# Criticality in *in silico* and *in vitro* neuronal networks

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**Abstract:** Neuronal networks are hypothesized to operate near a critical state—an intermediate regime between order and disorder—where information processing is optimized. This thesis investigates criticality in neuronal systems using a threefold approach: (i) a branching process model to reproduce avalanche dynamics with power-law statistics; (ii) simulations of spiking activity in spatially embedded networks using Random Geometric Graphs (RGGs) together with the Izhikevich dynamic neuronal model, to explore how modular topology promotes critical behavior; and (iii) analysis of electrophysiological recordings from human induced pluripotent stem cell (hiPSC) derived neuronal cultures. Our findings reveal that both simulated and experimental data exhibit scale-invariant avalanche statistics and satisfy universal exponent relations characteristic of critical systems. Observed deviations from mean-field theoretical predictions are attributed to spatial constraints and connectivity density. These results support the hypothesis that criticality emerges robustly in structurally diverse neuronal architectures while preserving core dynamical features.
**Keywords:** Criticality, neuronal networks, scale invariance, branching process, universality
**SDGs:** 3. Good health and well-being, 4. Quality education

## I. INTRODUCTION

Understanding the collective dynamics of the human brain remains a major challenge in neuroscience. Increasing evidence suggests that the brain operates near a *critical state*—a delicate boundary between order and disorder—where computational efficiency is maximized. This regime has been linked to healthy brain function, while deviations from it are associated with conditions such as epileptic seizures and neurodegenerative diseases [1].

In the context of statistical physics, criticality refers to the behavior of systems at phase transitions, where a macroscopic observable (the order parameter) changes non-analytically in response to a control parameter. At the critical point, systems display hallmark features such as diverging correlation lengths, critical slowing down, and scale-invariant dynamics. These features reflect an underlying universality, which refers to the fact that structurally different systems can display the same collective behavior near criticality [1].

The *criticality hypothesis* proposes that neuronal networks exploit these properties to support complex computation. Near criticality, the system displays optimal dynamic range, enhanced sensitivity to stimuli, efficient information transfer, and long-range interactions. These features may underpin the brain's capacity for adaptability, learning, and robust information processing [2].

Detecting criticality in the brain requires identifying key statistical signatures, such as scale invariance —evidenced by power-law distributions of neuronal avalanche sizes and durations— and specific exponent relations. Together with the observation of long-range correlations and universal behavior across systems, these features form a consistent framework for diagnosing criticality in neuronal dynamics [1].

This project aims to investigate the presence of critical dynamics in neuronal systems through a combination of theoretical modeling, numerical simulations, and analysis of experimental data.

## II. THEORETICAL BACKGROUND

### A. The Branching Process as a Model of Neural Dynamics

The *branching process* is a stochastic model for the propagation of events through networks, with broad applications in fields such as population dynamics, epidemic spreading, and nuclear chain reactions. In neuroscience, it provides a minimal yet powerful framework to study how activity spreads through neuronal circuits, capturing their hierarchical and propagative nature with few assumptions. Despite its simplicity, the model aligns well with empirical observations in cortical systems [3], making it a valuable tool for investigating how ongoing neuronal activity propagates and self-organizes.

In this model (see Fig. 1A), neurons are binary units (active/inactive) connected by directed links with transmission probabilities. At each time step, a neuron activates if any presynaptic input is successful, with additional spontaneous activations modeling background noise. The system is governed by the *branching ratio* ($\sigma$), the expected number of activations per active neuron in the next time step. By tuning $\sigma$, one can drive the network across different dynamical phases: subcritical, critical and supercritical (see Fig. 1B) [3]. In our implementation, we adopt a simplified version: at each time step, every active neuron activates a Poisson-distributed number of others with mean $\sigma$ [2], preserving the core stochastic dynamics without explicit connectivity.

