



# A Multimodal Principle Organizing Critical Neural Dynamics Across States and Scales

Thea Ng<sup>1</sup>, Gemechu Tolossa<sup>2</sup>, Jordan Janzen-Meza<sup>2</sup>, Kiran Bhaskaran-Nair<sup>2</sup>, Caroline Hoyniak<sup>3</sup>, Rebecca Spencer<sup>4</sup>, Joan Luby<sup>3</sup>, Deanna Barch<sup>3</sup>, Keith Hengen<sup>2</sup>

<sup>1</sup>Neuroscience & Behavior Program, Mount Holyoke College, South Hadley

<sup>3</sup>Department of Psychiatry, Washington University School of Medicine, St. Louis

<sup>2</sup>Department of Biology, Washington University in St. Louis, St. Louis

<sup>4</sup>Department of Psychological & Brain Sciences, UMass Amherst, Amherst



## Background

- The brain is hypothesized to operate near a critical point between order and disorder, maximizing computational efficiency and long-range communication. While critical dynamics have been widely observed in humans, the existing methods to quantify criticality often face a trade-off between temporal precision and interpretability.
- Quantifying criticality from continuous EEG and LFP is challenging due to the presence of structured oscillations (e.g., slow waves, spindles) that deviate from scale-free distributions and can obscure critical signatures. Here, we combine modeling with empirical data to test whether a novel temporal metric  $d_2$  can reliably track criticality across scales.
- As the first systematic mapping of  $d_2$  onto empirical neural data, we asked: 1) Which parameter settings allow  $d_2$  to unbiasedly estimate criticality consistent with ground truth models? 2) Does  $d_2$  indicate consistent between- and within-stage changes across species and modalities including EEG, LFP, and spikes? 3) Does sleep restore criticality in humans as observed in rodents (e.g., Xu et al., 2024)? 4) How does criticality vary across development and aging?

