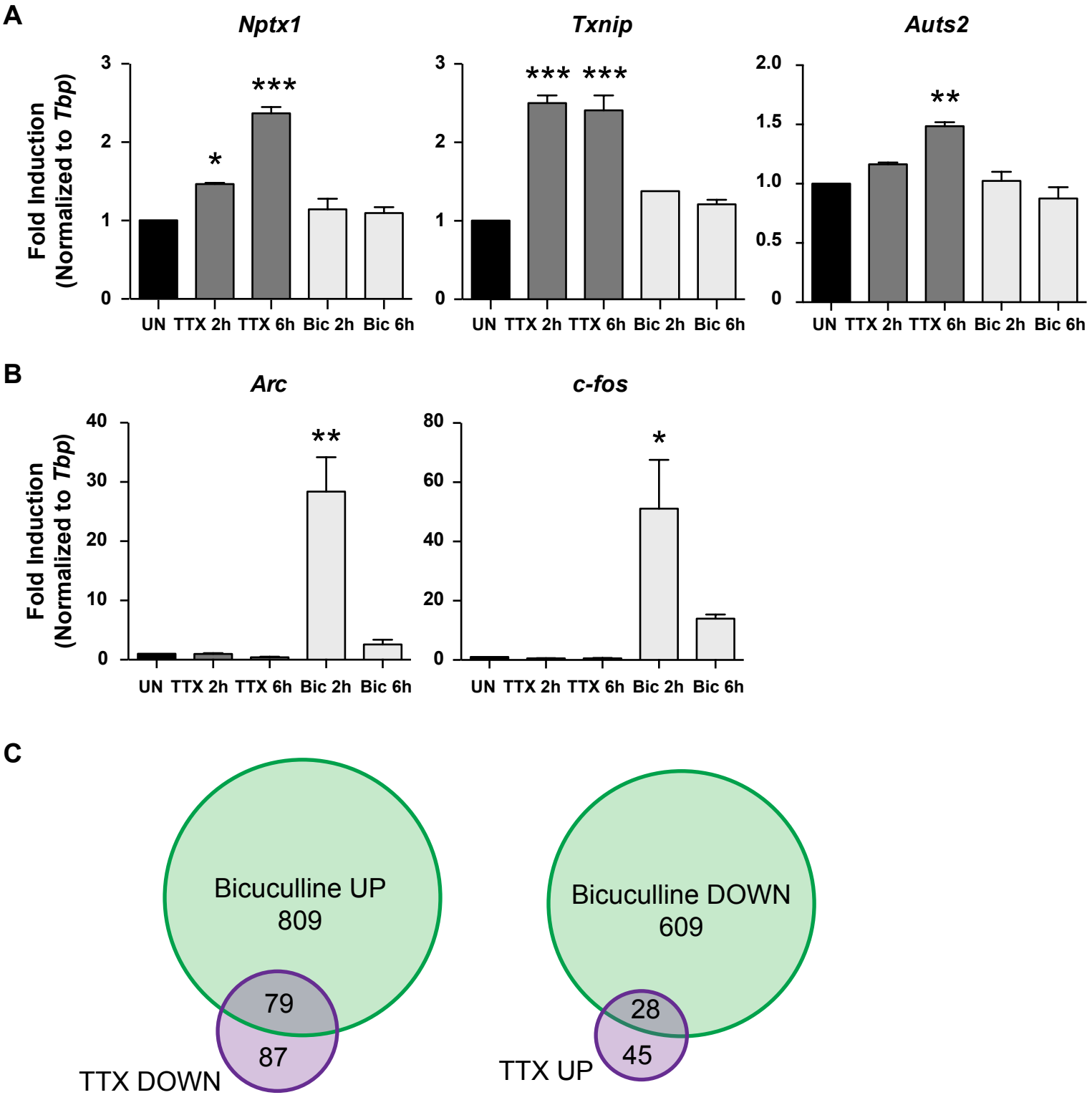

Figure S1. Confirmation of genome-wide data with RT-qPCR, Related to Figure 1. qRT-PCR results of TTX induced genes **(A)** and Bic induced genes **(B)** from cells that were treated with TTX or Bic for the indicated time points. n = 2 biological replicates. (C) Venn diagrams of bidirectional genes, comparing genes upregulated by bicuculline and downregulated by TTX (left) or genes downregulated by bicuculline and upregulated by TTX. *, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$. p values determined by a one-way ANOVA using Dunnett's multiple comparisons test.

