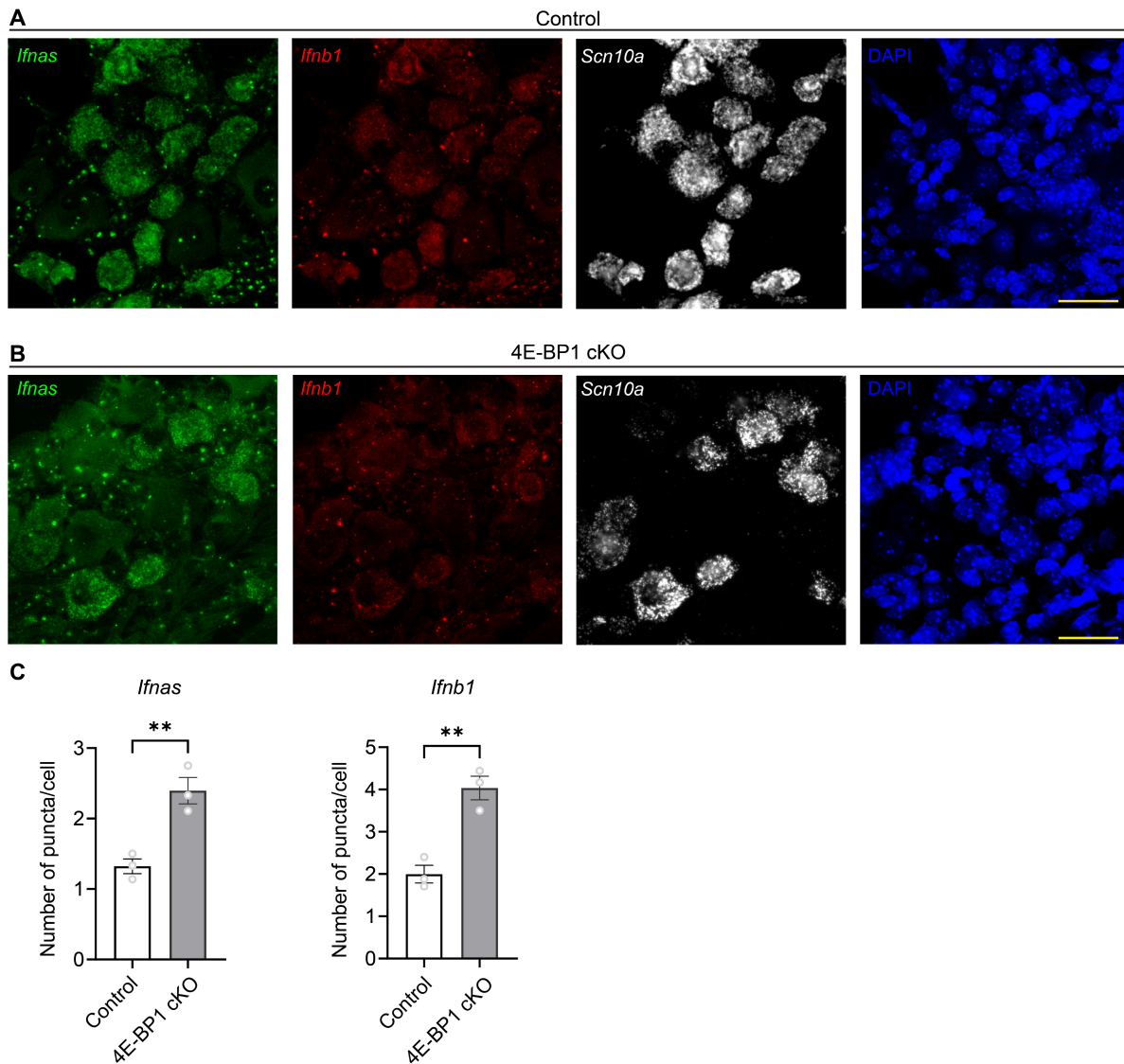


fig. S2. Type I interferon mRNAs are increased in 4E-BP1 cKO nociceptors. RNAScope images of DRG sections show increased abundance (# of puncta per cell) of *Ifnas* and *Ifnb1* mRNAs in *scn10a*-positive nociceptors of 4E-BP1 cKO mice (A) as compared to control (B) animals. (C) Quantification (n = 3 mice/group; Student's t-test, 2 tailed; scale bar: 30 μ m). All data are presented as mean \pm SEM. **P < 0.01.

