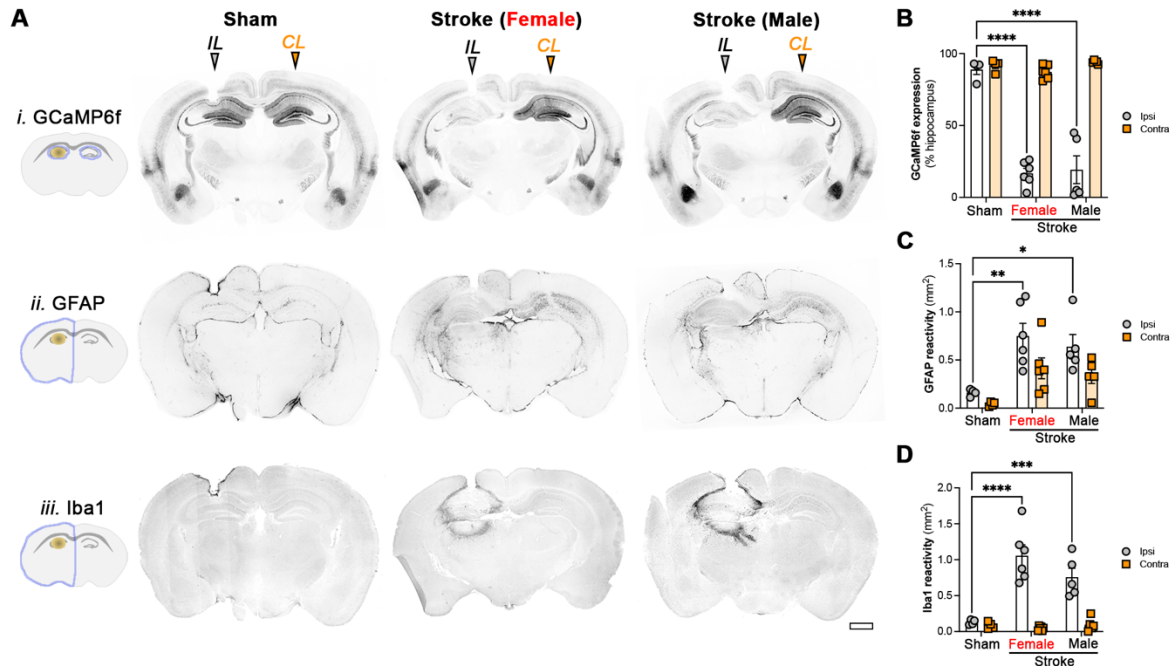
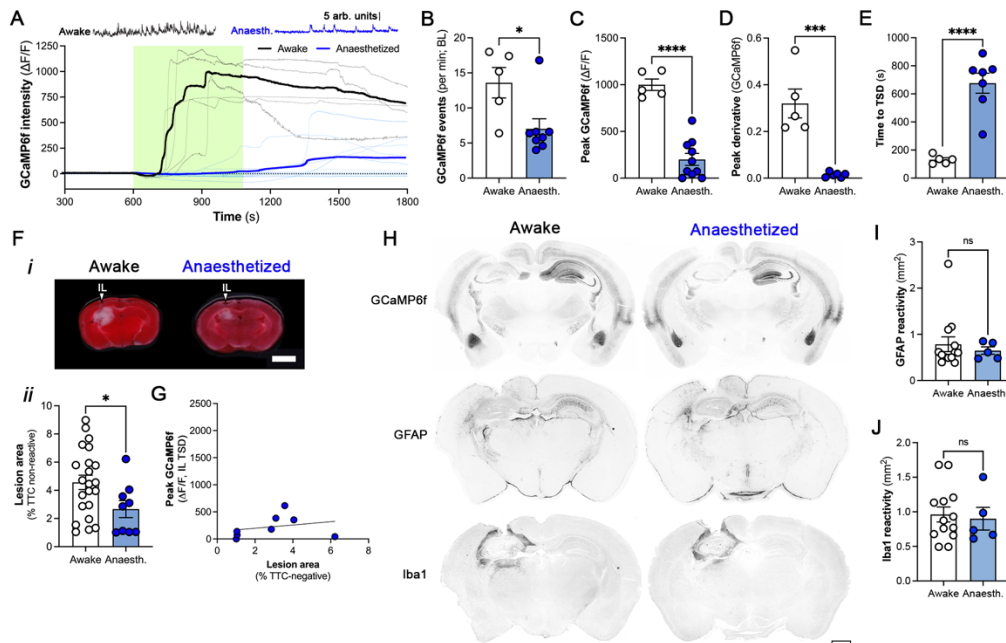


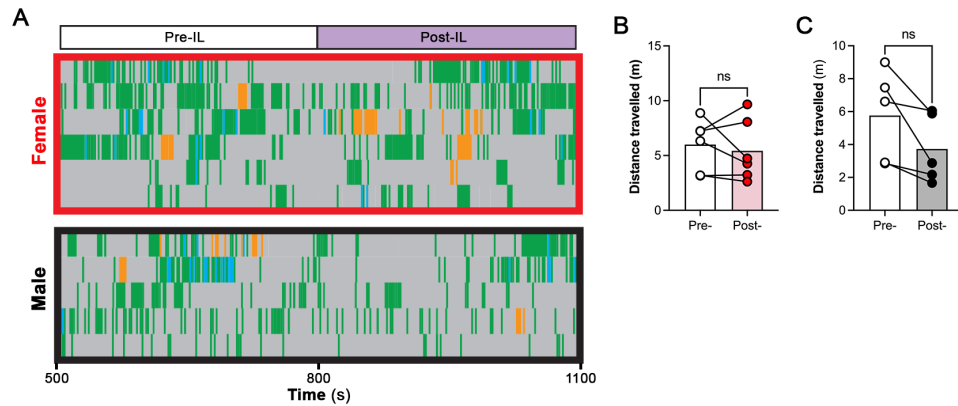
Supplementary Figure 1. Illumination power and time-dependent increases in ipsilesional TSD and lesion size. (A) Increasing laser illumination power and duration increases neuronal Ca^{2+} influx (GCaMP6f, $\Delta F/F$) during unilateral hippocampal photothrombosis in awake mice (mean $\pm$ s.e.m.). Distinct laser illumination parameters were used to initiate photothrombosis through the optical fiber also utilized for photometry (561 nm – black: 6 mW, 8 min, $n = 24$; green: 6 mW, 1.5 min, $n = 5$; orange: 5 mW, 1.5 min, $n = 8$; purple: 4 mW, 1.5 min, $n = 4$; grey: sham, $n = 4$). **(B)** Increasing illumination duration and power increased ipsilesional TSD (peak GCaMP6f, $\Delta F/F$; 6 mW, 8 min, 1258.0 ± 102.8 ; green: 6 mW, 1.5 min, $982.6 \text{ arb. units} \pm 128.5$; orange: 5 mW, 1.5 min, $323.4 \text{ arb. units} \pm 138.4$; purple: 4 mW, 1.5 min, $13.0 \text{ arb. units} \pm 13.0$, one-way ANOVA with Tukey *post hoc*, * $p < 0.05$, ** $p < 0.01$, **** $p < 0.0001$). **(C)** Photothrombotic stroke lesion scales with illumination duration (representative TTC-stained coronal sections). Scale bars, 2 mm. TTC-stained brain sections from independent mice were placed on a uniform black background. Stroke lesions (% TTC-negative brain area) are larger in 8 min illuminated mice. One-way ANOVA with Tukey *post hoc*, sham: $0.0\% \pm 0.0$, stroke (1.5 min, 6 mW): $0.86\% \pm 0.17$, stroke (8 min, 6 mW): $4.57\% \pm 0.17$ at 48 h post-stroke; **** $p < 0.0001$.