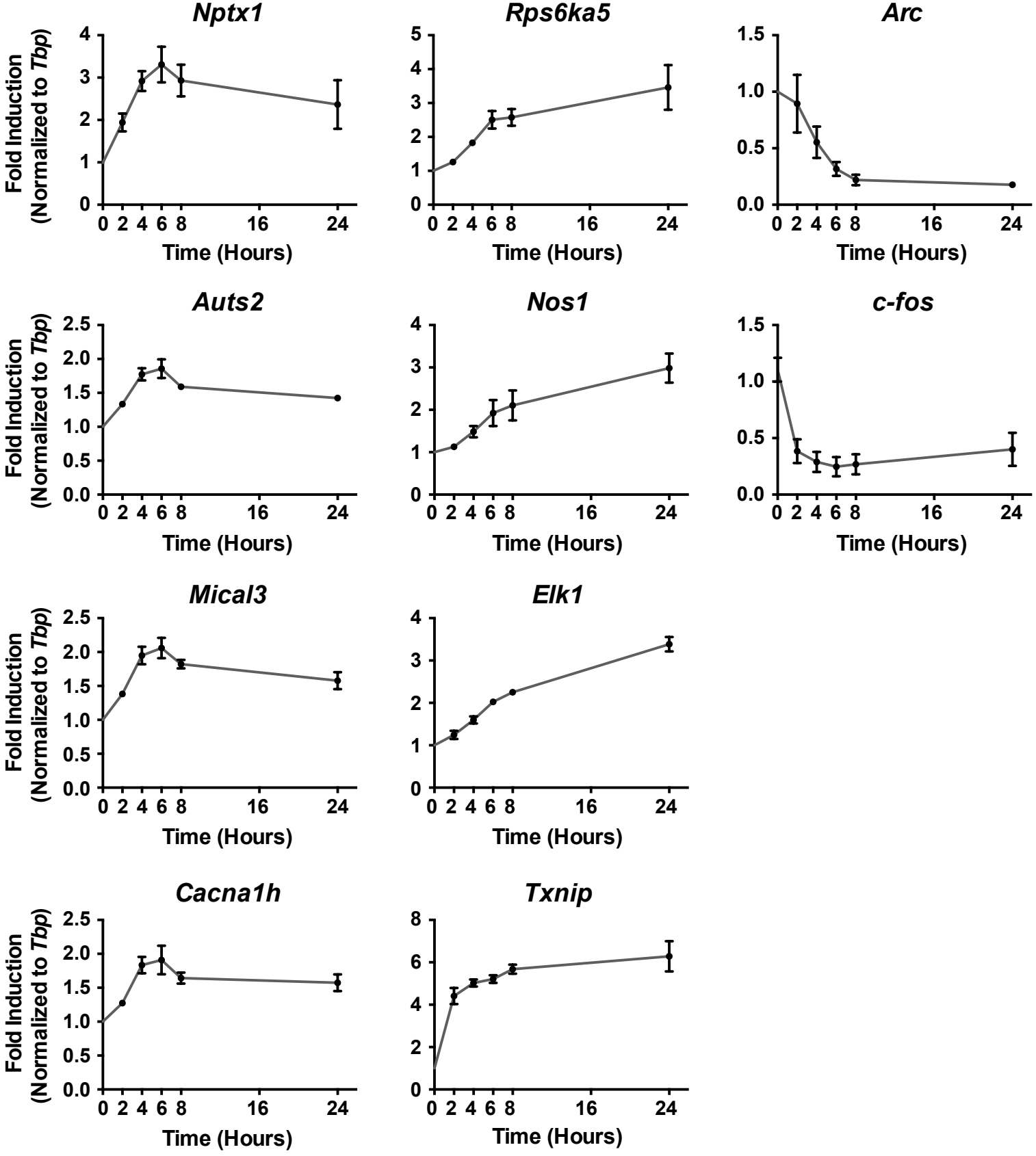

Figure S2. There are two waves of TTX-dependent gene induction, Related to Figure 1. qRT-PCR results from cells that were treated with TTX for the indicated time points. RNA levels were measured for the indicated genes. The columns represent distinct classes of genes based on their induction patterns. The left column contains genes that peak around 6 h of TTX treatment, while the middle column contains genes that continue to rise until 24 h. The right column contains immediate early genes that are induced in response to Bic, and decrease with TTX treatment. n = 3 biological replicates. Error bars represent SEM.

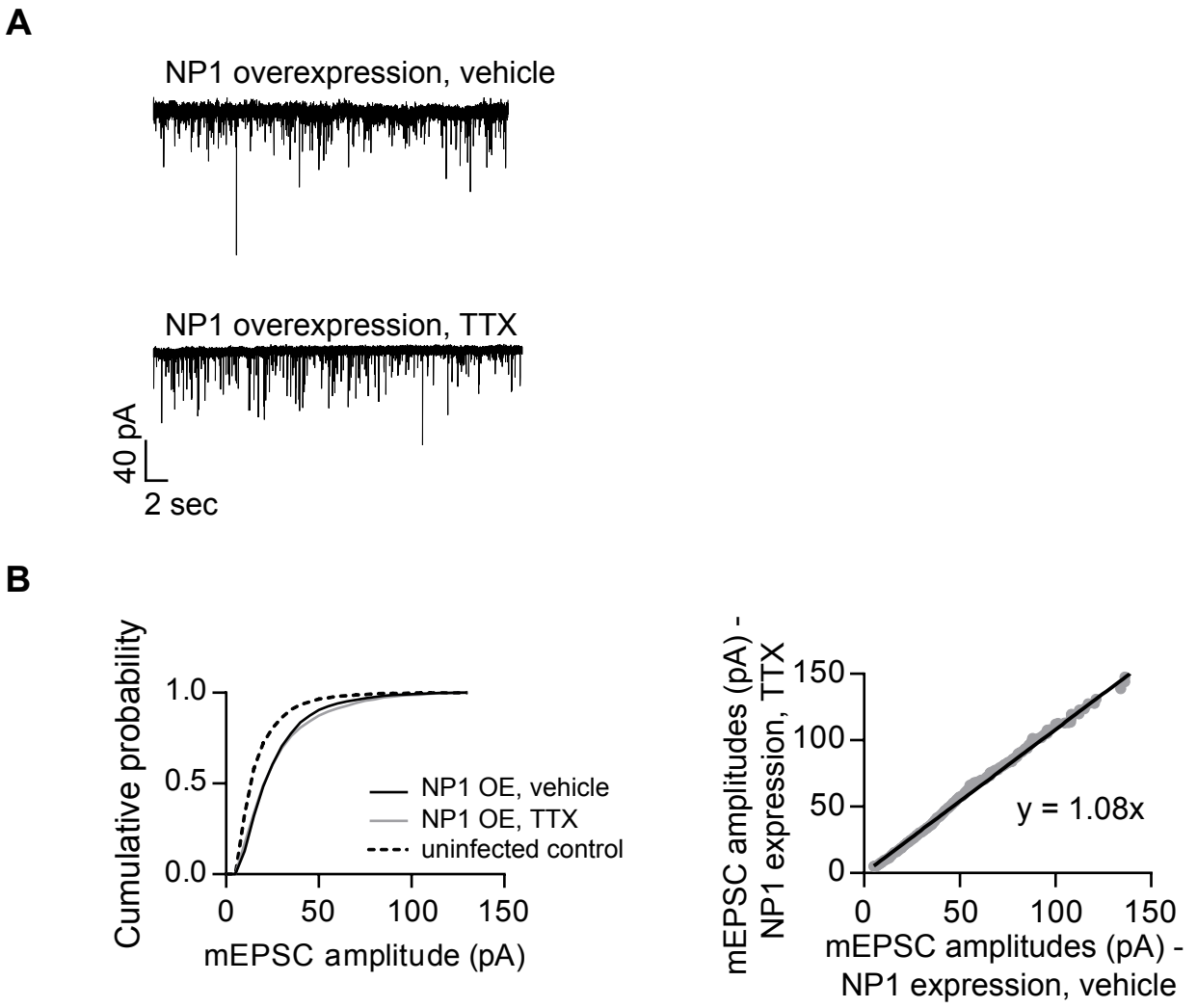

Figure S3. Overexpression of *Nptx1* occludes TTX-induced scaling, Related to Figure 3. (A) Example traces of AMPA-mediated mEPSCs from either vehicle or TTX treated cells. **(B)** Cumulative probability histograms of AMPA-mediated mEPSC amplitudes from cells overexpressing NP1 in either vehicle or TTX treated conditions indicate that TTX-induced scaling is occluded by high NP1 levels. NP1/TTX (n = 11) vs NP1/veh (n = 10), p = 0.005, D = 0.055.