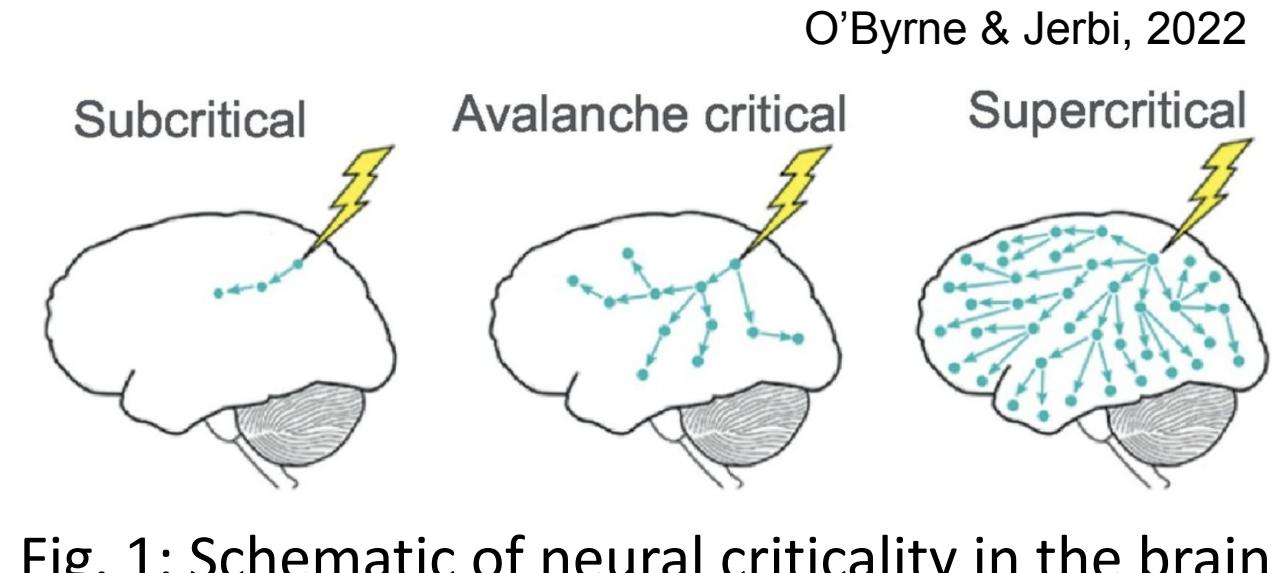


Fig. 1: Schematic of neural criticality in the brain

## Methods

- Subjects:** 1) **Human data** included overnight and nap EEG recordings from 101 healthy participants aged 9 months to 74 years. Four EEG datasets were included: Longitudinal – Infants (9, 12, 15 months) and toddlers (16, 21, 26, 31 months); Cross-sectional – Toddlers (3 years), young adults (mean = 20 years), and older adults (mean = 68 years). 2) **Rodent data** consisted of 24-hour local field potential (LFP) and single-unit spike recordings collected from 12 healthy adult rats.
- Electrophysiology preprocessing:** EEG was recorded with 21 to 122 electrodes during daytime naps and overnight sleep, including pre- and post-sleep wakefulness, preprocessed using a low-pass filter at 100 Hz, downsampled to 200 Hz, and cleaned for artifacts. Sleep stages were manually scored by sleep experts or YASA algorithm (for the 3 years dataset only). LFP data were preprocessed, downsampled to 200 Hz, and cleaned for artifacts. Artifact-free electrodes were selected for spike sorting. Recordings were obtained from four brain regions: CA1, RSPV (Retrosplenial cortex), V1, and ACad.
- Quantifying Criticality:** We implemented temporal renormalization group (tRG) framework (Sooter et al., 2025) to assess scale-invariant dynamics in continuous (EEG, LFP) and discrete (spikes, bin size = 10 ms) time series. Criticality distance metric  $d_2$  (Fig. 1C, where lower values indicate closer proximity to criticality) were defined as the Euclidean distance from the fitted AR model (Yule-Walker, order = 2) to the hyperplane of models associated with a critical power spectral exponent  $\beta = 2$ , where  $\rho_i$  are estimated AR coefficients.
- Dynamic System Models:** Five dynamic system models were implemented to generate synthetic neural-like activity with distinct aperiodic and oscillatory components across different data types: (1-2) the Ornstein-Uhlenbeck (OU) process and linear rate model simulated continuous dynamics with controlled ground truth criticality; (3) the coupled Hawkes process generated discrete spike-like activity; (4) the AR(2) model produced synthetic time series by varying two AR parameters to control criticality; and (5) the multivariate OU network expanded the simulations to include aperiodic and oscillatory modes.
- Computational Modeling:** We performed two sets of simulations. (1) Using EEG-like continuous data, we generated ultra-high sampling-rate signals and systematically downsampled them to test which sampling frequency best recovers  $d_2$  and separates different ground truth criticality. (2) Across both continuous and discrete datasets, we injected low-frequency (0.5-2 Hz) sinusoidal components to evaluate how combinations of bin size and AR order affect the ability of  $d_2$  to suppress oscillatory mode and capture underlying near-critical dynamics.
- Electrophysiology Data Analysis:**  $d_2$  was computed for each 1 minute segment (5 min smoothing window) of broadband raw EEG, LFP, and spiking data. The results were averaged across all clean electrodes for group comparisons. Baseline  $d_2$  were obtained from 2,000 surrogate datasets per segment by temporal shuffling. In addition, within-state changes were quantified by comparing  $d_2$  values from the first 10 and last 10 minute of cleaned segments of data from wake and NREM periods to assess directional changes over time.
- Statistical Analysis:** Group comparisons were assessed using the Mann-Whitney U test or the Wilcoxon signed-rank test, with significance estimated from 10,000 permutation resamples.