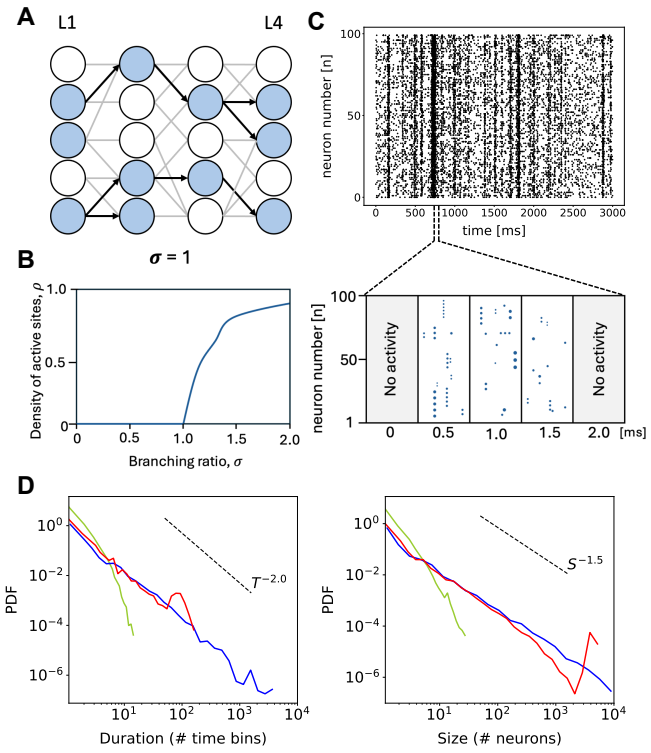


FIG. 1: **(A)** Branching model representation. When $\sigma = 1$, the system reaches a critical state where, on average, each active neuron (blue) activates exactly one neuron in the next layer, allowing information to be optimally propagated without amplification or loss. **(B)** Density of active states $\rho$ (order parameter) as a function of branching ratio $\sigma$ (control parameter) in a feedforward network. In the subcritical regime ($\sigma < 1$), activity quickly dies out, while in the supercritical regime ($\sigma > 1$), activity grows uncontrollably. At the critical point ($\sigma = 1$), the system balances between these extremes of extinction and explosion, leading to complex, scale-invariant dynamics. **(C) Top**: Representative raster plot of the branching model showing spatiotemporal activity patterns. **Bottom**: Avalanches are defined as consecutive frames with activity, bounded by silent bins. **(D)** Avalanche size and duration distributions for three branching ratios: subcritical ($\sigma = 0.8$, green), critical ($\sigma = 1.0$, blue), and supercritical ($\sigma = 1.2$, red). Only in the critical regime do the distributions follow power laws with characteristic exponents $\alpha$ and $\tau$, indicating sustained scale-invariant activity. Subcritical dynamics show premature decay, while supercritical ones exhibit runaway activation.

## B. Neuronal Avalanches and Scale Invariance

In 2003, Beggs and Plenz recorded local field potentials (LFPs) from cortical slices using multi-electrode arrays and identified cascades of neuronal activity that exhibited scale-invariant properties [2]. These cascades, termed *neuronal avalanches*, were defined by discretizing the continuous LFP signal into uniform time bins and detecting sequences of consecutive active bins bounded by silent bins, which marked the start and end of each event, as illustrated in Fig. 1C. Neuronal avalanches provided compelling evidence that brain activity may operate near a critical point. At criticality, systems exhibit scale-invariant dynamics, meaning there is no characteristic spatial or temporal scale governing the behavior. This absence of scale is typically reflected in power-law distributions. In neuronal networks, this is observed in the distributions of avalanche size $S$ and duration $T$, which follow:

$$P(S) \propto S^{-\tau}, \tag{1}$$

$$P(T) \propto T^{-\alpha}, \tag{2}$$

where the critical exponents $\tau$ and $\alpha$ capture the fundamental scaling behavior of the system. Beggs and Plenz [2] empirically reported $\tau \approx 1.5$ and $\alpha \approx 2.0$, values consistent with those predicted for systems in the universality class of critical branching processes (see Fig. 1D).