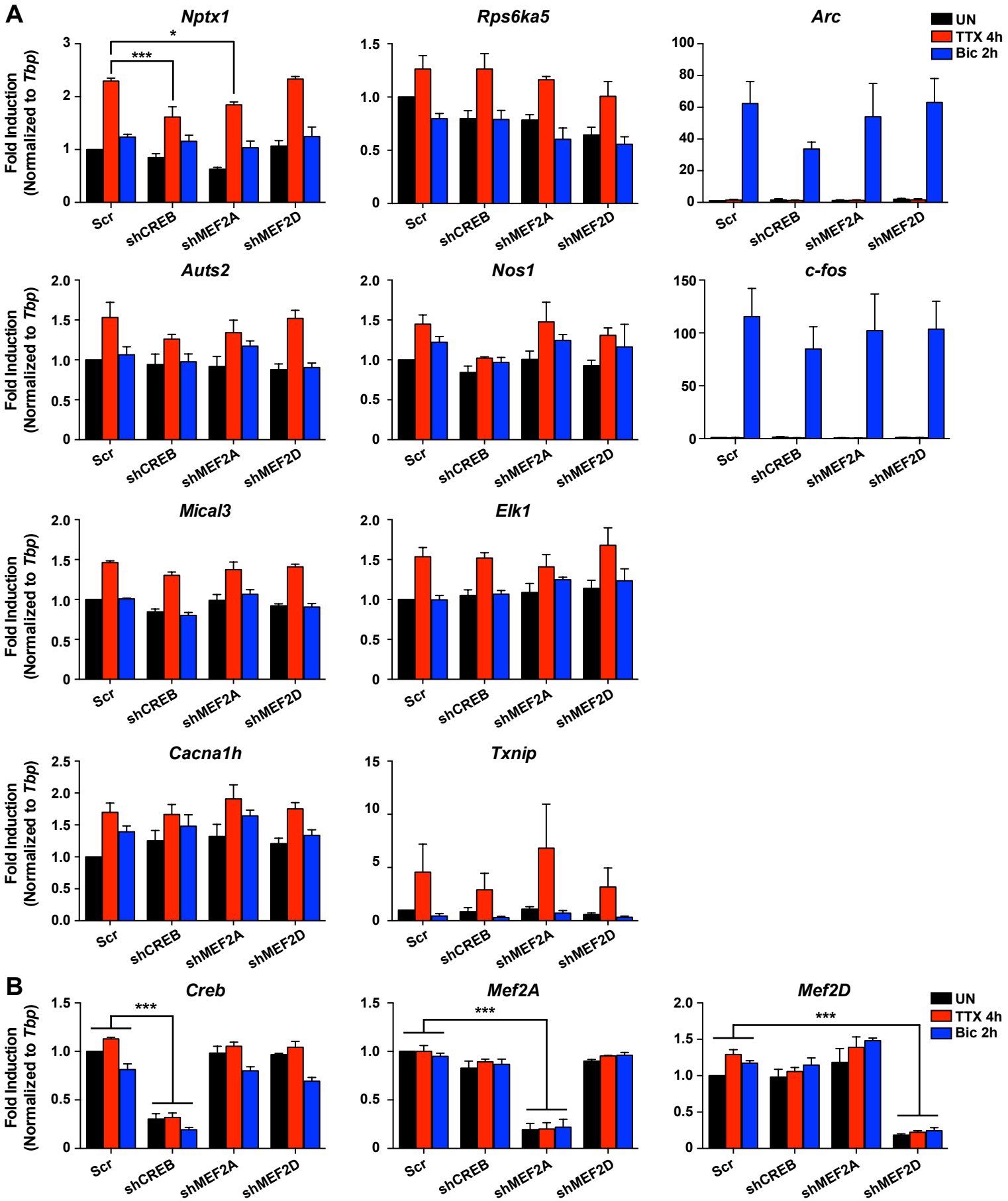

Figure S4. Effects of activity-regulated transcription factors in TTX-dependent induction, Related to Figure 4. Cortical neurons were infected with lentiviruses containing a scrambled shRNA control or an shRNA targeting CREB, MEF2A, or MEF2D. **(A)** RNA levels of the indicated TTX or Bic induced gnes were measured with qRT-PCR. **(B)** RNA levels of the indicated transcription factor genes were measured with qRT-PCR to test knockdown efficiency. n = 3 biological replicates. *, $p < 0.05$; **, $p < 0.01$; ***, $p<0.001$. p values determined by a two-way ANOVA with Tukey's multiple comparisons test using GraphPad Prism. Error bars represent SEM.

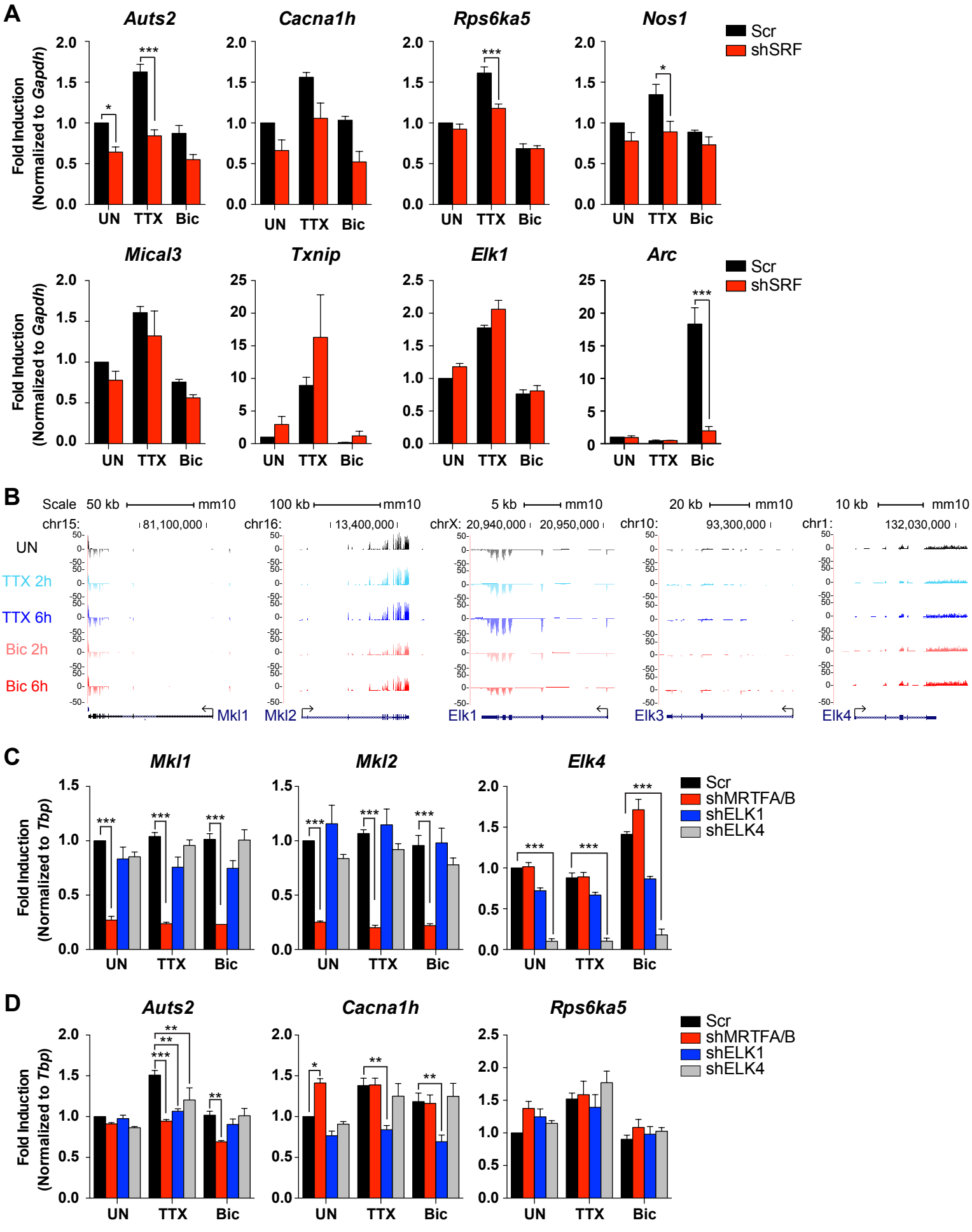
A
Auts2
Cacna1h
Rps6ka5
Nos1
Mical3
Txnip
Elk1
Arc
Fold Induction (Normalized to Gapdh)
UN
TTX
Bic
Scr
shSRF
B
Scale
50 kb
mm10
100 kb
5 kb
20 kb
10 kb
chr15:
81,100,000
chr16:
13,400,000
chrX:
20,940,000
20,950,000
chr10:
93,300,000
chr1:
132,030,000
UN
TTX 2h
TTX 6h
Bic 2h
Bic 6h
Mkl1
Mkl2
Elk1
Elk3
Elk4
C
Mkl1
Mkl2
Elk4
Fold Induction (Normalized to Tbp)
Scr
shMRTFA/B
shELK1
shELK4
D
Auts2
Cacna1h
Rps6ka5

