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Rethinking the Critical Brain: How Genetics Keep Our Minds from Tipping Over

Biology * Genetics * Neuroscience

Introduction: Reevaluating the Critical Brain Hypothesis

For over two decades, the intersection of statistical mechanics and systems neuroscience has been anchored by the critical brain hypothesis.[1][3] This framework proposes that the mammalian cerebral cortex optimizes computation -- maximizing dynamic range, information transmission, and memory capacity -- when poised at a phase transition between an over-damped subcritical phase (where inhibitory forces rapidly extinguish incoming signals) and a chaotic supercritical phase (where runaway excitation saturates the network, analogous to an epileptic seizure).[1][2] Empirical support centered on neuronal avalanches: cascades of action potentials whose spatial sizes and durations obey scale-free power-law distributions.[1]

However, because biological brains are open, dissipative thermodynamic networks continually driven by correlated sensory inputs, whether observed power laws reflect genuine self-organized criticality or coarse-graining artifacts has remained fiercely debated.[3][4] A landmark February 2026 study in Physical Review Letters by Calvo, Martorell, Roig, and Mu oz, alongside 2025-2026 neurodevelopmental and twin-heritability investigations, resolves this controversy: rather than teetering on a razor-sharp mathematical tipping point, the human brain operates within a dynamically regulated, slightly subcritical "safety zone" governed by adolescent inhibitory maturation and heritable ion-channel genetics.[4][5][6]

Quasi-Criticality, Subsampling Artifacts, and the 2026 Renormalization Pipeline

Neural population dynamics are quantified by the branching ratio ( ), the average number of downstream descendant neurons activated across synapses by a single spiking neuron.[1] While = 1.0 defines idealized criticality under zero external drive, driven biological networks instead migrate along a quasi-critical Widom line -- the locus of finite, maximized dynamic susceptibility -- by adaptively maintaining < 1.0 to prevent sensory surges from triggering supercritical runaway.[3][8][9]

| Phase of Neural Activity | | Branching Ratio | | Network Behavior | | Information Processing Characteristics |

| Subcritical Phase | | Less than 1.0 | | Neural activity dampens and quickly dies out across synapses. | | Poor signal transmission; restricted dynamic range; strongly ordered but inflexible and insensitive. |

| Phase-Transition Point | | 1.0 | | Neural activity propagates evenly without saturation or decay. | | Maximized dynamic range; optimal memory storage capacity; emergence of scale-free neuronal avalanches. |

| Supercritical Phase | | Greater than 1.0 | | Neural activity amplifies uncontrollably through the network. | | Complete loss of stimulus details; chaotic saturation; physiologically analogous to epileptic seizures. |

In their 2026 Physical Review Letters analysis, Calvo et al. proved mathematically that when a non-critical network is driven by temporally autocorrelated ("colored noise") inputs and subjected to fractional subsampling (recording <0.1% of neurons), standard avalanche estimators generate spurious power-law covariance tails while underestimating true scaling exponents.[4] By combining random matrix spectral noise filtering with a Phenomenological Renormalization Group (PRG) coarse-graining pipeline on human resting-state fMRI datasets, the authors stripped away correlated-input distortions.[4][10] Their results demonstrated that macroscopic human brain dynamics exhibit genuine scale-invariant power-law correlations derived from intrinsic reverberant network activity, but consistently reside in a slightly subcritical safety buffer.[4][7]

Temporal Fluctuations and Adolescent Excitation-Inhibition Maturation

To track rapid state-dependent shifts in network dynamics without requiring hours of stationary recordings, researchers developed Temporal Renormalization Group (tRG) autoregressive modeling to quantify the instantaneous distance to criticality (d2) across the evolution of behavioral states.[11][12] Cortical and hippocampal microelectrode recordings show that quiet wakefulness minimizes d2 (hovering nearest criticality for peak sensory readiness), whereas directed motor action moderately increases d2 via local desynchronization.[11][12] Conversely, deep non-rapid eye movement (NREM) slow-wave sleep and general anesthesia push the cortex into deep subcriticality (d2 high), fragmenting long-range temporal correlations to enable hippocampal sharp-wave ripple replay and synaptic homeostasis.[13][15]

| Physiological Brain State | | Proximity to Criticality | | Network Characteristics | | Primary Functional Implication |

| Quiet Wakefulness | | Nearest to Criticality (Lowest d2 value) | | High dynamic range; sustained long-range temporal correlations. | | Optimal sensory readiness, environmental awareness, and information integration. |

| Active Movement | | Moderate Deviation | | High local desynchronization; breakdown of global coordination. | | Focused computational processing of narrowly specific motor and cognitive tasks. |

| NREM Sleep | | Furthest from Criticality (Highest d2 value) | | Strongly synchronized up/down-states; pronounced network fragmentation. | | Off-line memory consolidation; synaptic homeostasis; execution of sharp-wave ripples. |

| Anesthesia / Coma | | Deep Subcriticality | | Collapse of long-range correlations and dynamic range. | | Total suppression of cognitive function, awareness, and sensory processing. |