However, the observation of power-law distributions in avalanche size and duration, while indicative, is not sufficient to conclusively demonstrate criticality. Similar heavy-tailed patterns can emerge from non-critical processes such as stochastic fluctuations, finite-size effects, or heterogeneous external inputs [4]. A more robust signature is the scaling of average avalanche size with duration:

$$\langle S \rangle(T) \propto T^{\gamma}, \tag{3}$$

which introduces a third critical exponent $\gamma$ that characterizes the dynamic coupling between both magnitudes. Importantly, at criticality, the three exponents $\tau$, $\alpha$, and $\gamma$ are not independent but are linked through a universal scaling relation derived from renormalization group theory and the assumption of scale invariance [4]:

$$\gamma = \frac{\alpha - 1}{\tau - 1}. \tag{4}$$

To analyse our data, temporal binning was applied using a time bin $\Delta t$ approximately matching the mean inter-spike interval (IEI) computed for each dataset [2]. This binning yielded a binary raster suitable for detecting neuronal avalanches. The distributions of avalanche size and duration were analyzed by computing empirical probability density functions (PDFs) with logarithmic binning to capture heavy-tailed behavior. We also tested the scaling relation between average avalanche size and duration as described by Eq. 4 [5].

Power-law and truncated power-law models were fitted using the `powerlaw` Python package [6], with the lower cutoff $x_{\min}$ optimized via Kolmogorov–Smirnov (KS) minimization. Fit quality and model selection were assessed through likelihood ratio tests and KS statistics. Uncertainties in the scaling exponents were estimated using non-parametric bootstrap resampling with $n = 100$ iterations.

## C. Modeling Distance-Dependent Connectivity

While classical branching models successfully reproduce key signatures of critical neuronal dynamics, they neglect

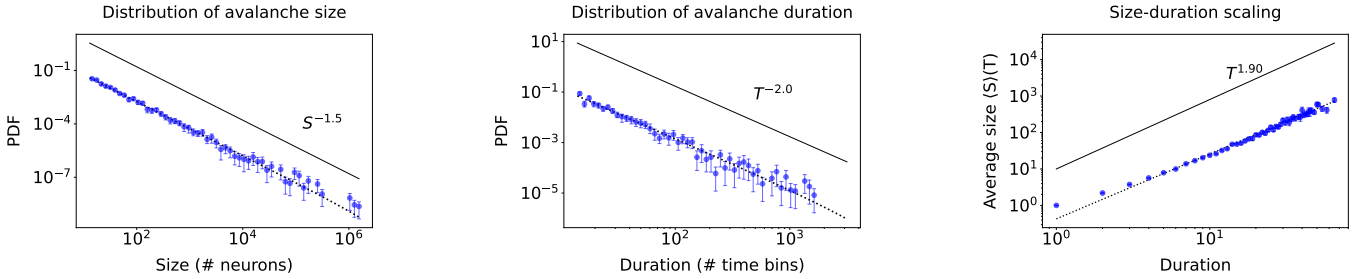


FIG. 2: Avalanche statistics using the branching model at criticality ($\sigma = 1$). Size and duration distributions follow power laws, and mean size scales with duration, consistent with predictions for critical branching processes.

the spatial constraints inherent to biological networks. In the brain, neurons are embedded in a three-dimensional space, and synaptic connections predominantly form between spatially proximal cells due to anatomical limitations. To capture the influence of spatial organization on network dynamics, we employ Random Geometric Graphs (RGGs)—spatial network models in which nodes are randomly distributed in a $d$-dimensional Euclidean space and connections are established between node pairs separated by less than a threshold distance $r$. This distance is set relative to system size, and thus is treated as dimensionless. Additionally, a fraction of connections can be randomly removed, introducing further variability and realism into the model.

Distance-dependent connectivity shapes the network's modular structure: small values of $r$ produce tightly clustered, locally connected modules, whereas larger values result in more homogeneous, globally integrated networks. We quantify this community structure using *modularity* $Q$ [7], a scalar metric that compares the observed density of intra-community connections to that expected in a random network with the same degree distribution. Values near 1 reflect strong modularity with dense intra-community and sparse inter-community links; values near 0 or negative indicate weak or no community structure. Such topological variations substantially affect the system's dynamical behavior and its proximity to criticality.