## References

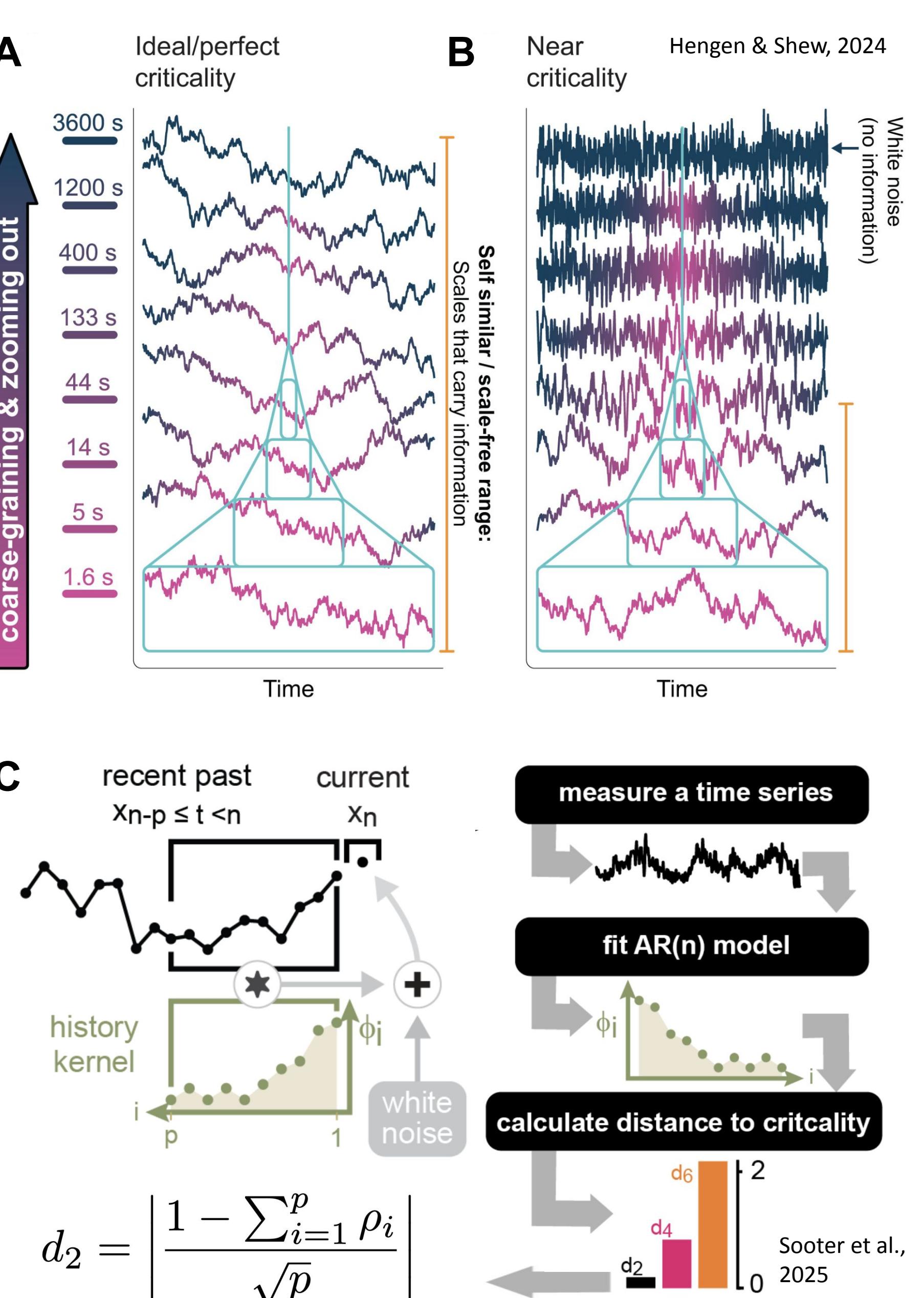


Fig. 2: Defining and quantifying temporal criticality in neural data

## Modeling Results

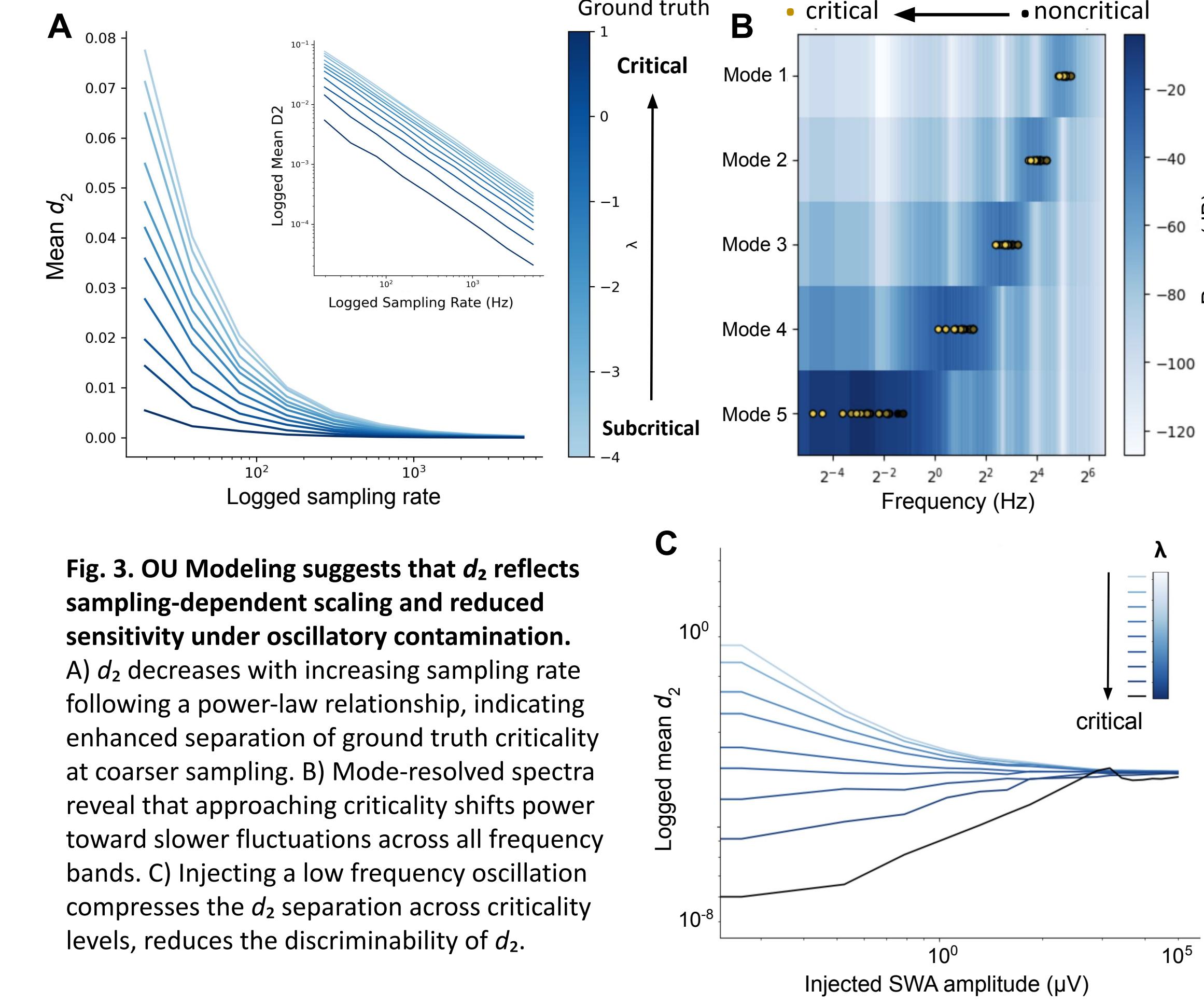


Fig. 3. OU Modeling suggests that  $d_2$  reflects sampling-dependent scaling and reduced sensitivity under oscillatory contamination. A)  $d_2$  decreases with increasing sampling rate following a power-law relationship, indicating enhanced separation of ground truth criticality at coarser sampling. B) Mode-resolved spectra reveal that approaching criticality shifts power toward slower fluctuations across all frequency bands. C) Injecting a low frequency oscillation compresses the  $d_2$  separation across criticality levels, reduces the discriminability of  $d_2$ .

