Supplementary Tables – General Parameters

Parameter	Value	Application
Reference temperature	37 °C	Nernst / GHK equations
Patch-clamp temperature	34 – 35 °C (measurements of V_{rest} and V_{thr})	Difference < 2 mV relative to 37 °C – negligible
$[Cl^-]_o$ (control)	130 mM	Standard ACSF
$[K^+]_o$ (control)	3 mM	Standard ACSF
Nernst constants (log10; 37 °C)	K^+ = +61.54 mV Cl^- = −61.54 mV	*RT/F × 2.303*

A Nernst constant of 61.54 mV (R = 8.314 J mol^{-1} K^{-1}, T = 310 K, F = 96 485 C mol^{-1}) was assumed for 37 °C.

Reducing the temperature to 35 °C lowers this constant to 61.12 mV, shifting all calculated equilibrium potentials by ≈ 0.68 % (≈ 0.57 mV) when Cl^-_i = 5.8 mM.

Throughout this supplement we use f_K = 0.15–0.25 and g_{Cl} = 0.15 based on (Booth and Rinzel 1995).

Supplementary Table 1. Calculated equilibrium potentials (E_{GABA}) and relative shifts (ΔE_{GABA}) for intracellular chloride concentrations between 5.0 mM and 8.0 mM at 37 °C

$[Cl^-]_i$ (mM)	E_{GABA} (mV)	ΔE_{GABA} (mV)
5.0	−87.02	−3.96
5.5	−84.48	−1.42
5.8 (control)	−83.06	0.00
6.0	−82.15	+0.91
6.5	−80.01	+3.04
7.0	−78.03	+5.02
7.5	−76.19	+6.87
7.9 (CRS)	**−74.80**	**+8.25**
8.0	−74.47	+8.59

Equation:

$$E_{GABA} = -61.54\ \text{mV} \times log_{10}\left(\frac{[Cl^-]_o}{[Cl^-]_i}\right)$$

Supplementary Table 2. Predicted shift of the resting membrane potential (ΔV_{rest}) produced by transient increases in extracellular potassium concentration ($\Delta[K^+]_o$) for three values of f_K (fractional K^+ conductance)

$\Delta[K^+]_o$ (mM)	ΔV_{rest} (mV) f_k=0.15	f_k=0.20	f_k=0.25
0.2	0.26	0.34	0.43
0.4	0.50	0.67	0.84
1.0	1.15	1.54	1.92
2.0	2.05	2.73	3.41
3.0	2.78	3.70	4.63
3.5	3.10	4.13	5.16

Equation:

$$\Delta E_K = 61.54\ \text{mV} \times log_{10}\left(\frac{3 + \Delta[K^+]_o}{3}\right)$$

For the cumulative depolarisation budget we used:

$$\Delta V_{rest} = f_K \times \Delta E_K$$

Supplementary Table 3. Sensitivity of the resting-potential shift (ΔV_{rest}) to graded reductions in membrane KCC2 triggered by interleukin-6

KCC2 loss (%)	ΔE_{GABA} (mV)	ΔV_{rest} (mV) (g_{Cl}=0.15)
20	+5.1	+0.8
30	+7.6	+1.1
40	**+10.1**	**+1.5**
50	+12.6	+1.9

Equation:

$$\Delta V_{rest} = 0.15 * \Delta E_{GABA}$$

ΔE_{GABA} calculated as in Supplementary Methods 1.

Supplementary Table 4. Resting-potential shift (ΔV_{rest}) for different ventral-to-dorsal ratios of GIRK conductance ($g_{GIRK\ v/d}$) and input resistance ($R_{in\ v/d}$)

$g_{GIRK\ v/d}$	$R_{in\ v/d}$	ΔV_{rest} (mV)
0.30	1.30	0.44
0.30	1.50	0.51
0.30	1.70	0.57
0.35	1.30	0.51
0.35	**1.50**	**0.59**
0.35	1.70	0.67
0.40	1.30	0.59
0.40	1.50	0.68
0.40	1.70	0.77

Equation:

$$\Delta V_{rest} = 0.90\ \text{mV} \times \left(\frac{R_{\text{in,v}}}{R_{\text{in,d}}}\right) \times \left(\frac{g_{\text{GIRK,v}}}{g_{\text{GIRK,d}}}\right) \times (1 + 0.25)$$

where 0.25 represents the TASK-channel contribution to the total leak current.

Supplementary Table 5. Predicted change in resting-membrane potential (ΔV_{rest}) produced by a chronic increase of extracellular potassium ($\Delta[K^+]_o$) under three values of the potassium-leak fraction (f_K)

$\Delta[K^+]_o$ (mM)	f_k = 0.15	f_k = 0.20	f_k = 0.25
0.25	0.321	0.428	0.534
0.30	0.382	0.509	0.636
0.35	0.442	0.589	0.737
0.40	0.501	0.669	0.836
0.45	0.560	0.747	0.933

Equation:

$$\Delta V_{rest} = f_K \times 61.54\ \text{mV} \times \log_{10}\left(\frac{3 + \Delta[K^+]_o}{3}\right)$$

Supplementary Table 6. Attenuation of local depolarisation at the soma (Att %) reported by multi-compartment models of CA1 pyramidal neurones

Publication	Model / figure	Att %
(Booth and Rinzel 1995)	Fig. 6B	11
(Doyon et al. 2011)	Fig. 4C	14

(Migliore et al. 2018)	Fig. 3A	12
(Currin and Raimondo 2022)	Suppl. Fig. S3	13

Supplementary Table 7. Supra-additive synergy (Synergy %) when two inhibitory-Cl^- manipulations are applied simultaneously

Publication	Experimental combination	Synergy %
(Doyon et al. 2011)	KCC2 ↓ 60 % **plus** $GABA_A$ frequency ↑ 200 % (Fig. 4C)	15
(Currin and Raimondo 2022)	Cl^- plus distal inhibition (Suppl. Fig. S3)	19

Supplementary Table 8. Summary statistics (mean ± SD) for the data in Supplementary Tables 6 & 7.

Metric	Mean	SD
Att %	12.5	1.29
Synergy %	17.0	2.83

(Att % = $100 \times [1 - \Delta V_{soma}/\Delta V_{injected}]$; Synergy % = $100 \times [(\Delta V_{combo} - \Sigma\,\Delta V_{single})/\Sigma\,\Delta V_{single}]$).

Supplementary Table 9. *GRM3* risk haplotype – prolongation of τ_{EPSP} via glutamate spill-over

Parameter	Value	Source / note
↓ mGlu3 mRNA	10–15 %	(Ghose et al. 2009)
↓ EAAT2 protein	−30 %	(Abdul et al. 2009) Fig. 2D AD hippocampus – used as upper bound
ΔGlu_{extra}	+35 %	−(ΔEAAT2 + 0.4 × ΔmGlu3)
Slope $\Delta\tau/\Delta Glu$	0.7	(Wild et al. 2015)
Modelled $\Delta\tau_{EPSP}$	+25 %	Table 2.13 (Methods)

35 % = 30 % (EAAT2) + 0.4 × 12.5 % (mGlu3)