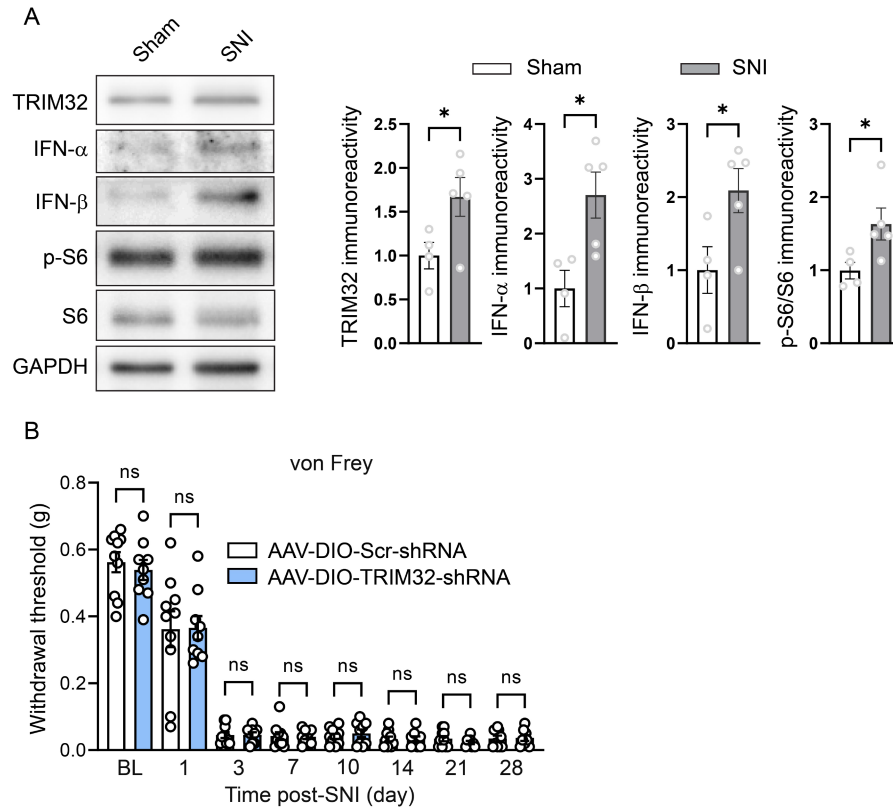


fig. S3. shRNA knockdown of TRIM32 in nociceptors does not rescue neuropathic pain. (A) Western blots and quantification showing increased TRIM32, IFN- α , IFN- β and p-S6 in DRG lysates of animals 4 days after SNI compared to sham control mice ($n = 4-5$ mice per group, Student's t-test, 2 tailed). (B) No difference in mechanical hypersensitivity in animals that received AAV-DIO-TRIM32-shRNAmir compared to scrambled control following SNI ($n = 8$ mice per group, two-way ANOVA mixed effects model followed by Bonferroni's post hoc comparison). All data are presented as mean \pm SEM. * $P < 0.05$.

Data S1. Analysis of TRAP-sequencing data. Tab A: Raw transcripts per million for INPUT and respective percentiles. **Tab B:** Raw transcripts per million for IP and respective percentiles. **Tab C:** Quantile normalized transcripts per million for IP and respective statistics. **Tab D:** Summary of differentially expressed genes between WT and 4E-BP1 cKO in IP samples.