



UNIVERSITY OF GENOA

MASTER'S PROGRAM IN BIOENGINEERING

Thesis submitted in partial fulfillment of the requirements for the title of
Master of Bioengineering

Analysis of Neuronal Avalanche Dynamics in Healthy and Pathological Brain States

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10 2026

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Abstract

The critical-brain hypothesis proposes that healthy neural networks operate close to a critical point, where information processing and dynamic range are enhanced. This work investigates signatures of criticality in the mouse hippocampus using in vivo Neuropixels 2.0 spike recordings, and examines whether they change in two pathological brain states: schizophrenia and epilepsy. A complete avalanche-analysis pipeline was developed, combining power-law fitting of avalanche size and duration, the deviation-from-criticality coefficient (DCC), and avalanche shape collapse, together with correlation and autocorrelation null models.

Healthy recordings showed robust near-critical dynamics, with mostly power-law or truncated power-law distributions, low DCC values, and significant shape collapse. The collapse disappeared when inter-neuronal correlations were removed, supporting the role of network interactions. No single measure was sufficient alone to identify criticality, but different complementary measures are needed.

In the schizophrenia model, exponents, DCC values and shape collapse were largely preserved, while avalanche size distributions more often shifted towards a lognormal form, particularly in ventral regions. In the epilepsy

model, status epilepticus showed heavier-tailed distributions and weaker, less consistent shape collapse, compatible with a temporary shift towards super-critical dynamics, whereas epileptogenesis remained closer to baseline.

Overall, pathological conditions did not simply move the network away from criticality. Instead, they produced subtle changes in avalanche statistics that depended on the measure and brain region. Despite the limited number of animals, these findings support the use of multiple avalanche measures across brain regions as candidate markers of altered network dynamics.

Chapter 1

Introduction

1.1 Aim of the study

This study aims to characterise the collective dynamics of the mouse hippocampus under the critical brain hypothesis and to test whether neuronal avalanche statistics can measure how close the brain operates to criticality. The hypothesis suggests that healthy networks self-organise near the point between ordered and disordered activity, where information transfer, storage, and dynamic range are jointly maximised [Kinouchi and Copelli, 2006]. Deviations from this point, toward subcritical or supercritical regimes, may signal pathology. A central aim is therefore to compare the critical dynamics of the healthy brain with those of models of induced epilepsy and schizophrenia, identifying how each pathology shifts the brain away from criticality and whether avalanche-based measures can serve as candidate biomarkers.

1.2 What is Criticality?

In physics, the concept of criticality was first elaborated to describe materials undergoing a phase transition. The term criticality refers to a distinctive state of complex systems poised at the transition between ordered and disordered dynamics. This transition point, where the system changes from one regime to the other, is called the critical point. Avalanche criticality is the form of critical phase transition most commonly discussed in the neuroscience literature [O’Byrne and Jerbi, 2022]. To better understand the meaning of this ‘critical point’, it is useful to consider the Ising model, a representative example of avalanche criticality. The model consists of a large lattice of interconnected nodes representing atoms in a piece of iron. Each node can occupy one of two possible spin states: spin-up or spin-down, represented in Figure 1.1 by the arrows. In particular, neighbouring atoms tend to keep the same spin direction [O’Byrne and Jerbi, 2022]. When the temperature T is low, these nearest-neighbour interactions dominate, causing most spins to align in the same direction. As a result, the material exhibits a net magnetisation and behaves like a magnet. On the other hand, when the temperature becomes very high, thermal fluctuations start to disrupt this alignment. Although neighbouring spins still tend to align locally, the thermal energy overwhelms their interactions, leading to a disordered configuration in which spins point in random directions. In this regime, the overall magnetisation disappears. At the critical temperature, however, the tendency of spins to align is exactly balanced by thermal fluctuations. The system no longer displays global order, but it is not completely disordered either. Instead, clusters of aligned spins emerge at different spatial scales: some regions contain mostly spin-up

states, while others contain mostly spin-down states. This creates a highly dynamic state characterised by a coexistence of order and disorder, with patterns continuously evolving over time [Beggs and Timme, 2012].

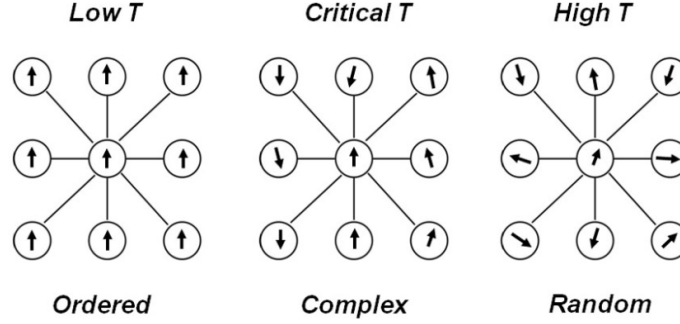


Figure 1.1: Simple diagram of spins in the Ising model

Importantly, in this case, temperature T acts as an external control parameter. The system reaches the critical state only when T is set to its critical value T_c , while any deviation from this value moves the system back to either the ordered or disordered regime. From the concept of criticality arises the phenomenon of self-organised criticality (SOC), where systems composed of many interacting components can spontaneously evolve into a state with properties similar to those of an equilibrium thermodynamic system near a second-order phase transition. Importantly, this behaviour emerges without the need for external parameter tuning, such as the temperature T , and this is where the term 'self-organised' comes from [Marković and Gros, 2013]. If SOC is interpreted as the self-organisation of a system toward an underlying ordinary critical point. One of its most important signatures is avalanche dynamics. Avalanching arises from the separation of timescales between the system's slow external driving and its fast internal relaxation processes

[Pruessner, 2012].

To better understand the concept of an avalanche it is relevant to introduce the sandpile model proposed by Bak, Tang, and Wiesenfeld [Bak *et al.*, 1987]. In this model, grains of sand are added one at a time to a lattice, and each site can only hold a certain number of grains. When this threshold is exceeded, the site becomes unstable and redistributes its grains to the neighbouring sites. This can cause the neighbouring sites to collapse as well, creating a chain reaction that spreads through the system until it stops. This cascade of events is called an avalanche. A key feature of avalanches is that they do not have a characteristic size. Most avalanches are small and stop after a few cascading events, while some can grow and spread across the entire system. Each avalanche can be described by two main quantities, its size S , defined as the total number of cascades, and its duration T , defined as the number of time steps it lasts. In the critical state, these quantities follow power-law distributions, $P(S) \sim S^{-\alpha}$ and $P(T) \sim T^{-\beta}$. This means that avalanches of many different sizes and durations can occur. Such scale-free power-law behaviour is a characteristic signature of avalanche criticality.

A similar phenomenon can be observed in the brain. When neuronal populations are recorded, their activity can form cascades of neuronal firing separated by periods of low activity. When their sizes and durations follow power-law distributions, they are referred to as neuronal avalanches, and the network is considered to operate close to a critical state [Beggs and Plenz, 2003].

1.3 The Critical Brain Hypothesis

The first ones to investigate whether the neural networks could self-organise to a critical state were Dunkelmann and Radons (1994) more than 30 years ago. In the following decades, in particular after the experimental work by John Beggs and Dietmar Plenz (2003), the hypothesis that the brain tends to operate near this critical state has gained a lot of popularity; in fact, not only is there considerable evidence in support of this hypothesis, but also criticality has been shown to maximise many properties that are considered optimal for computation including the range of stimulus intensities that can be processed and the amount of information that can be transferred [Shew *et al.*, 2009]. Therefore, if operating near criticality provides computational advantages, neural circuits may have a functional reason to reach and maintain this state. This could explain why signatures of criticality are commonly observed in neural activity. In this view, criticality is not simply a statistical feature of cortical activity, but could represent an operating state that the brain actively maintains because of its functional benefits. Homeostatic and plasticity mechanisms could help maintain this state and prevent the network from moving towards either an ordered or disordered regime. This view also makes departures from criticality clinically relevant, as pathological conditions may shift the network away from its optimal near-critical state[Zimmern, 2020]. However, there is still some controversy. On the experimental side, assessing criticality from neural recordings is difficult. Power-law distributions of avalanche size and duration are commonly used as evidence of criticality, but they are not sufficient to prove that a system is critical. Non-critical mechanisms can also produce

distributions that appear to follow power laws, while the estimated exponents can be strongly affected by methodological choices such as the temporal bin width, the detection threshold, and the spatial subsampling caused by the limited number of recording electrodes [Touboul *et al.*, 2017]. On the conceptual side, the critical brain hypothesis is challenged by the fact that criticality does not maximise only properties that are beneficial for computation. Moving a network towards criticality can also increase properties that may be disadvantageous for cortical function, such as the variability of the network response. This variability increases strongly near the critical point and can reduce the reliability and specificity of neural responses [Wilting and Priesemann, 2019]. The critical regime under which the brain is supposed to operate lies between two particular states, subcritical and supercritical, where a subcritical state leads to signal quenching, maximum specificity and maximum segregation, while a supercritical state leads to signal amplification, maximum sensitivity, and maximum integration [O’Byrne and Jerbi, 2022].

1.3.1 Neuronal Avalanches

In this case we are dealing with avalanche criticality, specifically neuronal avalanches, with neurons that influence the firing of other neurons through a network of axons, synapses, and dendrites, allowing neuronal activity to propagate throughout the network, leading to avalanches of activity [Friedman *et al.*, 2012]. Neuronal avalanches, as we can see in Figure 1.2, can be defined as cascades of neuronal activity in which neurons trigger subsequent neurons, leading to propagation of activity through the network

across multiple spatial and temporal scales [Beggs and Plenz, 2003].

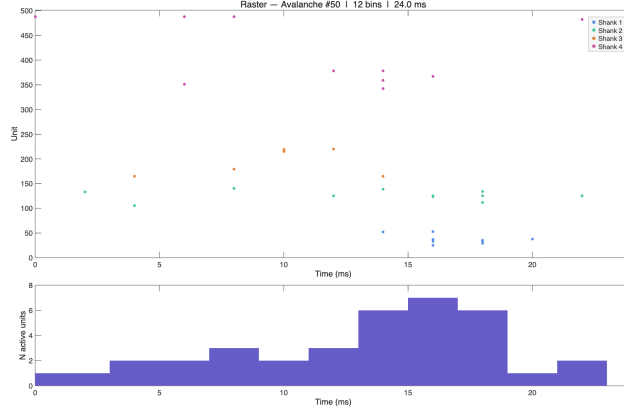


Figure 1.2: Raster and population activity profile of a representative neuronal avalanche. Top: spike raster during the avalanche, with each dot denoting a spike and units colour-coded by the shank on which they were recorded. Bottom: number of simultaneously active units per time bin, defining the temporal profile of the avalanche.

As in the sandpile model, neuronal avalanches at criticality show scale-free statistics. Their sizes and durations have no typical value and follow power-law distributions [Yaghoubi *et al.*, 2018]. Hence, the same statistical behaviour observed in the sandpile model is expected to occur in neural recordings when the network operates close to a critical point.

1.4 Evidences and signatures of Self-Organised Criticality

Neuronal avalanches have attracted so much attention from neuroscientific research because, when the neural network is in a critical state, they exhibit a series of peculiar characteristics. First and foremost, the most distinctive one is the power law scaling of avalanche sizes and distributions, where the size of an avalanche is defined as the number of electrodes or neurons that were activated during an avalanche. In particular, the probability distribution of avalanche size and duration follows a power law with exponent α :

$$P(s) \propto s^{-\alpha}; P(d) \propto d^{-\beta} \quad (1.1)$$

where $P(s)$ denotes the probability of an event of size s , and $P(d)$ denotes the probability of an event of duration d [Klaus *et al.*, 2011]. From the experimental work by Beggs and Plenz (2003, 2004) in rat cortical slices, the power-law exponents were assessed in a critical regime with $\alpha = 1.5$ and $\beta = 2$ [Pasquale *et al.*, 2008]. A power law distribution indicates that neuronal activity is correlated across units, without the formation of strongly interconnected subgroups. This specific pattern arises when the system operates near criticality, making power law scaling a hallmark signature of the self-organised critical state [Priesemann *et al.*, 2013]. However, power law scaling alone is not sufficient to determine whether a system is truly operating at criticality, as power law distributions can emerge from a variety of non-critical mechanisms [Stumpf and Porter, 2012]. Therefore, other signatures must be taken into account. For

example, a natural framework to quantify this critical state is the branching process, where one active node activates, on average, exactly one other node at the next time step. In neural systems, this translates to one active neuron triggering, on average, the activation of exactly one other neuron at the subsequent time step [Hartley *et al.*, 2014]. This condition is formally captured by the *branching ratio* σ , which is observed experimentally as the ratio of the number of descendant electrodes to the number of ancestor electrodes [Haldeman and Beggs, 2005]:

$$\sigma = \frac{n_{\text{descendants}}}{n_{\text{ancestors}}} \quad (1.2)$$

Therefore, we can interpret it as answering a simple but yet fundamental question: when a neuron fires, how many downstream neurons does it trigger on average? The branching ratio σ is interpreted differently depending on its value: $\sigma < 1$ indicates a subcritical system where activity dies out quickly, $\sigma > 1$ indicates a supercritical system where activity overflows the entire network, and finally $\sigma \approx 1$ indicates a critical state, allowing activity to propagate through the network without dying out [Heiney *et al.*, 2021]. If a neural system is in a critical state, another phenomenon also emerges: the mean temporal profiles of avalanches should be identical across scales. In other words, a small avalanche and a large avalanche are just scaled versions of each other. This phenomenon is known as *shape collapse*. Specifically, the mean number of spiking neurons s at time t in an avalanche of duration T is related to the universal scaling function for the avalanche temporal profile F [Marshall *et al.*, 2016].

$$s(t, T) \propto T^\gamma F\left(\frac{t}{T}\right) \quad (1.3)$$

The scaling parameter γ determines the relationship between avalanche duration and size, specifically quantifying how much larger long-duration avalanches are compared to short-duration ones. Crucially, if the system operates near criticality and the appropriate value of γ is selected, avalanches of all durations should collapse onto a single universal mean profile when plotted against the rescaled time t/T and scaled by $s(t, T)T^{-\gamma}$. One of the most demanding indicators of criticality is the exponent relation, which can help to separate a truly critical system from chaos models that generate power laws. It can be defined as the relationship between size (α) and duration (β) distributions power law exponents:

$$\gamma = (\beta - 1)/(\alpha - 1) \tag{1.4}$$

Derived from the exponent relation is the deviation from criticality coefficient (DCC), which is a scalar measure of how close to criticality a system is operating. The empirical β is obtained by fitting the mean avalanche size as a function of duration, $\langle S \rangle(D) \sim D^\beta$. The predicted β is calculated from the crackling-noise scaling relation, $\beta_{\text{pred}} = (\alpha - 1)/(\tau - 1)$, where τ and α are the avalanche size and duration exponents, respectively. The DCC is then defined as the absolute difference between these two values, $\text{DCC} = |\beta - \beta_{\text{pred}}|$. At criticality, the scaling relation is expected to hold, resulting in $\text{DCC} < 0.2$ [Ma *et al.*, 2019].

1.5 The Importance of Neuronal Avalanche Analysis in Clinical Neuroscience

Since, according to the SOC brain theory, which supposes that neural systems are part of self-organised criticality systems, the critical state indicates a maximisation of the efficiency with which neurons communicate with one another, a departure from this state should therefore indicate a breakdown of the system, pointing to the presence of pathologies.

Epilepsy

One of the main diseases to which criticality analysis through neuronal avalanches has been applied is epilepsy [Corsi *et al.*, 2024]. Epilepsy is one of the most widespread neurological conditions, affecting more than 50 million people worldwide and representing a significant burden on health services [Collaborators, 2025]. It is the condition of recurrent, unprovoked seizures, suggesting that the brain has become permanently altered, either pathophysiologically or structurally, to sustain abnormal, hypersynchronous neuronal firing. In particular, it is characterised by hyperexcitability of neurons and hypersynchrony of neural circuits [Stafstrom, 2006]. This is just what happens in a system considered supercritical, where activity propagates without bound. In particular, in a disease such as epilepsy, two distinct pathological states can be distinguished. The first is the seizure itself, that, as mentioned previously, is a hypersynchronous and runaway propagation of neural activity where activity propagates excessively rather than remaining confined. The second, and less obvious, is the interseizure

state, namely the period between seizures. Despite the lack of observable seizure activity, the epileptic brain keeps a pathological propensity to transition into seizure activity. From the perspective of criticality, this state can be interpreted as a chronic reduction in the distance from the 'critical point', making transitions towards uncontrolled activity more likely [Arviv *et al.*, 2016]. Epileptogenesis instead, which is the process through which the brain acquires this pathological propensity, has been associated with alterations in network connectivity and neuronal excitability arising from genetic conditions, such as ion channel mutations, or acquired conditions, such as stroke [Terry *et al.*, 2012]. Understanding whether and how these alterations lead to epileptogenesis has motivated the development of measures of network excitability and synchrony and, more recently, the application of criticality-based approaches [Meisel, 2016].

Schizophrenia

Schizophrenia represents another major neurological condition in which criticality analysis has been applied [Alamian *et al.*, 2022]. Schizophrenia is a severe psychiatric disorder with a lifetime prevalence of approximately 1% of the global population [McGrath *et al.*, 2008], characterised by a complex and heterogeneous symptomatology that includes symptoms such as hallucinations, delusions, and disorganised thinking, as well as symptoms such as social withdrawal, affective flattening, avolition, and cognitive deficits, including impairments in working memory, attention, and executive function [Owen *et al.*, 2016]. Its aetiology remains incompletely understood, but converging evidence implicates disruptions in synaptic transmission and

an imbalance between excitation and inhibition in cortical circuits as core pathophysiological mechanisms [Liu *et al.*, 2021]. Unlike epilepsy, which is associated with supercritical dynamics driven by neuronal hyperexcitability and hypersynchrony, schizophrenia has been characterised by subcritical dynamics, with reduced long-range temporal correlations and disrupted avalanche patterns [Nikulin *et al.*, 2012]. This subcritical signature is consistent with the E/I imbalance thought to underlie the disorder, and has been directly linked to altered spatiotemporal propagation of cortical activity in animal models of NMDA receptor hypofunction [Seshadri *et al.*, 2018]. Therefore, while epilepsy and schizophrenia both represent departures from the critical state, they appear to do so in opposite directions, the first drifting toward supercriticality and the latter toward subcriticality.