Supplementary Table 10. *GABRA1* mRNA ↓ 40 % – effect on τ_{IPSC} and R_{in}

Parameter	Value	Source / note
GABRA1 mRNA (PFC, BA9)	−40 %	(Glausier and Lewis 2011) Fig 2B
$\Delta\tau_{mIPSC}$ w α1 KO	+55 %	(Bosman et al. 2005) Table 1
Scaling factor for partial loss	×0.45	Linear interpolation: 40 / 90 (conservative)
mIPSC amplitude (A/A_{ctrl})	0.74	Chosen so that $Q/Q_{ctrl} \approx 0.93$ (see text)
mIPSC time constant (τ/τ_{ctrl})	1.25	1 + 0.55 × 0.45
IPSC charge (Q/Q_{ctrl})	0.93 (−7 %)	0.74 × 1.25
Modelled $\Delta\tau_{IPSC}$	+25 %	Table 2.13 (Methods)
Modelled g_{inh}	−7 %	$g_{inh} \propto Q$
ΔR_{in} (from Σg_{tot})	+1.4 %	$g_{inh} \approx 20\ \% \Sigma g_{tot}$; 0.20 × 7 % ≈ 1.4 %

Supplementary Table 11. *COMT* Val158Met – increase in F–I gain

Parameter	Value	Source / note
CSF ΔHVA (meta-analysis)	−16 %	(Saloner et al. 2020) (n = 132)
DA → gain coefficient	0.9	(Vijayraghavan et al. 2007)
Modelled Δgain F–I	+14 %	Table 2.13 (Methods)

Supplementary Table 12. *CACNA1C* rs1006737 A – increase of τ_{EPSP} and rheobase shift

Parameter	Value	Source / note
$\Delta I_{Ca,L}$	+30 %	(Mertens et al. 2015) Fig 3C (hiPSC neurons)
Slope $\Delta\tau / \Delta Ca_{post}$	0.5	(Wild et al. 2015)
Modelled $\Delta\tau_{EPSP}$	+15 %	—
Ratio $\alpha = \tau_m/(\tau_m + T)$	0.4	(Tuckwell 1988); CA1
Lapicque/Tuckwell formula	$\Delta rheo = -\alpha \cdot \Delta\tau_{EPSP}$	—
Modelled Δrheobase	−6 %	Exported to Table 3.3

For a representative rheobase of 150 pA (CA1 pyramidal, T = 5 ms) a 6 % decrease corresponds to ≈ 9 pA, well below the 10–20 pA step size used in typical current-step protocols.

Supplementary Table 13. *NRG1* HapICE – reduction of g_{inh}

Parameter	Value	Source / note
Loss of PV interneurons	30 %	(Fazzari et al. 2010)
N_{PV} / $N_{PV,ctrl}$	0.70	—
Booth–Rinzel rule (power 0.3)	—	(Booth and Rinzel 1995)
Modelled g_{inh}	−10 %	Table 2.13 (Methods)
IPSC measurement	−25 %	(Yin et al. 2013)

Supplementary Table 14. C4A over-expression – synaptic connectivity loss

Parameter	Value	Source / note
Synapse pruning (microglia engulfment, PSD-95$^+$)	↑ ≈ 35 %	(Yilmaz et al. 2021) Fig 4C–E
Spine density (apical dendrites, L2/3)	↓ 25 %	(Yilmaz et al. 2021) Fig 5B
mEPSC frequency	↓ 20 %	(Yilmaz et al. 2021) Fig 6B
mEPSC amplitude	↓ 15 %	(Yilmaz et al. 2021) Fig 6C

Fewer E → PV ⇒ γ-power ↓ inputs; weaker "internal noise" facilitates injection-locking of ELF 7-30 Hz, raising the probability of phase convergence (P_{phase}) and self-replays.

Supplementary Table 15. *SCN2A* R1882Q – reduced rheobase

Parameter	Value	Source / note
$\Delta I_{Na,pers}$	+40 %	(Ben-Shalom et al. 2017)
ΔV_{thr}	−3 mV	(Ben-Shalom et al. 2017) Fig. 1E
Modelled Δrheobase	−15 %	Table 3.3

V_{thr} measured in HEK293/Nav1.2 was transferred 1 : 1 to CA1 model (Ben-Shalom et al. 2017).

Supplementary Table 16. Amphetamine (2 mg kg^{-1} i.p.) – reduction of rheobase

Parameter	Value	Source / note
WT threshold (BLA)	120 ± 5 pA	(Rosenkranz and Grace 2002) Fig 3D
Threshold after AMPH	105 ± 5 pA	(Rosenkranz and Grace 2002) Fig 3D
Δrheobase	−12 %	Exported to Table 2.15 (Methods)

Supplementary Table 17. Chronic-intermittent ethanol (CIE, 5 weeks) – changes in NMDA/AMPA ratio and mIPSC frequency

Parameter	Value	Source / note
Δ(NMDA / AMPA)	+30 %	(Kroener et al. 2012)
$\Delta mIPSC_{freq}$	−15 % (trend, p = 0.08)	(Kroener et al. 2012)

Supplementary Table 18. Alcohol withdrawal (72 h) – change in R_{in} and IPSC frequency

Parameter	Control	72 h WD	Δ (%) / Source
R_{in} PV interneurons (MΩ)	155	186	+20 % —(Quadir et al. 2024) bioRxiv preprint
$IPSC_{freq}$ (Hz)	7.8	5.9	−24 % —(Quadir et al. 2024) bioRxiv preprint

Supplementary Table 19. IL-6 ↑ → KCC2 ↓ → ΔE_{GABA} → ΔV_{soma}

Parameter	Value	Source / note
↓ $g_{KCC2,surf}$	−40 %	(Jin et al. 2022) Fig. 5C
↑ $[Cl^-]_i$	5.8 → 8.6 mM	At 40 % KCC2 loss the model of (Doyon et al. 2011) predicts a 48 % rise (5.8 → 9.7 mM); 8.6 mM is chosen conservatively below this upper limit
E_{GABA}	−72.6 mV	−61.54 mV × $\log_{10}$(130 / 8.6)

ΔE_{GABA}	+10.5 mV	(Rivera et al. 2004) (40 % KCC2 loss)
g_{Cl} fraction in V_{rest}	0.15	(Booth and Rinzel 1995)
ΔV_{soma}	+1.6 mV	0.15 × 10.5 mV
Baseline E_{GABA}	−83 mV	Calculated for $[Cl^-]_i$ = 5.8 mM (see Suppl. Table 1)

Minimal scenario 5.8 → 7.9 mM

Supplementary Table 20. P2X7R ↑ + slowed $[K^+]_o$ clearance

Parameter	Value	Source / note
Extracellular ATP (microglia)	+210 %	(Shan et al. 2022) Fig. 4B
$t_{½}$ of $[K^+]_o$ clearance	× 1.7	(Shan et al. 2022) Fig. 4D
Excess $[K^+]_o$	+0.12 mM	Diffusion model (conservative)
$[K^+]_o$ absolute	3 → 3.12 mM	—
ΔE_K	+1.05 mV	61.54 mV × $\log_{10}$(3.12/3)
g_K fraction in V_{rest}	0.15	(Booth and Rinzel 1995)
ΔV_{soma}	≈ 0.16 mV	0.15 × 1.05 mV

Supplementary Table 21. ROS → 40 % Kir2.1 block

Parameter	Value	Source / note
↓ $g_{Kir2.1}$	−40 %	(Wang et al. 2022) Fig 3D
g_{Kir} / g_K	0.30	(Ding et al. 2016)
g_K / g_{total}	0.15	(Booth and Rinzel 1995)
Net Δg_{Kir} / g_{total}	0.018 (= 0.40 × 0.30 × 0.15)	—
Baseline V_{rest}	−71 mV	(Cembrowski et al. 2016)
Baseline E_K	−90 mV (3 mM $[K^+]_o$)	Nernst
ΔV_{soma} (Kir block)	+0.34 mV	0.018 × $\lvert V_{soma} - E_K \rvert$
Time window	24 – 72 h	(Wang et al. 2022)

† The TRPM2 effect (+5–10 pA inward current, (Wang et al. 2022)) is negligible relative to Kir2.1 and was not included in ΔV.

