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Supplementary Materials for

An autophagy-related protein Becn2 regulates cocaine reward behaviors in the dopaminergic system

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Supplementary Figure Legends and Table

Figure S1. Cocaine dose-response and food CPP in WT and Becn2 mutant mice.

(A) Dose response of cocaine-induced locomotor stimulation. Open field test on WT ($Becn2^{+/+}$), $Becn2^{+/-}$ KO, and $Becn2^{S97L}$ KI mice in response to injection of low (5 mg/kg) or high (30 mg/kg) doses of cocaine. 5 mg/kg cocaine injection: WT, N=10, $Becn2^{+/-}$ KO, N=8; $Becn2^{S97L}$ KI, N= 5. 30 mg/kg cocaine injection: WT, N=9; $Becn2^{+/-}$ KO, N=12; $Becn2^{S97L}$ KI, N=7. **(B)** Western blot analysis of Becn1 in the NAc of $Becn2^{+/-}$ KO mice and WT littermates. N=5 mice. T-test. **(C)** WT and $Becn2^{+/-}$ KO mice show similar CPP to food, a non-drug reward. WT, N=8; $Becn2^{+/-}$ KO, N=6. **, P<0.01; ***, P<0.001; NS, not significant.

Figure S2. Physiological and neurotransmitter profiling of Becn2 KO mice upon cocaine treatment.

(A-B) Quantification of cocaine-induced extracellular accumulation of neurotransmitters ACh, 5-HT, and glutamate **(A)** and DA metabolites DOPAC and HVA **(B)** in the nucleus accumbens of $Becn2^{+/-}$ KO mice and WT littermates by microdialysis and UPLC. Area under the curve (AUC) is quantified for 120 min after cocaine injection. WT, N=7; $Becn2^{+/-}$ KO, N=8. T-test. **(C)** Percentage of baseline dopamine and GABA in the prefrontal cortex of WT and $Becn2^{+/-}$ KO mice at the indicated time points before and after cocaine injection. AUC is quantified for 120 min after cocaine injection. WT, N=7; $Becn2^{+/-}$ KO, N=9. **(D)** Cocaine-induced kinase activation is blunted in $Becn2^{+/-}$ KO mouse striatum. Western blot analyses and quantification of cocaine-induced MEK and ERK phosphorylation in the striatum of WT and $Becn2^{+/-}$ KO mouse brain 15 min after cocaine (15 mg/kg) or vehicle i.p. injection. N=5 mice. *, P<0.05; NS, not significant.

Figure S3. $Becn2^{+/-}$ KO and WT mice show comparable anxiety levels.

Quantification of distance, crossings, and time in open areas (upper) and the traveling path (lower) of WT and Becn2$^{+/-}$ KO mice in the elevated zero maze. WT, N=13; Becn2$^{+/-}$ KO, N=13. T-test. NS, not significant.

Figure S4. Deletion of Becn2 in DA neurons using Becn2$^{flox/flox}$ (Becn2$^{f/f}$) mice.

(A) Generation and validation of Becn2$^{flox/flox}$ mice. (Left) Genomic structure of Becn2 and Becn2 conditional KO targeting vector. (Right) Southern blot analyses of genomic DNA from Becn2$^{+/+}$ and Becn2$^{flox/+}$ ES cells. Probes hybridizing to both 5' and 3' regions of the targeting vector were used. **(B)** RT-PCR analysis of Cre expression in the VTA and the prefrontal cortex of Becn2$^{flox/flox}$ mice sham treated or stereotaxically microinjected with AAV-TH-Cre. **(C)** Loss of Becn2 immunofluorescence in TH-expressing DA neurons in the VTA of Becn2$^{flox/flox}$ mice stereotaxically microinjected with AAV-TH-Cre. Scale bar: 100 μm (upper); 25 μm (lower).

Figure S5. AAV2/9-directed rescue of Becn2 expression in DA neurons of Becn2$^{+/-}$ KO mice.

(A) RT-PCR analysis of GFP expression in the VTA and the prefrontal cortex of Becn2$^{+/-}$ KO mice sham treated or stereotaxically microinjected with AAV2/9-TH-GFP. **(B)** Immunofluorescence of GFP in the VTA, but not the SN (substantia nigra), of Becn2$^{+/-}$ KO mice stereotaxically microinjected with AAV2/9-TH-GFP. TH immunostaining indicates DA neurons. Scale bar: 100 μm (upper); 25 μm (lower).

Figure S6. Autophagy is partially affected in neuronal cells by loss of Becn2.

(A) Fluorescence microscopy (upper) and quantification (lower) of GFP-LC3 puncta in the whole cell, soma, and major neurites of primary DA neurons isolated from WT or Becn2$^{+/-}$ GFP-LC3 reporter mice. The lysosomal inhibitor bafilomycin A1 (BafA1) was applied to compare the autophagy flux. N=21-30. Scale bar: 10 μm. **(B)** Western blot analysis of LC3 and p62 in SH-SY5Y neuroblasts transfected with scrambled control or Becn2 shRNA. The lysosomal inhibitor

bafilomycin A1 (BafA1) was applied to compare the autophagy flux. **(C)** Immunostaining of endogenous LC3 and TH in the VTA of WT and Becn2$^{+/-}$ KO mice injected with PBS or 50 mg/kg lysosomal inhibitor chloroquine (CQ) 4 hr prior to sample collection. N=3 mice (50 cells/mouse). Scale bar: 10 μm. *, P<0.05; **, P<0.01; ***, P<0.001; NS, not significant.

Figure S7. Loss of Becn2 does not cause proteostasis toxicity or morphological or growth abnormality of DA neurons.

(A) Proteostat fluorescence of SH-SY5Y neuroblast cells transfected with scrambled or Becn2 shRNA. Cells treated with 10 μM MG132 for 4 hr were used as positive control. **(B)** Quantification of proteostat fluorescence of SH-SY5Y neuroblast cells transfected with scrambled (sh-scrambled) or Becn2 (sh-Becn2) shRNA. Cells treated with 50 μM chloroquine for 16 hr were used as positive control. N=3 experiments. **(C)** Immunofluorescence of the DA neuron marker TH in primary DA neuron cultures isolated from WT or Becn2$^{+/-}$ KO mice. Scale bar: 10 μm. **(D)** Dendrite Sholl analysis showing average intersection numbers at indicated distances from the soma in primary DA neurons isolated from WT and Becn2$^{+/-}$ KO mouse embryos. **(E)** Immunostaining of TH in the NAc of WT and Becn2$^{+/-}$ KO mice. Quantification of the TH density is shown. N=3 mice (9 areas/mouse). Scale bar: 25 μm. One-way ANOVA with Bonferroni post hoc test. *, p<0.05; **, p<0.01; NS, not significant.

Figure S8. Dopamine-related receptor analysis in brains regions of Becn2 KO mice.