Figure S5. SRF and its cofactor ELK1 regulate the expression of TTX-induced genes, related to Figure 4.
(A) qRT-PCR results of TTX-induced genes from cells that were infected with the scrambled control or shSRF knockdown. Cells were left untreated (UN) or treated with TTX or Bic. n = 3 biological replicates. The Bic induced gene *Arc* was tested as a control of knockdown efficiency. **(B)** UCSC genome browser views of the SRF cofactor family members: *Mkl1*, *Mkl2*, *Elk1*, *Elk3*, and *Elk4* (*left to right*). **(C)** qRT-PCR results of the other cofactor family members after knockdown of the indicated family member (shMRTFA/B or shElk4) compared to a scrambled control. Cells were either left untreated or treated with TTX or Bic. **(D)** qRT-PCR results of TTX-induced genes after the indicated cofactor knockdown. n = 3 biological replicates. *, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$.
p values determined by a two-way ANOVA with Tukey's multiple comparisons test. Error bars represent SEM.

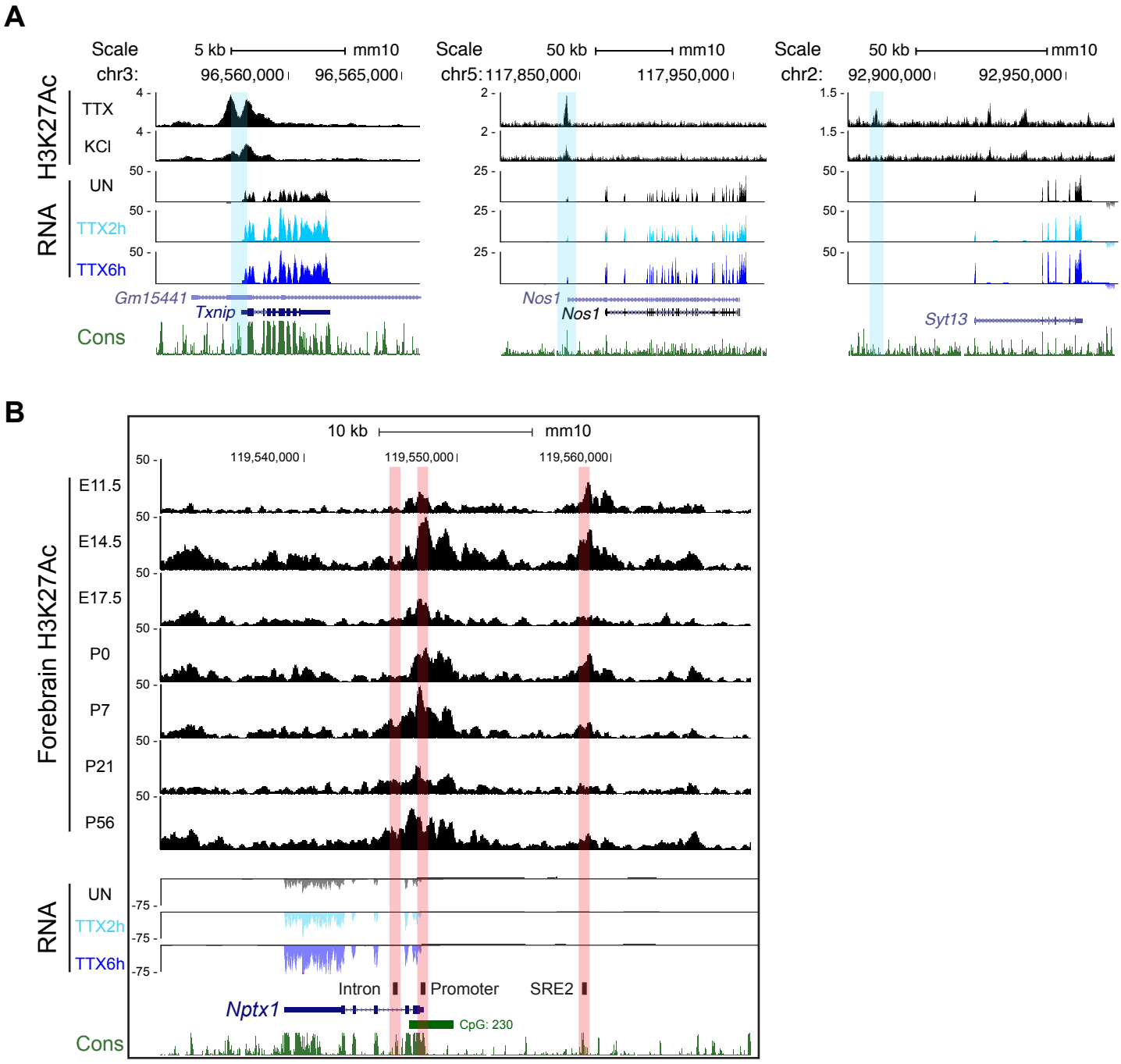

Figure S6. H3K27Ac peaks surround TTX up regulated genes, Related to Figure 4. (A) UCSC genome browser views of genes with a decreasing H3K27Ac peak nearby: *Txnip*, *Nos1*, and *Syt13*. The H3K27Ac data from Malik et al., 2014 is aligned with the RNA-seq data for the idicated conditions. Blue bars represent the decreasing H3K27Ac peaks. **(B)** A UCSC genome browser view of the *Nptx1* gene aligned with RNA-seq data from the indicated conditions and H3K27Ac ChIP data from Nord et al., 2013. The H3K27Ac ChIP was performed with forebrain tissue at the indicated time points throughout development. A potential SRE (SRE2) is located within a peak that decreases throughout development, while the peak around the promoter expands into the intronic region (Intron) as develpment progresses. Red bars highlight the regions amplified in ChIP experiments.