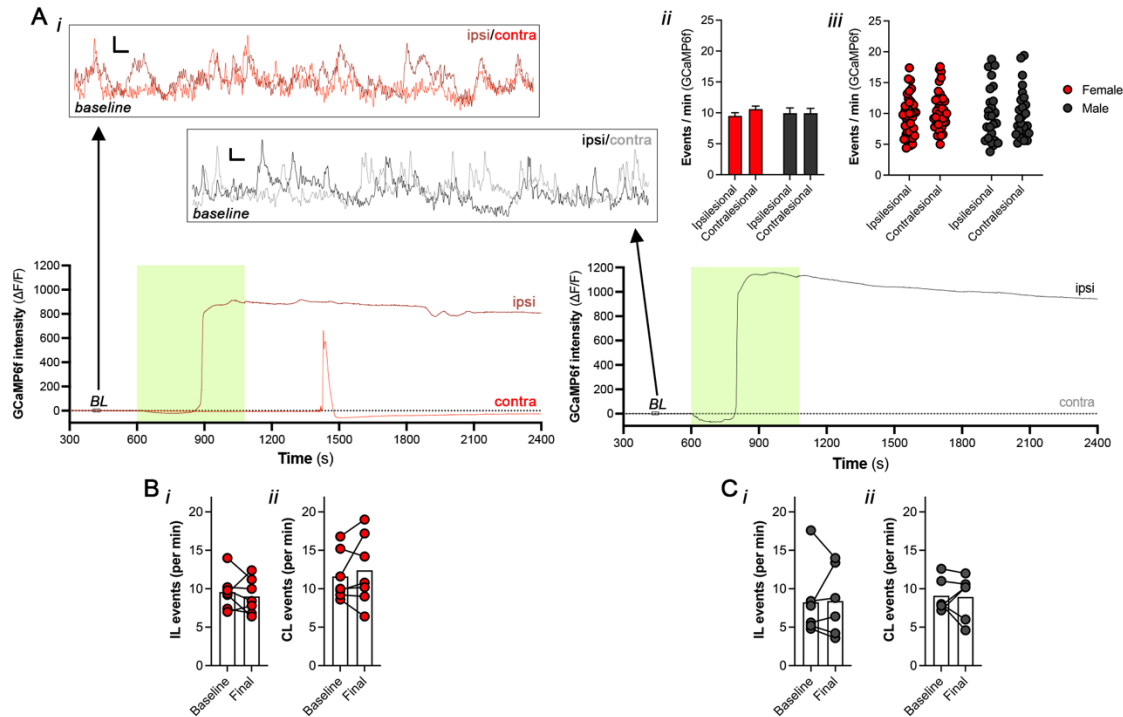
Supplementary Figure 2. Neuronal loss and glial reactivity are indistinguishable between males and females after unilateral hippocampal stroke. (A) Representative coronal sections highlight reduction of (i) hippocampal GCaMP6f expression and increased (ii) GFAP and (iii) Iba1-immunoreactivity in the ipsilesional hemisphere of female ($n = 6$) and male ($n = 5$) mice below the optical fiber tract one week following unilateral hippocampal photothrombosis relative to mice experiencing sham illumination ($n = 4$). Scale bars, 1 mm. (B) GCaMP6f expression (% of hippocampus positive for GCaMP6f) is reduced in the ipsilesional hippocampus after stroke (sham, ipsi: $88.91 \pm 3.39\%$, sham, contra: $91.67 \pm 2.07\%$; stroke – female, ipsi: $16.78\% \pm 3.5$, stroke – female, contra: $87.23 \pm 1.97\%$; stroke – male, ipsi: $19.24 \pm 9.7\%$, stroke – male, contra: $94.10 \pm 0.53\%$; two-way ANOVA with Bonferroni *post-hoc*, stimulus: $F_{2,24} = 36.65$, $p < 0.0001$, hemisphere: $F_{1,24} = 167.3$, $p < 0.0001$, interaction: $F_{2,24} = 33.64$, $p < 0.0001$; **** $p < 0.0001$). (C) GFAP reactivity in the ipsilesional hemisphere is increased following unilateral hippocampal photothrombosis (sham, ipsi: $161.3 \pm 18.5 \mu\text{m}^2$, sham, contra: $42 \pm 12 \mu\text{m}^2$; stroke – female, ipsi: $749.7 \pm 131.3 \mu\text{m}^2$, stroke – female, contra: $414 \pm 108 \mu\text{m}^2$, stroke – male, ipsi: $517.3 \pm 47.3 \mu\text{m}^2$, stroke – male, contra: $335 \pm 79 \mu\text{m}^2$; two-way ANOVA with Bonferroni *post-hoc*, stimulus: $F_{2,24} = 10.66$, $p = 0.0005$, hemisphere: $F_{1,24} = 8.52$, $p = 0.0075$, interaction: $F_{2,24} = 0.56$, $p = 0.58$; * $p < 0.05$, ** $p < 0.01$). (D) Iba1 reactivity (mm^2) in the ipsilesional hemisphere is increased following unilateral hippocampal photothrombosis (sham, ipsi: $127.3 \pm 17.0 \mu\text{m}^2$, sham, contra: $85 \pm 24 \mu\text{m}^2$; stroke – female: $1057.0 \pm 150.1 \mu\text{m}^2$, stroke – female, contra: $44 \pm 12 \mu\text{m}^2$, stroke – male: $982.6 \pm 206.9 \mu\text{m}^2$, stroke – male, contra: $96 \pm 42 \mu\text{m}^2$; two-way ANOVA with Bonferroni *post-hoc*, stimulus: $F_{2,24} = 11.13$, $p = 0.0004$, hemisphere: $F_{1,24} = 55.49$, $p < 0.0001$, interaction: $F_{2,24} = 13.03$, $p = 0.0001$; *** $p < 0.001$, **** $p < 0.0001$).