Supplementary Table 22. ECS shrinkage (Δf_K = −0.03)

Parameter	Value	Source / note
↓ f_K	−0.03	(Syková and Nicholson 2008) Fig. 5A–B
Burst $[K^+]_o$	6 mM (CRS)	—
ΔE_K (burst)	≈ 18.7 mV	61.5 mV × $\log_{10}$(6/3)
ΔV_{soma}	≈ 0.56 mV	0.03 × 18.7 mV

Δf_K adapted from (Syková and Nicholson 2008) Fig. 5A–B (chronic astrocytic swelling).

Supplementary Table 23. Caffeine – GIRK block

Dose (p.o.)	$[Caf]_{CSF}$ (µM)	GIRK block (%)	ΔV_{raw} (mV)	ΔV_{soma} (mV)	Source / note
100 mg	4 – 6	10 – 15	0.67 – 1.00	0.8 – 1.1	(Blanchard and Sawers 1983; Nehlig 2018)slope 2.5 % μM^{-1}(Lopes et al. 2019)
400 mg	12 – 16	25 – 35	1.7 – 2.4	2.0 – 2.8	GIRK saturation above 10 µM
10 µM slice	—	25	1.66	1.9	Acute hippocampal slice; (Lopes et al. 2019)

ΔV_{raw} = 0.35 × block × $\lvert E_K - V_{rest} \rvert$, with $\lvert E_K - V_{rest} \rvert$ ≈ 19 mV.

$\Delta V_{soma} = \Delta V_{raw} \times k_{R(\theta)} \times k_{PV} = \Delta V_{raw} \times 1.10 \times 1.10$.

The block-versus-concentration slope flattens above 10 µM; we therefore used 25–35 %.

The slope of 2.5 % μM^{-1} was obtained by linear regression of the 0–10 µM data in (Lopes et al. 2019) ($R^2 = 0.94$); own calculations.

Supplementary Table 24. "Low-PV" window (25 % drop in g_{inh})

Parameter	Value	Source / note
↓ g_{inh} (PV)	−25 %	(Donato et al. 2013), Fig. 3E
g_{inh} / g_{total}	0.20	(Booth and Rinzel 1995)
ΔE ($V_{rest} - E_{GABA}$)	12 mV	−71 − (−83)
ΔV_{soma}	+0.6 mV	0.20 × 0.25 × 12 mV

† PV → pyramidal IPSPs act perisomatically; there is no dendritic attenuation, hence the attenuation coefficient (*Att*) = 1.

Supplementary Table 25. Nicotine – somatic depolarisation in vCA1

Scenario	$[Nic]_{CSF}$ (µM)	ΔV_{raw} (mV)	ΔV_{soma} (mV)	Source / note
4 cigarettes h^{-1}	0.20 – 0.30	0.076 – 0.114	0.09 – 0.13	(Ji and Dani 2000; Rose et al. 2010), Fig. 2B (0.38 mV μM^{-1})
2 cigarettes h^{-1}	0.10 – 0.15	0.038 – 0.057	0.04 – 0.07	same as above
Peak after 1 cigarette	0.25	0.095	0.12	$t_{½} \approx 45$ min (Picciotto et al. 2008)
10 µM slice	—	3.8	3.8	Acute hippocampal slice; (Ji and Dani 2000), Fig. 2C

† $\Delta V_{raw} = 0.38$ mV $\mu M^{-1} \times [Nic]_{CSF}$

‡ $\Delta V_{soma} = \Delta V_{raw} \times k_{R(\gamma)} \times k_{PV} = \Delta V_{raw} \times 1.15 \times 1.10$

Supplementary Table 26. Baseline equation and input parameters for a magnetite nanocrystal chain

Parameter	Nominal value	Tested range	Source
α_{ds}	0.50	0.45 – 0.55	(Golding et al. 2005) Fig. 3
κ	8.27 µV × μT^{-1}	± 7 % †	(Kirschvink 1996)
Φ (16.7 Hz)	1.0×10^2	80 – 120	(Kirschvink et al. 1992); see Methods §2.11.2
Φ (7.83 Hz)	40	30 – 60 (± 25 %)	(Kirschvink et al. 1992); see Methods §2.11.2
$B_{rms-city}$	0.15 µT	0.10 – 0.20 µT	(Schüz et al. 2000; Brix et al. 2001; Bundesamt für Strahlenschutz (BfS) 2023 Fig 3.2; Loizeau et al. 2024)
$B_{rms-countryside}$	1 pT	0.5 – 2 pT	(Nickolaenko and Hayakawa 2014; Han et al. 2023)
Q_θ	30	—	(Buzsáki and Draguhn 2004; Zemankovics et al. 2010), Fig. 2

Baseline equation – see Methods § 2.11.3.

Φ values taken directly from (Kirschvink et al. 1992) (chain ≈ 10^3 crystals, $R \approx 100$ MΩ).

† A ± 7 % spread in κ changes ΔV_{soma} by ≈ 6 %, so κ is treated as constant in the sensitivity analysis.

Supplementary Table 27. Sensitivity matrix – urban environment (16.7 Hz)

Φ \ α	0.45	0.50	0.55
80	44 µV	49 µV	55 µV
100	56 µV	62 µV	68 µV
120	67 µV	74 µV	82 µV

Nominal prediction: ΔV_{soma} = 62 µV (0.062 mV)

Extreme scenario (0.55; 120) → 0.082 mV.

Supplementary Table 28. Sensitivity matrix – rural environment (7.83 Hz)

Φ \ α	0.45	0.50	0.55
30	0.11 nV	0.12 nV	0.14 nV
40	0.15 nV	0.17 nV	0.19 nV
60	0.22 nV	0.25 nV	0.28 nV

Nominal prediction: ΔV_{soma} = 0.17 nV

Supplementary Table 29. ΔV_{soma} versus Johnson noise (band 0 – 5 kHz)

Compartment	C [pF]	V_{rms}	ΔV_{soma} (city)	$\Delta V / V_{rms}$
Dendritic branch	0.5	120 µV	62 µV	0.52×
Whole cell	150	5 µV	62 µV	12×

The rural ΔV (0.17 nV) is > 10^5 × weaker than the thermal noise of a single dendrite.

Supplementary Table 30. One-second traction-field bursts (16.7 Hz)

Scenario	B_{rms}	ΔV_{soma}
City – 24 h median	0.15 µT	0.062 mV
City – peak × 3	0.45 µT	0.19 mV → 0.14 mV after RC (– 25 %)
Countryside – median	1 pT	0.17 nV
Countryside – peak × 3	3 pT	0.50 nV

One-second bursts are further attenuated by ≈ 25 % owing to the membrane RC filter (τ ≈ 20 – 50 ms).

Supplementary Table 31. Matrix of percentage changes (layer × axis) – CRS + CACNA1C A + hot-spot variant

Axis / Layer	Layer 1 (CRS ≥ 14 d)	Layer 2 (allele / drug)	Layer 3 (CREB hot-spot)	Layer 4 (minute–hour bias)
Rheobase	−44 %	−6 %	−15 %	—
R_{in}	+29 %	—	—	—
τ_{EPSP}	+15 %	+15 %	+25 %	—
$IPSC_{PV→pyr}$	−16 %	—	—	—
ΔV_{rest}	+11.3 mV	0 mV	+3.2 mV	+0.5–3 mV

Layer 4 acts purely additively on V_{margin}; it does not modify passive parameters.

Combination rule (Fricker and Miles 2000; Magee and Cook 2000):

$$Overall\ factor = \prod_{i=1}^{n} \left(1 + \frac{\Delta_i}{100}\right).$$

(Spruston and Johnston 1992; Dougherty et al. 2012; Kim and Johnston 2015; MacKenzie and Maguire 2015; Pignatelli et al. 2019; Zhang et al. 2021).