(A) The striatal level of D1R or DAT is unaffected in Becn2$^{+/-}$ KO mice. Western blot analysis of D1R and DAT in the striatum of WT, Becn1$^{+/-}$ KO, and Becn2$^{+/-}$ KO mice. N=3 mice. NS, not significant. **(B)** (Left) Scheme of a biochemical method we adapted to isolate presynaptic membranes from pooled mouse striatum tissues. (Right) Western blot validation of isolated synaptosomes using 2 presynaptic markers (SVP38 and DAT) and 4 postsynaptic markers (Homer1, PSD95, GluR1 and NR1). WCL, whole cell lysates; Crude, crude synaptosomes; Pre,

presynaptic membrane; PNS, post-nucleus supernatant; S2, supernatant fraction 2. **(C)** Synaptosomes and the presynaptic membrane fraction were isolated from the NAc or the prefrontal cortex tissues pooled from 10 WT (+/+) or $Becn2^{+/-}$ KO (+/-) mice. The level of D2R in crude synaptosomes and presynapses (Pre) in the two brain regions was analyzed by Western blot studies. Synaptophysin (SVP38), presynaptic marker; PSD95, postsynaptic marker. Quantification is shown above the protein bands. **(D)** Semi-quantitative RT-PCR of Becn1 and Becn2 in HEK293 cells transfected with scrambled control or Becn2 shRNA. **(E)** Becn2 depletion does not affect the levels of endosomes and lysosomes. Corrected total cell fluorescence (CTCF) and Integrated density of immunostained EEA1 and LAMP1 in HEK293 cells transfected with scrambled or Becn2 shRNA, quantified by ImageJ software. N=20-29. NS, not significant. **(F)** PNS (post-nucleus supernatant) and S2 (supernatant fraction 2) were collected during synaptosome isolation from the NAc pooled from 10 $Becn2^{flox/flox}$ ($Becn2^{f/f}$) mice sham-treated or stereotaxically microinjected with AAV expressing TH-Cre. The level of D2R in these fractions was analyzed by Western blot studies. Synaptophysin (SVP38), presynaptic marker; PSD95, postsynaptic marker. Quantification is shown above each protein band. f/f, $Becn2^{flox/flox}$; f/f TH, $Becn2^{flox/flox}$; AAV-TH-Cre.

Figure S9. Generation of Becn2 point mutant knock-in (KI) mice that lose GASP1 interaction by the CRISPR technique.

(A) Amino acids 69-88 deletion or the I80S point mutation in human Becn2 blocks agonist-induced D2R degradation. Biotin protection degradation assay in Flag-D2R HEK293 cells expressing WT Becn2 or loss-of-GASP1 interaction mutants $Becn2^{\Delta 69-88}$ and $Becn2^{I80S}$. NTm, shRNA non-targeting mutant. **(B)** Protein sequence alignment of the GASP1-binding region in human and mouse Becn2, analyzed by NCBI BLAST. **(C)** Targeting strategy and Southern blot analysis of CRISPR/Cas9 $Becn2^{S97L}$ KI clones. **(D)** Sequencing of $Becn2^{S97L}$ (TCT-->TTG) KI mice generated by CRISPR/Cas9. **(E)** Genotyping of $Becn2^{S97L}$ F2 pups by PCR.

Figure S10. The upstream autophagy inhibitor SBI-0206965 increases presynaptic D2R in the NAc of WT mice, but not Becn2$^{+/-}$ KO mice. Western blot analyses of D2R in WCL (whole cell lysates), crude synaptosomes, and presynaptic membranes in the NAc pooled from 10 WT or 10 Becn2$^{+/-}$ KO mice intraperitoneally treated with either vehicle (DMSO) or SBI-0206965 (SBI) at 2 mg/kg once daily for 5 days. Synaptophysin (SVP38) or Synapsin I (SYN1), presynaptic marker; PSD95, postsynaptic marker. Quantification is shown above each protein band.

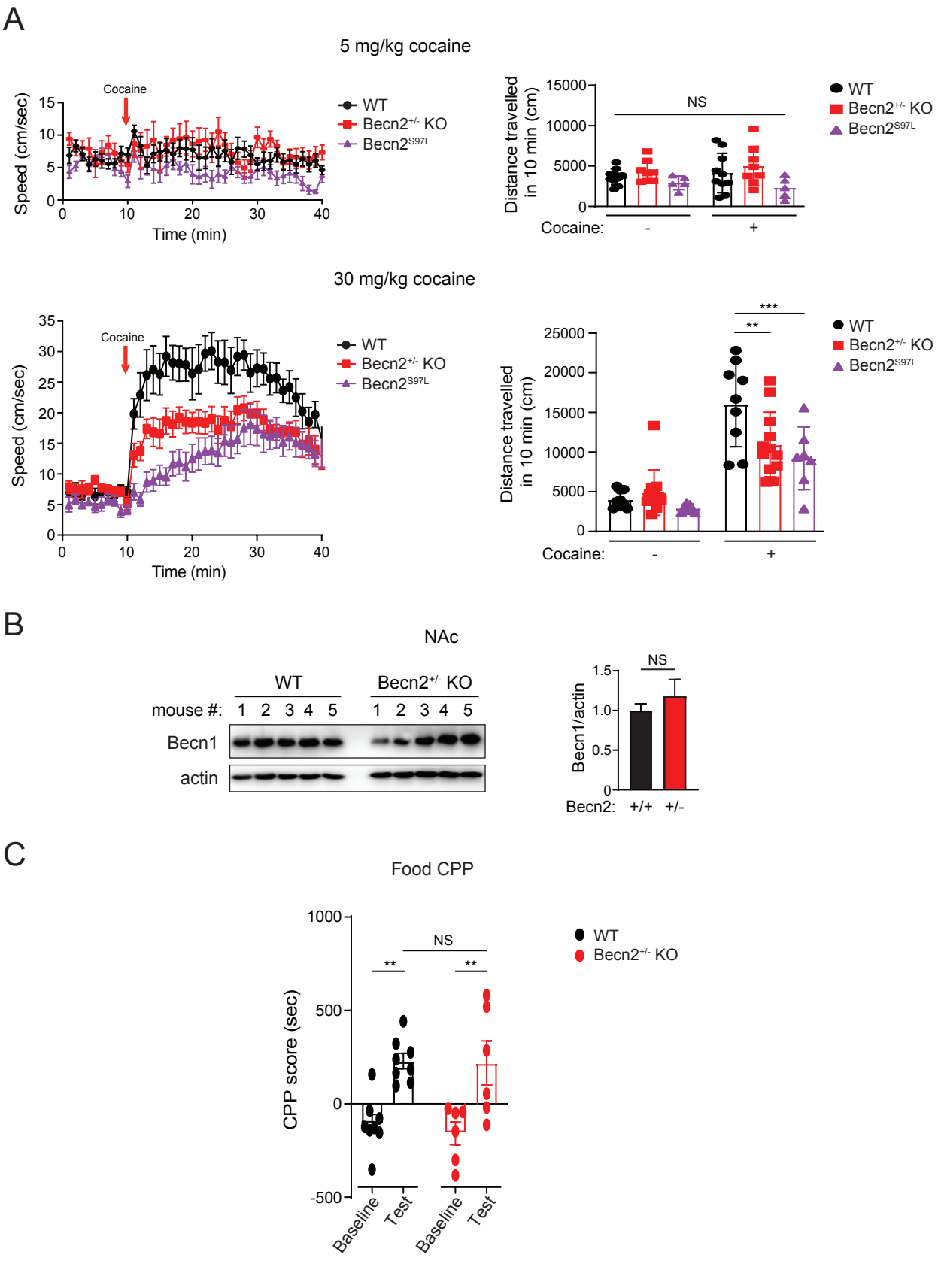
Table S1. Statistical analysis using ANOVA or Student's t-test.