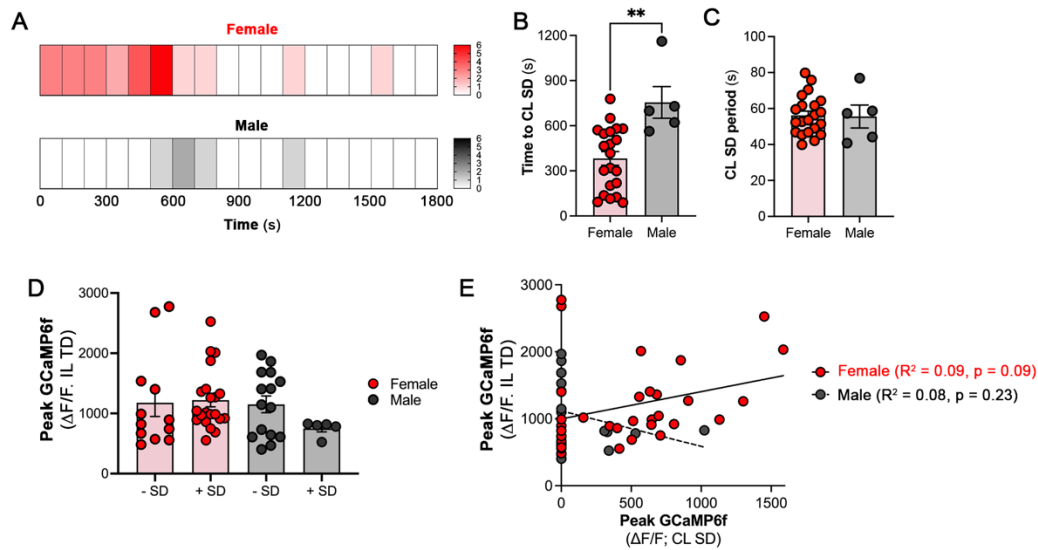
Supplementary Figure 3. Isoflurane anesthesia protects against focal hippocampal stroke. **(A)** Representative 60 s segments of baseline hippocampal GCaMP6f photometry recordings of awake (black) and anesthetized (blue) Thy1GCaMP6f mice. Neuronal Ca²⁺ dysregulation (ΔF/F; mean: thick lines; individual traces: thin lines) induced by unilateral hippocampal photothrombosis is distinct between awake (black) and anesthetized (blue) mice. Photothrombotic illumination (561 nm, 6 mW, 10-18 min). **(B)** Awake mice have more frequent Ca²⁺ events per min than anesthetized mice during the baseline epoch (awake: 13.6 ± 2.2 , n = 5; anesthetized: 7.0 ± 1.4 , n = 8; unpaired t-test, *p < 0.05). **(C)** Awake mice have larger TSD (peak GCaMP6f, ΔF/F) following hippocampal photothrombosis (awake: $999.7 \text{ arb. units} \pm 61.0$, n = 5; anesthetized: $200.5 \text{ arb. units} \pm 64.1$, n = 8; unpaired t-test, ***p < 0.001). **(D)** Slope of ipsilesional TSD (peak derivative of GCaMP6f) is larger in awake mice (awake: 0.32 ± 0.06 , n = 5; anesthetized: 0.04 ± 0.004 , n = 6; unpaired t-test, ***p < 0.001). **(E)** TSD onset (t_{peak derivative, GCaMP6f}) is more rapid in awake mice following onset of photothrombotic illumination (awake: $131.4 \text{ s} \pm 13.8$, n = 6; anesthetized: $676.2 \text{ s} \pm 71.7$, n = 7; unpaired t-test, ****p < 0.0001). **(F, i)** Representative TTC-stained coronal sections from awake and anesthetized mice 48 h after unilateral hippocampal photothrombosis reveal that **(ii)** awake mice have larger lesions than anesthetized mice (%TTC-negative brain area, awake: $4.57\% \pm 0.51$, anesthetized: $2.67\% \pm 0.6$, unpaired t-test, *p < 0.05; 48 h post-illumination). TTC-stained brain sections from independent mice were placed on a uniform black background. **(G)** No correlation exists between ipsilesional TSD and hippocampal stroke lesion size of anesthetized mice (R² = 0.02, p = 0.73). **(H)** Representative coronal sections at fiber implant site reveal that glial reactivity is not significantly different between awake (n = 13) and anesthetized mice (n = 5) one-week post-stroke. Scale bars, 1 mm. **(I)** Iba1 reactivity (mm²) is not different in awake and anesthetized stroke (awake: $962.0 \mu\text{m}^2 \pm 108.8$, anesthetized: $900.4 \mu\text{m}^2 \pm 163.7$, unpaired t-test). **(J)** GFAP reactivity (mm²) is not different in awake and anesthetized stroke (awake: $784.7 \mu\text{m}^2 \pm 159.8$, anesthetized: $647.4 \mu\text{m}^2 \pm 79.7$, unpaired t-test).



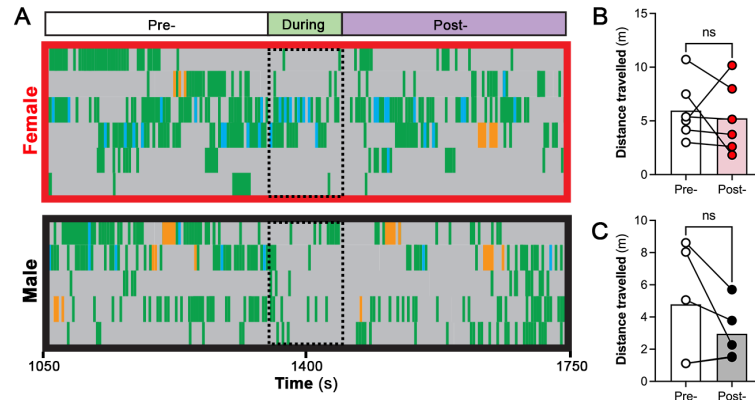
Supplementary Figure 4. Sham mice do not alter activity or locomotion in the approximate peri-TSD period. (A) Gantt behavioral plots for all analyzed female (red outline, $n = 6$) and male (black outline, $n = 5$) sham mice centered on the average time of TSD onset identified in the stroke group (800 s). **(B)** Distance travelled by female sham mice does not change following the average time of TSD onset in stroke mice (800 s, $n = 6$; pre-IL: $6.0 \text{ m} \pm 0.95$, post-IL: $5.43 \text{ m} \pm 1.15$). **(C)** Distance travelled by male sham mice does not change following the average time of TSD onset in stroke mice (800 s, $n = 5$, pre-IL: $5.77 \text{ m} \pm 1.24$, post-IL: $3.73 \text{ m} \pm 0.93$).