## $d_2$ flips between wake and sleep by varying temporal reaches

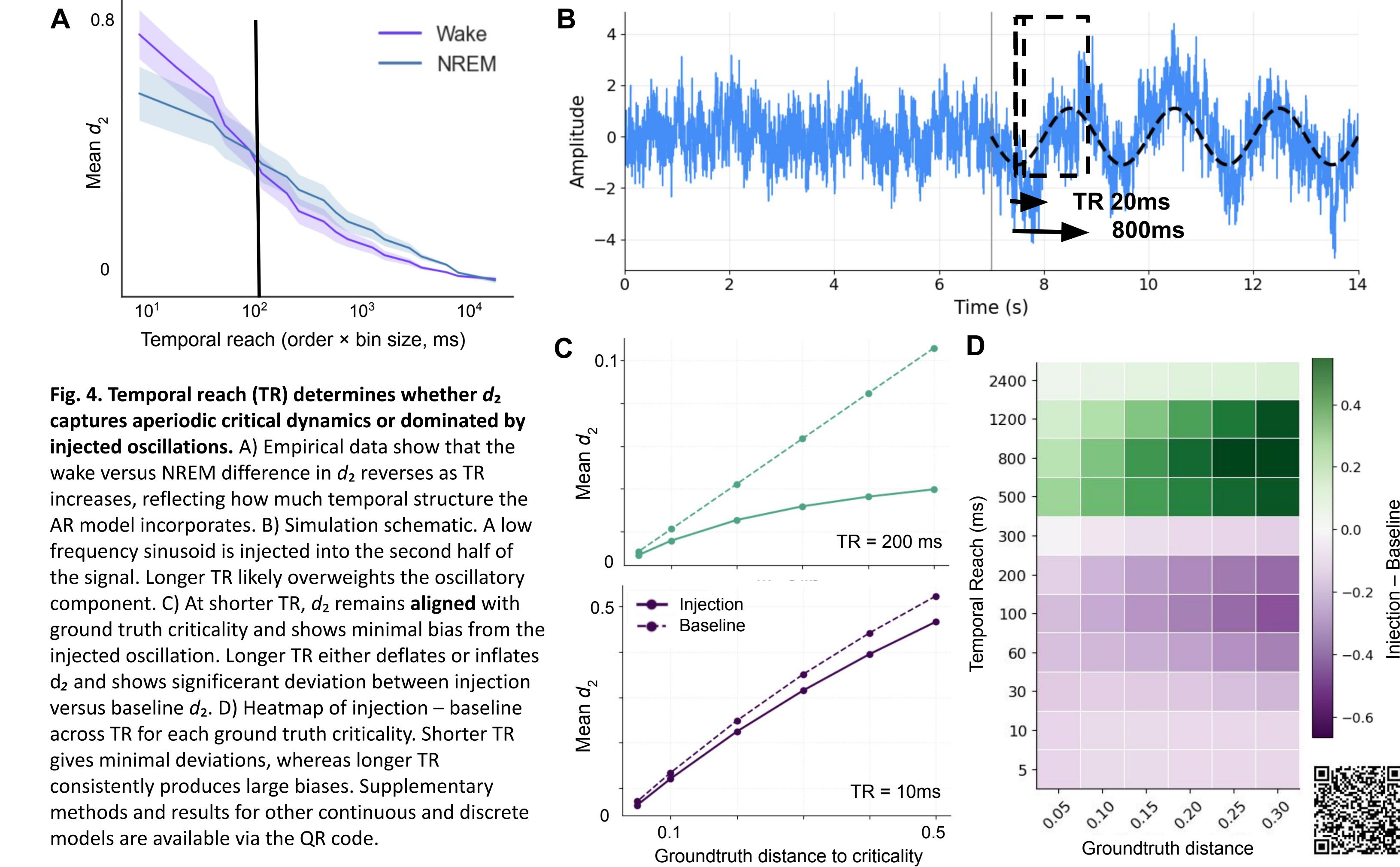


Fig. 4. Temporal reach (TR) determines whether  $d_2$  captures aperiodic critical dynamics or dominated by injected oscillations. A) Empirical data show that the wake versus NREM difference in  $d_2$  reverses as TR increases, reflecting how much temporal structure the AR model incorporates. B) Simulation schematic. A low frequency sinusoid is injected into the second half of the signal. Longer TR likely overweights the oscillatory component. C) At shorter TR,  $d_2$  remains aligned with ground truth criticality and shows minimal bias from the injected oscillation. Longer TR either deflates or inflates  $d_2$  and shows significant deviation between injection versus baseline  $d_2$ . D) Heatmap of injection – baseline across TR for each ground truth criticality. Shorter TR gives minimal deviations, whereas longer TR consistently produces large biases. Supplementary methods and results for other continuous and discrete models are available via the QR code.