Chapter 2

Methods

2.1 Neural Data Acquisition

In this study, the dataset consists of action potentials, acquired at 30 kHz, recorded in vivo from the mouse hippocampus using Imec Neuropixels probes. In particular, the recordings were performed using Imec Neuropixels 2.0 probes, which feature four shanks with a total of 5,120 recording sites, 384 of which can be recorded simultaneously through a single headstage [Steinmetz *et al.*, 2021].



Figure 2.1: Neuropixel 2.0 multishank probe front

Each mouse was implanted with three guide cannulae, a Neuropixels 2.0 four-shank probe, a ground pin, and a headplate. Anaesthesia was induced with isoflurane (5% in 2,L/min oxygen) and maintained at 1–2%, with hourly subcutaneous saline boluses (0.2,ml). Analgesia (SC meloxicam and buprenorphine) and the anti-inflammatory drug dexamethasone (SC) were administered at induction of anaesthesia. Following disinfection and exposure of the dorsal surface of the skull, burr holes were drilled for the guide cannulae and ground pin, and a craniotomy was made for the probe. The cannulae were lowered at approximately 0.01 mm/s to a depth of 6.00 mm for the basolateral amygdala and 2.03 mm for the hippocampus, and were secured in place with RelyX Unicem 2 (3M). The ground pin was lowered to contact the superficial cortex, and the headplate was cemented to the interparietal bone. The probe was then lowered at approximately 5 μ m/s to the target depth and secured with SuperBond cement. The implants were protected with successive layers of Parafilm, PTFE tape, and label tape. Animals recovered in a heated box and received once-daily SC analgesia and fluids for three days post-operatively, together with sweetened wet food. Each recording lasts approximately one and a half hours and includes two phases: a linear-track task, in which the mouse shuttles between the two ends of a track for small sucrose-water rewards, followed by a minimum of 30 minutes in a dark black bucket containing food pellets and bedding material to allow it to rest and sleep.



Figure 2.2: Example of Linear track recording. The mouse learns to shuttle between the two ends of a track for small sucrose-water rewards.

The dataset consists of multiple recordings across four mice. One is a healthy control that serves as a comparative baseline, illustrating the dynamics of a healthy brain; two are mice in which schizophrenia was induced through ketamine injection, and the last one is a mouse in which epilepsy was induced through the administration of kainic acid. Crucially, for the pathological mice, the first three linear-track recordings were acquired while the animals were still healthy, before disease induction. This provides a within-subject baseline for each animal, allowing the pathological state to be compared not only against the healthy control but also against each animal's own healthy recordings, therefore strengthening the comparison.

2.1.1 Spike Sorting and Neural Preprocessing

The whole dataset was then processed with Kilosort4 [Pachitariu *et al.*, 2024] to perform spike detection and spike sorting. Spike sorting extracts the firing times of individual neurons from recordings of the local electric field, a difficult task because recordings are non-stationary and the electric fields of nearby neurons overlap. Kilosort4 solves this with a graph-based clustering approach and performs drift correction. Drift correction is particularly relevant because vertical movements of the probe relative to the tissue change the waveform of a unit over time, and correcting for it substantially improves sorting performance in long recordings, as in the case of this study [Pachitariu *et al.*, 2024]. For each recording, clusters located deeper than 6500 μm from the probe tip along the shank were discarded (Fig. 2.3). Beyond this threshold, units were sparse, isolated, and confined almost entirely to a single shank, in contrast with the dense, banded depth distribution characteristic of genuine anatomical structures, and, in fact, the reconstructed position of these units places them outside of the brain, as the probe was only advanced 6400 μm in. These clusters were therefore attributed to noise and sorting artefacts rather than to real neuronal activity. Apart from spike detection and sorting, which were performed in Python with Kilosort4, all analyses were carried out using custom code written in MATLAB R2025b. Finally, after converting the spike times output from Kilosort from samples to seconds, the neuronal avalanche detection step can be performed.

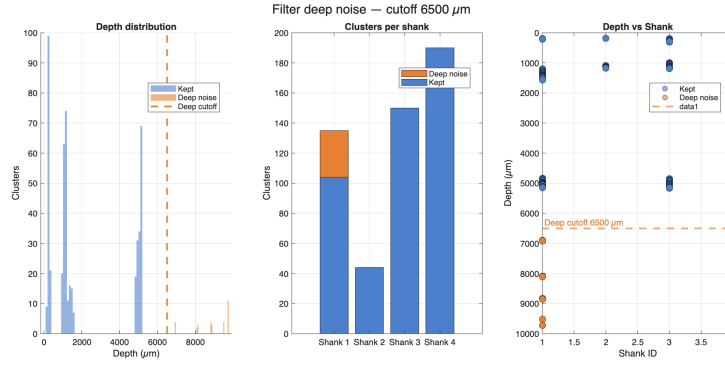


Figure 2.3: Depth-based filtering of noise clusters (representative recording). Clusters deeper than 6500 μm (dashed line) were discarded as noise: unlike retained units, they are sparse, lack banded structure, and are confined almost entirely to Shank 1. The same threshold was applied to all recordings.

2.2 Neuronal Avalanche Detection

Spike trains were first converted into a population activity time series. Spike times were divided into non-overlapping time bins of fixed width (Δt). For each time bin, the number of active units was counted, where an active unit is defined as a unit that fired at least one spike during that bin. If a unit fired more than once within the same bin, it was counted only once. Therefore, the population activity represents the number of neurons that were active at the same time, rather than the total number of spikes. To allow the use of small bin sizes without exceeding memory limits, the recording was processed in consecutive chunks of fixed duration, within which the binning was performed independently. The resulting activity vectors were then concatenated to reconstruct the population activity over

the entire recording. From the population activity, the neuronal avalanches were then identified following the definition provided by the study of [Beggs and Plenz, 2003], stating that an avalanche is defined as a sequence of consecutive bins with non-zero activity, preceded and ended by an empty bin. The duration T of an avalanche, also called its avalanche lifetime, is measured as the number of consecutive time bins (Δt) that it lasts, and the size S of an avalanche is defined as the sum of the population activity over all its bins. A unit active in several bins therefore counts once for each bin, while several spikes from the same unit within one bin count only once [Pasquale *et al.*, 2008]. The choice of bin size Δt is a critical parameter, as it directly determines which events are grouped into a single avalanche. The number of detected avalanches depends strongly on Δt : with small bins, single events tend to be split into several shorter ones, increasing the total count, whereas with large bins temporally adjacent but distinct events are merged into fewer, longer avalanches. The bin size must therefore be chosen carefully, as it directly affects the statistics of avalanche size and duration [Hahn *et al.*, 2010].

2.3 Analysis of Criticality signatures

Once neuronal avalanches have been identified, the next step is to investigate whether the system is close to the critical point. There is no single measure capable of assessing criticality on its own. For example, power-law distributions can arise even when the system is not at criticality; therefore, their presence is necessary but not sufficient [Plenz and Chialvo,

2009]. Therefore, a set of complementary features should be analysed, each of which captures a different aspect of neural dynamics. Three analyses were performed to assess different signatures of criticality. First, avalanche size and duration distributions were fitted with power-law models to evaluate whether the activity followed scale-free behaviour, with no preferred characteristic size or duration. Second, the branching parameter was calculated to estimate how activity propagates from one time bin to the next. This measure helps to understand whether the system operates in a subcritical, critical, or supercritical regime. Finally, avalanche shapes from different durations were rescaled and compared to test whether they collapsed onto a common universal curve. This provides a stronger test of criticality compared with power-law fitting alone. The scaling exponent obtained from the shape collapse was then compared with the value predicted from the size and duration distributions' exponents. The difference between these values was used to calculate the Deviation from Criticality Coefficient (DCC), which measures how far the system deviates from the critical state.

2.3.1 Power-law Fitting of Avalanche Size and Duration Distribution Curves

The first signature of criticality considered is the absence of a characteristic scale in avalanche statistics. In a critical system, avalanche sizes and durations are expected to follow scale-free distributions that can be

described by power laws:

$$P(S) \propto S^{-\alpha}; P(T) \propto T^{-\beta} \quad (2.1)$$

where (S) represents avalanche size and (T) represents avalanche duration. These distributions indicate that avalanches can occur over a wide range of sizes and durations, without a preferred characteristic scale, extending across multiple orders of magnitude. In a finite network, however, there is not an avalanche that can exceed the size of the system that generates it. Similarly, the finite recording duration and the limited number of recorded neurons constrain the range of observable avalanche sizes and durations. The distributions are therefore expected to follow a power law over an intermediate range and then deviate from it beyond a characteristic scale, resulting in a distribution known as a truncated power law. In recordings with a limited number of observed neurons, size and duration distributions may therefore be better described by a truncated power law than by a pure power law [Alstott *et al.*, 2014]. Power-law exponents were estimated following the approach proposed by [Clauset *et al.*, 2009]. Rather than fitting a straight line to a binned histogram in log-log space, which can introduce biases and strongly depends on the choice of bin size, the exponents were estimated directly from the unbinned data using maximum likelihood estimation (MLE). For a given lower cut-off $x_{\min}$, the exponent was calculated as

$$\alpha = 1 + \frac{n}{\sum_{i=1}^n \ln \left(\frac{x_i}{x_{\min}} \right)} \quad (2.2)$$

where n is the number of observations satisfying $x_i \geq x_{\min}$. The same estimator was applied to the duration distribution to obtain β . In empirical

neuronal avalanche distributions, power-law behaviour is typically observed only above a minimum value $x_{\min}$, while smaller events can be affected by finite-size effects and the discrete nature of the data. Therefore, $x_{\min}$ was not fixed beforehand but was estimated directly from the data. Each possible cut-off value was tested by recalculating the exponent using MLE and computing the Kolmogorov–Smirnov (KS) distance between the empirical cumulative distribution function (CDF) of the data above $x_{\min}$ and the theoretical power-law CDF,

$$F(x) = 1 - \left(\frac{x_{\min}}{x} \right)^{\alpha-1}, \quad (2.3)$$

with the KS statistic defined as the maximum absolute deviation between the two distributions,

$$D = \max_{x \geq x_{\min}} |F_{\text{emp}}(x) - F(x)|. \quad (2.4)$$

The value of $x_{\min}$ corresponding to the smallest KS distance was selected as the optimal lower bound, together with the associated exponent estimate. Candidate cut-offs were restricted to the lower portion of the distribution, and tails containing fewer than 30 observations were excluded to avoid unreliable fits. The goodness of fit was evaluated using the KS statistic, where lower values indicate a closer agreement between the empirical distribution and the fitted power law. Distributions were visualised as complementary cumulative distribution functions (CCDF), $P(X \geq x)$, rather than as probability density functions. The CCDF requires no binning and is therefore free from the arbitrariness and the tail noise affecting binned histograms in log-log space. For a power-law density $p(S) \sim S^{-\alpha}$, the CCDF follows directly by integration:

$$C(S) = P(S' \geq S) = \int_S^\infty p(S') dS' \sim \int_S^\infty S'^{-\alpha} dS' \sim S^{-(\alpha-1)}, \quad \alpha > 1, \quad (2.5)$$

so the fitted density exponent α maps to a CCDF exponent $\alpha - 1$. The same holds for durations, giving a CCDF exponent $\beta - 1$. To assess whether the distributions are genuinely scale-free rather than merely appearing linear over a limited range, the local slope of the CCDF was computed in log-log space using a sliding window of fixed width in decades. For a true power law, the local slope is constant across scales, whereas a systematic drift of the slope reveals curvature, the signature of an alternative distribution such as a lognormal. Finally, since the definition of an avalanche depends on the temporal bin size Δt , the entire fitting procedure was repeated across a range of bin sizes. The stability of the fitted exponents, of $x_{\min}$, and of the KS statistic across Δt was used to assess the robustness of the power-law characterisation. To confirm that a power law, whether pure or truncated, was indeed the best description of the healthy-baseline distributions, each avalanche size and duration distribution was subjected to a comparison between four candidate models: a power law, a truncated power law, a lognormal and an exponential. The comparison followed the framework of [Clauset *et al.*, 2009]. The four

discrete models were fitted to the tail ($x \geq x_{\min}$) by maximum likelihood:

$$\text{power law: } p(x) \propto x^{-\alpha}, \quad (2.6)$$

$$\text{truncated power law: } p(x) \propto x^{-\alpha} e^{-\lambda x}, \quad (2.7)$$

$$\text{lognormal: } p(x) \propto \frac{1}{x} \exp\left(-\frac{(\ln x - \mu)^2}{2\sigma^2}\right), \quad (2.8)$$

$$\text{exponential: } p(x) \propto e^{-\lambda x}. \quad (2.9)$$

The models were then ranked by the Bayesian Information Criterion (BIC),

$$\text{BIC} = -2 \ln \hat{\mathcal{L}} + k \ln n, \quad (2.10)$$

where $\hat{\mathcal{L}}$ is the maximised likelihood, k the number of parameters and n the number of tail observations. The $k \ln n$ term penalises the extra parameter of the two-parameter models, so that they are preferred only when the improvement in fit outweighs their added flexibility. The model with the lowest BIC was taken as the best description.

Two complementary significance tests were then applied. The truncated power law contains the pure power law as the special case $\lambda = 0$, so the two are nested and were compared with a likelihood-ratio test,

$$\text{LR} = 2 \left(\ln \hat{\mathcal{L}}_{\text{trunc}} - \ln \hat{\mathcal{L}}_{\text{PL}} \right), \quad (2.11)$$

which follows a χ^2 distribution with one degree of freedom; a significant LR indicates that an exponential cut-off is required.

The lognormal and exponential distributions are instead non-nested with respect to the power law and were therefore compared using Vuong's normalised likelihood-ratio test [Vuong, 1989]. For two candidate densities p_1 and p_2 , the log-likelihood ratio is given by

$$R = \sum_{i=1}^n \ln \frac{p_1(x_i)}{p_2(x_i)}, \quad (2.12)$$

and is normalised by the estimated standard deviation of the pointwise log-likelihood differences:

$$Z = \frac{R}{\sqrt{n}, \sigma}. \quad (2.13)$$

Under the null hypothesis that the two distributions provide an equally good fit, Z is asymptotically standard normal. The sign of R indicates which model is favoured, with positive values that favour p_1 , and negative values that favour p_2 . The corresponding p -value indicates whether this preference is statistically significant or whether the two models cannot be distinguished statistically.

2.3.2 Branching Parameter

The branching parameter quantifies how neural activity propagates through time and provides an estimate of the dynamical regime of the network. In the context of neuronal avalanches, the branching parameter λ answers a simple but fundamental question: when a neuron fires, how many downstream neurons does it trigger on average? [Heiney *et al.*, 2022] This metric helps to classify the network dynamics as subcritical, critical, or supercritical. In particular, a branching parameter smaller than one indicates that activity tends to die out rapidly, whereas values larger than one indicate that activity grows over time. At the critical point, activity is expected to propagate without systematic amplification or decay, leading to

a branching parameter close to 1 [Beggs and Plenz, 2003]. After defining N_T as the number of spikes occurring in the t_{th} time bin. The branching parameter of the i_{th} avalanche was then estimated as

$$\lambda_i = \frac{\sum_{t=1}^{L-1} N_{t+1}}{\sum_{t=1}^{L-1} N_t} \quad (2.14)$$

where L is the duration of the avalanche in temporal bins. Avalanches containing fewer than two bins were excluded from the analysis because no temporal propagation could be defined. The branching parameter of the recording was finally obtained by averaging the values computed across all valid avalanches,

$$\lambda = \frac{1}{M} \sum_{i=1}^M \lambda_i \quad (2.15)$$

where M is the total number of avalanches included in the analysis. Under this definition, $\lambda < 1$ indicates that, on average, activity decreases as the avalanche evolves, corresponding to subcritical dynamics. Conversely, $\lambda > 1$ indicates a progressive increase in activity, characteristic of a supercritical regime. Values close to one are consistent with critical dynamics, where activity is approximately preserved from one generation to the next.

2.3.3 Deviation From Criticality Coefficient and Shape Collapse

A stronger assessment of criticality was performed by analysing the scaling relation between avalanche size and duration through the avalanche shape collapse. In a critical branching process, avalanches of different durations are expected to exhibit a universal temporal profile after appropriate

rescaling. The average avalanche shape follows a scaling relation of the form

$$\langle S(t, T) \rangle = T^{\gamma-1} f\left(\frac{t}{T}\right), \quad (2.16)$$

where $S(t, T)$ represents the population activity at a normalised time t/T within an avalanche of duration T , $f(t/T)$ is the universal scaling function, and γ is the scaling exponent relating avalanche size and duration.

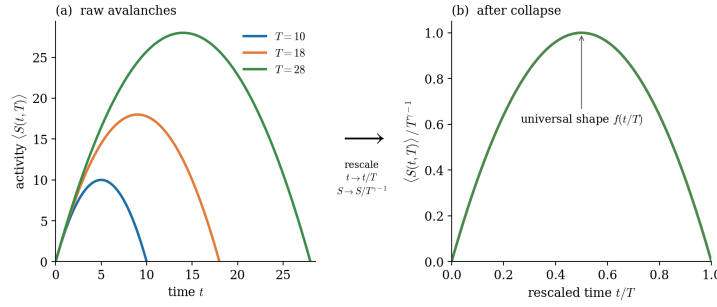


Figure 2.4: Left: Mean profiles $\langle S(t, T) \rangle$ of avalanches of increasing duration T . Under critical scaling the amplitude grows as $T^{\gamma-1}$. Right: After rescaling time by t/T and amplitude by $T^{\gamma-1}$, the profiles collapse onto a single universal shape $f(t/T)$ – the signature of scale-free critical dynamics. A symmetric $f(u) = 4u(1 - u)$ with $\gamma = 2$ is used for illustration.