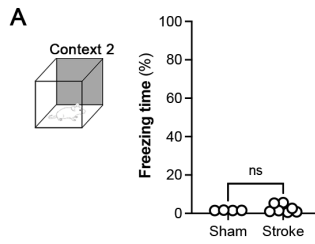
Supplementary Figure 5. Basal ipsi- and contralesional Ca²⁺ transients are unchanged over recording period. (A) Baseline ipsilesional (dark) and contralesional (light) GCaMP6f photometry traces (scale bar: 1 arb. units, 1 s) from awake and freely-behaving (i) female (red) and (ii) male (black) mice preceding photothrombosis (see Fig. 2 for SD analyses). (iii) No significant differences between Ca²⁺ events in ipsilesional and contralesional hippocampi between females (red, IL: 9.51 ± 0.52 , CL: 10.6 ± 0.49) and males (black, IL: 9.95 ± 0.85 , CL: 9.94 ± 0.78), displayed as mean $\pm$ s.e.m. (i, bar graph) or individual values (ii, scatter plot). Two-way ANOVA with Bonferroni *post hoc*, sex: $F_{1,63} = 0.02$, $p = 0.88$; hemisphere: $F_{1,63} = 1.02$, $p = 0.32$; interaction: $F_{1,63} = 1.02$, $p = 0.32$. (B) No change in Ca²⁺ events (per min) between baseline (300 - 600 s) and final (2100 - 2400 s) epochs in (i) ipsilesional (BL: 9.57 ± 0.87 , final: 9.0 ± 0.86 , $n = 7$) and (ii) contralesional GCaMP6f (BL: 11.6 ± 1.2 , final: 12.4 ± 1.72 , $n = 7$) from female shams (paired t-tests). (C) No change in Ca²⁺ events (per min) between baseline and final epochs in (i) ipsilesional (BL: 8.23 ± 1.97 , final: 8.4 ± 1.84 , $n = 6$) and (ii) contralesional GCaMP6f (BL: 9.1 ± 0.89 , final: 8.93 ± 1.2 , $n = 6$) from male shams. Unpaired t-tests.



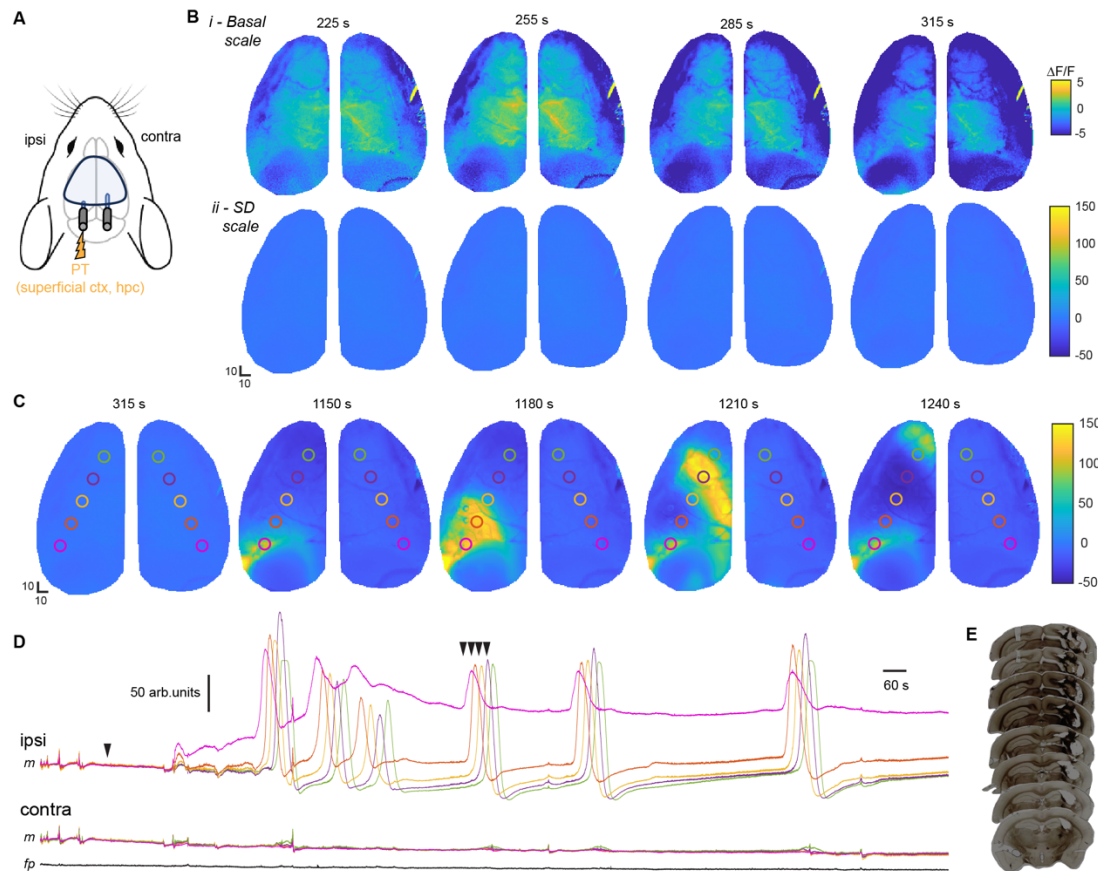
Supplementary Figure 6. Sex-specific contralesional SD characteristics. (A) Contralesional SD occur more rapidly in females than in males. Frequency plot (100 s bins) for CL SD onset following ipsilesional TSD (female: n = 21, male: n = 5). **(B)** Onset of CL SD is more rapid in females (females: 382.6 ± 45.71 , n = 21; males: 755.0 ± 105.6 , n = 5; unpaired t-test, ** p = 0.002). **(C)** No significant difference in CL SD period (females: 56.22 ± 2.40 , n = 21; males: 55.58 ± 6.40 , n = 5; unpaired t-test). **(D)** Ipsilesional TSD amplitude has no impact on the presence of CL SD. Two-way ANOVA with Tukey *post-hoc* (sex: $F_{1,49} = 1.88$, p = 0.18; $\pm$ SD: $F_{1,49} = 0.97$, p = 0.33; interaction: $F_{1,49} = 1.50$, p = 0.23). **(E)** Ipsilesional TSD and contralesional SD amplitudes ($\Delta F/F$) are not correlated in females (red) or males (black). Linear regression (female: solid line; $R^2 = 0.09$, p = 0.09; male: dotted line; $R^2 = 0.08$, p = 0.23).