Figure	Test	Statistical data
1A	Two-way repeated-measures ANOVA with Bonferroni post hoc test	Genotype X cocaine injection interaction: $F_{(2,50)}$=13.06, p<0.001; Genotype: $F_{(2,50)}$=8.692, p<0.001; Cocaine injection: $F_{(1,50)}$=89.40, p<0.001
1B	Two-way repeated-measures ANOVA with Bonferroni post hoc test	Genotype X cocaine injection interaction: $F_{(1,41)}$=4.414, p=0.0418; Genotype: $F_{(1,41)}$=39.16, p<0.0001; Cocaine injection: $F_{(1,41)}$=5.823, p=0.0204
1C	Two-way repeated-measures ANOVA with Fisher's Least Significant Difference (LSD) post hoc test	Active nose-poke×days interaction: $F_{(9,198)}$=5.618, p<0.0001; inactive nose-poke×days interaction: $F_{(9,198)}$=1.777, p=0.0746; infusion×days interaction: $F_{(9,198)}$=3.508, p=0.0016
1D	Two-way repeated-measures ANOVA with Fisher's Least Significant Difference (LSD) post hoc test	Active nose-poke: $F_{(2,28)}$ = 11.72, p=0.0002; infusion: $F_{(2,28)}$ = 14.40, p<0.0001; intake: $F_{(2,28)}$ = 47.83, p<0.0001
2A	T-test	t=2.46, df=15
2B	One-way ANOVA with Bonferroni post hoc test	MEK: $F_{(3,16)}$ = 5.712, p=0.0074; ERK: $F_{(3,16)}$ = 28.37, p<0.0001
2C	One-way ANOVA with Bonferroni post hoc test	$F_{(3,12)}$ = 8.0632, p=0.0033
3A	Two-way ANOVA with Bonferroni test	Genotype: $F_{(1,24)}$=5.068, p=0.034; Cocaine injection: $F_{(1,24)}$=24.363, p<0.0001; Interaction: $F_{(1,24)}$=5.404, p=0.029

3B	Two-way ANOVA with Bonferroni test	Interaction: $F_{(1,48)} = 7.251$, $p=0.0097$; CPP: $F_{(1,48)} = 23.58$, $p<0.0001$; Genotype: $F_{(1,48)} = 2.461$, $p=0.1232$
3C	Two-way ANOVA with Bonferroni test	Cocaine injection: $F_{(1,60)} = 81.47$, $p<0.0001$; Genotype: $F_{(2,60)} = 6.945$, $p=0.0019$; Interaction: $F_{(2,60)} = 9.120$, $p=0.0003$
3D	Two-way ANOVA with Bonferroni test	CPP: $F_{(1,42)} = 42.46$, $p<0.0001$; Genotype: $F_{(2,42)} = 5.078$, $p=0.0106$; Interaction: $F_{(2,42)} = 3.981$, $p=0.0261$
4B	T-test	$t=4.717$, $df=4$, $p<0.01$
4C	One-way ANOVA with Bonferroni test	LAMP1: $F_{(5,89)}=22.13$, $p<0.0001$; EEA1: $F_{(5,83)}=41.51$, $p<0.0001$
5A	Three-way ANOVA with Bonferroni post hoc test	D2R antagonist treatment: $F_{(1,\ 45)} = 5.444$, $p=0.0242$; Genotype: $F_{(1,\ 45)} = 10.11$, $p=0.0027$; Cocaine injection: $F_{(1,\ 45)} = 181.8$, $p<0.0001$; D2R antagonist treatment x Genotype: $F_{(1,\ 45)} = 6.321$, $p=0.0156$; D2R antagonist treatment x Cocaine injection: $F_{(1,\ 45)} = 20.27$, $p<0.0001$; Genotype x Cocaine injection: $F_{(1,\ 45)} = 20.08$, $p<0.0001$; D2R antagonist treatment x Genotype x Cocaine injection: $F_{(1,\ 45)} = 8.279$, $p=0.0061$
5B	Three-way ANOVA with Bonferroni post hoc test	Cocaine CPP: $F_{(1,\ 34)} = 71.11$, $p<0.0001$; Genotype: $F_{(1,\ 34)} = 0.000304$, $p=0.9862$; D2R antagonist: $F_{(1,\ 34)} = 0.05688$, $p=0.8129$; Cocaine CPP x Genotype: $F_{(1,\ 34)} = 3.488$, $p=0.0705$; Cocaine CPP x D2R antagonist: $F_{(1,\ 34)} = 2.488$, $p=0.1239$; Genotype x D2R antagonist: $F_{(1,\ 6)} = 11.29$, $p=0.0152$; Cocaine CPP x Genotype x D2R antagonist: $F_{(1,\ 6)} = 2.315$, $p=0.1790$
5C	T-test	$t=2.129$, $p=0.0659$
6C	Two-way ANOVA with Bonferroni test	Cocaine injection: $F_{(1,52)} = 75.41$, $p<0.0001$; Genotype: $F_{(1,52)} = 20.91$, $p<0.0001$; Interaction: $F_{(1,52)} = 27.80$, $p<0.0001$
6D	Two-way ANOVA with Bonferroni post hoc test	Cocaine injection: $F_{(1,52)} = 153.7$, $p<0.0001$; Genotype: $F_{(2,52)} = 7.592$, $p=0.0013$; Interaction: $F_{(2,52)} = 7.601$, $p=0.0013$
6E	One-way ANOVA with Bonferroni test	MEK: $F_{(3,16)} = 9.455$, $p=0.0008$; ERK: $F_{(3,16)} = 5.559$, $p=0.0083$
7A	Two-way ANOVA with Bonferroni's multiple comparisons test	Interaction: $F_{(5,122)} = 5.489$, $p=0.0001$; Cocaine injection: $F_{(1,122)} = 80.88$, $p<0.0001$; Drug treatment: $F_{(5,122)} = 8.789$, $p<0.0001$

7B	Two-way ANOVA with Bonferroni's multiple comparison test	Interaction: $F_{(4,88)}$ = 6.171, p=0.0002; Cocaine injection: $F_{(1,88)}$ = 40.61, p<0.0001; Treatment: $F_{(4,88)}$ = 4.142, p=0.0040
8A	T-test	t=2.698, df=10, p=0.0224
8B	T-test	MEK of cocaine-injected group, t=2.813, df=6, p=0.0307; ERK of cocaine-injected group, t=6.950, df=6, p=0.0004
S1A	Two-way ANOVA with Bonferroni post hoc test	Cocaine 5 mg/kg: Interaction: $F_{(2,40)}$=0.4458, p=0.6434; Cocaine injection: $F_{(1,40)}$=0.1033, p=0.7496; Genotype: $F_{(2,40)}$=4.118, p=0.0237. Cocaine 30 mg/kg: Interaction: $F_{(2,50)}$=4.316, p=0.0187; Cocaine injection: $F_{(1,50)}$=72.36, p<0.0001; Genotype: $F_{(2,50)}$=5.132, p=0.0094
S1C	Two-way ANOVA with Bonferroni post hoc test	Food CPP: $F_{(1,24)}$ = 27.24, p<0.0001; Genotype: $F_{(1,24)}$ = 0.2020, p=0.6572; Interaction: $F_{(1,24)}$ = 0.08755, p=0.7699
S2C	T-test	Dopamine: t=2.176, df=14, p=0.0471; GABA: t=0.1175, df=14, p=0.9081
S2D	T-test	MEK in Becn2 WT: t=2.949, df=8, p=0.0184; MEK in $Becn2^{+/-}$ KO: t=0.9550, df=8, p=0.3675; ERK in Becn2 WT: t=2.878, df=8, p=0.0206; ERK in $Becn2^{+/-}$ KO: t=1.231, df=8, p=0.2533
S6A	Two-way ANOVA with Bonferroni's multiple comparisons test	Whole cell - Interaction: $F_{(1,99)}$=20.95, p<0.0001; BafA1: $F_{(1,99)}$=284.4, p<0.0001; Genotype: $F_{(1,99)}$=23.29, p<0.0001. Soma - Interaction: $F_{(1,99)}$=26.63, p<0.0001; BafA1: $F_{(1,99)}$=210.4, p<0.0001; Genotype: $F_{(1,99)}$=24.68, p<0.0001. Neurite - Interaction: $F_{(1,99)}$=5.310, p=0.0233; BafA1: $F_{(1,99)}$=136.9, p<0.0001; Genotype: $F_{(1,99)}$=7.649, p=0.0068.
S6B	Two-way ANOVA with Bonferroni post hoc test T-Test	LC3-II/actin: shRNA: $F_{(1,8)}$ = 5.11345, p=0.0532; BafA1: $F_{(1,8)}$ = 41.1953, p=0.00020; shRNA X BafA1: $F_{(1,8)}$ = 6.1838, p=0.0377. p62/actin: shRNA: $F_{(1,8)}$ = 0.4806, p=0.5078; BafA1: $F_{(1,8)}$ = 2.4158, p=0.1514; shRNA X BafA1: $F_{(1,8)}$ = 7.6163, p=0.0247. BafA1[LC3-II/actin]/Basal[LC3-II/actin]: T=2.9417, df=4, p=0.0423. Becn2/actin: t=5.952, df=10, p=0.0001.
S6C	Two-way ANOVA with Bonferroni post hoc test	Genotype: $F_{(1,8)}$ = 95.6016, p<0.0001; CQ injection: $F_{(1,8)}$ = 59.3139, p<0.0001; Genotype X CQ injection: $F_{(1,8)}$ = 35.3898, p=0.0003