The scaling exponent γ was estimated from the relationship between the average avalanche size and duration. After selecting avalanches above the minimum cut-offs obtained from the power-law fitting procedure $S \geq x_{min,S}$ and $T \geq x_{min,T}$, avalanches were grouped according to their duration. For each duration group, the mean avalanche size was computed, and the following power-law relation was fitted:

$$\langle S(T) \rangle \propto T^{\gamma}. \quad (2.17)$$

The exponent γ was obtained from a linear regression in logarithmic space:

$$\log(\langle S(T) \rangle) = \gamma \log(T) + c. \quad (2.18)$$

For systems operating at criticality, the scaling exponent is not independent but is related to the exponents of the avalanche size and duration distributions:

$$\gamma_{\text{expected}} = \frac{\beta - 1}{\alpha - 1}, \quad (2.19)$$

where α and β are the exponents of the avalanche size and duration distributions, respectively.

The deviation from criticality coefficient (DCC) was then calculated as the absolute difference between the experimentally estimated scaling exponent and its theoretical prediction:

$$DCC = |\gamma - \gamma_{\text{expected}}|. \quad (2.20)$$

A value of DCC close to zero indicates consistency with the scaling relations expected for a critical system, whereas larger values indicate a deviation from critical dynamics.

To further evaluate the presence of universal avalanche dynamics, avalanche shape collapse was performed. Avalanches with different durations were first normalised to a common temporal scale by interpolating their activity profiles onto a normalised time axis:

$$\frac{t}{T} \in [0, 1]. \quad (2.21)$$

The corresponding avalanche profiles were then rescaled according to their duration:

$$S_{\text{scaled}}(t/T) = \frac{S(t, T)}{T^{\gamma-1}}. \quad (2.22)$$

If the system follows critical scaling, avalanche profiles with different durations should collapse onto a common function:

$$S_{\text{scaled}}(t/T) = f(t/T). \quad (2.23)$$

The quality of the collapse was quantified using the coefficient of variation (CV) computed across the rescaled profiles:

$$CV = \left\langle \frac{\sigma(S_{\text{scaled}})}{\mu(S_{\text{scaled}})} \right\rangle, \quad (2.24)$$

where μ and σ represent the mean and standard deviation of the rescaled profiles across duration groups. Lower CV values indicate a stronger collapse and therefore better agreement with universal scaling.

To test whether the observed collapse was statistically significant, a surrogate distribution was generated by randomly shuffling the association between avalanche profiles and duration values. Each shuffled profile was rescaled using the randomly assigned duration, destroying the temporal-duration relationship while preserving the original avalanche statistics. The p-value was obtained by comparing the CV of the experimental data with the distribution of CV values obtained from shuffled datasets.

2.4 Null Models

To increase the robustness of the analysis and to rule out that the observed dynamics could just be caused by chance, two complementary null models were computed.

2.4.1 Autocorrelation null model

The first is an autocorrelation null model, which means that it destroys the between-neuron temporal structure but keeping at the same time its marginal distribution. In order to build it, the population activity was binned at the chosen temporal resolution and the resulting sequence of bins was randomly permuted across the entire recording.

$$A_b^{\text{surr}} = A_{\pi(b)}, \quad \pi \sim \text{Unif}(\mathcal{S}_N), \quad (2.25)$$

where π is a permutation drawn uniformly from the symmetric group $\mathcal{S}_N$. This preserves the marginal distribution of population activity, which means how frequently a given level of activity occurs, while disrupting the temporal ordering of the bins, and so any autocorrelation between successive time points. Because the avalanches are defined as uninterrupted sequences of active bins, this null model tests whether the observed avalanche size and duration distributions reflect genuine temporal structure in the population dynamics, rather than being a trivial consequence of the instantaneous activity distribution alone [Ribeiro *et al.*, 2010].

2.4.2 Correlation null model

The second is a correlation null model, which means that it interferes with the cross-neuronal spatial correlation, while still preserving the temporal statistics of each individual neuron. For each unit i , its spike times $\{t_{i,k}\}$ were shifted by an independent random offset τ_i and wrapped circularly within the recording bounds:

$$\tau_i \sim \text{Unif}(0, T), \quad t_{i,k}^{\text{surr}} = [(t_{i,k} + \tau_i - t_{\min}) \bmod T] + t_{\min}. \quad (2.26)$$

Since a circular shift leaves each neuron’s firing rate, Inter spike interval distribution and autocorrelation unchanged, this type of shuffle preserve the individual firing statistics of every unit while randomising the relative timing between units, therefore eliminating the coordinated activity across the population. Thanks to this null model, it’s possible to evaluate if the observed avalanche dynamics is caused by genuine coordination between neurons, rather than from the superposition of independently firing units [Louis *et al.*, 2010].

2.5 Region-specific Critical Dynamics

2.5.1 Hippocampal anatomy

The hippocampal formation is not a single homogeneous structure but a set of interconnected subregions with distinct architecture and connectivity. Its principal subregions include the dentate gyrus (DG), the cornu ammonis fields CA3, CA2 and CA1, and the subiculum. These structures form the canonical trisynaptic circuit (Figure...), with input from the entorhinal

cortex (EC) that reaches the DG via the perforant path, the DG that projects to CA3 through the mossy fibres, CA3 which projects to CA1 via the Schaffer collaterals, and CA1 that provides output to the subiculum, which in turn projects to the EC and other target regions. Each subregion therefore occupies a distinct connectivity pattern, most importantly the strongly recurrent architecture of CA3 compared with the predominantly feedforward organisation of CA1, which may shape local network dynamics [Amaral and Witter, 1989].

Since the hippocampus is not a homogeneous structure, with dorsal and ventral poles that are functionally distinct, the critical dynamics might not be uniform across the formation. To address this, the avalanche and criticality analysis was also performed independently within each anatomically defined subregion [Strange *et al.*, 2014].

2.5.2 Anatomical subdivision

For each recording, the probe trajectory was reconstructed with Pinpoint and registered to the Allen Mouse Brain Common Coordinate Framework (CCF). Hippocampal samples were identified using their atlas IDs (CA1 = 382, CA3 = 463, DG = 726) [Birman *et al.*, 2023]. Pinpoint’s channel map is not rendered to physical scale, so the channel index cannot be converted to a metric depth by the probe geometry alone. The channel index was therefore mapped onto the depth axis of the sorted units through a linear scaling factor,

$$d_{\text{pin}}(k) = d_{\text{tip}} + k s, \quad (2.27)$$

where k is the channel index (with $k = 0$ at the probe tip), d_{tip} is the tip position in the spike-sorting coordinate frame, in which raw depth 0 denotes the tip and increases toward the surface, and s is a scaling factor. The factor was set empirically so that the Pinpoint-derived hippocampal boundaries coincided with the banded depth distribution of the recorded units, $s = 2.5 \mu\text{m}$ per channel index.

For each sorted unit, its depth was taken as the median depth of its spikes and its lateral position as the median lateral coordinate, from which the physical shank was identified as the nearest recording channel. Each unit was then matched to the closest Pinpoint sample and assigned its CA1/CA3/DG label, only if the depth mismatch fell below a tolerance θ :

$$\text{region}(c) = \text{region}\left(\arg \min_k |d_{\text{pin}}(k) - d_c|\right), \quad \text{if } \min_k |d_{\text{pin}}(k) - d_c| < \theta. \quad (2.28)$$

Units farther than θ from any hippocampal sample were left unmatched. The tolerance was chosen per recording, from a fixed set of candidate values, as the one maximising the number of successfully matched units, and capped at $500 \mu\text{m}$ to prevent spurious long-range assignments. Each region was further split along the dorsoventral axis. Taking the surface reference d_{surf} as the most superficial unit, a cutoff was placed at $d_{\text{surf}} - c_{\text{dv}}$:

$$\text{unit } c \rightarrow \begin{cases} \text{dorsal} & \text{if } d_c > d_{\text{surf}} - c_{\text{dv}}, \\ \text{ventral} & \text{otherwise,} \end{cases} \quad (2.29)$$

producing six subregions: dCA1, vCA1, dCA3, vCA3, dDG and vDG. The dorsoventral cutoff c_{dv} was calibrated per animal.

Units near the cortical surface, above the hippocampus, were classified as

cortical ($d_c > d_{\text{surf}} - c_{\text{cortex}}$) and excluded from the analysis. The cortex cutoff was either set manually or detected automatically as the largest gap in the depth distribution within a plausible range. In addition, since units at or beyond the surface were sparse, isolated and confined almost entirely to a single shank rather than forming the dense, banded depth distribution characteristic of genuine anatomical structures, they were attributed to noise or sorting artefacts and discarded (Fig.2.5).

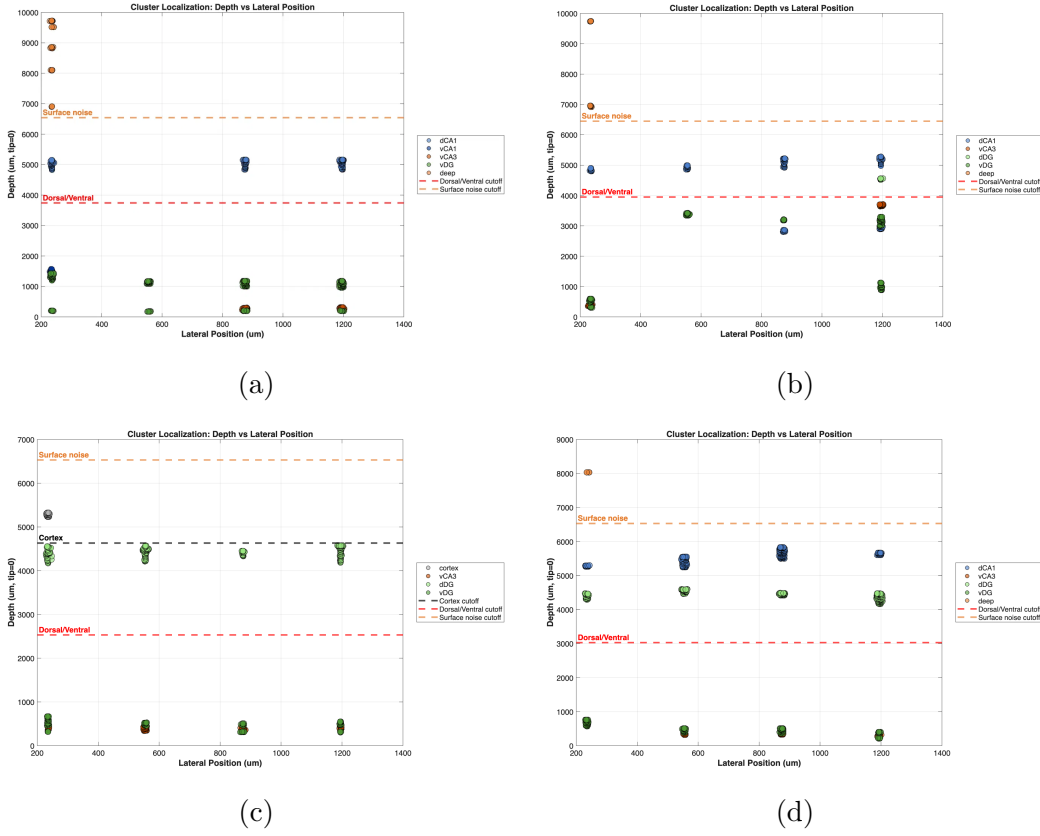


Figure 2.5: Subdivision into hippocampal subregions, shown separately for each animal. Each cluster is localised by depth (probe tip at zero) and lateral position and assigned to a subregion (dCA1, vCA3, dDG, vDG).

The full avalanche and criticality pipeline(avalanche detection, power-law fitting of size and duration distributions, shape collapse, DCC, and the null models) was then applied independently to each subregion, so that every critical signature was estimated for every single anatomical compartment.

Chapter 3

Results

3.1 Critical Dynamics in Healthy Recordings

The healthy dataset includes the six recordings from the healthy control animal, together with the first three linear-track recordings of each pathological mouse, which precede any manifestation of the disease and therefore serve as a healthy baseline. This provides a total of fifteen healthy recordings across four animals. Establishing that the healthy mouse hippocampus operates near a critical point requires more than the presence of the individual signatures of criticality in any single recording [Sethna *et al.*, 2001]. In fact, these signatures must be reproducible across repeated tracks of a given animal and across different animals. Consistency across trials and across mice is therefore seen as a necessary part of the evidence, rather than as a secondary check. Because the recordings span both the

dorsal and ventral hippocampus, which differ in connectivity and functional role, a further question is whether critical-like dynamics are a general property of the region or whether the two subregions behave differently.

3.1.1 Power-law

Whole hippocampus

Starting from the six linear track recordings of the healthy control and the first three recordings of each "pathological" animal, that is, their healthy baseline and focusing on the avalanche size and duration distributions, Fig.3.1 and Fig.3.2 show the complementary cumulative distribution functions together with the running exponent, one representative recording per animal is shown, for both avalanche size and duration.

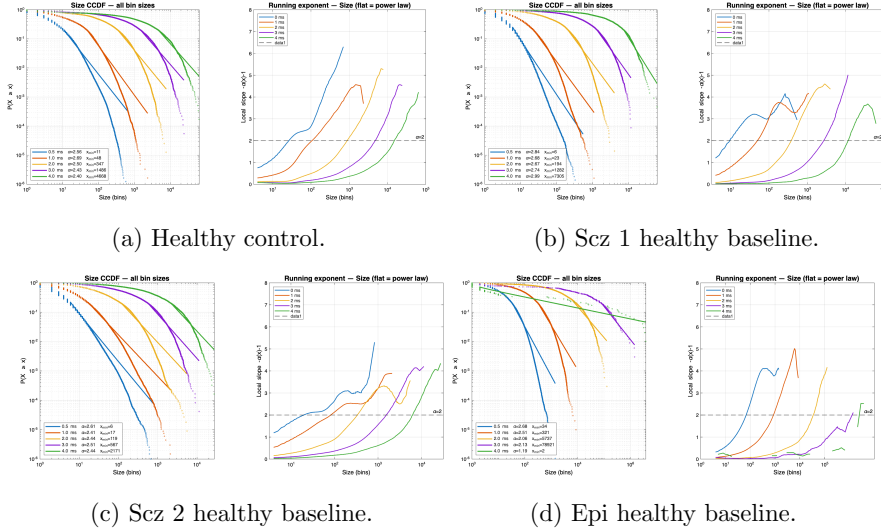


Figure 3.1: Avalanche size distributions for a single representative healthy recording from each animal, computed on the whole hippocampal dataset.

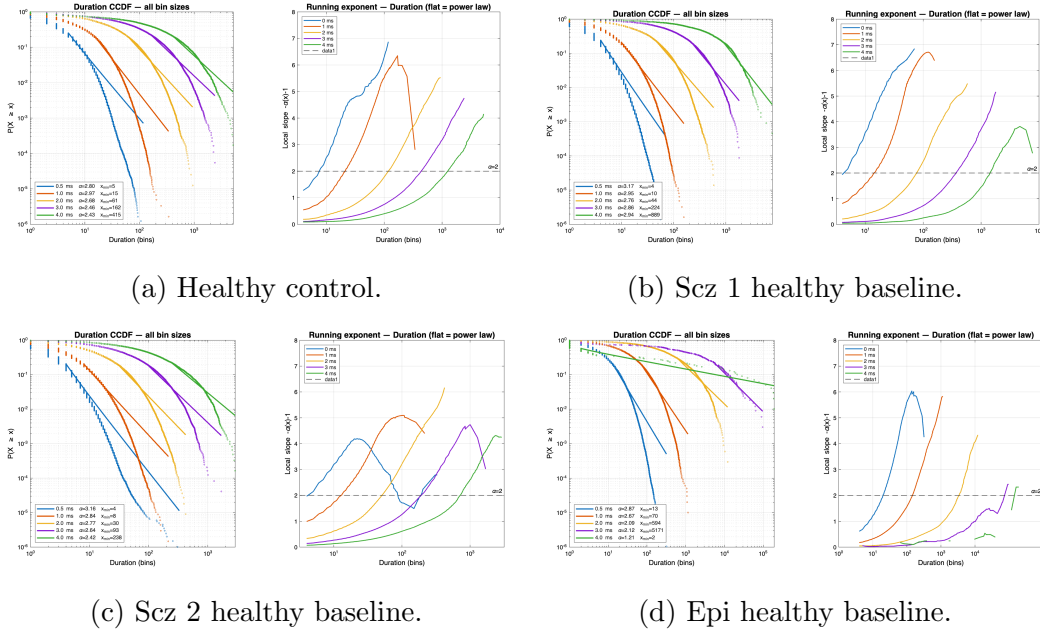


Figure 3.2: Avalanche duration distributions for a single representative healthy recording from each animal, computed on the whole hippocampal dataset.

The estimated exponents range from approximately 2 to 2.8 for avalanche size and from about 2.3 to 3 for duration (Table 6.1 and Fig. 3.3). These values do not match those originally reported by Beggs and Plenz [Beggs and Plenz, 2003], $\alpha \approx 1.5$ for size and $\beta \approx 2$ for duration. To address this discrepancy, it must be taken into account the big difference between the two datasets. Here the analysis is based on the action potentials of more than 400 neurons recorded in vivo, whereas Beggs and Plenz's work was investigating local field potentials in in vitro cortical slice cultures. In particular, it is interesting to note that, as the bin size varies, the curves and the values of the exponents change considerably. This highlights a

major limitation of neuronal avalanche analysis, which is the analysis being strongly dependent on the chosen bin size, which also undermines the reproducibility of the experiment (Fig. 3.4). In particular, the bin size that is usually chosen to carry out the analysis is the one closest to the mean ISI of the neuronal network. In this case, the entire analysis was performed using a bin size equal to 2 ms [Neto *et al.*, 2022].