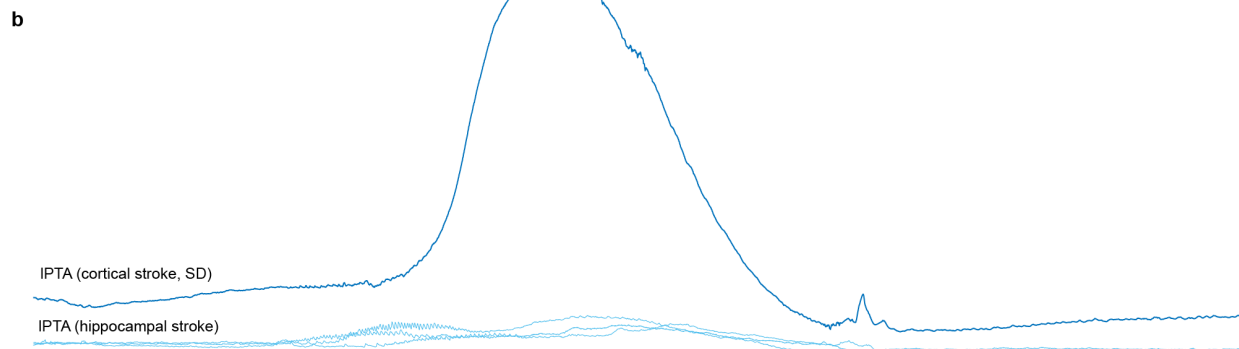
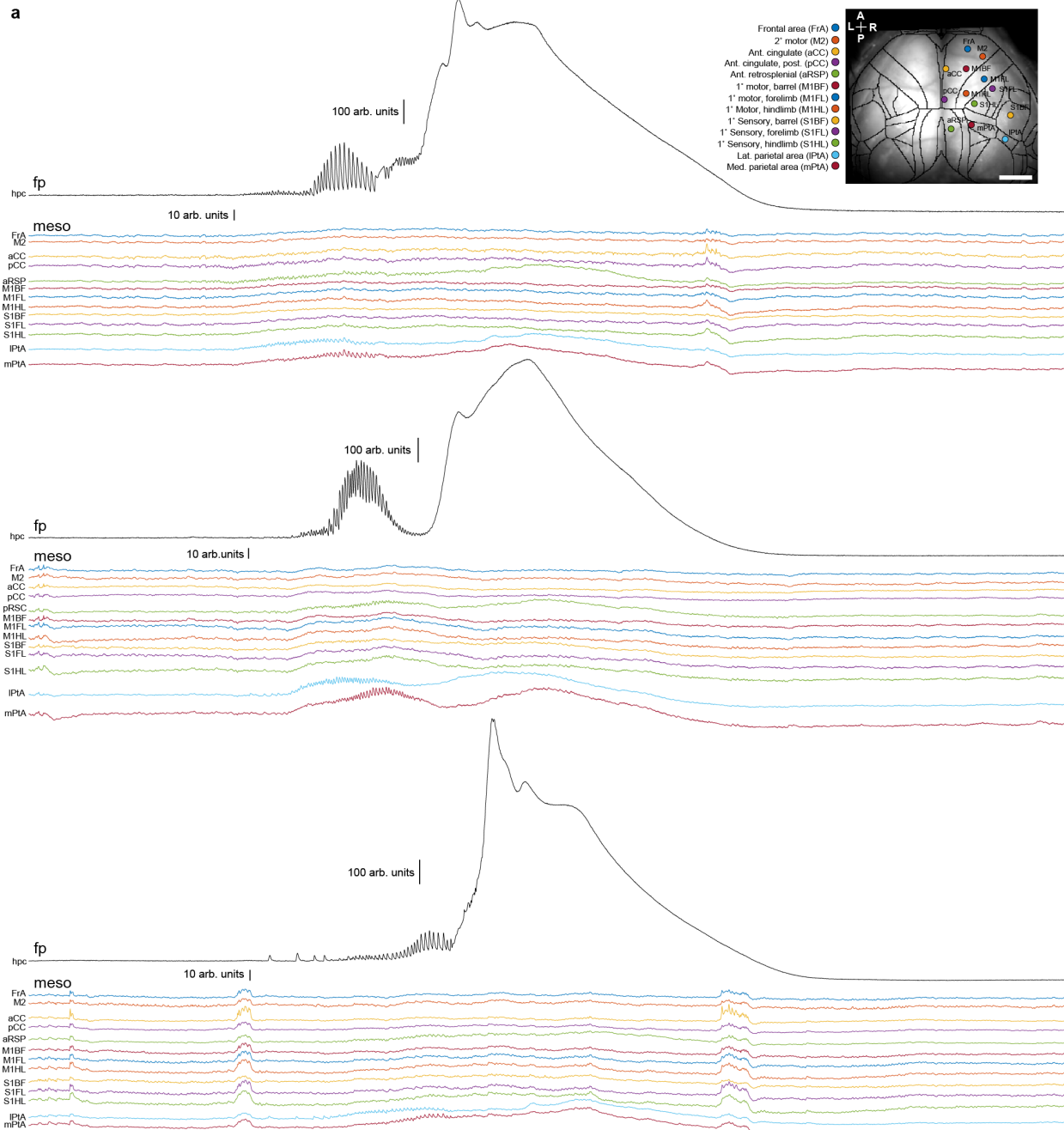
Supplementary Figure 7. Sham mice do not alter activity or locomotion in the approximate peri-CL SD epoch. (A) Gantt behavioral plots for all analyzed female (red outline, n = 6) and male (black outline, n = 4) sham mice centered on the average time of CL SD identified in the stroke group (1400 s). **(B)** Distance travelled by female sham mice does not change following the average time of CL SD in stroke mice (pre-: 5.96 m ± 1.13, post-: 5.24 m ± 1.33, paired t-test). **(C)** Distance travelled by male sham mice does not change following the average time of CL SD in stroke mice (pre-: 4.79 m ± 1.61, post-: 2.96 m ± 0.8, paired t-test).