Supplementary Table 32A. Propagation of uncertainty – *CACNA1C* A variant

Axis	Final value	σ_{base}	σ_{mult} (5 %)	σ_{tot}
Rheobase	**63 pA** (140 × 0.447)	10 pA	3.15 pA	≈ 10.4 pA

R_{in}	**142 MΩ** (110 × 1.29)	8 MΩ	7.1 MΩ	≈ 10.7 MΩ
τ_{EPSP}	**24.8 ms** (15 × 1.652)	1.0 ms	1.24 ms	≈ 1.59 ms
$IPSC_{PV\rightarrow pyr}$	0.84	—*	0.042	0.042

* For the normalised quantity (= 1.0) no published standard deviation is available; a ± 5 % multiplier uncertainty was applied instead.

$$\sigma_{tot} = \sqrt{\sigma_{base}^2 + \sigma_{mult}^2}$$

$\sigma_{mult} = 0.05 \times$ final value (5 % relative uncertainty).

Supplementary Table 32B. Combined factors (*SCN2A* R1882Q variant)

Axis	Overall factor	Change %
Rheobase	0.56 × 0.85 × 0.85 = 0.404	− 59.6 % (Rounded) − 60 %
R_{in}	1.29	+ 29 %
τ_{EPSP}	1.44	+ 44 %
$IPSC_{PV\rightarrow pyr}$	0.84	− 16 %

Layers 1 and 3 are identical to Supplementary Table 31; in Layer 2 a −15 % rheobase shift was assumed, all other axes 0 %.

Supplementary Table 33. Scaling constants used in transient-gain mode

Parameter	Value	Source / note
ΔR_{in}	+29 %	§ 3.11 (electrotonic cascade)
k_R (θ)	1.10 (after correction)	corrected from 1.29 by filtering
k_R (SWR)	1.29	R_{in} operates at full scale at 150 Hz
PV-shunt	−16 %	Hot-spot + *CACNA1C* A
k_{PV}	1.10	(Booth and Rinzel 1995), leak-divisive model

Supplementary Table 34. Sensitivity of ΔV_{soma} to ± 10 % changes in k_R (electrotonic gain) and k_{PV} (PV shunt)

Transient	k_R variation (± 10 %)	k_{PV} variation (± 10 %)	ΔV_{soma} range [mV]
θ	1.0 – 1.2	1.0 – 1.2	0.99 – 1.44
SWR	1.2 – 1.46	1.00 – 1.00	0.48 – 0.58

With the largest deviations (± 10 %), ΔV_{soma} changes by ≤ 20 %. This does not alter the qualitative conclusion that θ and SWR remain the leading candidates for ELF phase-locking.

Supplementary Table 35. Input parameters of the phase-coincidence model

Band	Fraction of sites f_i	Period T_i [ms]	Hit probability $p_i(\Delta t)$	Sources
7.83 Hz	1.00	128	$p_1 = \Delta t / T_1$	(Nickolaenko and Hayakawa 2014)
16 – 18 Hz	0.62	60	$p_{16} = \Delta t / T_{16}$	(Kirschvink 1996; Brix et al. 2001; Loizeau et al. 2024)
20 – 28 Hz	0.18	42.5 ± 7.5 (≈ 50 → 35)	$p_{20} = \Delta t / T_{20}$	(Paniagua et al. 2007; Gajšek et al. 2016)

For any specific Δt we use

$$p_i = \begin{cases} \frac{\Delta t}{T_i}, \Delta t < T_i \\ 1, \Delta t \geq T_i \end{cases}$$

Supplementary Table 36. Calculation of P_{phase} in an urban environment (p = 1)

Δt	p_1	p_{16}	p_{20}	$1 - f_1 p_1$	$1 - f_{16} p_{16}$	$1 - f_{20} p_{20}$	$P_{phase,city}$	± 10 % CI
20 ms	0.156	0.333	0.471	0.844	0.794	0.915	0.387	0.31 – 0.46
25 ms	0.195	0.417	0.588	0.805	0.7396	0.894	0.468	0.39 – 0.54
30 ms	0.234	0.500	0.706	0.766	0.690	0.872	0.539	0.45 – 0.61

$$P_{\text{phase}} = 1 - \prod_i (1 - f_i p_i)$$

Supplementary Table 37. Calculation of P_{phase} in a rural environment

Δt	p_1	$P_{phase,village}$	± 10 % CI
20 ms	0.156	0.156	0.14 – 0.17
25 ms	0.195	0.195	0.18 – 0.21
30 ms	0.234	0.234	0.21 – 0.26

Because only the 7.83 Hz band contributes in the rural scenario:

$$P_{\text{phase,village}} = f_1 p_1 = 1.00 \times p_1$$

Supplementary Table 38. Urban gradient ($G = P_{city} / P_{village}$)

Δt	Gradient	95 % CI (± 10 %)
20 ms	2.48	2.0 – 3.0
25 ms	2.40	1.9 – 3.0
30 ms	2.30	1.9 – 2.8

Shift in the correlation threshold *r*

If the frequency bands are partially correlated we introduce a reduction factor

$$\rho = 1 - r$$

For $r = 0.02 \ldots 0.10$;

Supplementary Table 39. Sensitivity to inter-band correlation ($\Delta t = 25$ ms)

r	ρ	$P_{phase,city}$ ($\Delta t = 25$ ms)	G
0.02	0.98	0.460	2.31
0.05	0.95	0.444	2.22
0.10	0.90	0.420	2.15

Conclusion: even at r = 0.10 the gradient remains > 2.1.

Supplementary Table 40. Variation of the 16–18 Hz band fraction (± 15 %)

f_{16}	P_{city}	Gradient
0.53 (−15 %)	0.441	2.26
0.62 (nominal)	0.468	2.40
0.71 (+15 %)	0.496	2.44

Supplementary Table 41. Theta + SWR coincidences in mice (RUN state, vCA1)

Variable	Nominal value	Unit	± 10 % range	Source / comment
f_θ	10	Hz	9 – 11	(Fernández-Ruiz et al. 2017) Fig. 2B
$\lambda_{\theta\text{-window}} = 4 f_\theta \times 60$	2 400	min^{-1}	2 160 – 2 640	calculated
$\Delta t_\theta = 1 / (4 f_\theta)$	25.0	ms	22.7 – 27.8	calculated
λ_{SWR}	1.90	min^{-1}	1.71 – 2.09	(Liu et al. 2022a)
t_{SWR}	100	ms	90 – 110	(Schieferstein et al. 2024)

$t_{eff,SWR}$	109.8	ms	99.8 – 119.8	+9.8 ms ($\tau_{EPSP,eff}$ − 15 ms)
$t_{sum} = t_{eff,SWR} + \Delta t_{\theta}$	135	ms	124.8 – 144.8	calculated
p_{hit}	0.00428	–	0.00356 – 0.00505	$\lambda_{SWR} \times t_{sum} / 60$
$N_{hit,raw} = p_{hit} \times \lambda_{\theta\text{-window}}$	10.26	min^{-1}	7.68 – 13.32	–
$N_{hit,city}$	4.80	min^{-1}	3.59 – 6.24	$\times P_{phase,city} = 0.468$
$N_{hit,village}$	2.00	min^{-1}	1.50 – 2.60	$\times P_{phase,village} = 0.195$

Supplementary Table 42. Theta + SWR coincidences in humans (vCA1)

Wake state	f_{θ} [Hz]	$\lambda_{\theta\text{-window}}$ [min^{-1}]	λ_{SWR} [min^{-1}]	t_{sum} [ms]	p_{hit}	$N_{hit,raw}$ [min^{-1}]	$N_{hit,city}$	$N_{hit,village}$	± 10 % CI (city / village)
Rest	7.0	1 680	1.2	146	0.00291	4.90	2.29	0.96	1.68 – 3.03 / 0.70 – 1.27
Slow walk	8.8	2 112	0.9	138	0.00207	4.38	2.05	0.85	1.51 – 2.72 / 0.63 – 1.13
β-arousal	16.0	3 840	0.8	126	0.00168	6.44	3.02	1.25	2.21 – 3.98 / 0.92 – 1.66

Confidence intervals were calculated as described in §S2.15.4 (simultaneous ± 10 % perturbation of all input parameters).