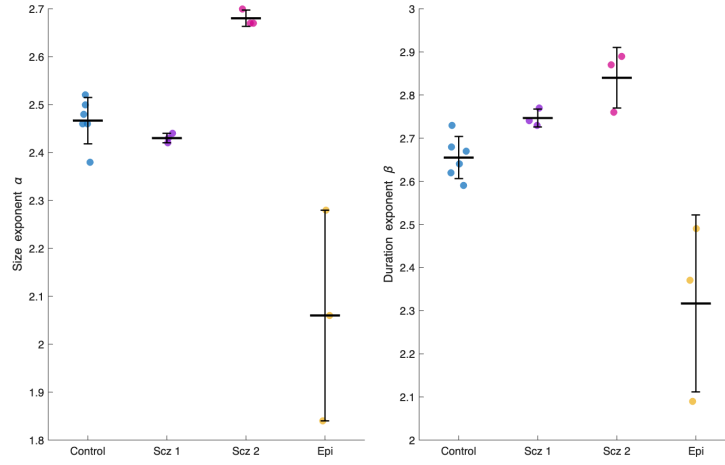


Figure 3.3: Power-law exponents across experimental groups at the 2 ms bin. Size exponent α (left) and duration exponent β (right) for each recording, grouped by animal (Control, Scz 1, Scz 2, Epi). Coloured dots are individual recordings; horizontal bars denote the group mean and whiskers the standard deviation.

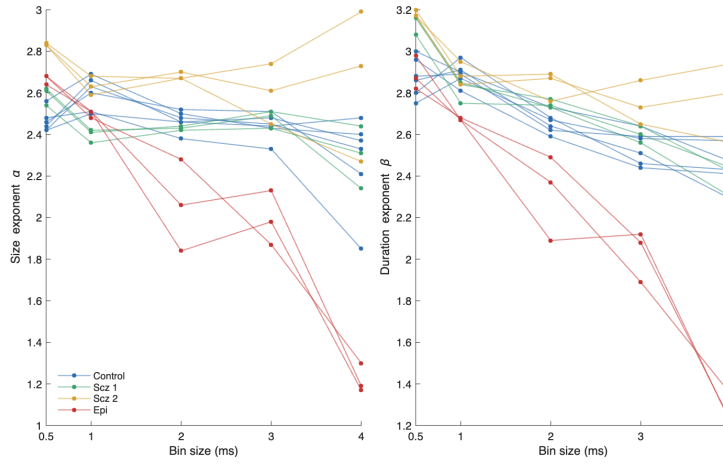


Figure 3.4: Dependence of power-law exponents on temporal bin size. Size exponent α (left) and duration exponent β (right) as a function of bin size (0.5, 1, 2, 3, 4 ms) for every recording, coloured by group (Control, Scz 1, Scz 2, Epi). Each line connects the exponents of a single recording across bins.

Null Models

To provide greater scientific rigour and rule out the possibility that the results are merely due to chance, it's important to analyse the correlation and autocorrelation null models as well. In Fig.3.5 and Fig.3.6, the CCDFs and running exponents for avalanche size and duration are shown. One feature that immediately stands out, particularly in the correlation null model, is that, compared to the curves obtained from the other “normal” models, these distributions are much more log-normal. This highlights the fact that the correlations between neurons have been disrupted, resulting in a distribution that resembles a Poisson-like distribution. In contrast, the avalanche size and duration distributions of the autocorrelation null model

are more similar to the ones of the "normal models", so since it shuffles the population-activity bins in time, keeping the overall activity level but removing any temporal structure, this shows that these distributions depend mostly on the overall activity level rather than on the precise temporal ordering of the bins[Priesemann *et al.*, 2014]. This can also be detected by inspecting Tables 6.4, 6.5 and Fig. 3.7, where the correlation null model returns substantially higher exponents, while the autocorrelation null model provides values close to those estimated from the recorded data.

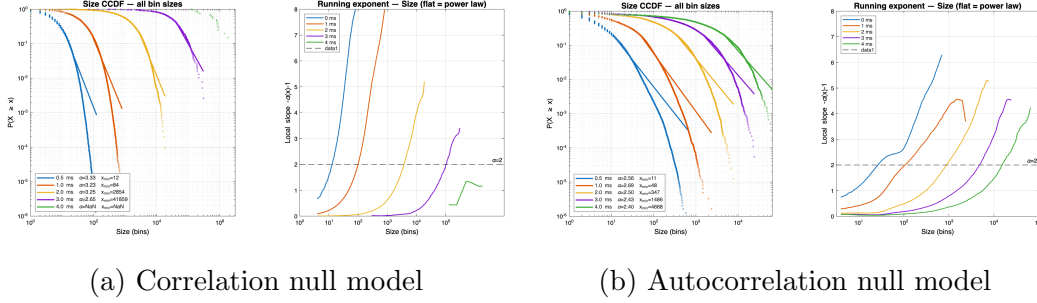


Figure 3.5: Correlation and autocorrelation null models for avalanche size, shown for a single representative healthy recording. CCDFs (left) and running exponents (right) are reported for (a) the correlation null model and (b) the autocorrelation null model.

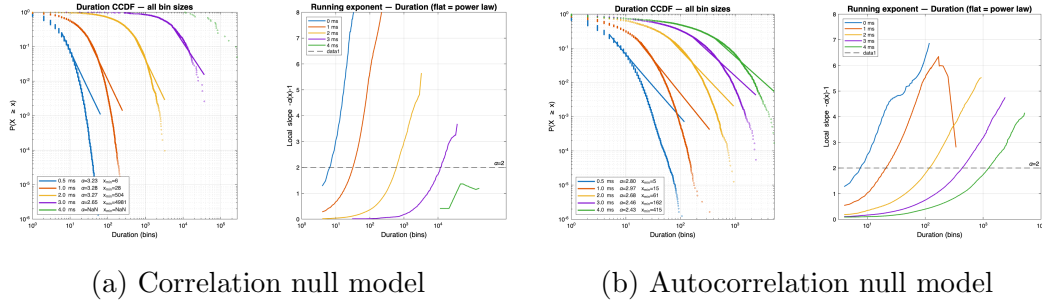


Fig.3.8, Fig.3.9, Fig.3.10 and Fig.3.11 show the complementary cumulative distribution functions (CCDF, left) together with the running exponent (right), for avalanche size and duration, respectively. One representative recording per animal is shown, with the dorsal and ventral hippocampus displayed separately for all mice. In addition, during the subdivision of the hippocampus into its ventral and dorsal parts, only some subregions were captured: dorsal Ca1 and ventral dentate gyrus for the healthy control, one schizophrenic mouse and the epileptic mouse, and dorsal and ventral dentate gyrus for the other schizophrenic mouse.

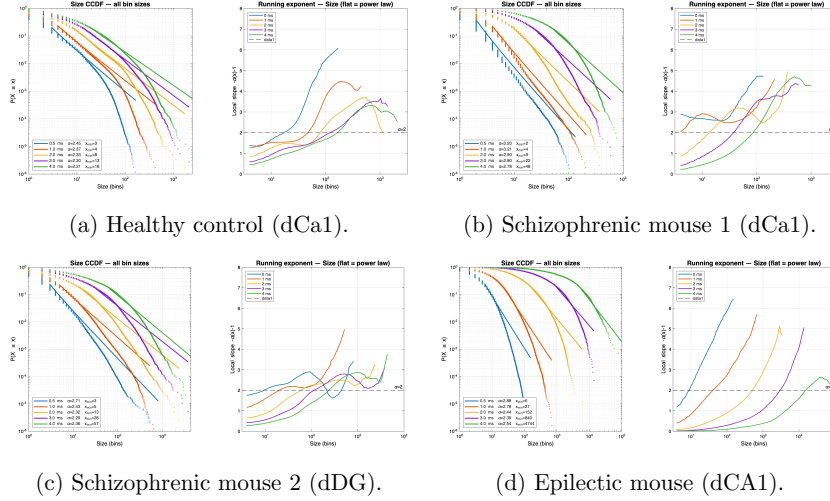


Figure 3.8: Avalanche size distributions (CCDFs and running exponents) for a single representative healthy recording from each animal, computed from the dorsal part of the hippocampus

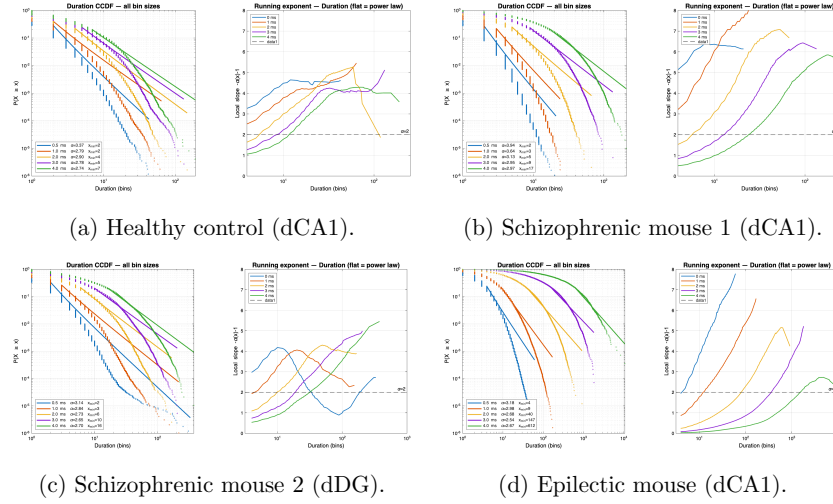


Figure 3.9: Avalanche duration distributions (CCDFs and running exponents) for a single representative healthy recording from each animal, computed from the dorsal part of the hippocampus

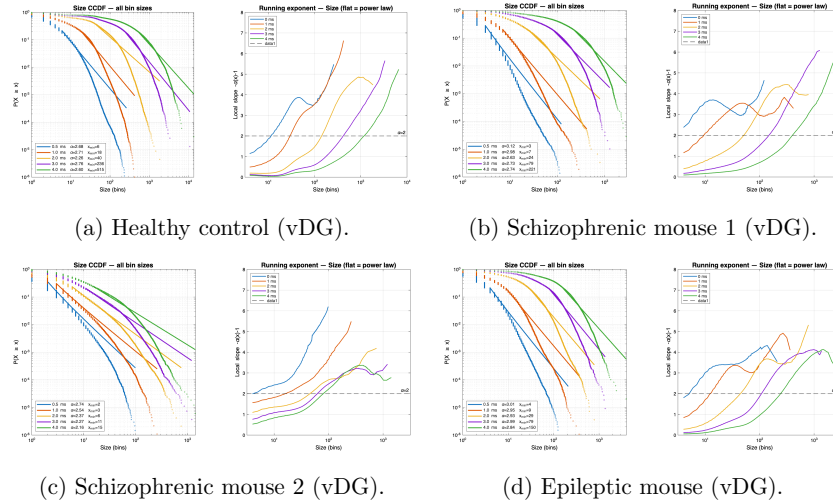
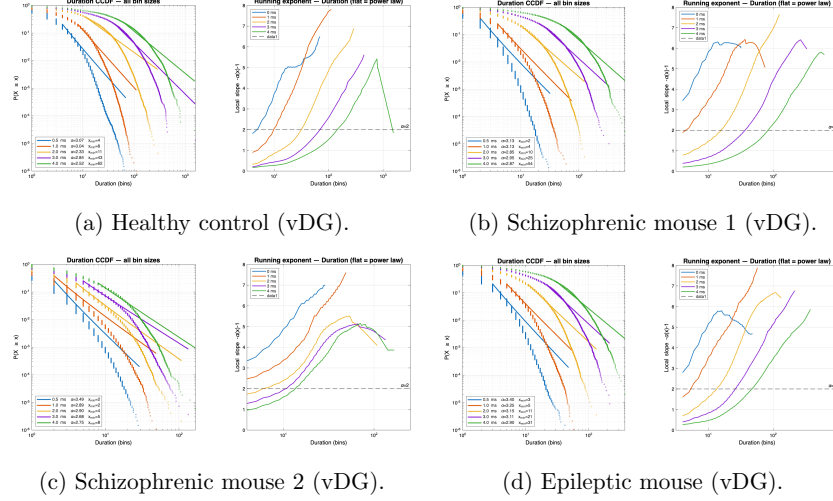


Figure 3.10: Avalanche size distributions (CCDFs and running exponents) for a single representative healthy recording from each animal, computed from the ventral part of the hippocampus



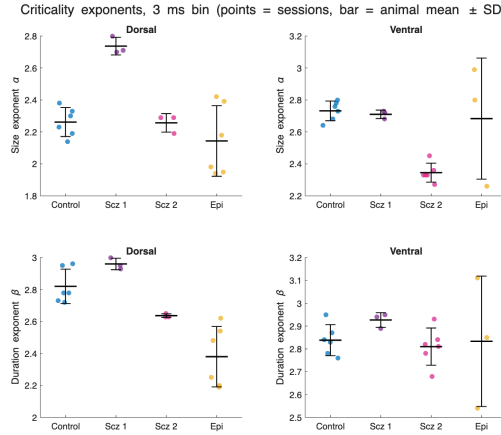


Figure 3.12: Criticality exponents by animal and hippocampal field at 3 ms. Size exponent α (top) and duration exponent β (bottom) for dorsal (left) and ventral (right) fields. Dots show individual sessions; bars show animal mean $\pm$ SD.

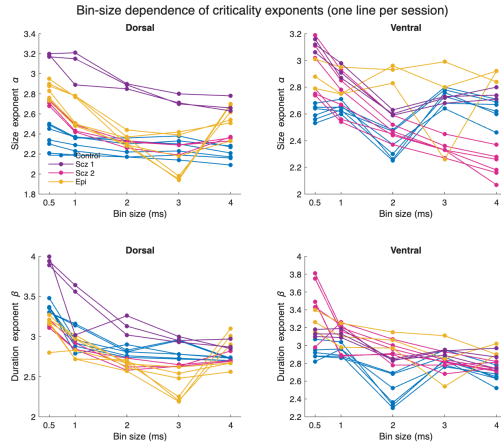


Figure 3.13: Bin-size dependence of criticality exponents by hippocampal field. Size exponent α (top) and duration exponent β (bottom) for dorsal (left) and ventral (right) fields. Lines show individual sessions, coloured by animal.

Figure 3.14 compares the dorsal and ventral estimates of the size (α) and duration (β) exponents at the 3 ms bin, averaged within each animal. Ventral estimates tend to be higher than dorsal estimates, with a mean dorsal/ventral difference of -0.27 for α and -0.15 for β . This pattern is observed in three of the four animals, while Scz 1 shows the opposite trend. A paired Wilcoxon signed-rank test at the animal level, which is a non-parametric test for paired samples that compares two measurements on the same unit using the signs and ranks of their differences, without assuming normality [Wilcoxon, 1945], is not significant ($p = 0.25$ for α , $p = 0.38$ for β), and significance cannot be reached with only $n = 4$ animals ($p_{\min} = 0.125$). The session-wise mixed model gives smaller p -values, but these may be affected by non-independent observations, so they are considered only as supporting evidence. Overall, the higher ventral exponents should be considered a descriptive trend that needs to be confirmed in a larger sample.

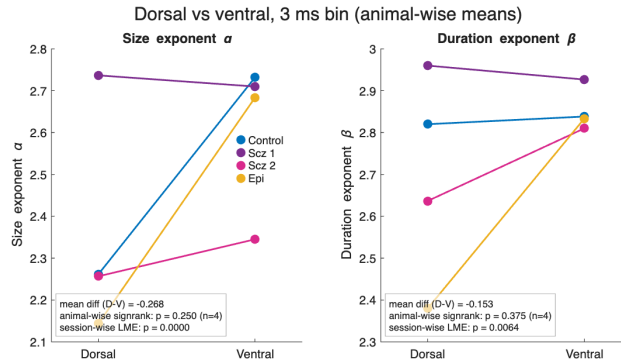


Figure 3.14: Dorsal vs ventral exponents at the 3 ms bin (animal-wise means). Size exponent α (left) and duration exponent β (right); each line is one animal, connecting its dorsal and ventral session-averaged mean.

Best-fit model classification

Now, it's possible to carry out the comparison between the lognormal, truncated power-law, power-law and exponential models, with the best fit selected via BIC and likelihood-ratio testing. From Fig.3.15, a clear predominance of truncated power-law and power-law fits over lognormal ones can be observed, especially in the case of duration. Nevertheless, it must still be noted that lognormal fits are present, particularly for size. One can therefore, because of the predominance of power law and truncated power law fits, argue that in a healthy brain avalanche size and duration distributions follow a power law, and that consequently a signature of criticality is preserved, but, at the same time, the presence of a significant number of lognormal fits, especially in the ventral subregion for the size, should not be overlooked.

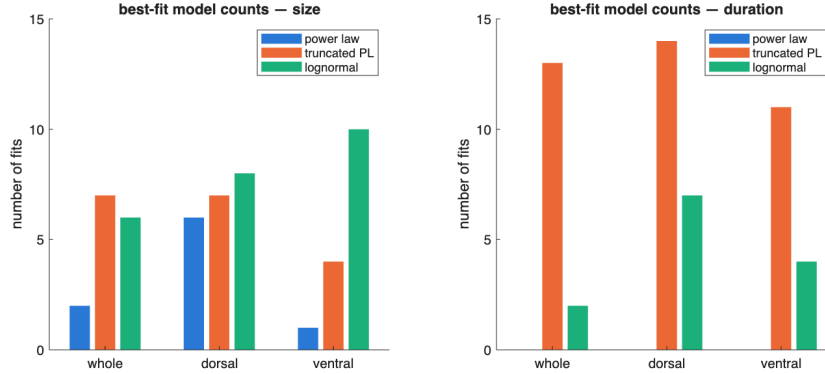


Figure 3.15: Best fit model counts for avalanche size (left) and duration (right) distributions across all healthy recordings. Each distribution was classified according to its best-fitting model, selected via BIC and likelihood-ratio testing. Counts are shown separately for the whole hippocampus, the dorsal and the ventral subregions.

Quantifying instead how lognormal the null-model distributions appear, especially when compared with the real recordings, Fig.3.16 shows a predominance of lognormal fits, particularly in the correlation null model, which destroys the correlations between the neurons of the network, confirming that neuronal correlations are necessary for avalanche dynamics.