S8A	One-way ANOVA with Bonferroni post hoc test	D1R: $F_{(2,6)} = 2.297$, p=0.1817; DAT: $F_{(2,6)} = 0.6348$, p=0.5623
S8E	T-test	CTCF EEA1: t=0.9168, df=55, p=0.3632; CTCF LAMP1: t=0.6446, df=43, p=0.5226; Integrated density EEA1: t=1.266, df=55, p=0.2107; Integrated density LAMP1: t=0.1518, df=43, p=0.88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Cocaine:
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***
**
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WT
Becn2+/- KO
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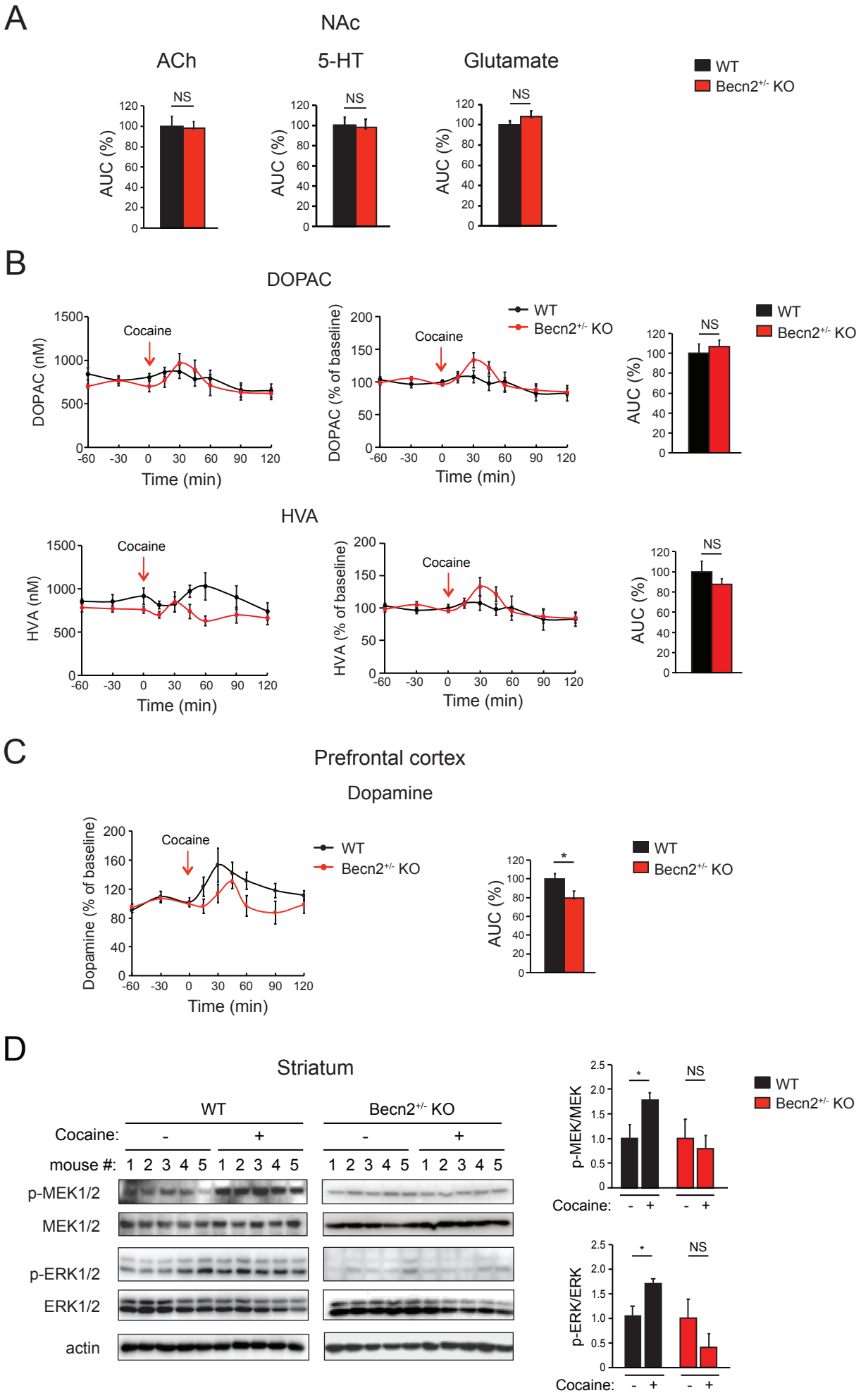
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Time (min)
HVA
HVA (nM)
HVA (% of baseline)
C
Prefrontal cortex
Dopamine
Dopamine (% of baseline)
*
D
Striatum
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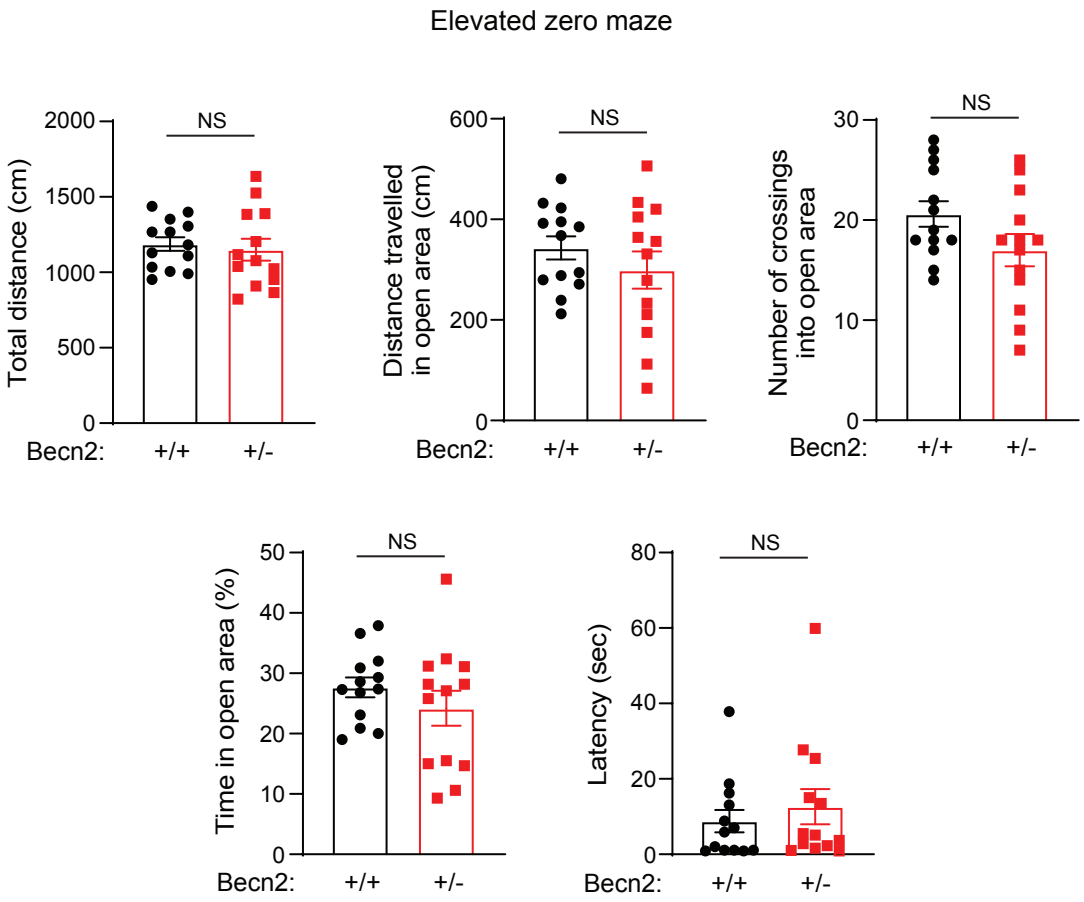
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NS
Becn2: +/+ +/-
Number of crossings into open area
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20
10
0
NS
Becn2: +/+ +/-
Time in open area (%)
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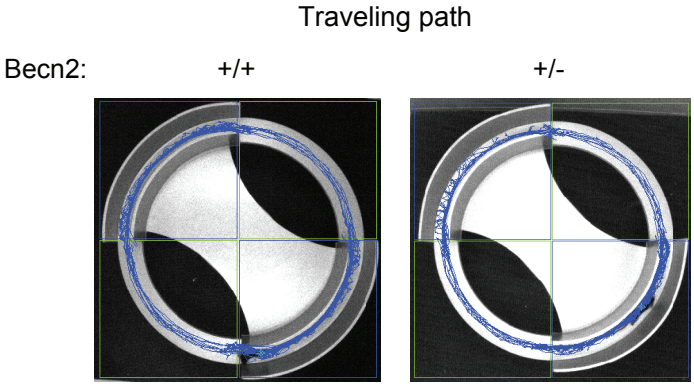
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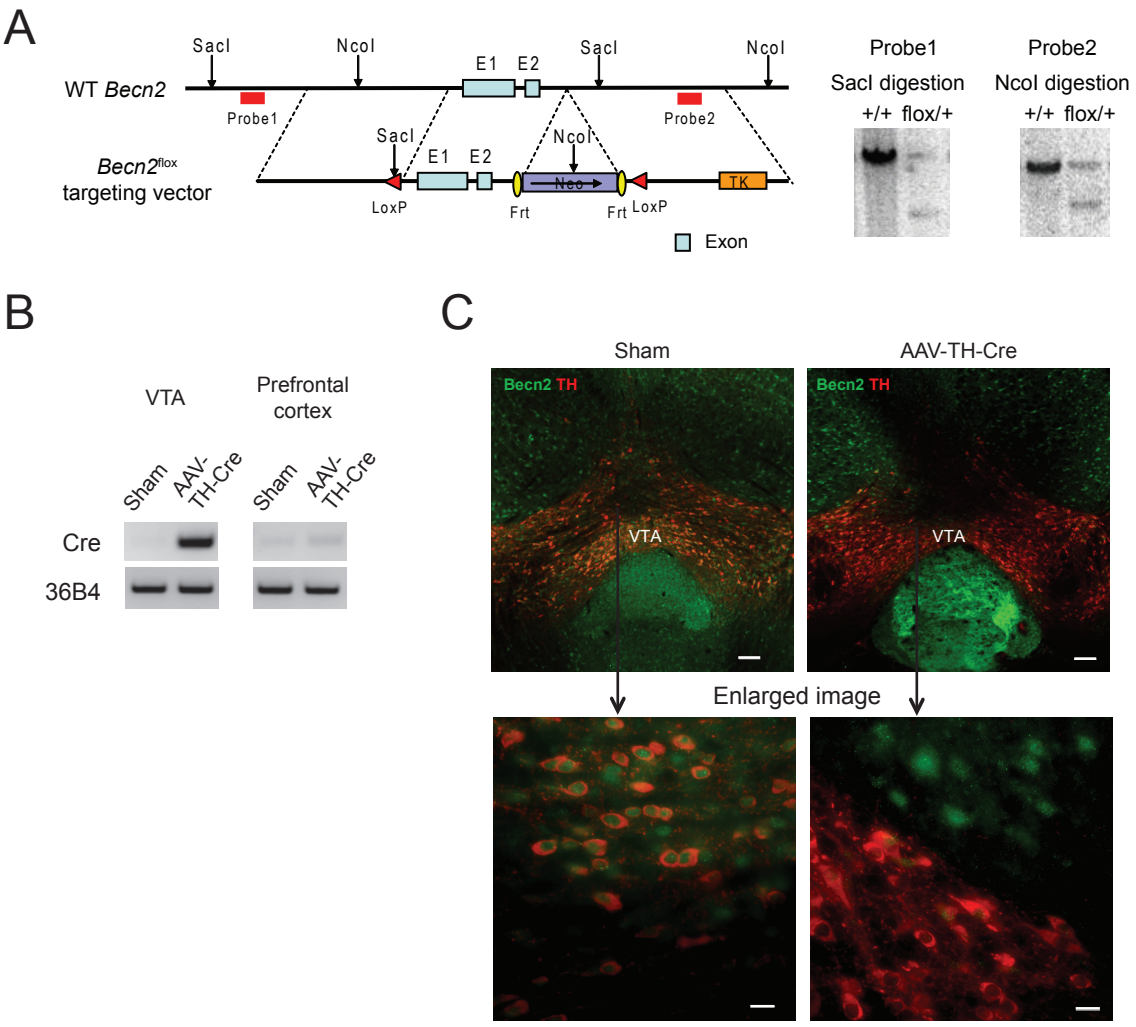
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Exon