To simulate neuronal activity on our RGG, we use the Izhikevich model [8], a two-dimensional system of ordinary differential equations. By tuning four key parameters ($a$, $b$, $c$, and $d$), this model accurately reproduces the spiking patterns of individual neurons as well as the bursting dynamics typical of cortical neuronal networks:

$$\frac{dv}{dt} = 0.04v^2 + 5v + 140 - u + I + \eta, \tag{5}$$

$$\frac{du}{dt} = a(bv - u), \tag{6}$$

where the after-spike reset is given by:

$$\text{if } v \geq 30 \text{ mV, then } \begin{cases} v \leftarrow c, \\ u \leftarrow u + d, \end{cases} \tag{7}$$

The variable $v$ denotes the neuron's membrane potential, and $u$ is a recovery variable. The input current $I$ represents synaptic inputs, computed as a weighted sum of presynaptic activity modulated by synaptic efficacy, which includes short-term synaptic depression (STD), and $\eta$ is a noise term to drive spontaneous activity. We set the Izhikevich neuron parameters to $a = 0.02$, $b = 0.2$, $c = -65$ mV, and $d = 6.5$, which reproduce regular spiking neuron dynamics.

### D. Experimental Data

To bridge the theoretical framework with empirical observations, we applied our analytical tools to high-resolution electrophysiological recordings. These cultures were grown on a high-density-CMOS-based microelectrode array (HD-MEA), which consists of 4,096 electrodes arranged in a $64 \times 64$ grid covering an area of approximately $3.8 \times 3.8$ mm$^2$, enabling simultaneous recording of electrical activity from multiple neurons at a temporal resolution of 50 $\mu$s. The dataset consists of hiPSC-derived neuronal cultures with a balanced 75% excitatory and 25% inhibitory neuron ratio. Recordings were performed using 2,048 electrodes (approximately half of the full array) over a duration of 5 minutes. The data was provided by Dr. Giulia Parodi (U. Genova, Italy) [9].

## III. RESULTS AND DISCUSSION

### A. Analysis of the Branching Model Dynamics

To characterize avalanche statistics across different dynamical regimes, we simulated synthetic synaptic activity using the branching process described in Section II.A, with varying branching ratios $\sigma$, spanning subcritical, critical, and supercritical regimes. Each simulation involved up to $N = 100$ neurons over $T_{\max} = 10{,}000$ time units. Temporal binning was applied using $\Delta t = 1.0$, approximately matching the IEI [2].

At $\sigma = 1$ (see Fig. 2), both avalanche size and duration distributions exhibited approximate power-law scaling. The estimated exponents were $\tau = 1.518 \pm 0.014$ for the size distribution and $\alpha = 1.99 \pm 0.04$ for the duration distribution. These values are in strong agreement with critical exponents reported in the literature [2, 5].

Additionally, the mean avalanche size as a function of duration scaled as a power law with exponent $\gamma_{exp} = 1.78 \pm 0.14$, which is consistent with the theoretical prediction $\gamma_{teo} = 1.91 \pm 0.09$. This third scaling relation further supports the criticality hypothesis of the model, confirming its ability to reproduce key signatures of critical neuronal dynamics. Simulations with $\sigma < 1$ and $\sigma > 1$ deviated from power-law behavior, reflecting subcritical (exponentially truncated) and supercritical (runaway) regimes, respectively. The observed scale-free distributions and consistent critical exponents validate the branching model as a robust framework for investigating criticality in neuronal systems.

## B. Impact of Network Topology on Dynamics

To investigate how network topology shapes the emergence of critical dynamics, we simulated spiking activity in a network of $N = 1{,}000$ Izhikevich neurons embedded in a 2D RGG over 5 minutes of simulation. The radius parameter ($r$) was varied to systematically control the network's modularity ($Q$) and connection density. Avalanche statistics were computed from population activity binned at 1 ms—matching synaptic transmission timescales for accurate avalanche detection (see Fig. 3A, B) [2].