## Empirical Results

### $d_2$ exhibits state-dependent changes in human EEG

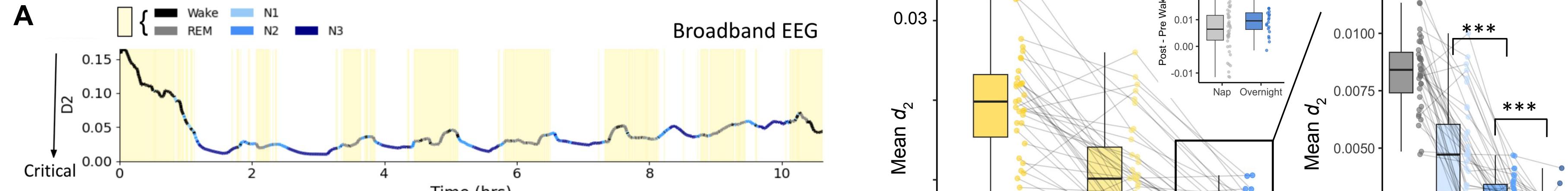


Fig. 5.  $d_2$  tracks state-dependent changes in human EEG across the sleep-wake cycle. A) Example overnight trajectory of broadband  $d_2$  from a healthy human participant, showing higher values during wake and a marked decline across NREM stages. B) Group-level comparison demonstrating reduced  $d_2$  in post-sleep wakefulness relative to pre-sleep, and indicating C) a progressive decrease in  $d_2$  from REM to deeper NREM 3.

### Simultaneous LFP-spike shows consistent direction of $d_2$ changes

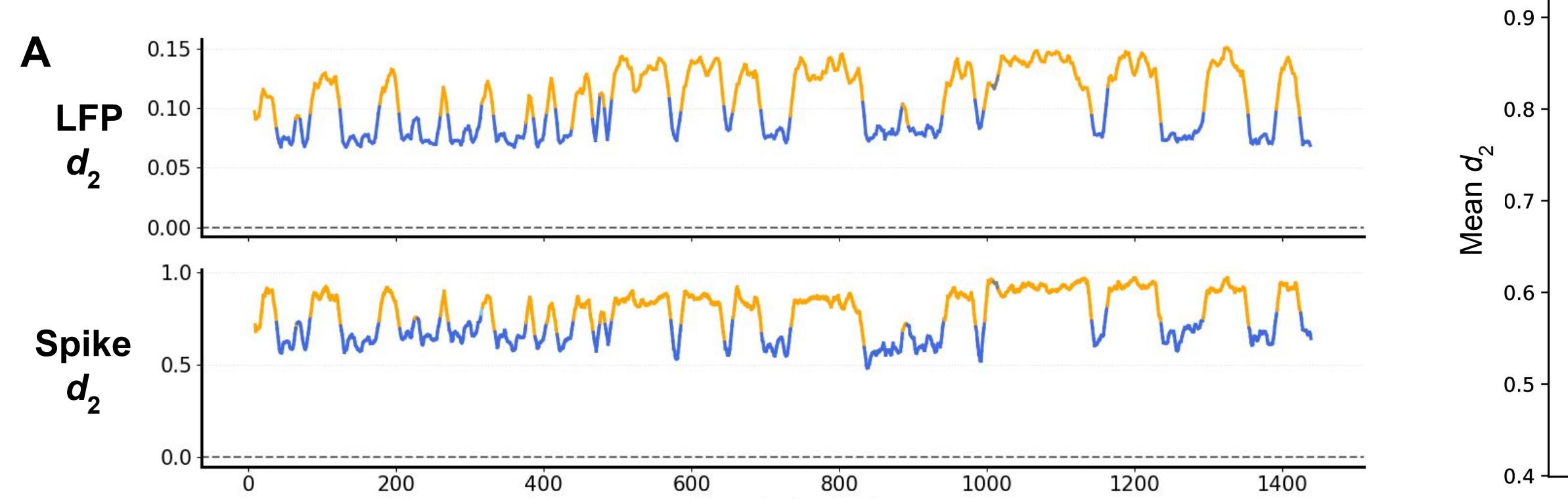


Fig. 7.  $d_2$  exhibits consistent state-dependent changes across sleep-wake cycles in simultaneous LFP and spike data. A) Example 24-hour trajectory of  $d_2$  from a healthy rat. B) Group-level analysis showing robust separation of wake, REM, and NREM in all individuals, with highest  $d_2$  during wake and lowest during NREM, consistent with patterns in human EEG.