Supplementary Table 43. Sensitivity analysis (± 10 %) – extreme values of N_{hit}

Species / state	$N_{hit,\ city}$ [min^{-1}]	$N_{hit,\ village}$ [min^{-1}]
Mouse (RUN)	3.59 – 6.24	1.50 – 2.60
Rest	1.68 – 3.03	0.70 – 1.27
Slow walk	1.51 – 2.72	0.63 – 1.13
β-arousal	2.21 – 3.98	0.92 – 1.66

Ranges span all 2^7 combinations of the input parameters (f_{θ}, λ_{SWR}, t_{SWR}) varied by ± 10 %.

Supplementary Table 44. Physical parameters for amplification of the 7.83 Hz signal

Parameter	Calm	Geomagnetic storm Kp ≥ 6	Unit	Source / formula
B_{rms}	0.3 pT	3 pT	pT	(Sátori et al. 2007)
κ	8.27	—	$\mu V\ \mu T^{-1}$	(Kirschvink 1996) chap. 12, tab. 2, p. 242
Φ (−25 dB)	40	40	–	(Winklhofer and Kirschvink 2010) Detuning −25 dB (midpoint of 15–30 dB range) ⇒ $\Phi \approx 40$
α_{ds}	0.50	0.50	–	(Golding et al. 2005)
ΔV_{soma}	5×10^{-8}	5×10^{-7}	mV	$\kappa \cdot B \cdot \Phi \cdot \alpha_{ds}$

−25 dB corresponds to $|H| \approx 0.056$ (18-fold amplitude attenuation for a single crystal). Serial coupling of two crystals (n = 2) therefore restores an *effective* amplitude gain $\Phi \approx 36 – 40$. A full 20-to-60 sensitivity sweep is reported in § S2.11.2.

Supplementary Table 45A. Calculation steps for P_{phase} (city / village) (g = 0.13)

Band	f_i	p_i (calm)	p_i (storm)	$1 - f_i p_i$ (calm)	$1 - f_i p_i$ (storm)
7.83 Hz	1.00	0.195	0.325	0.805	0.675
16–18 Hz	0.62	0.417	0.417	0.740	0.740
20–28 Hz	0.18	0.588	0.588	0.894	0.894
Product of the three	–	0.534	0.447	–	–
$P_{phase,city}$	–	0.466	0.553	–	–
$P_{phase,village}$	–	0.195	0.325	–	–

$$P_{phase} = 1 - \prod_i (1 - f_i p_i)$$

In the village scenario only the 7.83 Hz band contributes; therefore $P_{phase,village} = f_1\, p_1 = 1.00 \times p_1$

Supplementary Table 45B. Calculation steps for P_{phase} (city / village) (g = 0.22) full

Band	f_i	p_i (calm)	p_i (storm)	$1 - f_i p_i$ (calm)	$1 - f_i p_i$ (storm)
7.83 Hz	1.00	0.195	0.415	0.805	0.585
16–18 Hz	0.62	0.417	0.417	0.740	0.740
20–28 Hz	0.18	0.588	0.588	0.894	0.894
Product of the three	–	0.534	0.388	–	–
$P_{phase,city}$	–	0.466	0.612	–	–
$P_{phase,village}$	–	0.195	0.415	–	–
RR ≈ 1.33					

g = 0.22 corresponds to the full transfer of the mean peak narrowing (11 % ΔFWHM) into the phase domain.

Supplementary Table 46. Epidemiological studies and the β coefficient

#	Author (year)	Population	Endpoint	RR	95 % CI	log RR	SE² (log RR)	β = (RR−1)/0,28
1	(Nishimura et al. 2020)	Taiwan	Suicide attempts	1.15	1.05-1.25	0.140	0.00197	0.54
2	(Kay 1994)	Scotland	Depression (hospital)	1.36	1.12-1.66	0.308	0.00996	1.29
3	(Raps et al. 1992)	Israel	Psychiatric admissions	1.15	n/a	0.140	n/a	0.54
4	(Tada et al. 2014)	Japan	Suicides	1.18	1.05-1.32	0.165	0.00341	0.64
5	(Partonen et al. 2004)	Finland	Suicides	1.22	1.10-1.35	0.199	0.00265	0.79
6	(Feigin et al. 2014)	6 countries	First stroke	1.19	1.04-1.36	0.174	0.00468	0.68
7	(Shaposhnikov et al. 2014)	Moscow	Stroke hospitalisations	1.25	1.10-1.42	0.223	0.00424	0.89
8	(Villoresi et al. 1998)	Italy	Myocardial infarction	1.11	1.06-1.38	0.104	0.00453	0.39
Pooled (DL)	–	–	–	1.19	1.14-1.25	0.176	0.00052	0.69
Heterogeneity	–	–	–	Q = 4.25 (df = 6; p = 0.64)	–	–	–	I^2 = 0 %

Leave-one-out	–	–	–	pooled β range: 0.62 – 0.70	–	–	–	–

$$\log RR = \ln(RR)\,;\ \mathrm{Var} = SE^2,\ \text{where } SE = \frac{\ln(\mathrm{CI_{upper}}) - \ln(\mathrm{CI_{lower}})}{2 \times 1.96}$$

CI: 0,48 – 0,85

(Raps et al. 1992) does not report a 95 % confidence interval; the study was assigned a weight of 0 in the random-effects model.

Removing (Nishimura et al. 2020) raises β_{pooled} to 0.70, whereas removing (Kay 1994) lowers it to 0.62.

The β column has been recalculated using $\Delta = 0.28$ as the denominator.
The updated values are: 0.54 · 1.29 · 0.54 · 0.64 · 0.79 · 0.68 · 0.89 · 0.39.
The exact median is 0.66.

For consistency across the manuscript we retain β = 0.67 (rounded to two decimal places); the 0.01 difference is < 2 % in relative terms and has no material impact on β_{pooled} (0.69 vs 0.67) or on the final RR_{hosp} estimate (1.192 vs 1.188).

Supplementary Table 47. Sensitivity analysis – impact of uncertainty in Δ and β on RR_{hosp}

ΔN_{hit} / N_{hit}	β	$RR_{hosp} = 1 + \beta \cdot \Delta$
0.25	0.60	1.15
0.25	0.67	1.17
0.25	0.85	1.21
0.29	0.60	1.17
0.29	0.67	1.19
0.29	0.85	1.25
0.33	0.60	1.20
0.33	0.67	1.22
0.33	0.85	1.28

Legend: Δ – relative increase in N_{hit} (0.25 – 0.33, ± 15 %);

The span β = 0.60 – 0.85 covers the leave-one-out interval (0.62 – 0.70) and the upper section of the 95 % CI.
The lower 95 % bound (β = 0.48) would give RR ≈ 1.14 for Δ = 0.28 and can be obtained by linear interpolation.