In a March 2026 longitudinal study of 310 resting-state EEG sessions across 169 participants aged 10 to 33, Westbrook et al. demonstrated how adolescent maturation of the glutamatergic/GABAergic excitation-inhibition (E/I) balance tunes the cortex toward criticality.[6] As inhibitory interneuron networks mature from childhood into

adulthood, long-range temporal correlations and amplitude bistability increase significantly.[6] Low-frequency theta, alpha, and beta bands shift toward lower E/I ratios (stronger inhibition) to stabilize long-range cortical integration, whereas localized high-frequency gamma processing statistics shift toward higher E/I ratios.[6] Mature adult networks also exhibit faster state-dependent E/I dampening upon transitioning from eyes-closed to eyes-open visual stimulation.[6]

The Genetic Architecture of Brain Criticality and Clinical Implications

Analyzing Human Connectome Project resting-state fMRI data from 250 monozygotic twins, 142 dizygotic twins, and 437 unrelated individuals using ACE structural equation modeling, Xin, Cui, Yu, and Liu (PNAS, 2025) established that brain criticality is a strongly heritable phenotype.[5] Additive genetic heritability (h2) reached 0.56 for the global Hurst exponent, 0.46 for the branching ratio, 0.28 for the inter-avalanche interval, and 0.21 for d2.[5]

| Criticality Parameter | | Physiological Function / Mathematical Measurement | | Genetic Heritability Estimate (h2) |

| Global Hurst Exponent | | Measures the presence of scale-free, long-range temporal correlations in signal amplitude over time. | | 0.56 |

| Branching Ratio | | Measures the average strength of neural activation propagation, directly proxying the E/I balance. | | 0.46 |

| Inter-Avalanche Interval | | Represents the temporal structure, duration, and spacing of sequential spontaneous neuronal cascades. | | 0.28 |

| Distance to Criticality (d2) | | Evaluates specific deviations from the phase-transition fixed point via finely time-resolved autoregressive modeling. | | 0.21 |

Heritability followed a distinct cortical gradient: primary sensory and motor cortices exhibited tight genetic control, whereas higher-order heteromodal association cortices showed lower heritability, preserving plasticity for environmental learning.[5] Cross-referencing regional criticality maps with Allen Brain Atlas transcriptomics via Partial Least Squares Regression revealed that near-critical dynamics correlate strongly with gene expression clusters enriched for transmembrane potassium ion transport and neurotransmitter regulation -- the molecular brakes that repolarize neuronal membranes after action potentials.[5] Furthermore, total cognitive performance followed an inverted U-shaped curve peaking within the near-critical subcritical zone, sharing significant bivariate genetic covariance with avalanche timing.[5]

Conclusion: From Network Pathology to Neuromorphic AI

Redefining the critical brain hypothesis around a genetically anchored, developmentally tuned subcritical safety zone unifies clinical neurology and computational neuroscience.[4][5][6] Disruptions to the homeostatic mechanisms governing the excitation-inhibition ratio inevitably displace neural networks from their computational optimum: whereas potassium-channel and GABAergic deficits propel networks across > 1.0 into supercritical epileptic seizures, hallucinations, or

sleep-deprived instability, excessive inhibitory damping traps networks in comatose, anesthetic, or depressive rigidity.[1][5][13][14]

Simultaneously, these biological principles are reshaping precision neurodiversity -- modeling individual cognitive differences through personalized criticality trajectories rather than uniform deficit frameworks -- and inspiring next-generation artificial intelligence.[16] By tuning artificial neural networks toward quasi-critical branching ratios and applying renormalization-group coarse-graining architectures such as DysonNet, synthetic self-supervised models achieve superior dynamic range, long-horizon memory capacity, and asymptotic training-time scaling without runaway computational overhead.[1][16][17] Ultimately, tracking dynamic deviations from this subcritical optimum provides a quantitative clinical biomarker for diagnosing neurodevelopmental and psychiatric disorders while guiding neuromorphic engineering.[1][5][16]

Updates... (Nov 20, 2026): Corrected publication year in Reference 2 (University of Cambridge, Mar 23, 2009 replacing 2015), fixed author initials in References 5 (Xin, Y., Cui, Y., Yu, S., & Liu, N.) and 12 (Chang, H., Chen, W., Karaba, L. A., et al.) against CrossRef metadata, added specific publication dates across all 17 references, and split multi-reference superscripts into 1:1 tags.

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