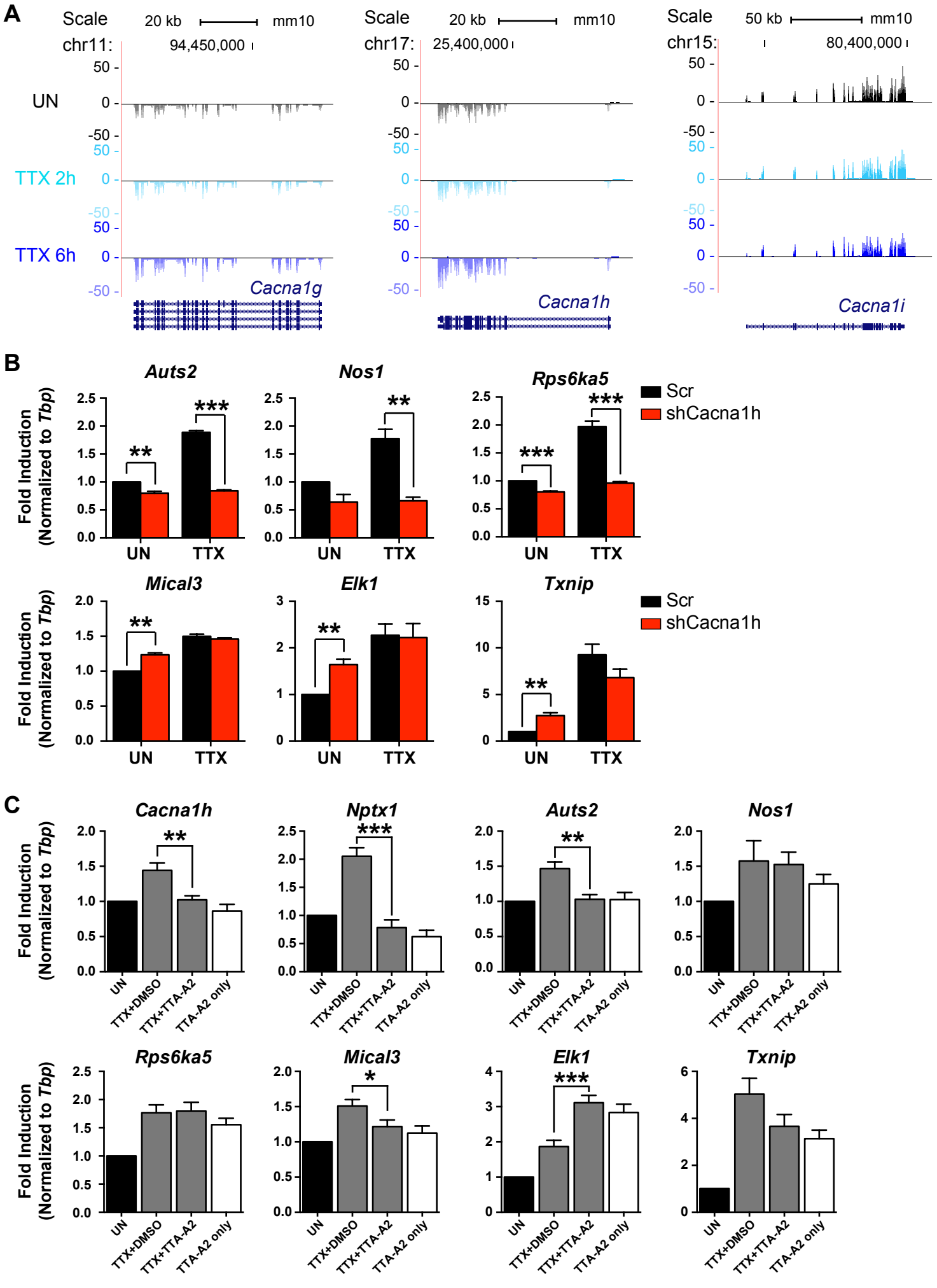
A
Scale
20 kb
mm10
chr11:
94,450,000
UN
TTX 2h
TTX 6h
Cacna1g
Scale
20 kb
mm10
chr17:
25,400,000
Cacna1h
Scale
50 kb
mm10
chr15:
80,400,000
Cacna1i
B
Auts2
Nos1
Rps6ka5
Scr
shCacna1h
Fold Induction (Normalized to Tbp)
Mical3
Elk1
Txnip
UN
TTX
C
Cacna1h
Nptx1
Auts2
Nos1
Rps6ka5
Mical3
Elk1
Txnip
UN
TTX+DMSO
TTX+TTA-A2
TTA-A2 only

Figure S7. Cortical neurons express all three isoforms of the T-VGCC, related to Figure 5. (A) UCSC genome browser views of the RNA-seq data from cells treated with TTX for 2 or 6 h, aligned with the genomic loci of the three T-VGCC genes: *Cacna1g*, *Cacna1h*, and *Cacna1i*. These genes encode the CaV3.1, CaV3.2, and CaV3.3 subtypes, respectively. **(B)** RNA expression levels of the indicated genes after knockdown of *Cacna1h* (CaV3.2) or infection with a scrambled control, in either untreated or TTX-treated conditions. n = 3 biological replicates. **(C)** RNA expression levels of the indicated genes in response to TTA-A2 treatment. Cells were either untreated (UN), treated with TTX for 6 h pretreated with DMSO or TTA-A2, or treated with TTA-A2 alone. n = 7 biological replicates. *, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$. p values for **(B)** were determined by multiple t tests, as performed, where statistical significance was determined using the Holm-Sidak method. p values for **(C)** were determined using a one-way ANOVA using Dunnet's multiple comparisons test. Error bars represent SEM.