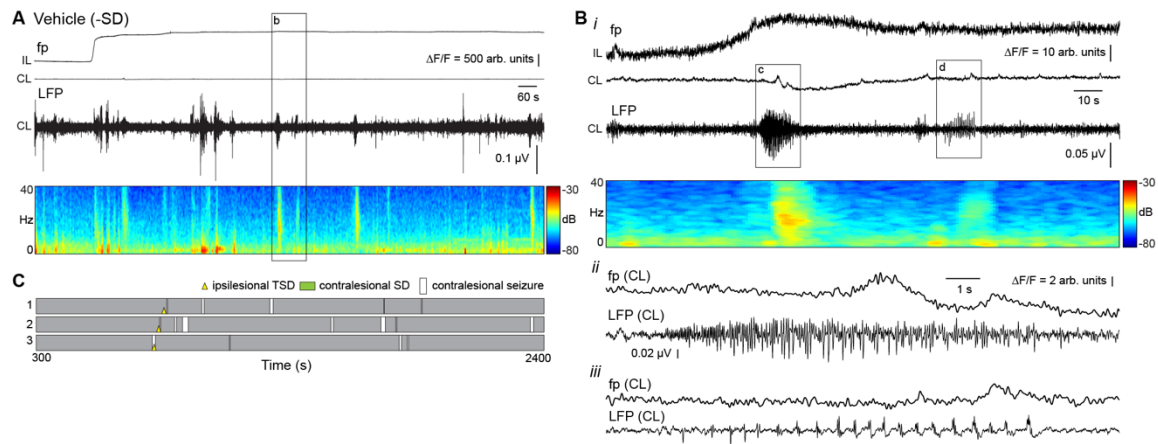
Supplementary Figure 8. Freezing behavior is specific to fear conditioned context. (A) No change in time spent freezing (%) by sham or stroke mice in a novel context independent of foot shock. Unpaired t-test, $p = 0.33$ (sham: $n = 4$, stroke: $n = 8$).



Supplementary Figure 9. Unilateral hippocampal-neocortical photothrombosis triggers SDs restricted to ipsilesional neocortex. **(A)** Schematic of mesoscale imaging window above superficial neocortex and bilateral fiberoptic implants (left: neocortex and dorsal hippocampus; right: dorsal hippocampus). **(B)** Baseline mesoscale neocortical GCaMP6f dynamics captured at **(i)** basal fluctuation and **(ii)** SD scales over 4 time points at 30 s intervals (see $\Delta F/F$, right). **(C)** Representative mesoscale neocortical GCaMP6f images (basal: 315 s, SD: 1150-1240, 30 s intervals) highlighting an ipsilesionally-confined SD from ischemic neocortex. Photothrombosis (561 nm: 6 mW, 8 min) occurred at 600 s. **(D)** Representative structure/ROI-based (11 px², 35 μ m/px) mesoscale imaging traces (n = 1, m) from female mouse for ROIs separated by 1.2 mm outward from the ischemic core in the SD propagation axis in ipsilesional (light, bold) neocortex, reflected in contralateral (dark) hemisphere following photothrombosis encroaching into neocortical tissue. Proximal ROI highlights TSD, while distal ROIs indicate up to 5 cortical SDs confined to the ipsilesional neocortex and absent from the time-locked contralateral hippocampal photometry recording. **(E)** Coronal sections of representative female mouse brain experiencing focal neocortical/hippocampal photothrombosis, extracted 7 d post-illumination, indicates a unilateral stroke lesion encroaching on neocortex.



Supplementary Figure 10. Cortical neurons demonstrate seizure-like Ca^{2+} dynamics without SD during ischemia-induced contralesional hippocampal SD. (A) 200 s epochs of contralesional recordings surrounding hippocampal SD ($\Delta F/F$, fiber photometry) following unilateral hippocampal photothrombosis highlight the presence of rhythmic GCaMP6f dynamics in neocortical mesoscale imaging ($\Delta F/F$) but the absence of a large slow component (cortical spreading depolarization) during hippocampal SD. **(B)** Contralesional mesoscale GCaMP6f traces from the lateral parietal area following hippocampal photothrombosis, normalized to the GCaMP6f response in the same region during a cortical stroke-induced SD.



Supplementary Figure 11. Contralesional electrographic seizures can occur in the absence of SD. (A) Example ipsilesional (IL) and contralesional (CL) GCaMP6f ($\Delta F/F$, red) paired with CL LFP (black trace) and time-varying frequency-power-time spectrogram in an awake, behaving female mouse (600 – 2400 s) pretreated with vehicle (PBS) via injection i.p. prior to baseline recording. **(B)** Window (180 s, *i*) surrounding representative seizures in the absence of SD. (*ii*, *iii*) Expanded sections (15 s) highlight differences in seizure waveform (LFP) relative to GCaMP6f dynamics. **(C)** Schematic overlay of contralesional recordings (LFP/FP) in vehicle-treated mice, highlighting incidence of electrographic seizure (white box) in relation to IL TSD (yellow triangle), but absent of CL SD (green box).

Supplemental Tables: Detailed Statistical Information

Supplementary Table 1. Detailed Statistical Information for Figure 1.

Figure Panel	Response variable	n	statistical analysis	Result
Figure 1e	Peak GCaMP6f ($\Delta F/F$)	sham - female = 7, stroke - female = 32, sham - male = 6, stroke male = 20	two-way ANOVA with Tukey <i>post hoc</i>	sex: F1, 61 = 0.20, p = 0.65; sham vs. stroke: F1, 61 = 43.74, **** p < 0.0001; interaction: F1,61 = 0.20, p = 0.65; female – stroke: 1205 arb. units $\pm$ 106.8, n = 32; male – stroke: 1051 arb. units $\pm$ 111.7
Figure 1f	Peak derivative (GCaMP6f)	stroke - female = 14, stroke - male = 10	unpaired t-test	p = 0.6887, female: 0.29 $\pm$ 0.04; male: 0.32 $\pm$ 0.07
Figure 1g	Time to TD	stroke - female = 31, male = 18	unpaired t-test	p = 0.9355 (female: 221.5 s $\pm$ 18.3; male: 219.2 s $\pm$ 18.3)
Figure 1h	Lesion size (mm2)	sham - female = 7, stroke - female = 13, sham - male = 6, stroke male = 10	Two-way ANOVA with Tukey <i>post hoc</i>	sex: F1, 32 = 0.47, p = 0.50; sham vs. stroke: F1, 32 = 31.65, **** p < 0.0001; interaction: F1,32 = 0.51, p = 0.48
Figure 1i	Peak GCaMP6f ($\Delta F/F$)-Lesion Size (mm2)	sham - female = 7, stroke - female = 13, sham - male = 6, stroke male = 10	Simple linear regressions	female: R ² = 0.36, ** p = 0.006; male: R ² = 0.54, ** p = 0.003