### Ongoing analysis: $d_2$ exhibits opposite within-state trajectories during wake and sleep

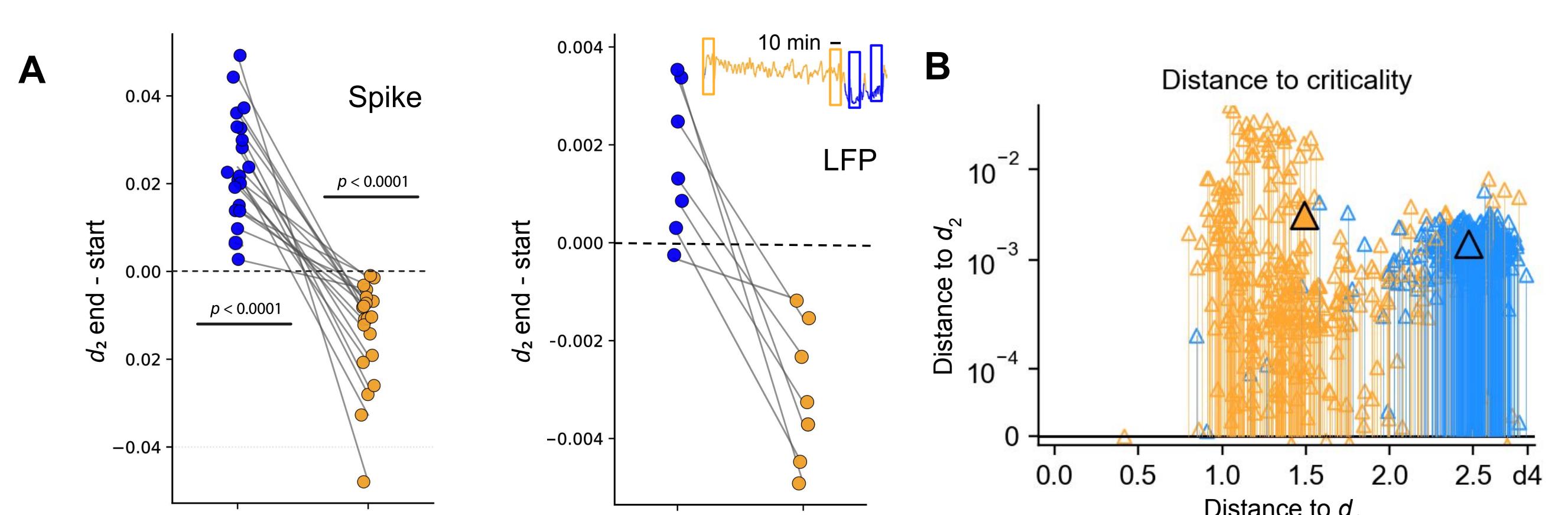


Fig. 8. Exploratory within-state dbeta dynamics. A) Across all datasets, both spikes and LFP show increasing  $d_2$  from the start to the end of NREM, and decreasing  $d_2$  from the start to the end of wake. B) Epoch level visualizations of wake and NREM demonstrate that  $d_2$  exhibits a clear separation between states not only at the aggregated but individual epoch levels.