Supplementary Table 48. Margins at V_{margin} = 5.7 mV (± 10 %)

Parameter	Nominal value	± 10 % range
V_{margin}	5.7 mV	5.13 – 6.27 mV
Margin after – 55 %	2.57 mV	2.31 – 2.82 mV
Caffeine (0.9 mV)	1.67 mV	1.50 – 1.84 mV

Supplementary Table 49. Matrix of parameter transfer – fear loop (schizophrenia)

#	Parameter / effect	Original region & species	Source (year)	Value in source	Rule†	k	Model value
1	DA ↑ (single recall)	mPFC, rat	(Yoshioka et al. 1996)	≈ +60 %	R4	1.0	+60 %†
2	Cortisol ↑	Saliva, human	(Schwabe and Wolf 2012)	≈ +55–60 %	R4	1.0	+55–60 %†

3	Glu ↓ ≈ 10 % / GABA ↓ ≈ 8 % (ratio ↑)	PFC, human	(Rowland et al. 2016)	−10 % / −8 %	R2	1.0	−10 % / −8 %†
4	PV IN −30 %	PFC & BLA, rat, DG, tree-shrew	(Czeh et al. 2005; Shepard et al. 2016)	−30 %	R1	1.0	−30 %†
5	γ shift (↓ PFC, ↑ BLA)	PFC & BLA, mouse	Steullet *et al*., 2017	qualitative	R2	—	↑ / ↓†
6	Apical dendritic length −20 %	dmPFC, rat	(Radley et al. 2004)	−20 %	R1	1.0	−20 %†
7	BDNF histone-H3/H4 acetylation ↑	vHPC, mouse	(Lubin et al. 2008)	↑	R1	—	↑†
8	AMPAR +25 %	BLA, mouse	(Rumpel et al. 2005)	+25 %	R1	1.0	+25 %†
9	Engram expansion ≈ +35 %	DG, mouse	(Stefanelli et al. 2016)	+35 %	descriptive	—	

† Parameter measured outside vCA1; transferred according to rules R1–R4 (Methods § 2.18.1).

All values are expressed as differences versus control and serve an illustrative purpose.

Supplementary Table 50. Matrix of parameter transfer – sadness loop (major depression)

#	Parameter / effect	Original region & species	Source (year)	Value in source	Rule†	k	Model value
1	HVA ↓ (g ≈ −0.30 SD); 5-HIAA n.s.	CSF, human	(Ogawa et al. 2018)	HVA g ≈ −0.30; 5-HIAA n.s.	R4	—	↓ (qual.)†
2	CRF ↑	CSF, human	(Nemeroff et al. 1984)	+45 – 80 % (mean ≈ +65 %)	R4	1.0	+65 %†
3	Glu ↑ 15 % / GABA ↓ 8 % (ratio ↑)	sgACC, human	(Godfrey et al. 2018; Hu et al. 2023)	+15 % / −8 %	R1	1.0	+15 % / −8 %†
4	Pyramidal depolarisation ≈ +5 mV	IL-mPFC, rat†	(McKlveen et al. 2016; Hu et al. 2023)	+4 – 6 mV (estimated from traces)	R2	—	+5 mV
5	PV IN −30 %	vHPC, tree-shrew & sgACC/PrL, rat	(Czeh et al. 2005; Yu et al. 2020)	−28 – 33 % (vHPC); −25 % (sgACC)	R1	1.0	−30 %†
6	ROS ↑ / GSH ↓	PFC, rat	(Cabungcal et al. 2013)	qual.	R1	—	↑ / ↓†
7	PNN ↓	sgACC/PrL, rat	(Yu et al. 2020)	qual.	R1	—	↓†
8	β-power ↑ (13–30 Hz) / α-power ↓ (8–12 Hz) (rumination)	Scalp, human	(Moon et al. 2018; Forner-Phillips et al. 2020; Benschop et al. 2021)	qual.	R4	—	↑ / ↓†
9	BDNF methylation ↑	mPFC, mouse	(Cheng et al. 2023)	↑ (qual.)	R1	—	↑†
10	Spine density −15 %	dlPFC, human	(Kang et al. 2012;	−12 – 18 % (mean −15 %)	R1	1.0	−15 %†

			Kassem et al. 2013)				
11	vHPC – sgACC connectivity ↑ (δ/θ)	MEG, human	(Hamilton et al. 2015; Higgins et al. 2021)	freq. ↑	R2	—	δ/θ coherence ↑ (qual.)

† Parameter measured outside vCA1; transferred according to rules R1–R4 (Methods § 2.18.1).

All values are differences versus control and are illustrative only.

Supplementary Table 51. Matrix of parameter transfer – trauma loop (PTSD)

#	Parameter / effect	Original region & species	Source (year)	Value in source	Rule†	k	Model value
1	NA ↑ ~3–4 × baseline (fast-scan voltammetry)	BLA, rat; LC-NE, mouse	(McCall et al. 2015; Ronzoni et al. 2016)	~3–4 × baseline	R4	1.0	~3–4 × baseline†
2	DA ↑ +60 %	BLA, rat	(Rosenkranz and Grace 2002; Giustino et al. 2020)	+50 – 70 %	R1	1.0	+60 %†
3	CSF-CRF ↑ +30–40 % (29 ± 8 vs 22 ± 6 pg ml^{-1})	CSF, human	(Bremner et al. 1997)	+30–40 % (29 ± 8 vs 22 ± 6 pg ml^{-1})	R4	1.0	+30–40 % (29 ± 8 vs 22 ± 6 pg ml^{-1}) †
4a	Glu ↑ +14 %	right hippocampus †	(Rosso et al. 2017)	+14 % (CI 9–18 %)	R1	1.0	+14 %†
4b	GABA ↓ −20 ± 10 %	anterior insula†	(Rosso et al. 2014)	−20 ± 10 %	R2 (δ/θ coupling)	1.0	−20 ± 10 %†
5	ΔE_{GABA} +5 ± 2 mV	dCA1, mouse	(Inoue et al. 2013)	+7 mV	R2	—	+5 ± 2 mV†
6	PV IN −30 %	BLA & mPFC, mouse	(Shepard et al. 2016)	−30 %	R1	1.0	−30 %†
7	EEG γ ↑ / α ↓	Scalp, human	(Dunkley et al. 2015; Shaw et al. 2023)	qual. (state-dependent)	R4	—	↑ / ↓†
8	BDNF promoter methyl ↑	mPFC, rat (SPS)	(Roth et al. 2009)	↑ (qual.)	R1	—	↑†
9	ROS ↑ / GSH ↓	mPFC, rat	(Cabungcal et al. 2013)	↑ / ↓ (qual.)	R1	—	↑ / ↓ (qual.)
10	LTP threshold ↓	BLA → vCA1, mouse	(Kim and Cho 2020)	qualitative (facilitated LTP)	R1	—	qualitative
11	PNN ↓	(BLA/PFC)	(Murthy et al. 2019)	↓ (qual.)	R1	—	↓ (qual.)

† Parameter measured outside vCA1; transferred according to rules R1–R4 (Methods § 2.18.1).

‡ Value expressed as difference versus control; illustrative only.

Percent changes refer to peak vs same-session baseline unless noted.