Supplementary Table 2. Detailed Statistical Information for Figure 2

Figure Panel	Response variable	n	statistical analysis	Result
Figure 2d	Open field activity (Δ)	sham - females = 7, stroke - females = 7, sham - males = 5, stroke - males = 9	Two-way ANOVA with Tukey <i>post hoc</i>	sex: F1, 26 = 0.15, p = 0.70; sham vs. stroke: F1, 26 = 22.32, **** p < 0.0001; interaction: F1, 26 = 0.11, p = 0.74
Figure 2f	Distance travelled (Δ)	sham - females = 6, stroke - females = 8	unpaired t-test	* p = 0.0186 (sham: -0.56 m $\pm$ 0.66; stroke: +5.01 m $\pm$ 1.69)
Figure 2h	Distance travelled (Δ)	sham - males = 5, stroke - males = 10	unpaired t-test	** p = 0.0039 (sham: -2.26 m $\pm$ 0.84; stroke: +3.70 m $\pm$ 1.11)

Supplementary Table 3. Detailed Statistical Information for Figure 3

Figure Panel	Response variable	n	statistical analysis	Result
Figure 3d	Peak GCaMP6f (CL SD)	n = 20 females, n = 5 males	Two-tailed Mann-Whitney	p = 0.097, female: 742.6 arb. units $\pm$ 84.3, male: 506.0 arb. units $\pm$ 135.2,
Figure 3g	CL events (per min)	n = 25	Two-tailed paired t-test	**** p < 0.0001., pre-CL SD: 7.3 $\pm$ 0.84, post CL-SD: 2.56 $\pm$ 0.66,
Figure 3h	Peak GCaMP6f (CL SD) - CL events/min	n = 25	simple linear regression	female: R ² = 0.24, * p = 0.01

Supplementary Table 4. Detailed Statistical Information for Figure 4

Figure Panel	Response variable	n	Statistical analysis	Result
Figure 4d	Open field activity (females)	sham, n = 6, stroke, n = 10	two-tailed unpaired t-tests	** p < 0.01, * p < 0.05, sham – during: -16.6% $\pm$ 4.75, stroke – during: +22.89% $\pm$ 11.0, sham – post: -12.14% $\pm$ 4.5, stroke – post: +20.4% $\pm$ 5.61,
Figure 4e	Open field activity (males)	sham n = 5, stroke n = 2	two-tailed unpaired t-tests	n.s.; sham – during: -9.31% $\pm$ 5.4, stroke – during: +9.1% $\pm$ 5.1, sham – post: -6.94% $\pm$ 6.43, stroke – post: +4.26% $\pm$ 10.34
Figure 4g	Speed (females)	sham, n = 6, stroke, n = 10	two-tailed unpaired t-tests	p = 0.0275, sham – during: -0.002 m/s $\pm$ 0.007; stroke – during: 0.006 m/s $\pm$ 0.006; sham – post: -0.002

				m/s $\pm$ 0.0005; stroke – post: 0.01 m/s $\pm$ 0.003
Figure 4h	Speed (males)	sham n = 5, stroke n = 2	two-tailed unpaired t-tests	sham – during: -0.01 m/s $\pm$ 0.003, stroke – during: -0.002 m/s $\pm$ 0.005, sham – post: -0.006 m/s $\pm$ 0.0003, stroke – post: 0.001 m/s $\pm$ 0.003
Figure 4i	Distance (post-TSD)	stroke - SD, n = 10; stroke + SD, n = 8	two-tailed unpaired t-test	** p < 0.01, -SD: 12.12 $\pm$ 1.87 m in 20 min; +SD: 28.71 $\pm$ 5.99 m

Supplementary Table 5. Detailed Statistical Information for Figure 5

Figure Panel	Response variable	n	statistical analysis	Result
Figure 5b	Freezing time (%)	sham, n = 4, stroke - SD, n = 3, stroke + SD, n = 3	Two-way RM ANOVA with Bonferroni post hoc	time: $F_{1,19} = 69.59$, **** p < 0.0001; insult: $F_{2,19} = 37.41$, **** p < 0.0001; interaction: $F_{2,19} = 34.68$, **** p < 0.0001)
Figure 5c	Freezing time (%)	sham, n = 6, stroke - SD, n = 7, stroke + SD, n = 8	Two-way RM ANOVA with Bonferroni post hoc	time: $F(1,7) = 18.44$, ** p < 0.01; insult: $F_{2,7} = 4.8$, * p < 0.05; interaction: $F_{2,7} = 4.92$, * p < 0.05]
Figure 5d	Peri-stroke: Peak GCaMP6f (IL TSD)-Freezing (Day 2)	females, n = 15	simple linear regression	$R^2 = 0.30$, p = 0.033
Figure 5e	Peri-stroke: Peak GCaMP6f (CL SD)-Freezing (Day 2)	females, n = 15	simple linear regression	$R^2 = 0.034$, p = 0.51
Figure 5g	Freezing time (%)	sham, n = 4	paired t-test	p = 0.0233
Figure 5h	Freezing time (%)	stroke - SD, n = 3, stroke + SD, n = 3	Two-way RM ANOVA with Bonferroni post hoc	time: $F_{1,11} = 60.82$, **** p < 0.0001; insult: $F_{1,11} = 23.94$, *** p = 0.0005; interaction: $F_{1,11} = 25.27$, *** p = 0.0004.
Figure 5i	Freezing time (%)	stroke - SD, n = 6, stroke + SD, n = 7	Two-way RM ANOVA with Bonferroni post hoc	time: $F_{1,4} = 16.5$, p = 0.02; insult: $F_{1,4} = 0.005$, p = 0.95; interaction: $F_{1,4} = 2.116$, p = 0.2195
Figure 5j	Post-stroke: Peak GCaMP6f (IL TSD)-Freezing (Day 2)	females, n = 14	simple linear regression	$R^2 = 0.0005$, p = 0.94
Figure 5k	Post stroke: Peak GCaMP6f (CL SD)-Freezing (Day 2)	females, n = 14	simple linear regression	$R^2 = 0.42$, p = 0.011