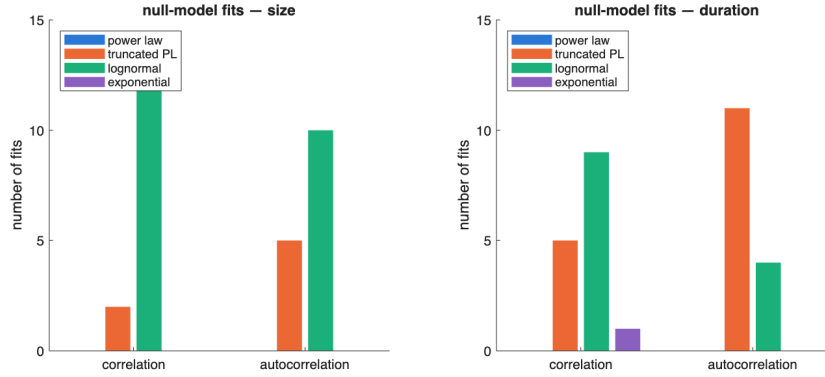


Figure 3.16: Best-fit model counts for avalanche size (left) and duration (right) distributions under the two null models, across all healthy recordings. Each distribution was classified according to its best-fitting model, selected via BIC and likelihood-ratio testing.

Figure 3.17 summarises the best-fit model classification for the whole-hippocampus recordings, combining size and duration. A power law or truncated power law was selected for 73% of the recorded distributions, compared with only 24% for the correlation null and 53% for the autocorrelation null. The difference between the real data and the correlation null is highly significant (chi-square test of proportions, $\chi^2(1) = 14.3$, $p = 2 \times 10^{-4}$), while the autocorrelation null does not differ significantly from the real data ($\chi^2(1) = 2.6$, $p = 0.11$). This is consistent

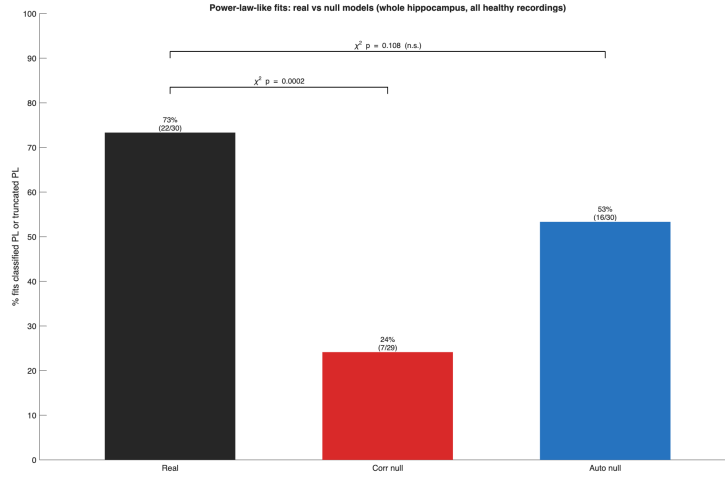


Figure 3.17: Percentage of avalanche distributions best fit by a power law or truncated power law (BIC with likelihood-ratio testing), pooled over size and duration across all healthy recordings.

with the exponent results. Removing inter-neuronal correlations shifts the avalanche distributions towards lognormal, non-power-law shapes, whereas shuffling the population activity while preserving its overall level leaves the classification largely unchanged. Together, these results provide further evidence that the recorded avalanche statistics are more power-law-like than the activity-matched controls, and that inter-neuronal correlations contribute to this pattern. However, because the test treats each fit as independent, these results should be considered as supporting evidence.

3.1.2 Branching parameter

The branching parameter did not provide a statistically meaningful criterion in this analysis. Both the real recordings and the two null models consistently produced values very close to one, with no clear difference between the real data, where critical dynamics may be present, and the surrogate data, where temporal correlations had been removed. Since a branching parameter close to one is expected for a critical system, obtaining similar values in the surrogate data suggests that the estimator was not effectively capturing the collective dynamics of the system. This result can be explained by a known limitation of branching-ratio estimation in spatially subsampled recordings. The branching parameter represents the average number of events generated by one preceding event, and a value of one is expected at criticality when the whole system is observed. However, electrophysiological recordings capture only a very small fraction of the neurons in the underlying network. Even when hundreds of neurons are recorded simultaneously, as in this research, they represent only a small fraction of the total number of neurons in the same brain region. As a result, many of the neurons involved in the propagation of an avalanche are not observed. This causes avalanches to appear artificially truncated and can bias the estimated branching parameter toward one, even when the underlying system is not critical [Priesemann *et al.*, 2009]. The same effect is present in the surrogate data, which can explain why the null models also produce values close to one. The branching parameter is also strongly affected by the temporal bin size used to define avalanches. Since different bin sizes were considered in this analysis, small changes in the bin width

can lead to changes in the estimated branching parameter, even when the underlying dynamics remain unchanged [Levina and Priesemann, 2017]. Figure 3.18 shows the branching parameter m for every healthy recording across the four animals and the two null models. All values are within 0.058 of unity, indicating that all conditions are effectively near-critical ($m \approx 1$). Differences between the four animals were tested using a Kruskal–Wallis test, a non-parametric test that compares three or more independent groups without assuming normally distributed data [Kruskal and Wallis, 1952]. The test is significant ($p = 0.016$), mainly because the Epi values are slightly lower (≈ 1.009) and the correlation null values are grouped slightly above 1. However, these differences are very small and do not distinguish between critical and non-critical dynamics. The correlation null provides a clear example of this limitation. Removing correlations between neurons should disrupt coordinated avalanche propagation, yet it still gives $m \approx 1.005$, which would suggest near-critical dynamics if m were considered alone. Since the correlation null removes inter-neuronal correlations by design, its near-unity value shows that the estimator can return $m \approx 1$ even when the underlying dynamics are not expected to be critical. Importantly, the recorded values also almost completely overlap with those of the autocorrelation null. Therefore, m does not clearly distinguish the animals, the conditions, or the real data from the activity-matched shuffles. Overall, the fact that the branching parameter remained close to one in all conditions, both in the real recordings and in the null models, suggests that it cannot be considered a reliable measure of criticality for the heavily subsampled populations analysed here.

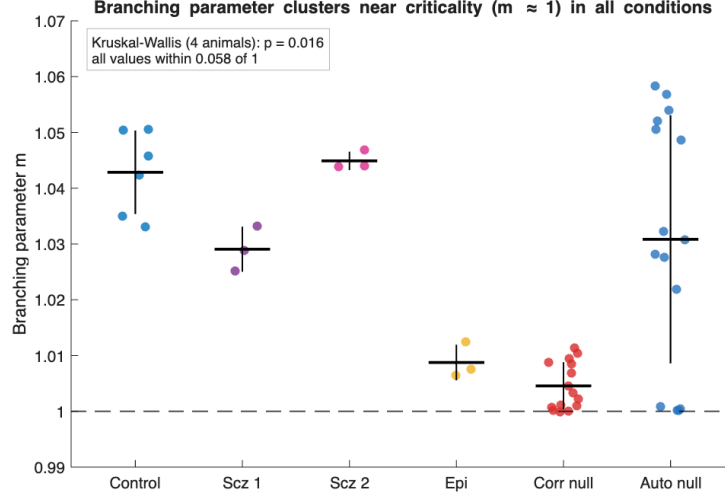


Figure 3.18: Branching parameter m per healthy recording for each animal and null model. All conditions are near-critical ($m \approx 1$, within 0.058 of unity); bars denote mean $\pm$ SD.

3.1.3 DCC and Shape Collapse

The presence of power-law distributions is a necessary, but not sufficient, condition for criticality. In fact, at a critical point, the size and duration must satisfy the crackling noise scaling relation:

$$\gamma_{\text{expected}} = \frac{\beta - 1}{\alpha - 1}, \quad (3.1)$$

where α and β are the exponents of the avalanche size and duration distributions, respectively. In addition, γ , also known as scaling parameter, also describes how the mean avalanche size scales with its duration:

$$\langle S(T) \rangle \propto T^\gamma. \quad (3.2)$$

To test this analysis, two complementary analyses were used: the DCC (Deviation from Criticality Coefficient) which measures the difference between the value of γ predicted from the fitted distributions and the value directly obtained from the relationship between avalanche size and duration, and the shape collapse, which instead tests whether avalanches with different durations follow a similar temporal profile when appropriately rescaled. In order to be at criticality, the DCC must stay below 0.2 [Ma *et al.*, 2019]. Across the healthy recordings, the DCC showed a clear U-shaped dependence on bin size.

Whole hippocampus

As shown in Fig.3.19, Fig. 3.20 and Table 6.6, it remained low between 0.5 and 2 ms, reaching its minimum around 2 ms, then increased rapidly for larger bin sizes. Two of the three Epi recordings are an exception, with the DCC already high at 2 ms (1.03 and 1.78). This animal has a lower ISI and therefore fires more densely. At 2 ms, separate avalanches may merge into single events, affecting the fits. In fact, these sessions are close to criticality at 0.5–1 ms ($\text{DCC} \leq 0.02$), therefore, their deviation at ms likely reflects a bin that is too large rather than a genuine loss of criticality. The presence of a bin size at which the DCC is minimised suggests that, at the appropriate temporal scale, the independently estimated size and duration exponents are consistent with each other and satisfy the crackling-noise scaling relation.

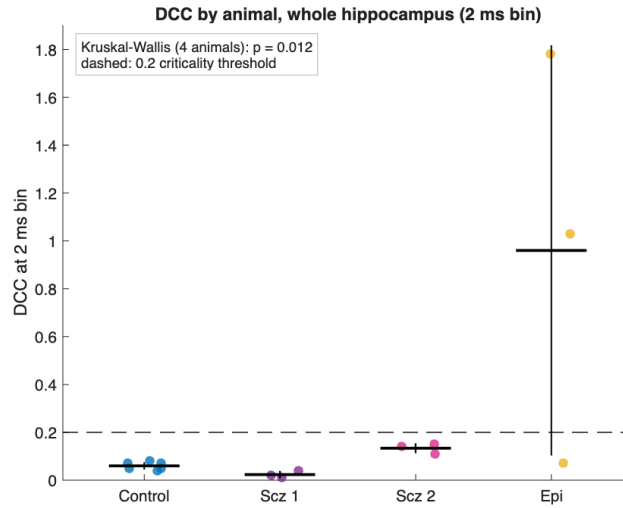


Figure 3.19: DCC at the 2 ms bin for the whole hippocampus, per healthy recording, grouped by animal; bars denote mean $\pm$ SD.

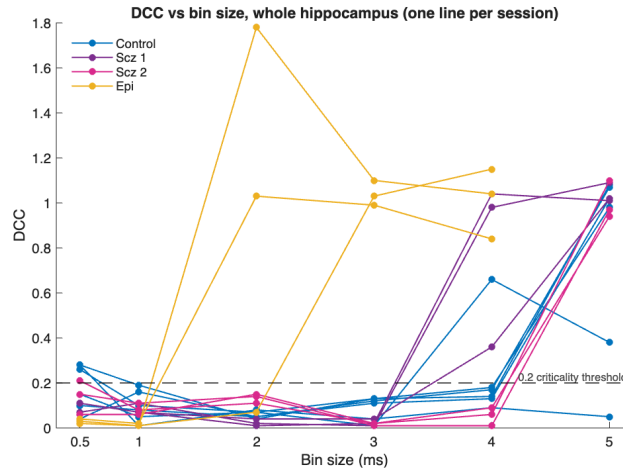


Figure 3.20: DCC as a function of temporal bin size for the whole hippocampus, one line per healthy recording, coloured by animal.

Looking at Fig. 3.21, which shows the DCC values for the null models and the recordings at a 2 ms bin, both null models and the real dataset show

very low DCC values, often below 0.02 and sometimes close to zero. This happens even though the surrogate data have lost their original spatiotemporal structure. Both the correlation null and the autocorrelation null satisfy the crackling-noise scaling relation at the optimal bin size, similarly to the real data. Therefore, the DCC should not be considered a stand-alone criterion, but rather as part of a combined test with avalanche shape collapse. The DCC checks the consistency of the scaling exponents, while shape collapse tests whether the full avalanche profiles follow a common universal shape. Therefore, only when both tests are satisfied can they provide stronger evidence for critical dynamics [Touboul *et al.*, 2017]. In any case, the high dependence on the bin size of the DCC can still be observed in the null models (Fig. 3.22 and Table 6.7).

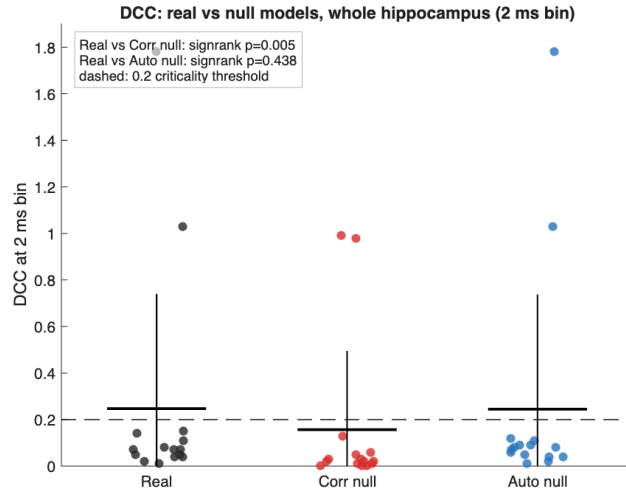


Figure 3.21: DCC at the 2 ms bin for the whole hippocampus in the recorded data and the two null models; each point is a session, bars denote mean $\pm$ SD.

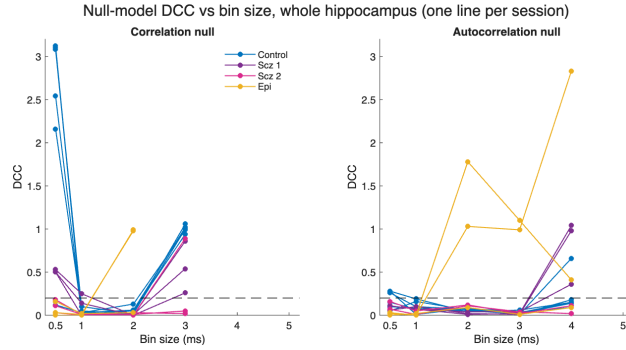


Figure 3.22: DCC as a function of temporal bin size for the two null models (whole hippocampus), one line per healthy recording, coloured by animal.

Dorsal and Ventral hippocampus

Calculating also the DCC separately for the dorsal and ventral subregions, not all subregions were present in every animal because of differences in probe placement. Overall, the results show a clear difference between dorsal and ventral regions in how the DCC changes with bin size. As shown in Table 6.8 and Fig. 3.24, in dorsal CA1 the DCC is generally high at the smallest bin size, it then decreases and falls below the 0.2 criticality threshold around 2-3 ms, before increasing at larger bin sizes. In contrast, the ventral dentate gyrus remains mostly below the threshold across different bin sizes. The dorsal dentate gyrus shows an intermediate behaviour, with relatively low DCC values across most bin sizes but without the clear U-shaped pattern observed in dCA1. These results are still suggesting that, even in the healthy brain, the agreement with the crackling-noise scaling relation depends strongly on the temporal scale used to define avalanches. However, these results should be interpreted with caution. In fact, the different subregions contain different numbers of

neurons and have different firing properties. Since the DCC depends on the temporal scale used to define avalanches, the narrow window observed in dCA1 could simply reflect the scale at which the relatively sparse and temporally precise CA1 activity is best captured, rather than a true difference in criticality between regions.

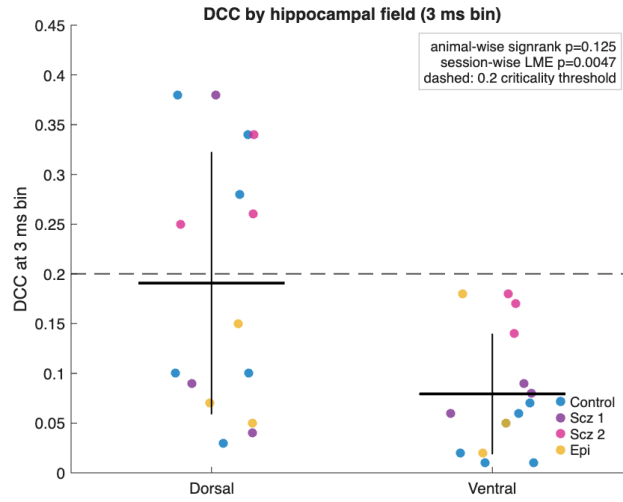


Figure 3.23: DCC at the 3 ms bin for the dorsal and ventral hippocampal fields; each point is a session, coloured by animal, bars denote mean $\pm$ SD.

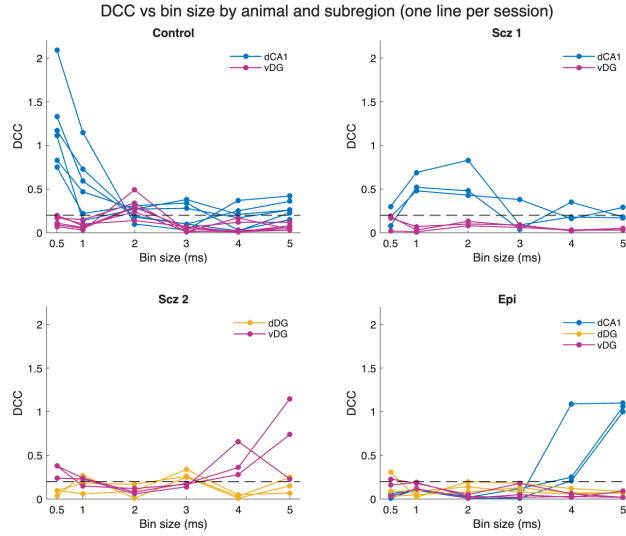
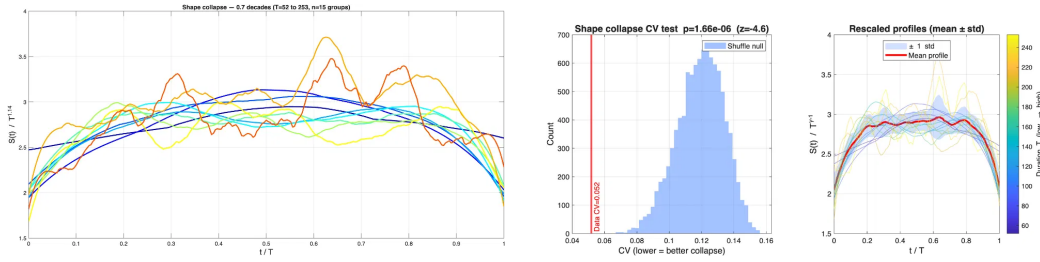


Figure 3.24: DCC versus temporal bin size, one panel per animal, one line per session, coloured by subregion (dCA1, dDG, vDG).