## The distance to criticality may shift across the lifespan

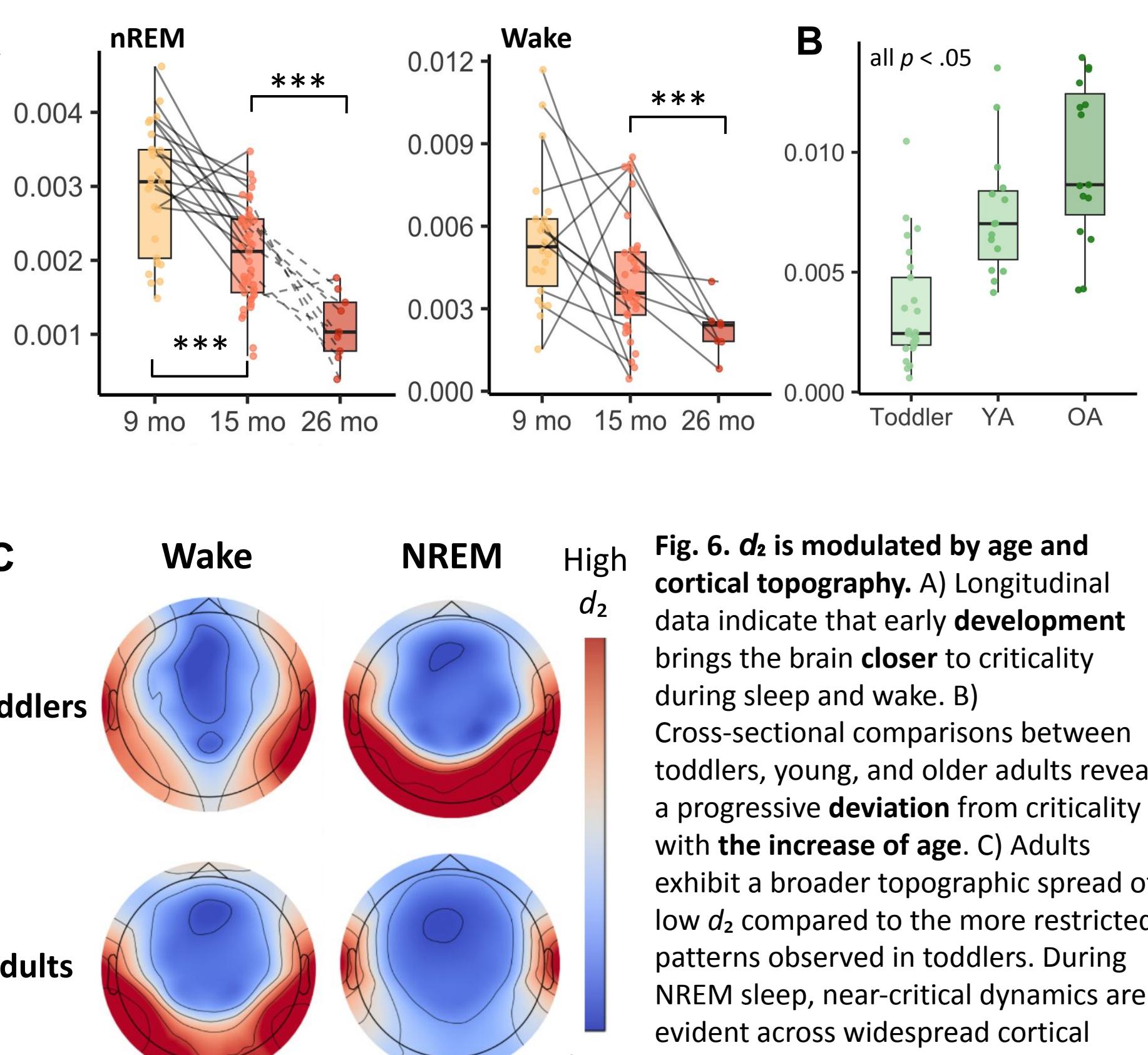


Fig. 6.  $d_2$  is modulated by age and cortical topography. A) Longitudinal data indicate that early development brings the brain closer to criticality during sleep and wake. B) Cross-sectional comparisons between toddlers, young, and older adults reveal a progressive deviation from criticality with the increase of age. C) Adults exhibit a broader topographic spread of low  $d_2$  compared to the more restricted patterns observed in toddlers. During NREM sleep, near-critical dynamics are evident across widespread cortical regions, whereas in wakefulness, low  $d_2$  are confined to frontal-central areas.

## Discussions

### Across recording modalities, we observe that with a short temporal reach:

- Cross-species consistency:**  $d_2$ -measured criticality shows consistent state-dependent changes across human EEG & sEEG, and rodent LFP & spike data.
- Temporal reach effect:** Simulations indicate that  $d_2$  accurately reflects ground truth criticality only at short temporal reach (< 25 ms), capturing underlying near-critical dynamics without bias from low-frequency oscillations.
- State dependence:** Wakefulness deviates from criticality, whereas with deeper nREM sleep showing lower  $d_2$ .
- Within-state changes:** With sleep progress,  $d_2$  gradually increases; during wake,  $d_2$  decreases as a group effect.
- Developing brains:** Become progressively more critical, whereas **aging** is associated with deviation from criticality.
- Summary:** Criticality is dynamically regulated by species, recording modality, brain state, cortical region, and age. Therefore, we believe that more work need to be done to build a unified cross-modal framework.
- Ongoing directions:** 1) Evaluate  $d_2$  as a predictor of wake-sleep transitions and arousal states. 2) Extend analyses to additional datasets with longer recordings, light-dark cycles, and wake/sleep-rich segments for cross-validation. 3) Examine associations between  $d_2$  and neurocognitive metrics (e.g., IQ, pre- and post-sleep changes of memory performance). 4) Investigate relations between empirical criticality measured by  $d_2$  and quasi-criticality theory.