We report results for three values of $r$, corresponding to distinct dynamical regimes (Fig. 3C). For small $r = 0.05$ ($Q = 0.905$), avalanche size and duration distributions decay rapidly, lacking power-law tails indicative of subcritical dynamics. In contrast, large $r = 0.3$ ($Q = 0.457$) yields heavy-tailed distributions, consistent with supercritical behavior and runaway excitation. At an intermediate radius $r = 0.1$ ($Q = 0.771$), power-law scaling emerges, consistent with critical dynamics. The measured exponents $\tau = 2.08 \pm 0.03$ (size) and $\alpha = 2.64 \pm 0.04$ (duration) yield a scaling relation $\gamma_{\text{exp}} = 1.54 \pm 0.04$, closely matching the theoretical prediction $\gamma_{\text{teo}} = 1.52 \pm 0.05$ . Although the critical exponents differ from branching model predictions (further discussed in next section), the presence of robust scaling and consistent exponent relations confirms the emergence of criticality in spatially structured networks. These results highlight the role of spatial embedding and modularity in shaping neural activity regimes.

## C. Comparison with Experimental Neuronal Data

To assess model relevance, we compared simulated dynamics with avalanche statistics from neuronal cultures. The optimal bin size estimated was 0.4 ms—consistent with spike-based studies of dissociated cultures[10] but notably smaller than the 4 ms used for cortical slices [2], reflecting differences in signal type and preparation. Power-law scaling was robust near this bin size, while larger bins ( >1 ms) produced bimodal distributions indicative of supercriticality.

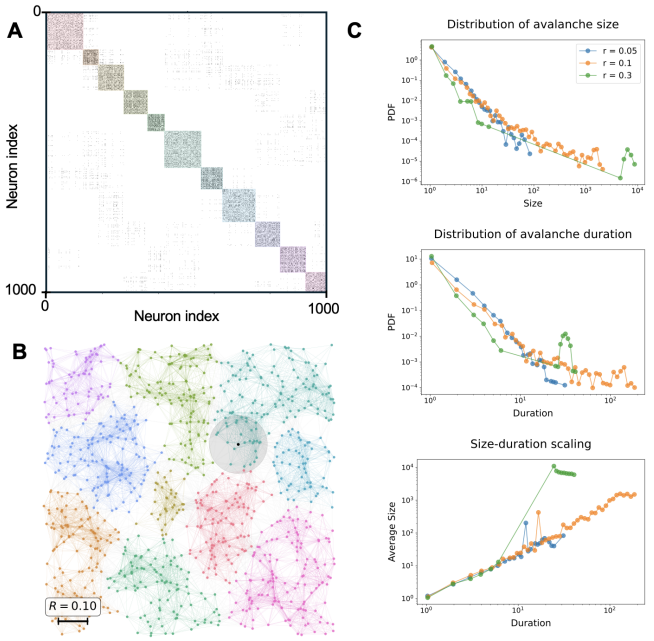


FIG. 3: Impact of network modularity on neuronal avalanche dynamics. **(A)** Adjacency matrix of a RGG with radius $r = 0.1$, showing modular structure (modularity $Q = 0.771$) computed using the Louvain algorithm. **(B)** Spatial layout of the network with nodes color-coded by module, highlighting clustered connectivity. A representative node is marked with a black dot and its connection radius $r = 0.1$, illustrating the local neighborhood within which nodes are connected. **(C)** Avalanche size and duration distributions, along with their scaling relation, for three connection radius: $r = 0.05$ (blue, $Q = 0.905$), $r = 0.1$ (orange, $Q = 0.771$), and $r = 0.3$ (green, $Q = 0.457$). Critical-like power-law behavior is observed only for $r = 0.1$.