As mentioned above, the DCC alone is not sufficient to demonstrate criticality, so it's important also to analyse the avalanche shape collapse. Unlike the DCC, which compares a few scalar exponents, shape collapse provides a more rigorous test, in fact it checks whether the average temporal profiles of avalanches with different durations, after rescaling by their duration and peak amplitude, collapse onto the same universal curve. As stated before, the rescaling is made by the exponent γ , with the time axis of each profile normalised by its duration T , and its amplitude is rescaled by $T^{\gamma-1}$, where $\gamma = (\alpha - 1)/(\beta - 1)$ through $\langle S \rangle \sim T^\gamma$ and that underlies the DCC. Fig 3.25 shows an example of the avalanche shape collapse obtained for the healthy recordings and illustrate the form that it should take to represent a signature of criticality. It can be seen that the rescaled temporal profiles of avalanches of different durations collapse onto

an approximate symmetric parabolic curve. This universal shape is a distinguishing feature of criticality, the more accurately the profile overlap, the stronger the support for criticality. Despite the individual profiles are still noisy, especially for the shortest durations where fewer avalanches are present, the mean profile is well defined and clearly parabolic. Importantly, the quality of the collapse is not due to chance, with a coefficient of variation of the rescaled profiles ($CV = 0.052$) much lower than the shuffled null distribution ($z = -4.6, p = 1.66 \times 10^{-6}$). This confirms that avalanches with different durations share a common rescaled shape. Together with the estimated exponent $\gamma \approx 1.14$, these results provide a significant evidence of criticality.



(a) Rescaled avalanche profiles for durations $T = 52$ – 253 bins (15 groups), with amplitude scaled by $T^{\gamma-1}$ ($\gamma = 1.14$). Colour encodes duration.

(b) Left: collapse quality (Coefficient of variation CV) compared to a shuffled null; the data ($CV = 0.052$) collapse significantly better than chance ($p = 1.7 \times 10^{-6}$). Right: rescaled profiles with the mean shape (red) and ± 1 std band.

Figure 3.25: Avalanche shape collapse for the healthy hippocampus. Profiles of avalanches of different durations rescaled onto a common shape.

Table 6.9 and Fig. 3.26 reveal that, for the whole hippocampus, all healthy recordings show a highly significant avalanche shape collapse compared with the shuffled null model (all $p < 10^{-3}$, with most values around $p \sim 10^{-6}$ – 10^{-8}). This confirms that avalanches with different durations share a common rescaled temporal profile. The collapse exponent γ is very stable across the fifteen recordings, ranging from 1.06 to 1.14 (mean ≈ 2.10), with no clear differences between the four animals. The dynamic range of the collapse is relatively narrow, between 0.6 and 1.0 decades, but this is mainly due to spatial subsampling, which limits the detection of the longest avalanches [Srinivasan *et al.*, 2024].

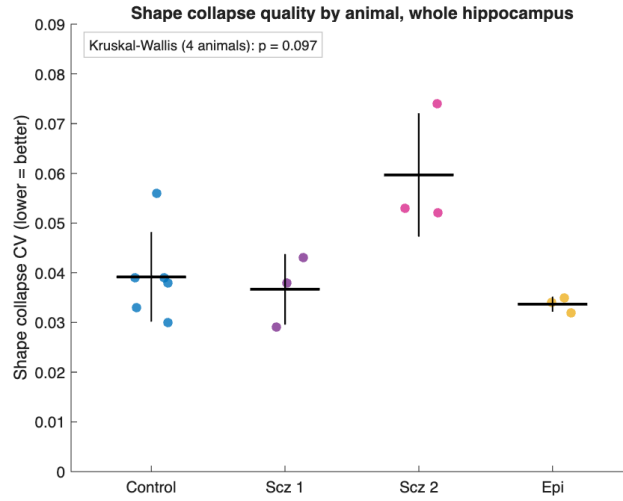


Figure 3.26: Shape collapse quality (CV of the rescaled profiles, lower = better collapse) per healthy recording, grouped by animal, whole hippocampus; bars denote mean $\pm$ SD.

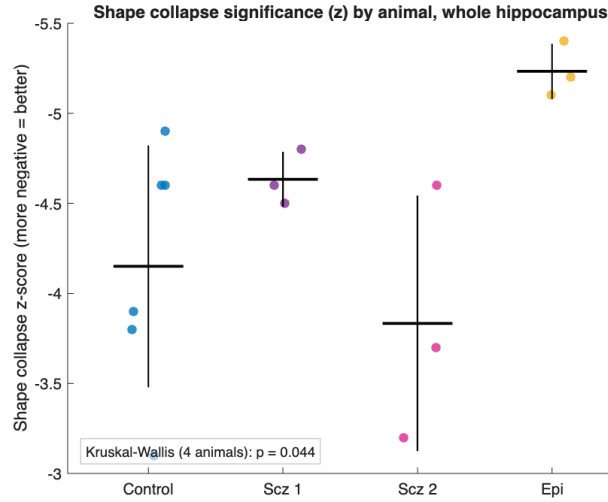


Figure 3.27: Shape collapse significance (z-score of the data CV against a shuffled null) per healthy recording, grouped by animal, whole hippocampus; bars denote mean $\pm$ SD. More negative z indicates a stronger, more significant collapse.

Null Models

Table 6.10 and Fig 3.28 show the shape-collapse analysis for the two null models. Comparing these results with the animal models (Table 6.9), it is evident that neither null model shows a clear shape collapse, with the rescaled profiles losing the parabolic shape observed in the real recordings and becoming flat and featureless. The two null models behave in different ways. The autocorrelation null preserves each neuron's firing rate and internal temporal structure, but it does not produce a collapse better than chance. In all recordings, the p-values are well above the significance level ($p \sim 0.7$, with $z > 0$). This shows that, when the temporal alignment between neurons is removed, the population activity no longer has a

common avalanche shape. The correlation null gives a more complex result. In some recordings, the CV is very low, and the p-value is significant, however, this does not indicate a real shape collapse. The low CV is caused by the rescaled profiles being almost completely flat, meaning that they overlap simply because they have no clear structure. Overall, the null-model results show that the avalanche shape collapse observed in the hippocampus disappears when neuronal correlations are removed and cannot be reproduced by either surrogate. This supports the conclusions from the avalanche distributions and the DCC, stating that the critical dynamics observed in the healthy hippocampus probably depend on interactions between neurons [Morales *et al.*, 2023].

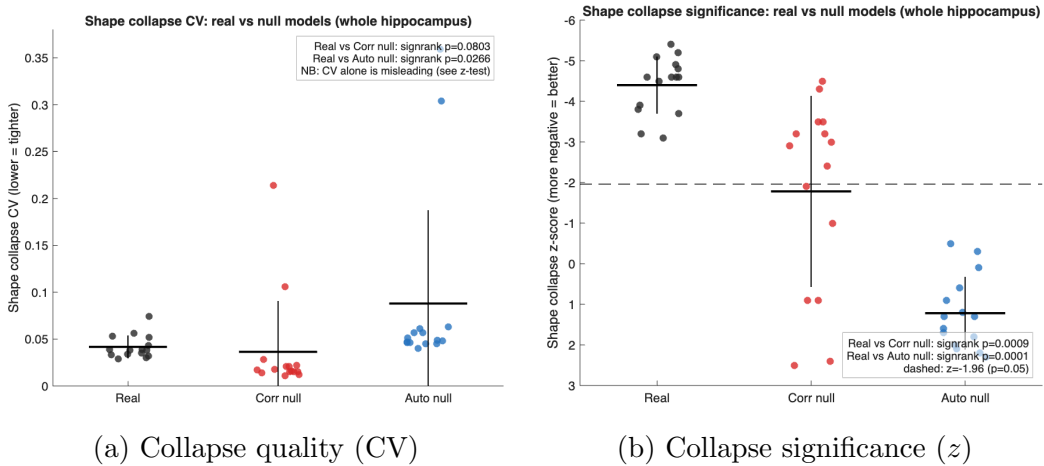
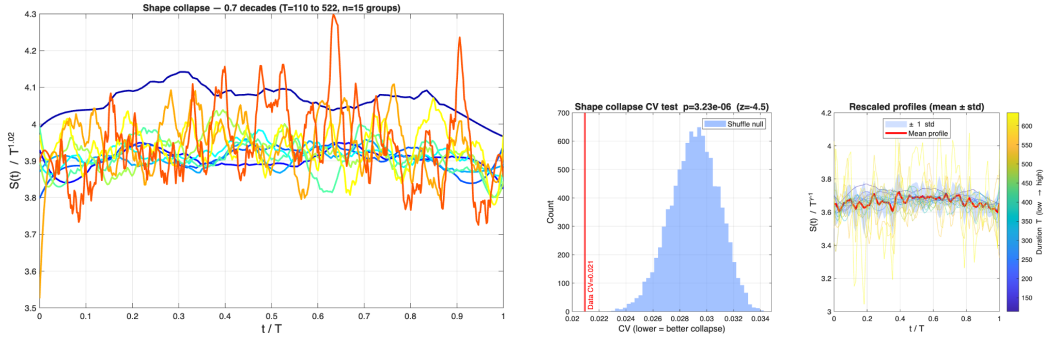
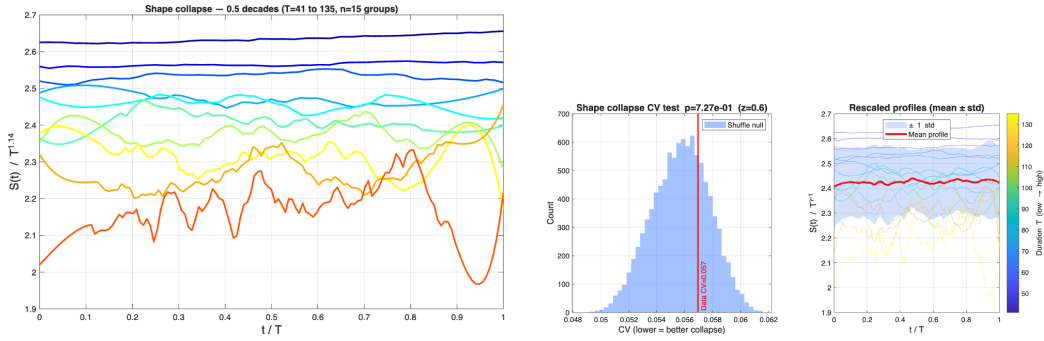


Figure 3.28: Avalanche shape collapse, real vs the two null models (whole hippocampus; one point per healthy recording, bars mean $\pm$ SD). (a) CV (lower = tighter): misleading, lower for the smoother correlation null. (b) Significance z : the data collapse far better than both nulls (signrank $p = 9 \times 10^{-4}$ vs corr, 1×10^{-4} vs auto).



(a) Correlation null model - Rescaled profiles

(b) Correlation null model - CV test



(c) Autocorrelation null model - Rescaled profiles

(d) Autocorrelation null model - CV test

Figure 3.29: Avalanche shape collapse under the two null models (representative healthy recording, whole hippocampus).

Table 6.11 and Fig. 3.30 extend the shape collapse analysis to the individual hippocampal subregions and show a clear difference along the dorsoventral axis. The ventral dentate gyrus (vDG) shows consistent results across all four animals, with a low and stable rescaling exponent ($\gamma \approx 1.03$ -1.08), and the collapses that cover a wide range of durations (up to $T \sim 400$ –560 bins), also being almost always highly significant (p as low

as 10^{-7}). These results are very similar to those obtained for the whole hippocampus, suggesting that the ventral region is a big contributor to the overall collapse. The dorsal CA1 (dCA1) shows a different pattern. Its rescaling exponent is generally higher ($\gamma \approx 1.1$ -1.7), and the collapse is limited to a narrow range of short durations ($T \approx 20$ –75 bins). This may be related to the sparse and temporally precise firing in this region under spatial subsampling. As a result, the quality of the collapse is lower (higher CV). To interpret the results, it is important to consider that all subregional collapses are limited by the short dynamic range available under subsampling, especially in dorsal CA1, where the T range covers less than half a decade. Therefore, the estimated exponents should be considered indicative rather than precise. Also, the sampled subregions are different across animals (dCA1 and vDG in Control and Scz 1, vCA3, vDG, and dDG in Scz 2, all three dorsal and ventral regions in Epi), making a balanced comparison between animals difficult. Despite these limitations, the analysis shows that a significant avalanche shape collapse can be observed in every subregion with a sufficient number of avalanches, with the most robust results found in the ventral dentate gyrus.

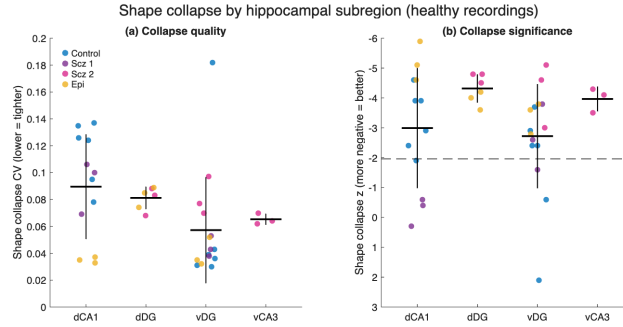


Figure 3.30: Avalanche shape collapse by hippocampal subregion (healthy recordings; one point per session, coloured by animal, bars mean $\pm$ SD). (a) Collapse quality (CV, lower = tighter). (b) Collapse significance (z vs a shuffled null; more negative = better, dashed $z = -1.96$). dCA1 has the loosest and most variable collapse (highest CV, most heterogeneous z), while dDG, vDG and vCA3 collapse more tightly and consistently.

3.2 Comparison with Pathological Brain States

Having shown that the healthy hippocampus exhibits the main features of critical avalanche dynamics, heavy-tailed size and duration distributions, consistent scaling exponents, and a significant avalanche shape collapse that disappears when neuronal correlations are removed, it is crucial now to investigate whether these features change in pathological brain states. The critical-brain hypothesis suggests that healthy neural circuits are regulated to operate close to a critical point, where information transmission, dynamic range, and computational capacity are optimised [Shew and Plenz,

2012]. Therefore, changes in the balance between excitation and inhibition may lead to a shift away from criticality. Excessive excitation or hypersynchrony may push the network towards a supercritical state, while excessive inhibition may result in a subcritical state [Shew *et al.*, 2011].

3.2.1 Schizophrenia

Schizophrenia has been associated with changes in the balance between excitation and inhibition and with reduced long-range neural synchronisation. At the cellular level, this imbalance has been linked to reduced NMDA receptor function and to a loss of parvalbumin-positive GABAergic interneurons. These changes reduce inhibition of pyramidal cells and disrupt the gamma-band synchrony normally supported by these interneurons [Lewis *et al.*, 2005]. Within the critical-brain framework, this combination of increased excitation and reduced neuronal coordination could move the network away from the critical state, without completely disrupting organised activity [Alamian *et al.*, 2022]. The same criticality analysis pipeline used for the healthy hippocampus was applied to the schizophrenia recordings. In particular, for both the two schizophrenia animal models 6 tracks are available, where 3 correspond to the time when the mice received the ketamine injection, 3 coincide to the onset of the disease, and an additional 3, only for one of the mice, were recorded during the chronic stage of schizophrenia.

Recordings were collected while the animals moved along a linear track to receive a sucrose-water reward. Three sessions were recorded each week, on Tuesday, Wednesday, and Thursday, with one recording per session.

Therefore, every three consecutive linear tracks correspond to one week. The twelve linear tracks represent four successive conditions in the same animals: linear tracks 1 to 3 are the healthy baseline, recorded before any intervention; linear tracks 4 to 6 were recorded after an acute ketamine injection, linear tracks 7 to 9 correspond to the acute stage of the schizophrenia model and linear tracks 10 to 12, available only for Scz 1, correspond to the chronic stage. Since only two animals were available, comparisons between conditions are based on sessions rather than animals. These results should therefore be considered preliminary.

Power-law

Whole Hippocampus

The analysis started with the avalanche size and duration distributions, which were fitted with the same four models: power law, truncated power law, lognormal, and exponential. The best-fitting model was selected for each distribution, following exactly the same procedure used for the healthy recordings. Fig. 3.31 shows representative avalanche size and duration distributions after ketamine administration, while the exponents for all tracks are reported in Table 6.12.