Supplementary Table 52. Rescue E⁺ protocol (90-min precision window, minimal core)

Time (min)	Activated axes	Intervention	Biophysical / network target
–15 → 0	–	Premedication: ECG, SpO_2, electrolyte panel (K^+ / Mg^{2+})	Cardio-electrolyte safety
0 → 40	C (+ partly D)	Ketamine 0.5 mg kg^{-1} i.v. *or* esketamine 56–84 mg intranasal	Peak BDNF release & rapid spine turnover
+10 → 30	D (phenotype-specific)	SZ: iTBS 40 Hz, 600 pulses, 80 % RMT MDD: rTMS 2 Hz *or* dTMS α 10 Hz PTSD: dTMS θ 2 Hz	γ/θ phase-reset; ↑ PV gain
+10 → 90	F* (optional)	μ-metal booth ≥ 35 dB if β-loop 0-1 (+)	Reduce ELF injection-locking
+60 → 90	E	45-min EMDR or CBT-p + propranolol 40 mg p.o. (taken 60 min before session)	Extinction of "hot" engrams
> 90	–	Hydration, light meal	Return to baseline
24 h	–	PET-KCC2 (TOF, ROI vCA1) + 5-min ECG	Assess ↑ KCC2 & HRV

Activation criteria

Protocol is launched **only if** $\Delta V_{margin} < 3$ mV after 4 weeks (or < 5 mV after 8 weeks) **and** B-loop 0 / 1 +.

Repeat schedule

- One session → biomarker check at 4 weeks.
- If $\Delta V_{margin} < 5$ mV → next session after 4 weeks (max 3 sessions yr^{-1}).
- With $\Delta V_{margin} \geq 5$ mV return to "hygiene" E.

Safety notes

- Exposure per session < 2 h; continuous CO_2 / T / RH monitoring (ISO 14117).
- No prior studies on ketamine + μ-metal shielding → full safety assessment in Pilot / RCT required.
- Concept protocol; IRB / Bio-Ethics approval mandatory before any in-vivo start.

Supplementary Table 53. β-loop (ELF → clinic): three-stage causal test before activating axis F

Stage	Research question	Data / analysis	Success criterion
0 — Geomagnetic storms (retrospective 5–10 yr)	Does $K_p \geq 6$ (± 48 h) or ΔFWHM 7.83 Hz ≥ 13 % increase daily ICD-10 F + I admissions?	ΔFWHM from global SRN stations (35°–65° N/S); admissions from WHO / Eurostat / HCUP; quasi-Poisson GAM (nspline DOY + DOW + T_{max} + RH).	95 % CI of β_{macro} excludes 0
1 — Daily noise (prospective 12 mo)	Do fluctuations ΔFWHM < 20 pT (lags	1–300 Hz magnetometer (≤ 1 pT	β_{micro} ($p < 0.05$) with the same sign as β_{macro}

	0–7 d) correlate with admissions?	RMS); 1-h FFT windows; DLNM.	
2 — Bayesian hierarchy (after 24 mo)	Does $\beta_{pooled} \neq 0$ after adjusting for season / weather / smog?	Hierarchical model (countries / cities); prior N(0, 1); $\hat{R} < 1.1$.	95 % CrI of β_{pooled} excludes 0
3 — 48 h alert	Do *Kp*, ΔFWHM + weather predict admissions?	GBM (XGBoost) + LSTM.	ROC AUC ≥ 0.80

* Stage 3 is launched only if stages 0–2 succeed; it is used solely for early warning and does not affect falsification.

Assumptions & quality control

- ELF data: SRN stations within ± 10° geomagnetic latitude; urban magnetometer calibrated in a μ-metal chamber.
- Hospital admissions: ICD-10 F00–F99 + I00–I99, UTC day 00–24 h.
- Control variables: DOY, DOW, T_{max}, RH; sensitivity analyses include $PM_{2.5}$ (CAMS EU) and a "COVID dummy."
- Statistical power (α = 0.05): stage 0 > 0.99; stage 1 ≈ 0.85 (β ≈ 0.04, N = 365).

Supplementary Table 54. PET KCC2 → ΔE_{GABA} → ΔV_{margin} (ROI vCA1; SUVR normalised to cerebellum)

Step	Equation / Assumption	Input data	Result (+15 % SUVR)	Sources
1	$\Delta[Cl^-]_i$ (%) ≈ – 0.90 × ΔSUVR (%)	+15 % SUVR	–13.5 %	(Keramidis et al. 2023)
2	ΔE_{GABA} (mV) ≈ 0.267 mV × $\lvert\Delta[Cl^-]_i$ (%)$\rvert$ (full form: 26.7 mV × ln (1 − Δ[Cl⁻]/100) @ 37 °C)	–13.5 %	–3.60 mV	Nernst equation @ 37 °C
3	ΔV_{margin} (mV) ≈ 0.60 × ΔE_{GABA}	–3.60 mV	+2.16 mV	§ 2.19

Supplementary Table 55. MEG γ burst → $\Delta gain_{dend}$ → ΔV_{margin}

Step	Equation / assumption	Input data	Result (–35 % γ burst)	Source / note
1	$\Delta gain_{dend}$ (%) ≈ 0.90 × Δburst (%)	–35 %	–31.5 %	NEURON simulation (*gamma_gain.hoc*)
2	$\Delta V_{effEPSP}$ (mV) ≈ 0.032 mV × $\Delta gain_{dend}$ (%)	–31.5 %	–1.0 mV	(Kim and Johnston 2015; Malik and Johnston 2017)
3	ΔV_{margin} (mV) ≈ – $\Delta V_{effEPSP}$	–1.0 mV	+1.0 mV	Multicompartment model

95 % confidence interval for the coefficient 0.032 mV %⁻¹: 0.028 – 0.036 mV %⁻¹ (patch-clamp, n = 48).

Supplementary Table 56. HRV (rMSSD) → ΔV_{rest} → ΔV_{margin}

Step	Equation / assumption	Input data	Result (+5 ms rMSSD)	Source / note
1	ΔV_{rest} (mV) ≈ – 0.08 mV·ms^{-1} × ΔrMSSD	+5 ms	–0.40 mV	(Rowland et al. 2016; Keerthy et al. 2021)
2	$\Delta V_{margin} \approx -\Delta V_{rest}$	–0.40 mV	+0.40 mV	Margin definition

The −0.08 mV·ms^{-1} coefficient was estimated from a meta-regression of HRV versus excitability (patch-clamp n = 54 plus TMS-MEP/MRS studies) and will be re-validated empirically in the pilot phase.

Supplementary Table 57. Projected sensitivity of HRV (rMSSD) and its contribution to ΔV_{margin} in the pilot study

Sub-cohort*	N (planned)	rMSSD baseline (mean ± SD, ms)	Δ rMSSD at $T_{4\ weeks}$ ± SE (ms)‡	p (paired t)	Estimated contribution ΔV_{margin} (mV)§
A – "low stim" (caffeine < 200 mg day^{-1} & nicotine < 5 cig day^{-1})	17	27.8 ± 7.1	+5.6 ± 1.2	0.001	+0.45
B – "high stim" (caffeine ≥ 200 mg day^{-1} or nicotine ≥ 5 cig day^{-1})	13	24.2 ± 8.4	+2.1 ± 1.5	0.09	+0.17
Combined (A + B)	30	26.2 ± 7.8	+4.1 ± 1.0	0.002	+0.33

* Split by daily caffeine and nicotine intake; cohort B has lower planned power (~ 0.56) and is considered exploratory.

† Power calculation for α = 0.05, expected effect +5 ms, SD = 5 ms: power ≈ 0.82 (package pwr v 1.3, R 4.4).‡ SE = SD / √N; $SD_{low\ stim}$ = 5 ms, $SD_{high\ stim}$ = 7 ms) (Shaffer and Ginsberg 2017).

§ Conversion uses the coefficient 0.08 mV·ms^{-1}, derived from published data (patch-clamp CA1: (Rowland et al. 2016); HRV-MEP correlation: (Keerthy et al. 2021)).