Supplementary Table 6. Detailed Statistical Information for Figure 6

Figure Panel	Response variable	n	statistical analysis	Result
Figure 6e	Power (dB)	n = 5 females	RM two-way ANOVA with Bonferroni post-hoc	(frequency: $F_{3.62, 58.0} = 189.6$, **** p < 0.0001; epoch: $F_{3,16} = 23.14$, **** p < 0.0001; interaction: $F_{366, 1952} = 6.95$, **** p < 0.0001)
Figure 6f	Power (dB)	n = 5 females	RM two-way ANOVA with Bonferroni post-hoc	(frequency: $F_{2.61, 57.36} = 80.25$, **** p < 0.0001; epoch: $F_{2,22} = 1.88$, p = 0.18; interaction: $F_{256, 2816} = 2.53$, **** p < 0.0001)
Figure 6i	Power (dB)	n = 4 males	RM two-way ANOVA	(frequency: $F_{128, 1536} = 80.25$, **** p < 0.0001; epoch: $F_{1,12} =$

			with Bonferroni <i>post-hoc</i>	3.61, $p = 0.082$; interaction: $F_{128, 1536} = 2.65$, **** $p < 0.0001$)
Figure 6k	Time (s, FP) vs. Time (s, LFP)	$n = 5$ CL SDs from 4 female mice	Linear regression	onset: $R^2 = 0.99$, **** $p < 0.0001$; end: $R^2 = 0.99$, **** $p < 0.0001$
Figure 6l	Cumulative seizure duration	female, $n = 5$; male, $n = 4$	Two-tailed unpaired t-test	$p = 0.32$, female: $78.32 \text{ s} \pm 23.9$, $n = 5$; male: $44.4 \text{ s} \pm 19.31$

Supplementary Table 7. Detailed Statistical Information for Figure 7

Figure Panel	Response variable	n	statistical analysis	Result
Figure 7d	Power (normalized)	$n = 4$ females	two-way RM ANOVA with Bonferroni <i>post hoc</i>	time: $F_{119, 357} = 1.72$, **** $p < 0.0001$; hemisphere: $F_{1, 3} = 347.2$, *** $p = 0.0003$; interaction: $F_{119, 357} = 1.59$, *** $p = 0.0006$,
Figure 7e	Power (dB) - ipsilesional	$n = 4$ females	two-way RM ANOVA with Bonferroni <i>post hoc</i>	time: $F_{119, 357} = 44.37$, $p < 0.0001$; epoch: $F_{1, 3} = 31.26$, $p = 0.0113$; Interaction: $F_{119, 357} = 6.783$, $p < 0.0001$
Figure 7f	Power (dB) - contralesional	$n = 4$ females	two-way RM ANOVA with Bonferroni <i>post hoc</i>	time: $F_{119, 357} = 13.77$, $P < 0.0001$; epoch: $F_{1, 3} = 0.8182$, $P = 0.4324$; interaction: $F_{119, 357} = 1.207$, $P = 0.0966$

Supplementary Table 8. Detailed Statistical Information for Figure 8

Figure Panel	Response variable	n	statistical analysis	Result
Figure 8i	Peak GCaMP6f (IL TSD)	vehicle, $n = 7$; MK801, $n = 6$	two-tailed unpaired t-test	vehicle: $1360 \text{ arb. units} \pm 174.7$; MK-801: $879.6 \text{ arb. units} \pm 97.11$; * $p = 0.0429$
Figure 8k	Peak GCaMP6f (CL SD)	vehicle, $n = 6$; MK801, $n = 6$	two-tailed unpaired t-test	vehicle: $724.1 \text{ arb. units} \pm 76.5$; MK-801: $246.1 \text{ arb. units} \pm 20.84$; **** $p < 0.0001$
Figure 8l	Seizure amplitude	vehicle - SD, $n = 4$; vehicle + SD, $n = 5$; MK801 $n = 6$	one-way ANOVA with Bonferroni <i>post hoc</i>	vehicle - SD: $0.054 \mu\text{V} \pm 0.016$; vehicle + SD: $0.044 \mu\text{V} \pm 0.007$; MK-801: $0.15 \mu\text{V} \pm 0.041$; $F_{2,12} = 4.49$, * $p = 0.035$
Figure 8m - pre	Power (dB)	vehicle, $n = 5$; MK801, $n = 9$	RM two way ANOVA with Bonferroni <i>post hoc</i>	frequency: $F_{4.44, 53.3} = 74.7$, **** $p < 0.0001$; treatment: $F_{1, 12} = 10.91$, ** $p = 0.0063$; interaction: $F_{128, 1536} = 6.70$, **** $p < 0.0001$
Figure 8m - seizure	Power (dB)	vehicle, $n = 5$; MK801, $n = 10$	RM two way ANOVA with Bonferroni <i>post hoc</i>	frequency: $F_{4.35, 52.2} = 105.7$, **** $p < 0.0001$; treatment: $F_{1, 12} = 14.77$, ** $p = 0.0023$; interaction: $F_{128, 1536} = 4.42$, **** $p < 0.0001$
Figure 8m - SD	Power (dB)	vehicle, $n = 5$; MK801, $n = 11$	RM two way ANOVA with Bonferroni <i>post hoc</i>	frequency: $F_{4.64, 55.7} = 39.74$, **** $p < 0.0001$; treatment: $F_{1, 12} = 10.91$, ** $p = 0.0063$; interaction: $F_{128, 1536} = 4.46$, **** $p < 0.0001$
Figure 8m - post	Power (dB)	vehicle, $n = 5$; MK801, $n = 12$	RM two way ANOVA with	frequency: $F_{3.60, 43.14} = 63.25$, **** $p < 0.0001$; treatment: $F_{1, 12} = 17.96$, ** $p = 0.0012$; interaction: $F_{128, 1536} = 8.24$, **** $p < 0.0001$

			Bonferroni post hoc	
Figure 8o	Cumulative seizure duration	vehicle - SD, n = 4; vehicle + SD, n = 5, MK801, n = 6	one-way ANOVA with Bonferroni post-hoc	vehicle - SD: 40.6 s ± 19.6, n = 4; vehicle + SD: 87.5 s ± 6.93, n = 5; MK-801: 295.7 s ± 73.7, n = 6; , F _{2,12} = 6.895, * p = 0.01
Figure 8q	Freezing %	vehicle - SD, n = 4; vehicle + SD, n = 7; MK801 - sham, n = 4; MK801 - stroke, n =7	Two way ANOVA with Bonferroni post hoc	time: F _{1,18} = 20.28, *** p = 0.0003; treatment: F _{3,18} = 2.66, p = 0.08; interaction: F _{3,18} = 2.98, p = 0.06