Avalanche size and duration in experimental data follow power-law distributions with exponents $\tau = 1.86 \pm 0.02$ and $\alpha = 2.14 \pm 0.02$, respectively. The size-duration scaling yielded $\gamma_{\text{exp}} = 1.37 \pm 0.09$, which is in good agreement with the predicted value (Eq. 4) $\gamma_{\text{teo}} = 1.32 \pm 0.04$.

To rule out stochastic artifacts, we applied surrogate data methods based on spike train shuffling: single-electrode (SE) and all-electrode (AE), using the same bin size as in the avalanche detection [10]. The loss of power-law behavior in the shuffled data confirms that the observed distributions reflect genuine neuronal dynamic.

The extracted critical exponents, though broadly consistent with prior studies [1, 5], deviate from the branching model predictions ($\tau = 1.5$, $\alpha = 2.0$, $\gamma = 2.0$). These deviations can be interpreted through the exponent $\gamma$, which quantifies the spatiotemporal spread of neuronal avalanches [1]. Specifically, $\gamma = 1$ implies chain-like propagation, while $\gamma = 2$ indicates widespread activation in dense networks. Our measured intermediate value, $\gamma_{\text{exp}} = 1.37$, suggests activity spread over sparse, fractal-like subnetworks—consistent with the modular, spatially constrained nature of cortical circuits. This interpretation is supported by our simulations using RGGs, where

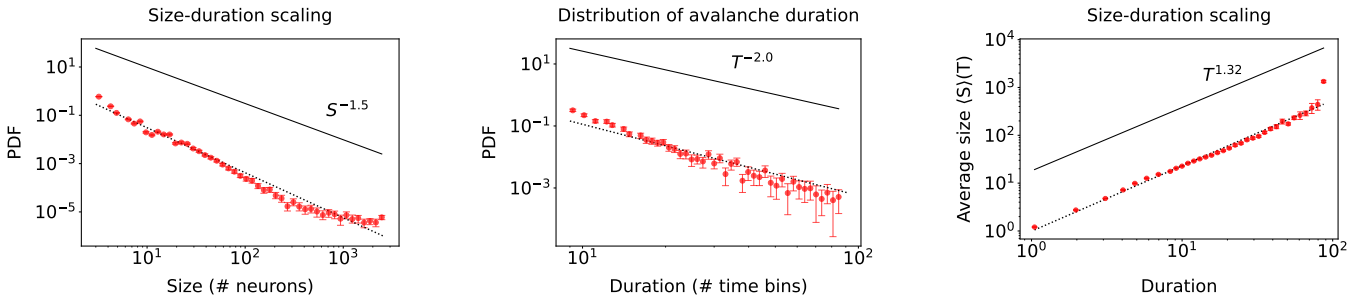


FIG. 4: Experimental avalanche data show power-law distributions for size and duration, and a size-duration relation consistent with Eq. (4), Power laws for theoretical exponents ($\tau = 1.5$, $\alpha = 2.0$, $\gamma = 2.0$) are shown. Error bars reflect statistical uncertainty in distribution tails.

intermediate $\gamma$ values naturally emerged in topologies with limited spatial connectivity and modular organization. These findings reinforce the idea that connectivity density critically shapes avalanche dynamics [1].

The branching model exponents studied belong to the universality class of *mean-field directed percolation* [1], which assumes homogeneous, dense connectivity. Systematic deviations from its critical exponents in experimental data can thus be attributed to structural features of real neuronal networks, such as sparsity and spatial embedding. Therefore, such variations in critical exponents do not necessarily imply different universality classes; rather, they may result from finite-size effects, synaptic heterogeneity, measurement noise, imperfect timescale separation, or methodological factors such as temporal binning and coarse-graining [1, 5].

We propose that incorporating connectivity density into the branching model, while maintaining the critical condition ($\sigma = 1$), yields critical exponents that deviate from mean-field values yet still satisfy the universal scaling relation. This supports the view that criticality does not require finely tuned, homogeneous architectures, but can emerge across structurally diverse configurations.

Consequently, the brain may operate not at a sharply defined critical point, but within a broader *critical regime*—a flexible region in parameter space where scale invariance and exponent relations are preserved despite anatomical and physiological variability.