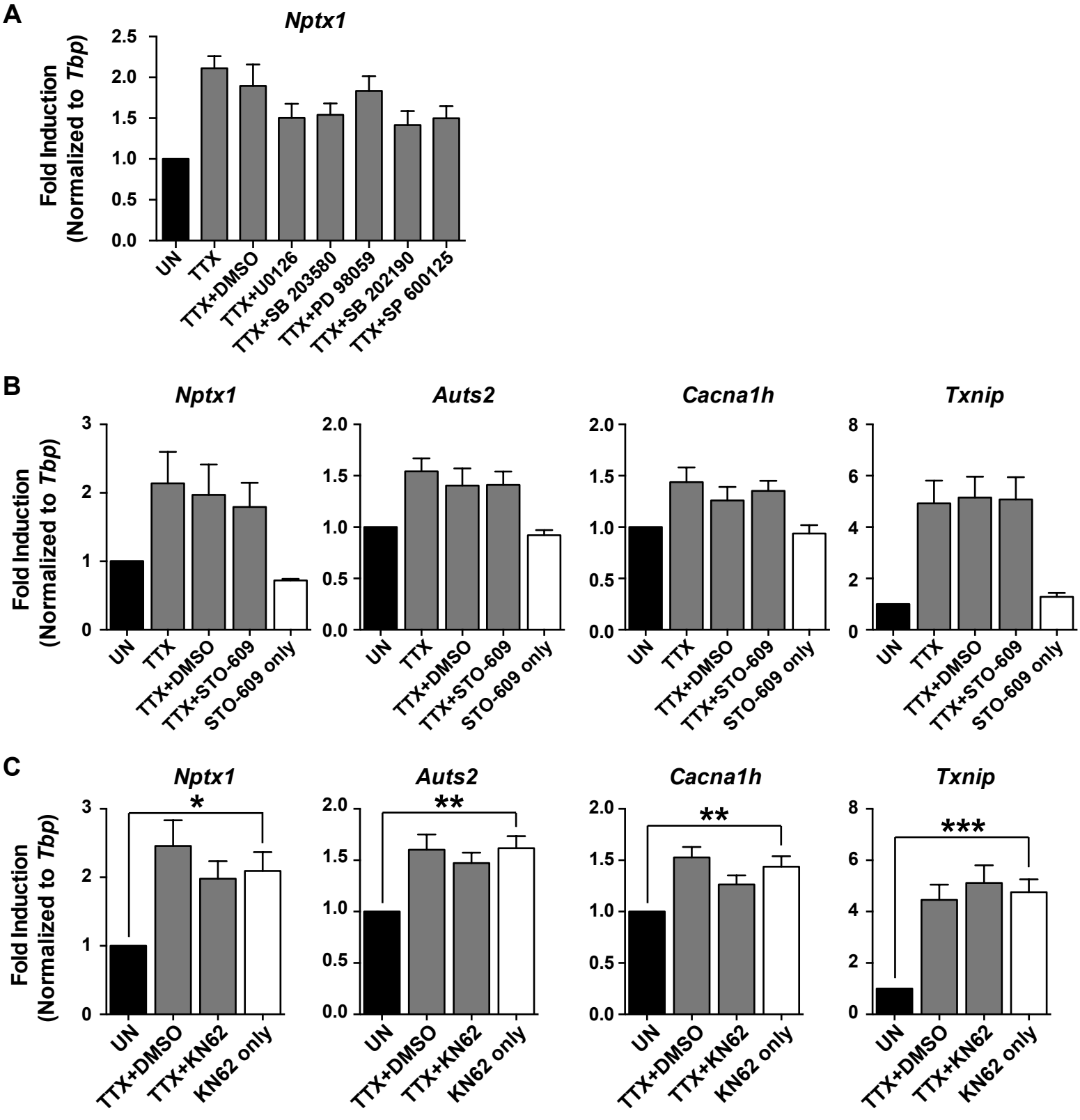

Figure S8. Calcium signaling pathways mediating induction, Related to Figure 7. (A) qRT-PCR results of cells treated with TTX for 6 h and pretreated with the indicated blockers of comoponents of the MAPK signaling pathway. n = 5 biological replicates. **(B)** qRT-PCR results of cells treated with TTX and/or STO-609. n = 4 biological replicates. **(C)** qRT-PCR results of cells treated with TTX and/or KN-62. DMSO was used as a vehicle when indicated. n = 5 biological replicates. In the above bar graphs, black bars represent untreated cells. Grey bars represent cells treated with TTX and pretreated with the indicated compounds. White bars represent cells treated only with the indicated compound. *, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$. p values determined by a one-way ANOVA using Dunnett's multiple comparisons test. Error bars represent SEM.

Supplemental Table 1, related to Figure 1: Genes that are differentially regulated by TTX and BIcuculline, attached as an excel file.

Supplemental Table 2, related to Figure 1: TTX-induced genes that have been associated with neurodevelopmental disorders (Gene information from SFARI and other published sources)

Gene ID	Gene Name	Associated Disorder	Reference
Auts2	*Autism susceptibility candidate 2*	ASD, ADHD, epilepsy	Sultana et al., 2002; Kalscheuer et al., 2007
Cacna1h	*Calcium channel, voltage-dependent, alpha 1H subunit*	ASD	Splawski et al., 2006
Bbs4	*Bardet-Biedl syndrome 4*	Bardet-Biedl syndrome, ASD	Girirajan et al., 2013
Fan1	*FANCD2/FANCI-associated nuclease 1*	ASD and schizophrenia	Ionita-Laza et al., 2013
Ogt	*O-linked N-acetylglucosamine (GlcNAc) transferase*	Maternal stress marker	Howerton et al., 2013
Smg6	*Smg-6 homolog, nonsense mediated mRNA decay factor (C. elegans)*	ASD, neurodevelopmental disorders	Nguyen et al., 2013
Upf2	*UPF2 regulator of nonsense transcripts homolog (yeast)*	ASD, neurodevelopmental disorders	Nguyen et al., 2013
Nos1	*Nitric Oxide Synthase I*	Schizophrenia	Shinkai et al., 2002
Elk1	*Elk1, member of ETS oncogene category*	Schizophrenia	Sanders et al., 2013
Nptx1	*Neuronal pentraxin 1*	Bipolar Disorder	Rajkumar et al., 2015
Myt1	*Myelin transcription factor 1*	Schizophrenia	Fernandez-Enright et al., 2013
Dbp	*D site of albumin promoter binding protein 1*	Schizophrenia	Sanders et al., 2013

Supplemental Experimental Methods

Lentiviral infections

Lentiviral constructs containing the indicated shRNAs in the pLLX vector were transfected into HEK293T cells with the helper plasmids Δ8.9 and VsVg using Fugene HD (Promega) before incubating for 48-72 h. Viral supernatant was then added directly to the cultures to infect the neurons. shRNA sequences were designed using GE Dharmacon siDesign Center or as previously described (Abad et al., 2006; Joo et al., 2015; Lee et al., 2010; Rodriguez-Gomez et al., 2012). The shRNA sequences used are: *Scrambled,* 5'-GCCCTGCCACCGTAATTTA-3'; *shNP1*, 5'-GTACAGCCGCCTCAATTCT-3'; *shSRF*, 5'-AAGATGGAGTTCATCGACAAC-3'; *shCREB*, 5'-GGAGTCTGTGGATAGTGTA-3'; *shMEF2A*, 5'-GTTATCTCAGGGTTCAAAT-3'; *shMEF2D*, 5'-GTAGCTCTCTGGTCACTCC-3'; *shMRTFA/B*, 5'-CATGGAGCTGGTGGAGAAGAA-3'; *shELK1*, 5'-GGGATGGTGGTGAGTTCAAGT-3'; *shELK4*, 5'-GCAACGAGCCCTAGTCTTTCT-3'; *shCacna1h*, 5'-GGGCTTCCTTTAGTAGCAA-3'.

Pharmacological Inhibitors

To induce scaling, 1 μM TTX (Tocris) or 50 μM bicuculline (Sigma) was used. Cells were also pretreated with the following inhibitors at the indicated concentrations: 5 μM NBQX (Tocris), 100 μM AP-5 (Tocris), 10 μM Nimodipine (Tocris), 5 mM EGTA (Bio-world), 10 μM EGTA-AM (Life Technologies), 2 μM ω-Conotoxin GVIA (Alomone labs), 2 μM ω-Agatoxin IVA (Alomone labs), 2 μM SNX-482 (Alomone labs), 10 μM NNC 55-0396 dihydrochloride (Alomone labs), 1 μM Mibefradil (Alomone labs), 50 μM TTA-A2 (Alomone labs), 10 μM KN-62 (Tocris), 3 μM STO-609 (Tocris), and 10 μM each of the MAPK inhibitors: U0126, SB 203580, PD 98059, SB 202190, SP 600125 (Tocris). 5 μg/mL Actinomycin D was added to block transcription. EGTA-AM, Nimodipine, TTA-A2, STO-609, KN-62, Actinomycin D, and the MAPK inhibitors were dissolved in DMSO to make stock solutions. Cells were pretreated with EGTA-AM for 30 min before exchanging the media and being stimulated with TTX. Cells were pretreated with EGTA for 5 min before stimulation, and for all other inhibitors, cells were pretreated for 15 min before stimulation by either TTX or bicuculline.