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Prefrontal cortex
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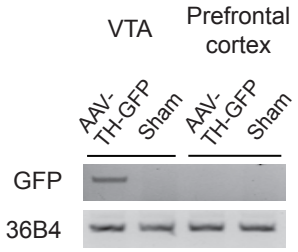
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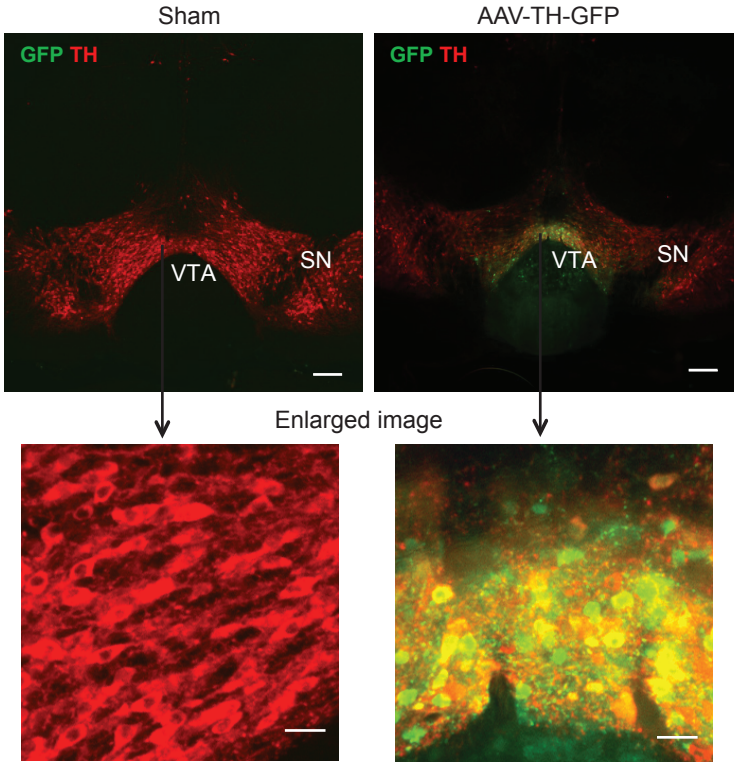
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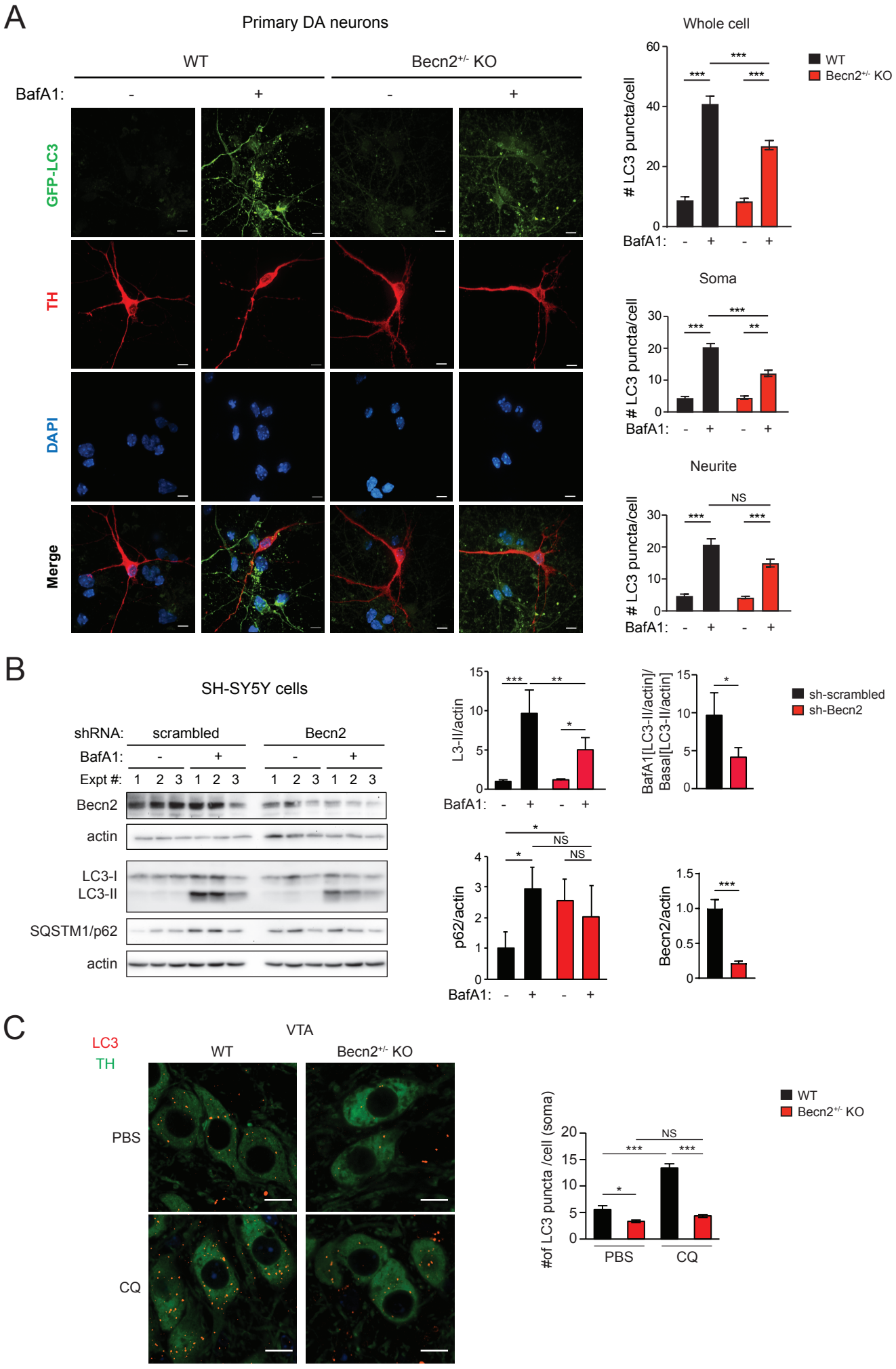
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Soma
Neurite
NS
B
SH-SY5Y cells
shRNA:
scrambled
Becn2
Expt #:
Becn2
actin
LC3-I
LC3-II
SQSTM1/p62
L3-II/actin
BafA1[LC3-II/actin]/ Basal[LC3-II/actin]
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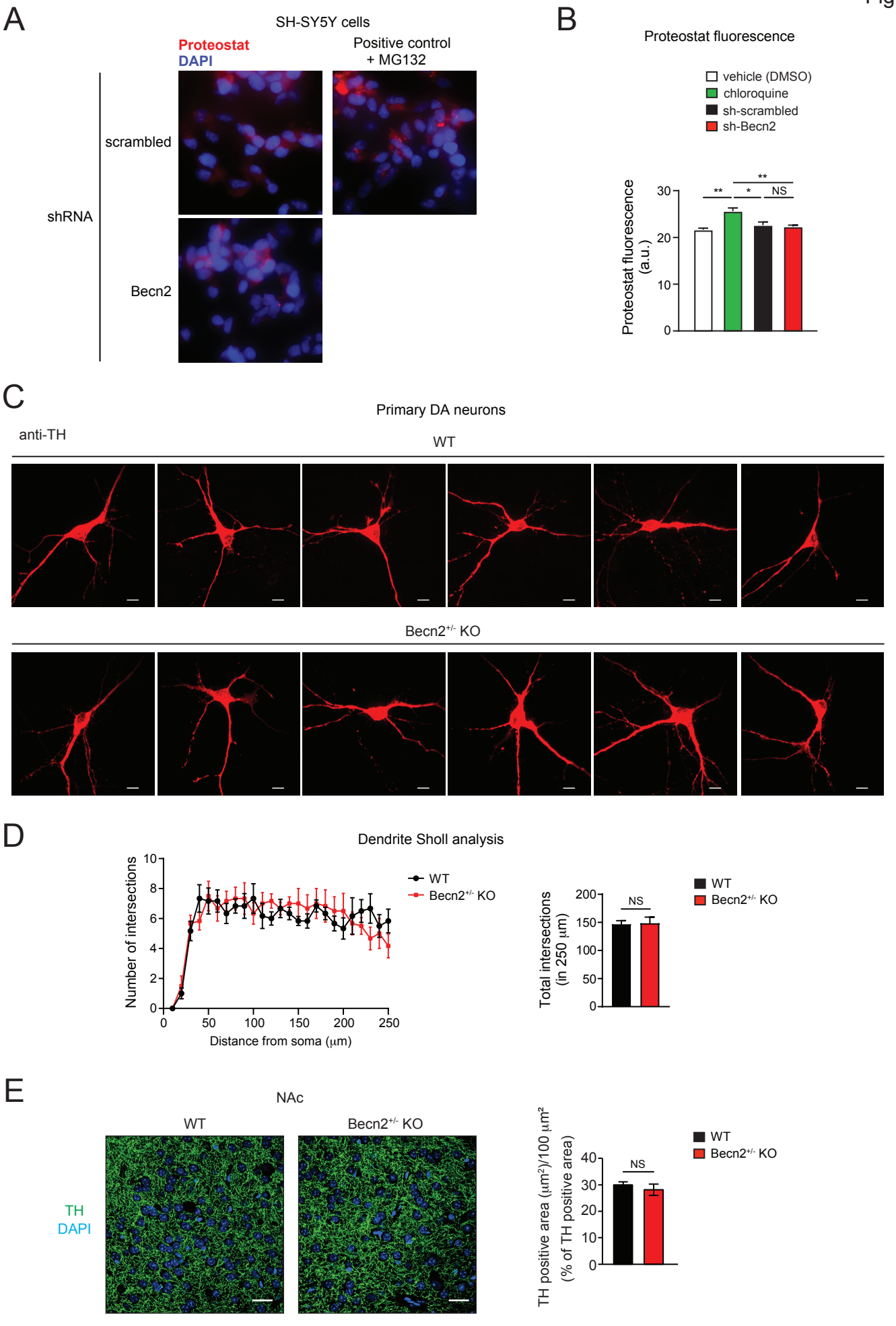
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30
20
10
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NS
C
Primary DA neurons
anti-TH
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Becn2+/- KO
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Dendrite Sholl analysis
Number of intersections
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6
4
2
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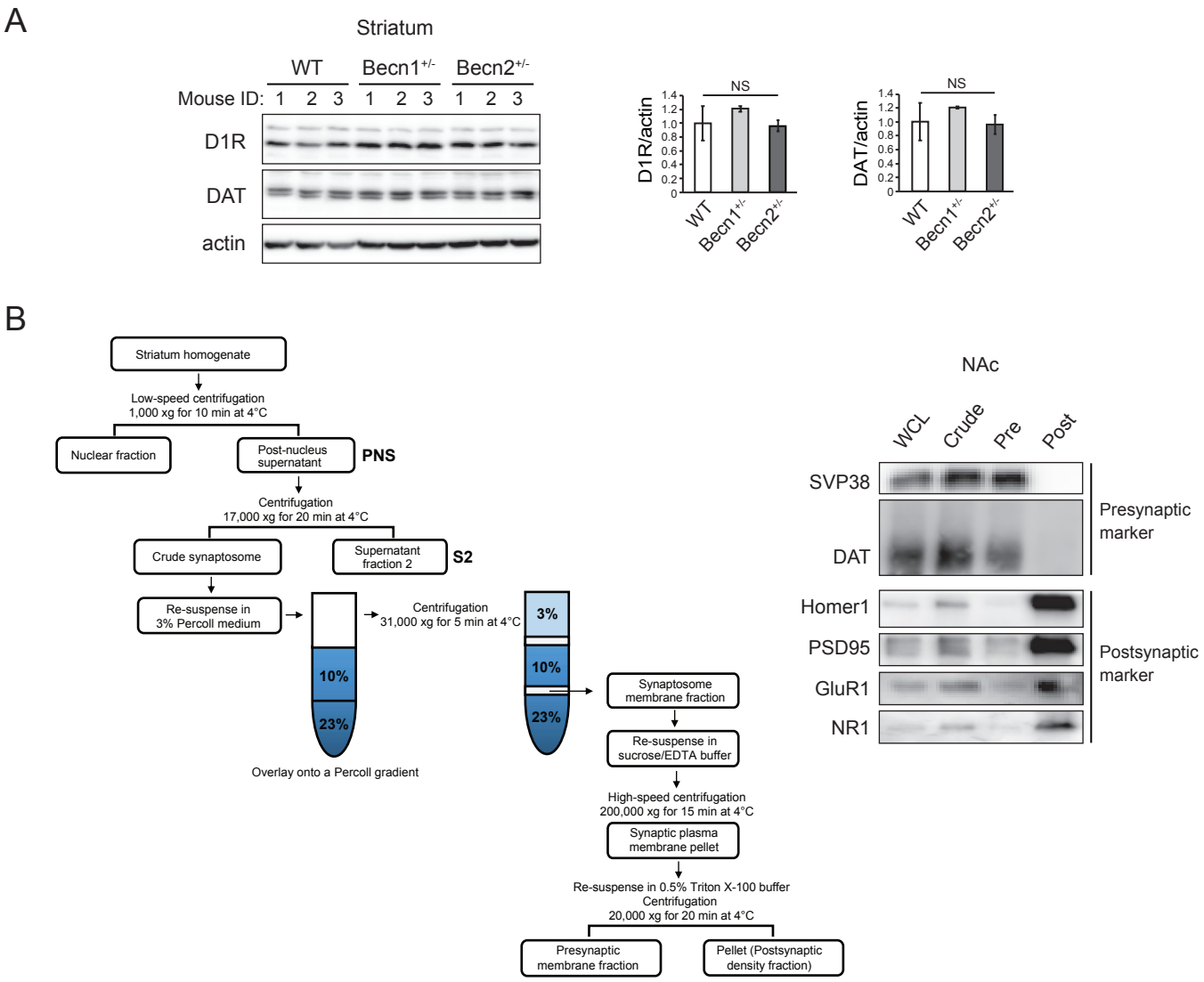
100
150
200
250
Distance from soma (μm)
WT
Becn2+/- KO
Total intersections (in 250 μm)
200
150
100
50
0
NS
WT
Becn2+/- KO
E
NAc
WT
Becn2+/- KO
TH
DAPI
TH positive area (μm²)/100 μm² (% of TH positive area)
40
30
20
10
0
NS
WT
Becn2+/- KO