## IV. CONCLUSIONS

This study demonstrates that neuronal networks exhibit hallmark signatures of criticality, including scale-invariant avalanche dynamics and power-law distributions with consistent critical exponents. Using a branching process model and simulations on spatially constrained networks, we show that criticality emerges near modular configurations and is robust to structural variability. Experimental data from hiPSC-derived neuronal cultures confirm these findings, revealing exponent values that align with predictions and satisfy known scaling relations. Together, these results support the hypothesis that criticality constitutes a fundamental organizing principle of brain dynamics, enabling universality across anatomically diverse systems.

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# Criticitat *in silico* i *in vitro* en xarxes neuronals

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**Resum:** Es planteja la hipòtesi que les xarxes neuronals operen prop d'un estat crític —un règim intermedi entre l'ordre i el desordre— on el processament de la informació és òptim. Aquesta tesi investiga la criticitat en sistemes neuronals mitjançant un enfocament triple: (i) un model de procés de ramificació per reproduir dinàmiques d'allaus amb estadístiques de llei de potència; (ii) simulacions de l'activitat de descàrrega neuronal en xarxes amb estructura espacial, utilitzant Gràfics Geomètrics Aleatoris (RGG) conjuntament amb el model neuronal dinàmic d'Izhikevich, per explorar com la topologia modular afavoreix el comportament crític; i (iii) l'anàlisi de registres electrofisiològics de cultius neuronals derivats de cèl·lules mare pluripotents induïdes humanes (hiPSC). Els resultats obtinguts mostren que tant les dades simulades com les experimentals presenten estadístiques d'allaus invariants a l'escala i compleixen relacions d'exponents universals pròpies dels sistemes crítics. Les desviacions observades respecte a les prediccions teòriques de camp mitjà s'atribueixen a les restriccions espacials i a la densitat de connectivitat. Aquests resultats donen suport a la hipòtesi que la criticitat emergeix de manera robusta en arquitectures neuronals estructuralment diverses, tot preservant propietats dinàmiques fonamentals.
**Paraules clau:** Criticitat, xarxes neuronals, invariància d'escala, procés ramificat, universalitat.
**ODSs:** 3. Salut i benestar, 4. Educació de qualitat

**Objectius de Desenvolupament Sostenible (ODSs o SDGs)**

| | | | |
|---|---|---|---|
| 1. Fi de la es desigualtats | | 10. Reducció de les desigualtats | |
| 2. Fam zero | | 11. Ciutats i comunitats sostenibles | |
| 3. Salut i benestar | X | 12. Consum i producció responsables | |
| 4. Educació de qualitat | X | 13. Acció climàtica | |
| 5. Igualtat de gènere | | 14. Vida submarina | |
| 6. Aigua neta i sanejament | | 15. Vida terrestre | |
| 7. Energia neta i sostenible | | 16. Pau, justícia i institucions sòlides | |
| 8. Treball digne i creixement econòmic | | 17. Aliança pels objectius | |
| 9. Indústria, innovació, infraestructures | | | |

Aquest treball de fi de grau s'alinea principalment amb l'ODS 3: Salut i benestar, ja que la comprensió de la criticitat en xarxes neuronals pot contribuir a entendre millor el funcionament del cervell tant en condicions sanes com en patològiques. La identificació de signatures dinàmiques pròpies d'estats crítics pot ajudar a millorar el diagnòstic de trastorns neurològics, com l'epilèpsia o les malalties neurodegeneratives, i obrir noves vies cap a estratègies terapèutiques més precises i eficients.

A més, el treball també es vincula amb l'ODS 4: Educació de qualitat, concretament amb la fita 4.4, ja que fomenta l'adquisició de competències científiques i tècniques en l'àmbit universitari. A través del modelatge matemàtic, l'anàlisi de dades experimentals i la simulació de sistemes complexos, es potencia una formació transversal orientada a la recerca i la innovació en neurociència computacional.

## GRAPHICAL ABSTRACT