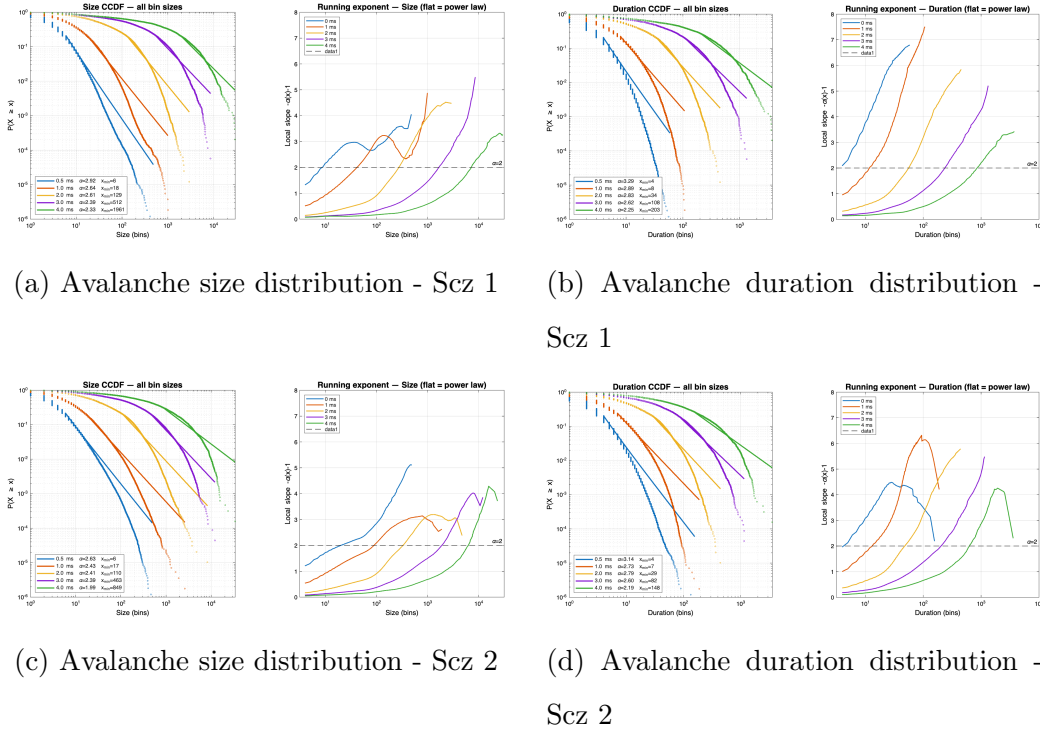
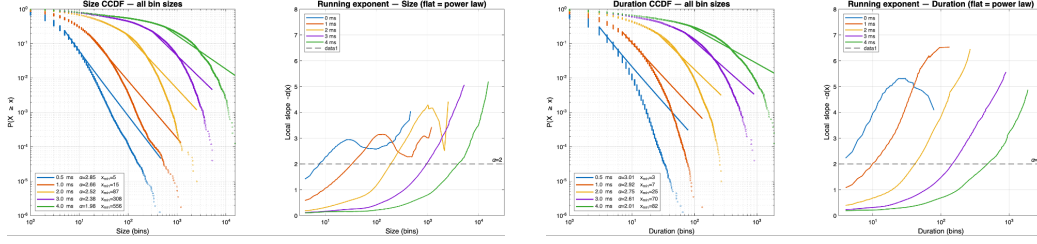
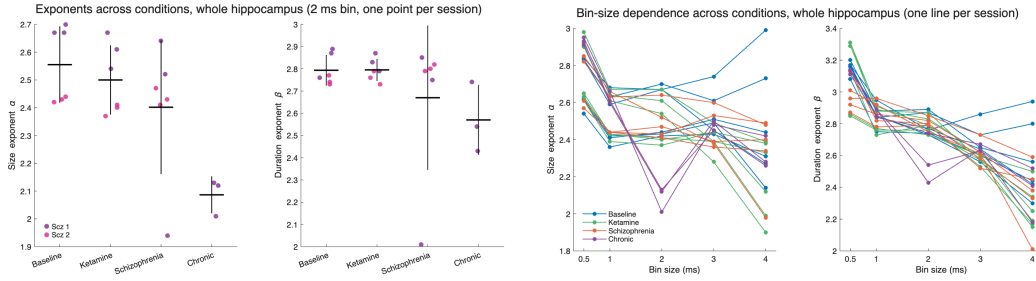


Figure 3.31: Avalanche size and duration distributions under the ketamine condition (whole hippocampus), for a representative track of each animal. Left: complementary cumulative size distributions; right: complementary cumulative duration distributions.

Figure 3.33a summarises the ketamine injection and acute and chronic schizophrenia conditions, with the chronic condition available only for one animal. By eye, these distributions look very similar to those observed in the healthy condition. The CCDFs remain heavy-tailed across all bin sizes, while the running exponents slowly increase towards the finite-size cutoff. The fitted exponents also remain within a similar range across conditions, with $\alpha \approx 2.0-3.0$ for avalanche size and $\beta \approx 2.2-3.1$ for duration. As in the

healthy recordings, the exponents generally decrease as the bin size increases. One recording, Scz 1 track 7, shows unusually low exponents due to unstable fits at the largest bin sizes and is therefore not considered further.





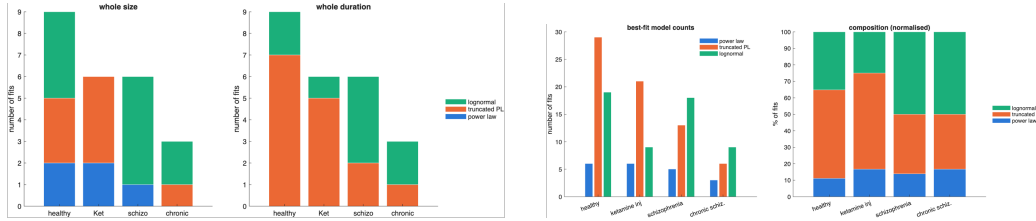
(a) Exponents at the 2 ms bin, one point per session, coloured by animal (bars mean $\pm$ SD). (b) Bin-size dependence, one line per session, coloured by condition.

Figure 3.33: Avalanche size (α) and duration (β) exponents across the four conditions (whole hippocampus, Scz 1 and Scz 2).

This similarity is only apparent. The difference becomes clear when we look at the best-fitting model rather than only at the exponent values (Fig. 3.34). Both schizophrenia conditions show a shift from power-law and truncated power-law fits towards lognormal fits. This suggests a move away from scale-free dynamics, which is not observed in the ketamine condition. Therefore, the departure from criticality is more evident in the shape of the distributions than in the value of a single exponent.

Figure ?? shows the proportion of whole-hippocampus fits best described by a lognormal model across the four conditions. Lognormal fits are relatively uncommon in the healthy baseline (33%, 6/18) and even less common after ketamine (8%, 1/12). In contrast, they become much more common in the schizophrenia condition (75%, 9/12) and remain frequent in the chronic stage (67%, 4/6). This is consistent with the decrease in power-law-like fits described above and suggests a shift away from scale-free avalanche

statistics in the schizophrenia model. Ketamine shows the opposite pattern, with the distributions tending, if anything, towards power-law behaviour. A chi-square test of proportions supports this pattern. The healthy versus schizophrenia comparison shows a strong trend ($p \approx 0.06$), becomes significant when the two schizophrenia stages are combined ($p = 0.045$), and the ketamine-versus-schizophrenia comparison is clearly significant ($p = 0.003$). Since only two animals are available, these tests are based on individual sessions and should therefore be considered preliminary.



(a) Number of best-fitting models for avalanche size (left) and duration (right) across the four conditions. Each bar shows the number of recordings for which each model provided the best fit. (b) Number of best-fitting models for avalanche size and duration combined (left) and their normalised composition (right), expressed as the percentage of fits corresponding to each model.

Figure 3.34: Comparison of the best-fitting models for avalanche size and duration distributions across healthy, ketamine, schizophrenia, and chronic schizophrenia conditions. The figure highlights the shift from power-law and truncated power-law fits towards lognormal fits in the schizophrenia conditions.

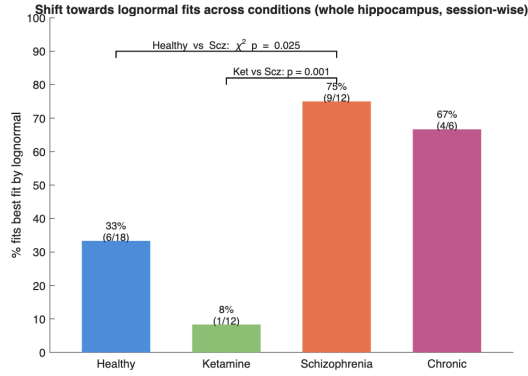


Figure 3.35: Proportion of whole-hippocampus fits best described by a lognormal model across conditions (session-wise, size and duration pooled; counts shown on each bar).

Dorsal and Ventral Subregions

The analysis is now extended from the whole hippocampus to the individual subregions, with size and duration exponents estimated separately for each sampled dorsal and ventral field.

Figure 3.36 summarises the size (α) and duration (β) exponents for each hippocampal subregion across all conditions. Figure 3.37 shows how these exponents change with bin size.

Under ketamine (Table 6.15), the distributions remain heavy-tailed across both animals and all sampled regions. The exponents decrease as the bin size increases (Fig. 3.37), following the same pattern seen in the healthy hippocampus. No subregion shows a clear loss of avalanche activity across different timescales after ketamine injection.

There is, however, a small but consistent difference between regions. In Scz 1, dorsal CA1 has the highest exponents, especially for duration at the

smallest bin size ($\beta \approx 4.0\text{--}4.2$ at 0.5 ms). It also has very small cutoffs. This may be related to the sparse and temporally precise firing in this region. In contrast, the ventral dentate gyrus has lower exponents and a wider dynamic range [Strange *et al.*, 2014]. The higher exponents in dorsal CA1 and lower exponents in the dentate and ventral regions are also consistent with known differences along the hippocampal longitudinal axis, including differences in excitability, firing activity, and place-field size [Fanselow and Dong, 2010]. These differences may therefore reflect the properties of the different hippocampal regions rather than a specific effect of ketamine. Overall, the subregional exponents under ketamine are similar to those observed in the healthy recordings and, like the whole-hippocampus exponents, do not provide clear evidence of a departure from criticality. In the acute schizophrenia condition (Table 6.16), most fields remain within the healthy range ($\alpha \approx 2.2\text{--}3.4$ for size and $\beta \approx 2.5\text{--}4.3$ for duration). The exponents also show the same decrease with increasing bin size. The main exception is Scz 1 track 7, where the ventral dentate gyrus shows very low exponents at larger bin sizes ($\alpha = 1.30$, $\beta = 1.85$), together with degenerate cutoffs. This produces the low and non-monotonic trajectory seen in Figure 3.37 and is similar to the anomaly already observed for this recording at the whole-hippocampus level. In tracks 8 and 9, the exponents return to the healthy range. In Scz 2, no similar deviation is observed, with the vCA3, vDG and dDG fields remaining relatively stable across all three tracks (Fig. 3.36).

In the chronic condition (Table 6.17), dorsal CA1 and the ventral dentate gyrus show similar exponents across all three tracks. For dCA1,

$\alpha \approx 2.6\text{--}3.3$ and $\beta \approx 2.9\text{--}3.4$, while for vDG, $\alpha \approx 2.4\text{--}3.2$ and $\beta \approx 2.5\text{--}3.2$.

Both regions show the expected decrease with increasing bin size, with no clear anomalous field. These ranges are similar to those observed in the same animal under healthy, ketamine and acute schizophrenia conditions (Fig. 3.36). This suggests that there is no clear region-specific change in the fitted exponents during the chronic stage.

Overall, the subregional exponents alone do not show a consistent departure from criticality in either the acute or chronic schizophrenia conditions.

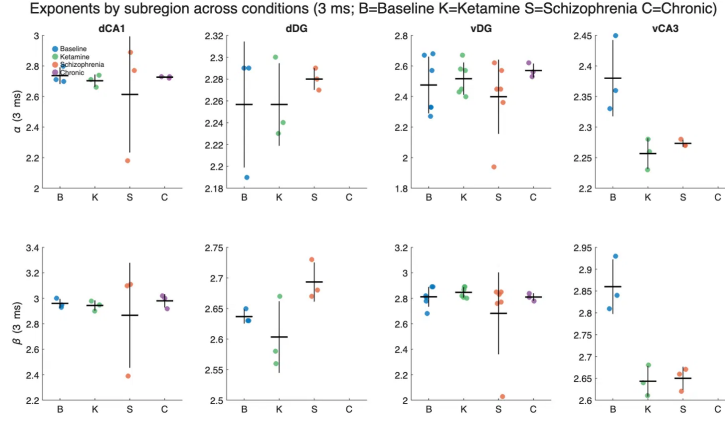


Figure 3.36: Size (α , top) and duration (β , bottom) exponents at the 3 ms bin by hippocampal subregion (dCA1, dDG, vDG, vCA3) across conditions (B = baseline, K = ketamine, S = schizophrenia, C = chronic; one point per session, coloured by condition, bars mean $\pm$ SD).

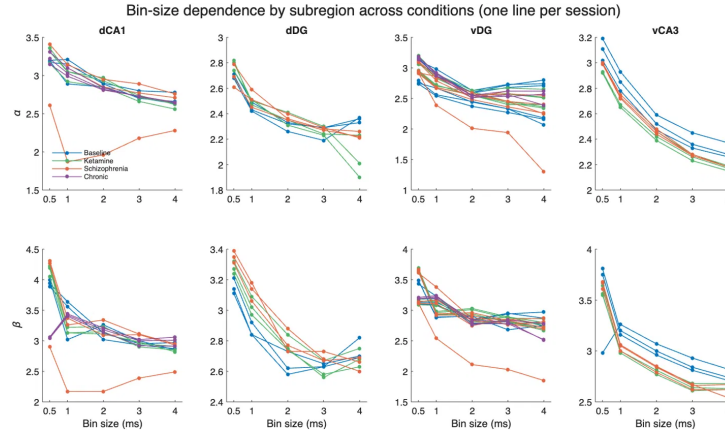


Figure 3.37: Bin-size dependence of the size (α , top) and duration (β , bottom) exponents by hippocampal subregion (dCA1, dDG, vDG, vCA3) across conditions (one line per session, coloured by condition).

Figure 3.38 shows the best-fit composition by subregion (dorsal vs ventral) and observable (size vs duration), revealing a clear difference between the two regions. In the dorsal fields, the size distributions maintain a substantial power-law component across healthy, ketamine, and schizophrenia conditions. In fact, six out of nine schizophrenia size fits are either pure power law or truncated power law. The duration distributions are also mostly described by truncated power laws. Thus, the dorsal hippocampus largely preserves scale-free behaviour across conditions. The ventral fields show a different pattern. Their size distributions contain less power-law weight even in the healthy condition, while lognormal fits become progressively more frequent. Lognormal fits account for roughly half of the distributions in healthy and ketamine conditions, five of six in schizophrenia, and two of three in the chronic condition. The duration

distributions show a different pattern from the size distributions. Rather than showing a clear shift toward lognormal fits, they remain largely dominated by truncated power-law fits across conditions in both dorsal and ventral fields. Lognormal fits remain a minority and do not increase as clearly as for size. This shift toward lognormal best fit models, also observed at the whole hippocampus level, is therefore more pronounced in the ventral fields, while the dorsal fields remain closer to a scale-free, near-critical regime.

This difference is shown in Figure 3.39, which reports, for each condition, the fraction of fits best described by a lognormal model in each subregion, separately for avalanche size and duration. For size, this fraction remains similar across conditions in the dorsal subregions, at around one third (healthy vs schizophrenia, χ^2 $p = 1.0$). In the ventral subregions, however, it increases from 44% in the healthy condition to 83% in schizophrenia and 67% in the chronic stage (healthy vs schizophrenia, $p = 0.13$). For duration, no similar increase is observed in either field. Lognormal fits remain a minority across conditions and become, if anything, less frequent in schizophrenia. Duration fits therefore remain mainly described by truncated power laws.

When all conditions are pooled, dorsal and ventral fields differ for size fits (64% vs 42% power-law-like fits), but not for duration fits (67% vs 67%). Thus, the increase in lognormal best-fit models, also observed at the whole-hippocampus level, is mainly seen for avalanche size and is more pronounced in the ventral fields. In contrast, the dorsal fields remain closer to a scale-free, near-critical regime.

These differences are not statistically significant. Since only two animals are available, the best-fit counts are compared across individual sessions, and the number of sessions in each condition is small. We therefore interpret these results as trends rather than significant effects. The difference between dorsal and ventral fields is also consistent with known physiological differences along the hippocampal longitudinal axis [Strange *et al.*, 2014]. Overall, these results suggest that the possible shift away from criticality in schizophrenia is not uniform across the hippocampus, but is more evident in the ventral fields.

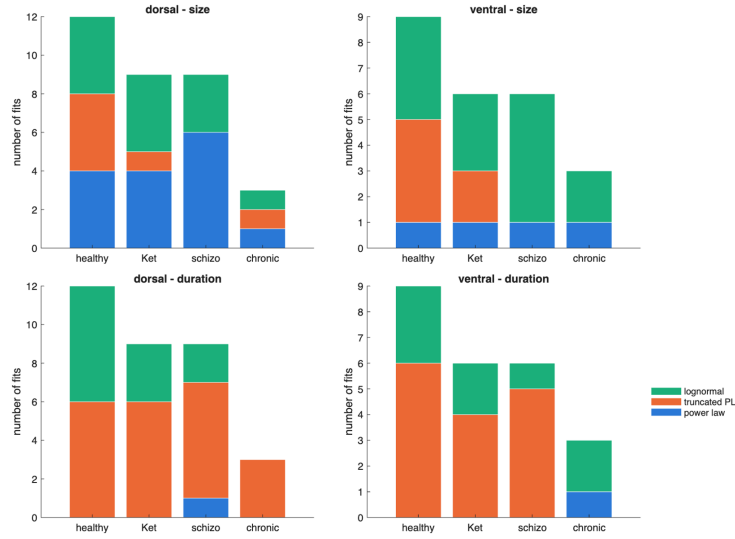


Figure 3.38: Best-fit model composition by subregion. Stacked counts of the best-fitting models, power law (blue), truncated power law (orange), and lognormal (green), for avalanche size (top) and duration (bottom) distributions in the dorsal (left) and ventral (right) hippocampus across the four conditions. Bar heights indicate the total number of fits, which varies depending on the subregions sampled in each condition.