A
Striatum
WT
Becn1+/-
Becn2+/-
Mouse ID: 1 2 3 1 2 3 1 2 3
D1R
DAT
actin
D1R/actin
DAT/actin
NS
WT
Becn1+/-
Becn2+/-
B
Striatum homogenate
Low-speed centrifugation
1,000 xg for 10 min at 4°C
Nuclear fraction
Post-nucleus supernatant
PNS
Centrifugation
17,000 xg for 20 min at 4°C
Crude synaptosome
Supernatant fraction 2
S2
Re-suspense in 3% Percoll medium
10%
23%
Overlay onto a Percoll gradient
Centrifugation
31,000 xg for 5 min at 4°C
3%
10%
23%
Synaptosome membrane fraction
Re-suspense in sucrose/EDTA buffer
High-speed centrifugation
200,000 xg for 15 min at 4°C
Synaptic plasma membrane pellet
Re-suspense in 0.5% Triton X-100 buffer
Centrifugation
20,000 xg for 20 min at 4°C
Presynaptic membrane fraction
Pellet (Postsynaptic density fraction)
NAc
WCL
Crude
Pre
Post
SVP38
DAT
Presynaptic marker
Homer1
PSD95
GluR1
NR1
Postsynaptic marker
C
NAc
Crude
Pre
Becn2: +/+ +/- +/+ +/-
D2R
1.0 3.0
50 kD
SVP38
PSD95
Prefrontal cortex
Crude
Pre
Becn2: +/+ +/- +/+ +/-
D2R
1.0 0.8
50 kD
SVP38
PSD95
D
shRNA: scrambled Becn2
Becn2
Becn1
GAPDH
E
sh-scrambled
sh-Becn2
EEA1
LAMP1
Corrected total cell fluorescence (CTCF)
Integrated density
NS
F
NAc
PNS
S2
f/f
f/f TH
f/f
f/f TH
D2R
1.0 1.4 1.0 0.5
SVP38
1.0 1.0 1.1 1.1
PSD95
1.0 1.1 0.3 0.2