Supplementary Table 58. Rescue F (ELF screen; 7 + 7 days)

Days	Exposure	Cabin parameters	Parallel procedures	Exit criterion
1 – 7	24 h day^{-1} (≤ 30 min break d^{-1})	μ-metal / active compensation DC < 0.05 μT; attenuation 7 – 30 Hz ≥ 35 dB	Fixed-time A–D cycle One EMDR / CBT-p session inside cabin (day 3)	Assessment on day 7 MEG γ-burst ↓ ≥ 35 % **or** PET KCC2 ↑ ≥ 15 % (if already available) **or** ΔV_{margin} increase ≥ 3 mV

8 – 14	Only if day-7 criterion unmet	μ-metal / active compensation DC < 0.05 μT; attenuation 7 – 30 Hz ≥ 35 dB	+ $MgSO_4$ 1 g i.v. over ≥ 20 min every 48 h + tVNS 2–10 Hz (30 min day^{-1}; frequency phenotype-specific)	Assessment on day 14 Criterion as above

Measurements

- MEG γ-burst and ΔV_{margin}: mandatory on days 7 and 14.
- PET KCC2: day 14 only.

Inclusion criteria for Rescue F

1. $\Delta V_{margin} < 3$ mV despite ≥ 1 E^+ session.
2. β-loop stages 0 – 2 positive (95 % CrI $\beta_{pooled} \neq 0$).
3. Voluntary consent to 7 – 14 days in isolation.

Maximum: 1 F-cycle per 6 months.

Safety

Exposure < 2 h day^{-1} in confined space; HEPA filtration, CO_2 sensor, IR camera, intercom; full AE/SAE monitoring in pilot.

Rescue F is not used in the pilot or RCT II; it will be evaluated in a separate RCT III only if the β-loop is confirmed and the DSMB rules the cabin's logistical cost clinically justified.

Supplementary Table 59. Secondary limitations

Area / module	Technical or secondary limitation
Magnetite parameters (κ, Q)	Variation ±7 % changes ΔV_{soma} by ≈ 6 %.
ELF–θ coherence	Stability of 7.83 Hz in the human hippocampus undocumented (to date only EEG α/θ).
ΔV_{ELF} vs Johnson noise	$\Delta V_{ELF} \approx 0.5 \cdot V_{rms}$ in a dendrite, $\sim 12 \cdot V_{rms}$ at whole-cell level (model).
"Low PV window"	Data (−25 % g_{inh}) from P14–P21; in adults the TASK/GIRK contribution may differ.
ELF exposure	No personal dosimeters; exposure estimated from geomagnetic modelling.
Brain temperature	Slices 34–35 °C → Nernst potentials over-estimated by ≈ 0.6 mV (2–3 %).
Monte-Carlo propagation	Total error estimated without full MC; 95 % CI may be slightly too narrow.
Heterothermia ± 0.3 °C	Regional variance omitted; adds < 0.2 mV.
Linearity of ΔV summation	Additivity assumed up to ≈ 10 mV; above that Nav/shunt nonlinearities may appear.
Effect durability > 12 wk	No long-term follow-up yet; scheduled checkpoints at 6 and 12 months.
Hormonal variability	Cycle / estradiol not analysed; potential differences in KCC2 / R_{in}.

Equipment drift	MEG / rTMS re-calibration required every 4 wk; procedure in protocol.
Multiple testing	> 50 exploratory hypotheses; FDR / hierarchy applied.
Extreme replay	No data for > 50 replays day^{-1} or > 1 month burst duration.

Supplementary Table 60. Detailed research directions and proposed methods

#	Research goal / knowledge gap	Key methods *
1	ELF biosensor (β +)	μ-metal slice demagnetisation; Cry1/2 KO; nano-SQUID; cryo-EM of magnetite chains; TRPC/V blockade + patch-clamp; DFT flavin radical ≤ 1 μT
2	Second-generation biomarkers	PET PV, PET Kir4.1, MRI CEST Cl^-, ^{23}Na MRI; automated MEG burstometry
3	Species / demographic validation	CRS ± CACNA1C A / SCN2A GoF (♂/♀, 6 & 18 mo); serial PET/MRI 18–80 yr
4	Non-linearities	Calcium imaging + patch-clamp in CREB clusters; NEURON / NetPyNE heterogeneity
5	Transient landscape	24 h on-scalp MEG + hippocampal LFP; CWT + ML clustering
6	Replay limits	Optogenetic "replay trainer" 50× day^{-1}, 3 mo; tele-EEG 12 mo; MRI CA1/BLA every 3 mo
7	Axis A–F deconvolution	Factorial RCT II (2^4 arms); adaptive Bayesian platform "drop-the-loser"
8	β-loop harmonisation	Time-series meta-regression; DLNM; ACS vs arrhythmia subgroups
9	Phenotype extrapolation	GAD, ChAD/BD, ASD, TLE, addictions, AD/MCI, MS + epilepsy, Dravet, Rett, Fragile X, SCN8A EIEE (SCN8A GoF), KCNQ2 EE (KCNQ2 LoF), OCD
10	Economic translation	RCT III (n ≈ 400, 18 mo); ICER/QALY; adherence analysis

Supplementary Table 61. Hypothetical triggers that may launch the ΔV_{margin} narrowing cascade in clinical phenotypes other than SZ, MDD, and PTSD

Phenotype	Chronic trigger	Dominant neuromod./cytokine axis	Expected network motif	Documentation of four markers*
GAD	"Worry loop" without extinction phase	CRF ↑ + tonic LC-NE	θ with β (15–25 Hz)	✓ τ_{EPSP}, PV, R_{in} ✗ V_{thr}
ChAD / BD	Repeated DA surges in mania	DA ↑ + glutamatergic cortex→vHPC	γ 35–50 Hz ↔ θ	✓ τ_{EPSP}, PV ~ R_{in} ✗ V_{thr}
ASD (FMR1, MECP2)	Early sensory-social overload	5-HT / OXT ↓, mGluR5 ↑	δ–θ dominant, γ deficit	✓ τ_{EPSP}, PV, R_{in} ✗ V_{thr}
TLE	Partial seizures – Glu/K^+ re-entry	ACh & NA bursts	δ–θ interictal, high γ ictal	✓ (full)
Addictions	Cue-induced craving (NAc↔vCA1)	DA ↑ + Glu potentiation	β–γ "salience burst"	✓ τ_{EPSP}, PV ~ R_{in}, V_{thr}
AD / MCI	Aβ-driven "memory wandering"	Aβ → ROS → IL-1β	θ–β dominant, γ deficit	✓ (full)
MS + epilepsy	Chronic IL-6 / TNFα	NKCC1 ↑, Kir ↓	δ–θ + epileptiform γ	✓ τ_{EPSP}, PV, R_{in} ~ V_{thr}
Dravet (SCN1A LoF)	Febrile seizures, Nav1.1 PV ↓	Homeostatic NA / ACh	high γ / fast ripples	✓ (full)
SCN8A EIEE	SCN8A GoF, Nav1.6 hyper	Nav-driven intrinsic burst	very high γ / FR	✓ τ_{EPSP}, PV, R_{in}, V_{thr}

KCNQ2 EE	KCNQ2 LoF (M-current ↓)	Intrinsic excitability ↑	broad γ shift	✓ τ_{EPSP}, PV ✗ R_{in}, V_{thr}
OCD	"Error loop" cortico-striatal	DA ↑ + 5-HT ↓	high β-γ in CSTC	~ τ_{EPSP}, PV; others weak

* ✓ = ≥ 1 patch-clamp or in vivo study; ~ = partial data; ✗ = gap highlighted in the text.

Although RMH posits that the critical "ignition" $\Delta V_{margin} < \approx 5$ mV occurs in ventral CA1 for SZ, MDD and PTSD, certain channelopathies or temporal-lobe epilepsies may breach this threshold first in CA3/dorsal CA1 or even in neocortical layer V, before the instability propagates to the wider limbic network.