Whole cell voltage clamp recordings

At DIV14-18, dissociated hippocampal cultures were voltage-clamped at -70 mV, as previously described (Reese and Kavalali, 2015), using an Axon Instruments Axopatch 200B amplifier. Access resistance was less than 25MΩ for each recording. The internal pipette solution contained (in mM): 120 K-Gluconate, 20 KCl, 10 NaCl, 10 HEPES, 0.6 EGTA, 4 Mg-ATP and 0.3 Na-GTP at pH 7.3. In order to isolate AMPA-mEPSCs, the extracellular solution contained 1 μM TTX, 50 μM picrotoxin (to block mIPSCs), 50 μM (2R)-amino-5-phosphonovaleric acid (AP5), 2 mM Ca^{2+} and 1.25 mM Mg^{2+}. All recordings were performed under continuous perfusion, and cells were perfused for 3 min before recording to achieve stable baselines. The AMPA-mEPSCs were quantified with Synaptosoft MiniAnalysis software. 4 minutes were analyzed per recording. To ensure that high frequency cells did not skew the amplitude comparisons by being overrepresented, 200 mEPSC amplitudes were randomly selected from each recording to generate the cumulative probability histograms and rank order plots. Significance was determined using the Kolmogorov-Smirnov test, which was performed using PAST 3.1 (http://folk.uio.no/ohammer/past/).

Chromatin Immunoprecipitation (ChIP)

ChIP was performed as previously described (Joo et al., 2015; Kim et al., 2010; Schaukowitch et al., 2014). Briefly, neurons were crosslinked with 1% formaldehyde for 10 min before quenching with 2M glycine (125 mM final concentration). Cells were washed with cold PBS and harvested in PBS with proteinase inhibitors (Roche). Cells were lysed with Buffer I (50 mM Hepes KOH, pH 7.5, 140 mM NaCl, 1 mM EDTA, pH 8.0, 10 % Glycerol, 0.5% NP-40, protease inhibitors) and nuclei were resuspended in Buffer II (300 mM NaCl, 1 % Triton-X 100, 0.1% Sodium deoxycholate, 1 mM EDTA, pH 8.0, 0.5 mM EGTA, pH 8.0, 10 mM Tris, pH 8.0, protease inhibitors) before being sonicated to shear the DNA on a Misonix sonicator. Lysates were incubated with 4 μg of the following antibodies overnight at 4°C: anti-SRF (H-300, Santa Cruz Biotechnology Cat# sc-13029, RRID:AB_2302440), anti-ELK1 (I-20 X, Santa Cruz Biotechnology, Cat# sc-355, RRID:AB_631429). The next day, Protein A/G beads (Santa Cruz) were added for 2 h at 4°C, and then the beads were washed with Low, High, and LiCl buffers. DNA was eluted with Elution buffer (10 mM Tris, pH 8.0, 1 mM EDTA, pH 8.0, 1 % SDS) at 65°C for 10 min, and then reverse crosslinked at 65°C for 5-8 h. DNA was incubated with RNAse A (Qiagen), followed by Proteinase K (NEB), and purified by Phenol:Chloroform extraction, followed by a commercial DNA purification kit (Qiagen). Regions were amplified using qPCR with the primers listed below.

Immunostaining
Hippocampal neurons from P3 rats were cultured until DIV14-17. Cells were treated with 1 μM TTX overnight. Cells were washed once with PBS before fixation with 4% paraformaldehyde in PBS for 20 min at room temperature. Cells were washed three times with PBS. To stain for surface NP1, neurons were left unpermeabilized, and blocked with 10% Normal Donkey Serum (NDS; Jackson ImmunoResearch Laboratories) in PBS for 1 h at room temperature. Primary antibody incubation was performed for 2 h at room temperature in 2% NDS in PBS at the following dilution: anti-NP1 (BD Biosciences Cat# 610369, RRID:AB_397754, 1:300). Cells were washed three times with PBS before secondary incubation for 1 h at room temperature. The secondary antibody Alexa-Fluor anti-Mouse 555 was diluted 1:1000 in 2% NDS in PBS. Cells were washed three times in PBS, and mounted using hard-set mounting solution (VectaShield). Images were taken on a Zeiss LSM 150 using a 63x objective, and were quantified using ImageJ. For immunohistochemistry, mice were perfused with 4% formaldehyde and brains were incubated in 4% paraformaldehyde overnight. After three washes with PBS, the brains were incubated with 30% sucrose and then mounted in Tissue-tek (Sakura). The brains were sliced at 40 μm, and the slices were incubated with blocking solution (10% NDS in PBS) for 1 h at room temperature. Primary antibody incubation was performed overnight at 4°C in 0.1% TX-100 in PBS at a dilution of 1:50. Cells were washed four times with 0.1% TX-100 in PBS, and incubated with secondary antibody Alexa-Fluor anti-Mouse 555 at a dilution of 1:200 in 0.1% TX-100 in PBS for 1 h at room temperature. Slices were again washed four times with 0.1% TX-100 and coverslips were applied with hard-set mounting solution (VectaShield).

RNA-seq analysis
The sequencing reads were mapped to the mm10 UCSC annotation using TOPHAT (Trapnell et al., 2009) with the parameters (-a 8 -m 0 -I 500000 -p 8 -g 20 --library-type fr-firststrand --no-novel-indels --segment-mismatches 2). RPKM calculations were done using the HOMER package by normalizing to 10 million reads. Tracks for visualization on UCSC browser are generated using HOMER. Heatmaps were generated in JavaTreeView from log transformed fold change values, normalized to the unstimulated condition, using hierarchical clustering in Cluster. Venn diagrams were created using BioVenn (Hulsen et al., 2008).

ChIP-seq analysis
H3K27Ac ChIP-seq (GSE60192) data was downloaded from GEO. Bowtie2 (Langmead et al., 2009) aligner was used to align the fastq reads to mm10 UCSC annotation. Duplicates were removed using Picard MarkDuplicates (http://broadinstitute.github.io/picard/) and visualization tracks were generated using HOMER. Using MACS, the KCl-K27ac peaks were identified by using the Unstimulated-K27ac samples as control at a p-value of 1E-5. Closest genes were identified near the negative peaks generated for KCl-K27ac through MACS by using in-house downstream scripts.