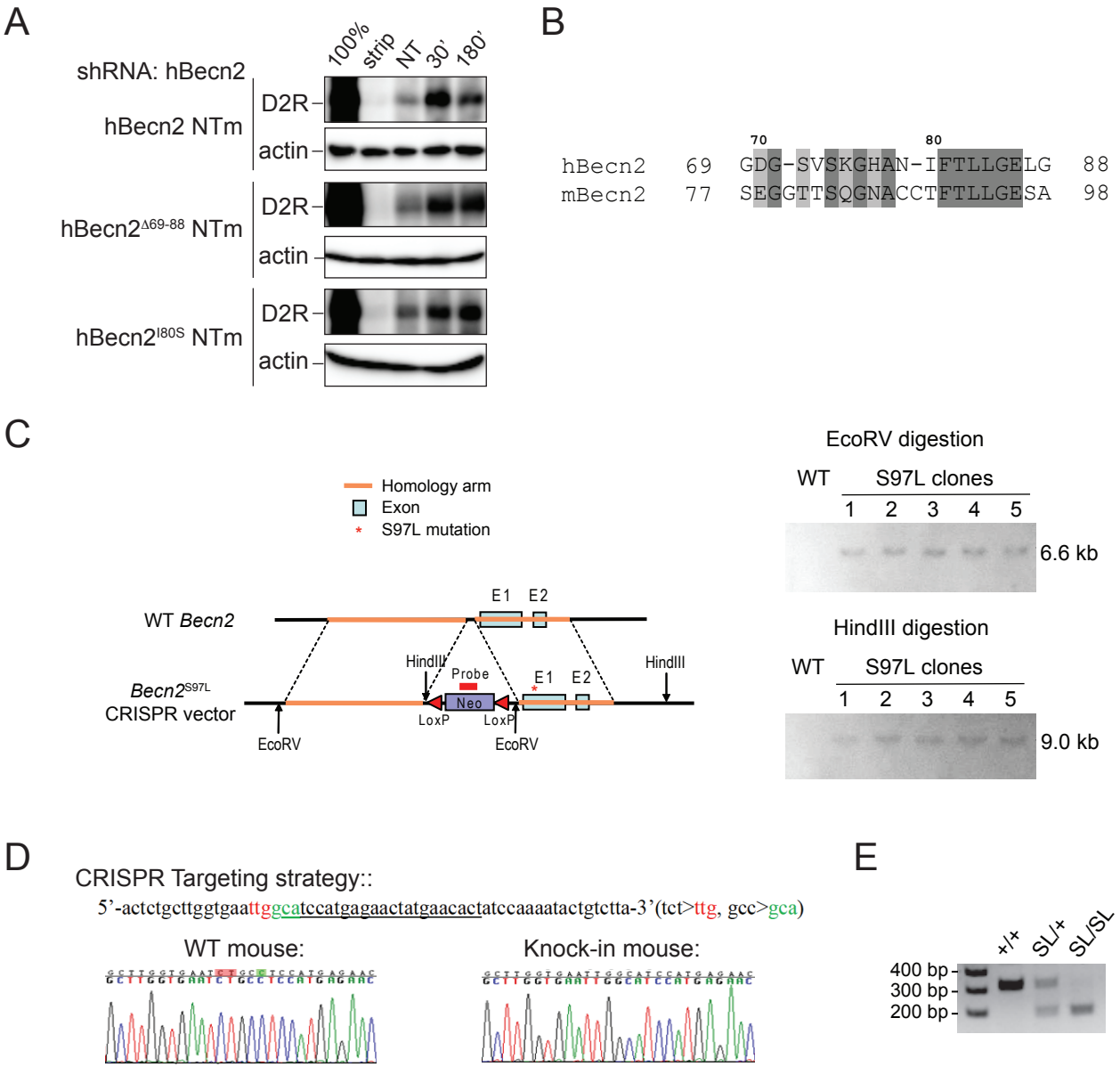
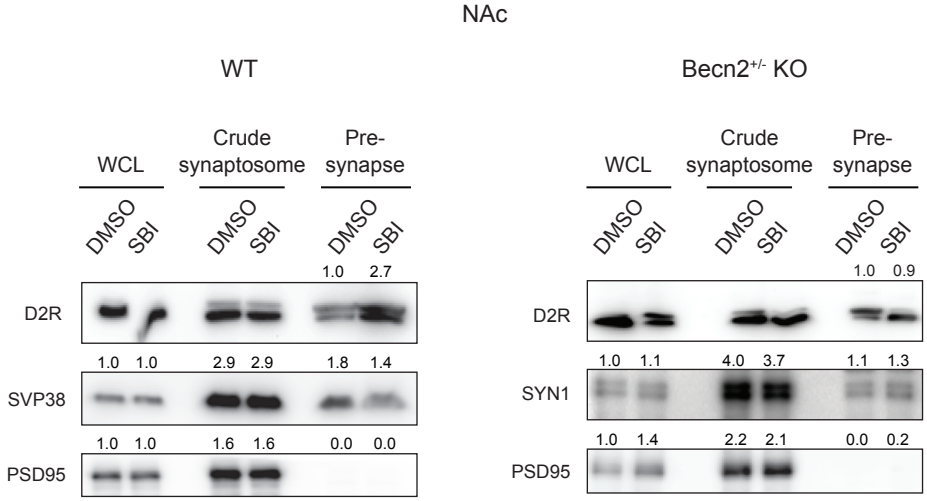

A
100%
strip
NT
30'
180'
shRNA: hBecn2
hBecn2 NTm
D2R
actin
hBecn2Δ69-88 NTm
hBecn2I80S NTm
B
70
80
hBecn2 69 GDG-SVSKGHAN-IFTLLGELG 88
mBecn2 77 SEGGTTSQGNACCTFTLLGESA 98
C
Homology arm
Exon
S97L mutation
WT Becn2
Becn2S97L
CRISPR vector
E1
E2
HindIII
Probe
Neo
LoxP
EcoRV
EcoRV digestion
WT
S97L clones
1 2 3 4 5
6.6 kb
HindIII digestion
9.0 kb
D
CRISPR Targeting strategy::
5'-actctgcttggtgaattggcatccatgagaactatgaacactatccaaaatactgtctta-3'(tct>ttg, gcc>gca)
WT mouse:
Knock-in mouse:
E
+/+
SL/+
SL/SL
400 bp
300 bp
200 bp

Figure S10


NAc
WT
Becn2+/- KO
WCL
Crude synaptosome
Pre-synapse
DMSO
SBI
D2R
SVP38
PSD95
SYN1
1.0 1.0 2.9 2.9 1.8 1.4
1.0 1.0 1.6 1.6 0.0 0.0
1.0 2.7
1.0 1.1 4.0 3.7 1.1 1.3
1.0 1.4 2.2 2.1 0.0 0.2
1.0 0.9