GRO-seq analysis
10 million nuclei per sample were extracted and used for global run-on, and base hydrolysis was performed as previously described (Core et al., 2008). Nascent RNAs were immunoprecipitated with anti-BrdU antibody-conjugated beads (Santa Cruz Biotech) twice. Between two immunoprecipitations, purified run-on RNAs were subjected to polyA tailing by using Poly(A)-polymerase (NEB). Subsequently, RNAs are subjected to first-strand cDNA synthesis using Superscript III Reverse Transcriptase (Invitrogen) with the oNTI223 primer. Extra oNTI223 primers were removed by Exonuclease I (NEB) and cDNAs were size-selected in an 8 % polyacrylamide TBE-urea gel. Purified cDNAs were subsequently circularized using CircLigase (Epicentre) and relinearized with ApeI (NEB). The relinearized DNA template were subjected to PCR amplification by using Phusion High-Fidelity DNA Polymerase (NEB) and Illumina TrueSeq small-RNA sample barcoded primers. Subsequently, PCR products were isolated by 6 % polyacrylamide TBE gel and purified. The final libraries were sequenced using an Illumina HiSeq per the manufacturer's instructions. The raw fastq reads were trimmed using CUTADAPT with parameters -a AAAAAAAAAAAAAAAAAAAA -z -e 0.10 -f fastq -m 32 (Chae et al., 2015). Then the surviving reads were submitted to BWA aligner for mapping to the mm10 UCSC annotation. Samtools and the HOMER package were used to make visualization tracks and RPKM calculations. RPKM was calculated by normalizing to 10 million reads.

Western blotting
Protein was extracted from neurons using sample buffer (60 mM Tris-HCl, pH 6.8, 2% SDS, 10% glycerol). Before loading, samples were mixed with loading buffer (5% 2-mercaptoethanol, 0.1% bromophenol blue) and denatured at 95°C. Proteins were run on a 12% SDS-PAGE gel, and analyzed by western blotting with the following antibodies:

anti-NP1 (BD Biosciences Cat# 610369, RRID:AB_397754, 1:1000) or anti-β-actin (Sigma-Aldrich Cat#A5441, RRID:AB_476744, 1:5000).

Visual Cortex experiments

For dark rearing experiments, male C57Bl6/J mice were placed into completely dark chambers at P21-25 at the end of their light cycle. The visual cortices were removed, and RNA was extracted in Trizol after 24 h in the dark. For measuring protein levels, mice were taken out of the dark after 3 d. Intraocular injections of TTX were performed as in (Frenkel and Bear, 2004). Briefly, 1 μL of 1 mM TTX or PBS was injected into each eye of the mouse using a glass pipette at a rate of 1 μL/1 min (Joo et al., 2015). RNA was then extracted from visual cortex of the opposite hemisphere with Trizol.

Primer Sequences

Primers for RT-qPCR

Nptx1-forward	GAAGAAGAGCCTGCCAGAGAT
Nptx1-reverse	CAGGCACAGCATAAGAGAAGG
Rps6ka5-forward	CTTGACGCACAATGGCTACG
Rps6ka5-reverse	CCTGCCCTGACAGCATTGTA
Auts2-forward	TGAGGGATCCTTACCGTGAC
Auts2-reverse	ATCGGTCGGCTTCATACAAC
Cacna1h-forward	GCCAGAAATGCTACCTGGGT
Cacna1h-reverse	CAGAGAGGCTCACGTTGTGT
Nos1-forward	GGTTTAAGGACCTGGGGCTC
Nos1-reverse	CCAGCCACTAAAGGGACAGG
Txnip-forward	TGCAGGAAATGAAGCATCTG
Txnip-reverse	GTTCCCCGCTGTAGAGACTG
Elk1-forward	CTCTGCATCCACCAACTTGA
Elk1-reverse	CTGCTGCAGCTTCTGAGAGA
Mical3-forward	TTCTCACCTTGCGCAAAAGC
Mical3-reverse	CGATGCCGCCATATCAGAGT
Arc-forward	GTGAAGACAAGCCAGCATGA
Arc-reverse	CCAAGAGGACCAAGGGTACA
c-fos-forward	ATCCTTGGAGCCAGTCAAGA
c-fos-reverse	ATGATGCCGGAAACAAGAAG
Nptx1-pre-mRNA-forward	AACACGCCTTAAGATGGCAC
Nptx1-pre-mRNA-reverse	CCCTGCTCGGACTGCAAAAT
Auts2-pre-mRNA-forward	CCATTTTCCCAGGGCTTTGC
Auts2-pre-mRNA-reverse	GCCTCTTGGGTGTGTTCTCA
Txnip-pre-mRNA-forward	TGCTATTCTCACAGGGTCAACA
Txnip-pre-mRNA-reverse	GGGGAATAGAGAGGAAATTTTCAG
Arc-pre-mRNA-forward	GTGCCAGTGCCCACTACAAT
Arc-pre-mRNA-reverse	CCAAGTCCACTTTCCAGGAG
Auts2-short-forward	CGGCTAGGTCCACAAGTCTG
Auts2-short-reverse	CTGCCGTTTAGGATCCGGTT
Srf-forward	GGTGCCAGGTAGTTGGTGAT
Srf-reverse	TGAAGCCAGCATTCACAGTC
Mkl1-forward	CTGTCTAAGCCCTGGTGCTC
Mkl1-reverse	GCTCCTCAATCTGCTTGTCC
Mkl2-forward	TCATGCAACTTTTCGCTCAC
Mkl2-reverse	GCATAGCACGTGGTCAAAGA
Elk4-forward	TGCAACGAGCCCTAGTCTTT
Elk4-reverse	TTCCAGGGAAGGTAGTGTGG
Creb-forward	AAACATACCAGATCCGCACA
Creb-reverse	CTCTCTCTTCCGTGCTGCTT

Mef2a-forward	AGGGCTGCTTGTCCTAGATG
Mef2a-reverse	ACCAGCGCTGACCTGTCT
Mef2d-forward	CTGGCACTAGGCAATGTCAC
Mef2d-reverse	AGTGGGGCTGTTGCTGAG
Tbp-forward	TGACTCCTGGAATTCCCATC
Tbp-reverse	TTGCTGCTGCTGTCTTTGTT
Gapdh-forward	AGGTCGGTGTGAACGGATTTG
Gapdh-reverse	TGTAGACCATGTAGTTGAGGTCA
eNptx1-forward (eRNA-F)	ACATGCTCACGGTTGTGGAT
eNptx1-reverse (eRNA-R)	TTCTCTAGCCAGCAGTTGCC

Primers for ChIP qPCR

Nptx1-SRE1-forward	CAGGCTGTGTGAGGTAGTGG
Nptx1-SRE1-reverse	CACCAGGGTTTCCACGATCA
Nptx1-E1-forward	AGCAAAACCAGGAGCCAAGA
Nptx1-E1-reverse	CCACGCTGTGAACTCGGTAG
Nptx1-Intron-forward	AACACGCCTTAAGATGGCAC
Nptx1-Intron-reverse	CCCTGCTCGGACTGCAAAAT
Nptx1-Promoter-forward	TGAGACCGGGATCGGGC
Nptx1-Promoter-reverse	GAAGACTGTGCGGCGTAGAA
Nptx1-Neg1-forward	CACAGCGGTGAGCTCTAACT
Nptx1-Neg1-reverse	AAGAGGAATGTGGGCTGGTG
Nptx1-SRE2-forward	AGGGACAGCTTTTCGGATGG
Nptx1-SRE2-reverse	CTTGCCAATGAGATGGGGGT
Neg2-forward	GCAGAGCGGACCATAAACAT
Neg2-reverse	CCGTGAAATGAGTTGCCTTT
c-fos-Promoter-forward	CCTCCCTCCTTTACACAGGA
c-fos-Promoter-reverse	ACCTTCCGCGTGTAGGATTT