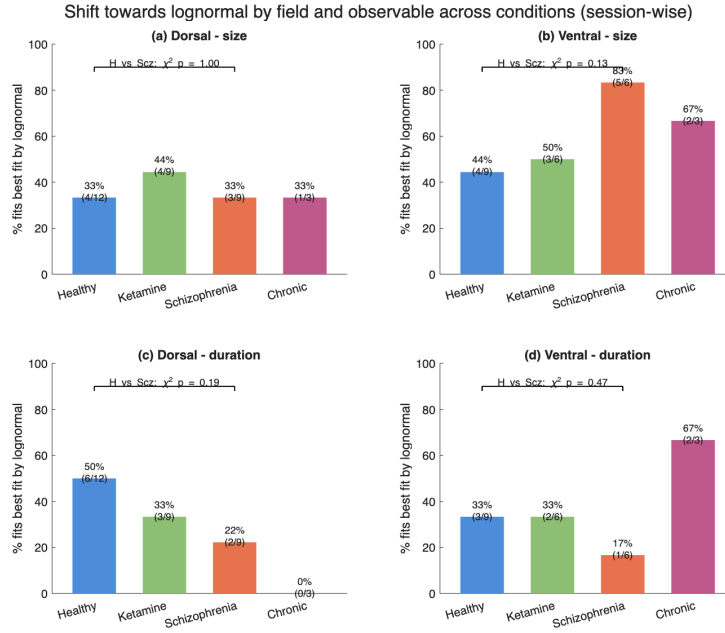


Figure 3.39: Percentage of fits best described by a lognormal model, per condition, by field (dorsal/ventral) and observable (size/duration); session-wise, counts on bars.

DCC and Shape Collapse

Because the avalanche distributions alone cannot establish a departure from criticality, we apply the same two complementary tests used for the healthy hippocampus to the pathological conditions, the DCC and the avalanche shape collapse. These tests assess whether the scaling exponents remain mutually consistent and whether the avalanche profiles still collapse onto a universal shape.

Whole Hippocampus

The DCC results for the pathological conditions are reported in Tables

6.18, 6.19 and 6.20, and summarised in Figures 3.40 and 3.41. In all conditions, the DCC shows the same pattern as in the healthy hippocampus. Each recording reaches a low minimum at an intermediate bin size, well below the 0.2 criticality threshold, usually between 1 and 3 ms, and then increases strongly at the largest bin sizes. At the 2 ms bin size, the DCC does not differ significantly between conditions (Kruskal–Wallis, session-wise, $p = 0.19$). The single high value in Scz 1 track 10 is explained by its minimum occurring at a different bin size and therefore does not indicate a clear loss of criticality. At the level of the DCC, neither the acute nor the chronic schizophrenia condition shows a clear departure from criticality, all recordings satisfy the criterion at an appropriate temporal resolution, as observed in the healthy tissue. This result, however, should not be interpreted in isolation. As discussed throughout this work, the DCC is a scalar consistency check between three exponents that can also be satisfied by decorrelated surrogates at a favourable bin size. More generally, scalar indicators of this kind can be satisfied by non-critical mechanisms as well [Touboul *et al.*, 2017, Beggs and Timme, 2012]. Therefore, the DCC alone cannot distinguish genuinely critical dynamics from non-critical ones or establish whether the pathological conditions preserve criticality. The near-critical DCC values observed across all conditions are therefore consistent with preserved critical dynamics, but do not constitute evidence of criticality on their own. For this reason, the DCC must be considered together with the avalanche shape collapse, a more stringent test that constrains the entire temporal

profile of the avalanches rather than only three summary exponents
[Sethna *et al.*, 2001, Friedman *et al.*, 2012], which will be examined next.

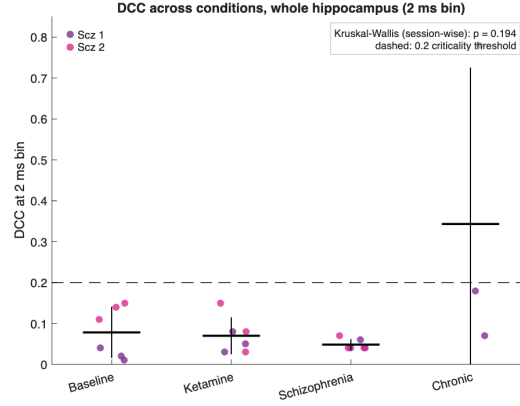


Figure 3.40: DCC at the 2 ms bin across conditions (whole hippocampus, one point per session, coloured by animal; bars mean $\pm$ SD).

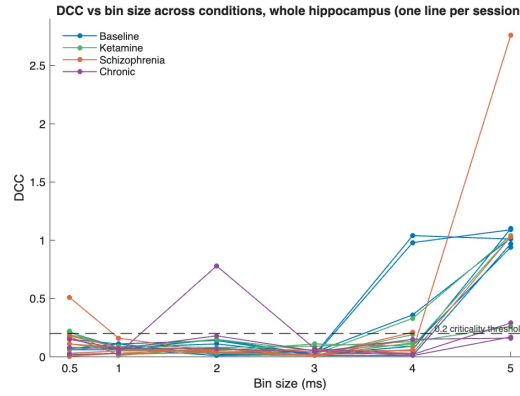


Figure 3.41: DCC as a function of temporal bin size across conditions (whole hippocampus, one line per session, coloured by condition).

Figure 3.42 and Table 6.21 report the avalanche shape collapse under ketamine injection. In every recording, the rescaled profiles converge onto a

single, roughly parabolic mean profile (red curve), and the collapse is highly significant against the shuffled null in all cases (p between 10^{-6} and 10^{-7} , $z \approx -4.5$ to -4.8). The rescaling exponent is stable across the two animals and close to that of the healthy hippocampus, ranging from $\gamma = 1.11$ – 1.20 , with CV values of 0.036 – 0.065 over a dynamic range of 0.7 decades. Therefore, ketamine preserves a clear avalanche shape collapse, consistent with the near-critical behaviour observed in the healthy tissue. Even in this case, looking at these results it is still important to address the fact that the dynamic range is narrow (0.7 decades), well below the decade conventionally required for a stringent collapse and, that the individual rescaled profiles remain noisy, particularly at the shortest durations.

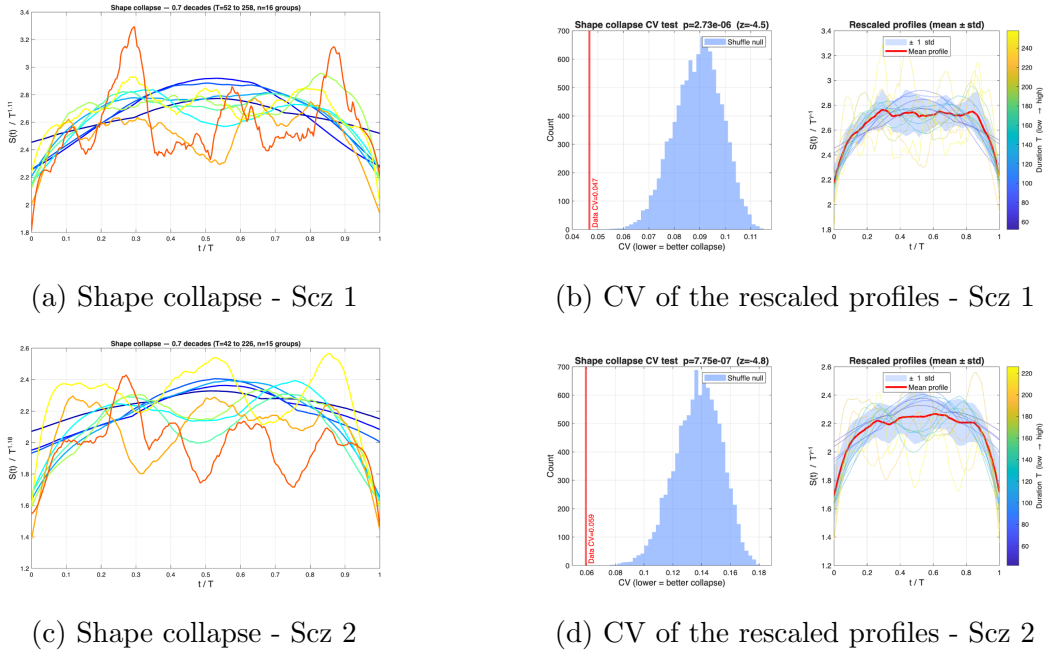


Figure 3.42: Avalanche shape collapse under the ketamine condition in the whole hippocampus, shown for a representative track from each animal.

Figure 3.43 and Table 6.22 report the avalanche shape collapse under schizophrenia. As observed under ketamine and in the healthy tissue, the rescaled profiles converge onto a common parabolic mean profile in every recording, and the collapse is significant against the shuffled null, with p -values ranging from 1.4×10^{-3} to 3.4×10^{-7} and z -scores from approximately -4.1 to -5.0 . The rescaling exponent remains stable and close to the healthy value, spanning $\gamma = 1.09$ – 1.23 , with CV values of 0.045 – 0.064 over dynamic ranges of 0.6 – 1.1 decades. The schizophrenic hippocampus therefore continues to satisfy the shape-collapse criterion, with no systematic degradation relative to the healthy or ketamine conditions. Taken together with the DCC, the shape collapse indicates that the schizophrenia condition retains signatures of near-critical avalanche dynamics according to the scaling tests. This may appear to contrast with the best-fit composition, where the schizophrenia condition showed a shift of the size distributions toward lognormal forms. The two observations can be reconciled by noting that the shape collapse constrains the temporal profile of avalanches, which, as also shown by the distribution analysis, remains broadly consistent with a truncated power law, whereas the shift toward lognormality was mainly observed in the size distributions. Therefore, the effects of schizophrenia on critical dynamics appear to be subtle and observable-dependent, rather than reflecting a wholesale loss of critical-like dynamics [Beggs and Timme, 2012, Touboul *et al.*, 2017].

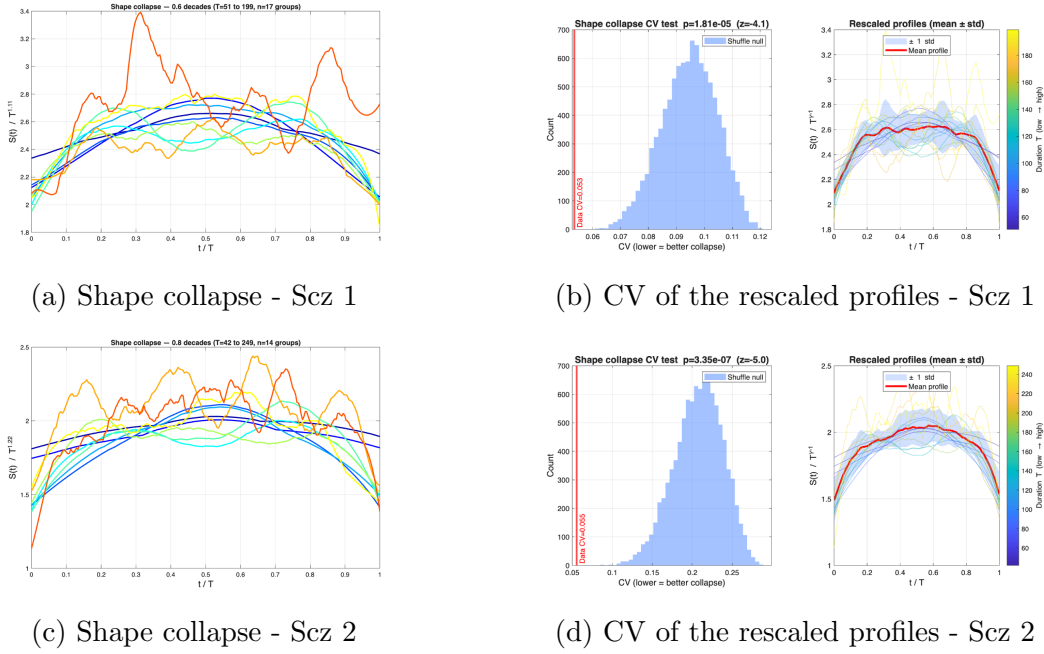


Figure 3.43: Avalanche shape collapse under the schizophrenia condition in the whole hippocampus, shown for a representative track from each animal.

During the chronic schizophrenia phase, the avalanche shape collapse remains significant across all three recordings (Table 6.23, Fig. 3.44). The rescaling exponent is highly consistent across tracks, ranging from $\gamma = 1.09$ to 1.12, while the CV remains low (0.038–0.049), indicating a good convergence of the rescaled avalanche profiles onto a common temporal shape. All recordings show statistically significant deviations from the shuffled null, with p -values ranging from 6.0×10^{-4} to 1.4×10^{-2} . However, the collapse is supported over only approximately 0.5 decades in all three recordings, representing a relatively limited dynamic range. Overall, these results indicate that the chronic schizophrenia condition preserves the

temporal scaling structure of avalanches. Nevertheless, these results should be interpreted with caution, as the chronic schizophrenia condition is represented by only three recordings from a single animal. Therefore, the observed preservation of the avalanche shape collapse cannot be generalised to the broader schizophrenia condition, and no strong conclusions about the effects of chronic schizophrenia on critical dynamics can be drawn from these recordings alone. The results should instead be regarded as preliminary evidence that would require confirmation in a larger sample of animals and recordings.

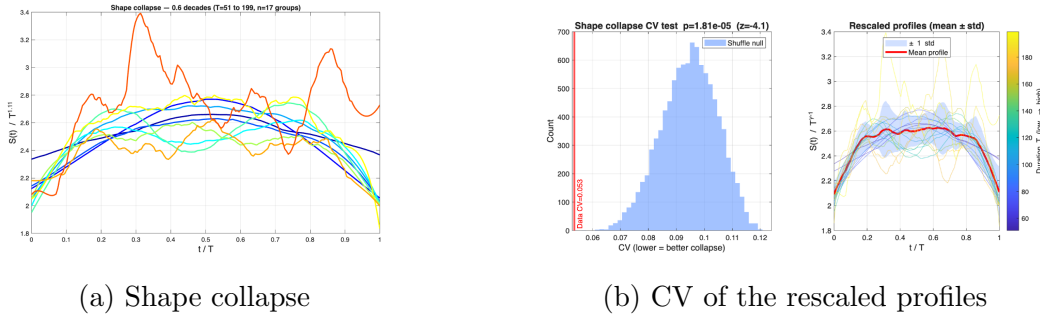


Figure 3.44: Avalanche shape collapse under the chronic schizophrenia condition in the whole hippocampus (Available only for one mouse), shown for a representative track.

Figure 3.45 summarises the avalanche shape collapse across all conditions. Both the quality of the collapse and its significance compared with the shuffled null are similar across conditions. All sessions show a good collapse ($CV \approx 0.03\text{--}0.07$), and all are significant ($z < -2$).

A Kruskal–Wallis test shows no significant difference between conditions for either measure (CV $p = 0.66$; z $p = 0.09$, session-wise). Overall, these

results show that the ketamine, acute schizophrenia and chronic schizophrenia conditions all retain a significant avalanche shape collapse, with no clear reduction compared with the healthy baseline.

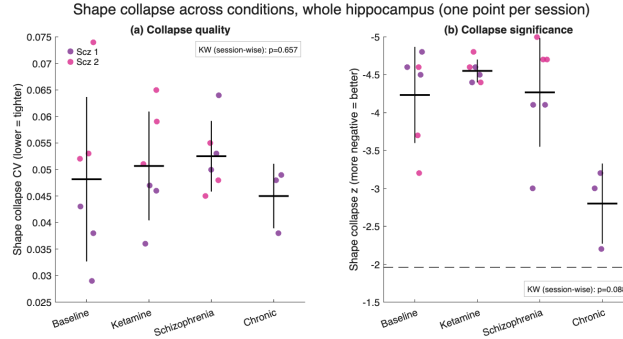


Figure 3.45: Avalanche shape collapse across conditions (whole hippocampus, one point per session, coloured by animal; bars mean $\pm$ SD).

Dorsal and Ventral subregions

Figures 3.46 and 3.47 show the DCC values for each hippocampal subregion under the three conditions. Overall, they show the same dorsoventral difference observed in the healthy hippocampus. The ventral dentate gyrus reaches low DCC values at intermediate bin sizes in almost all recordings. These values are well below the 0.2 criticality threshold. In contrast, the dorsal regions show higher DCC values. Dorsal CA1 in Scz 1 and the dorsal dentate gyrus in Scz 2 often reach values between 0.4 and 1.0 at the same intermediate bin sizes where the ventral regions show the lowest values. In the chronic condition, dorsal CA1 also remains relatively high, with a minimum of about 0.27 in track 12. Ventral CA3, when sampled, shows intermediate values, with low DCC values around 3 ms but a sharp increase at the largest bin size. This pattern suggests that the low DCC values

observed in the whole hippocampus are mainly driven by the ventral fields, while the dorsal fields, especially dorsal CA1, show higher DCC values across most bin sizes. Importantly, this difference is already present in the ketamine condition and does not show a clear progressive increase from the acute to the chronic schizophrenia condition. Therefore, the observed dorsoventral difference does not appear to be specifically related to disease progression, but may instead reflect differences between hippocampal subregions [Fanselow and Dong, 2010].

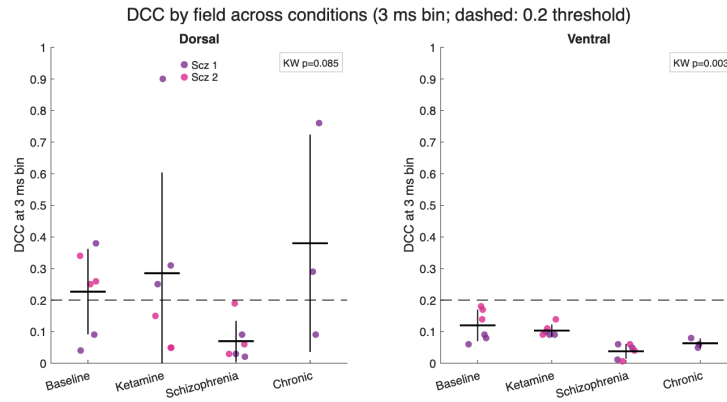


Figure 3.46: DCC at the 3 ms bin by hippocampal field across conditions (one point per session, coloured by animal; bars mean $\pm$ SD).

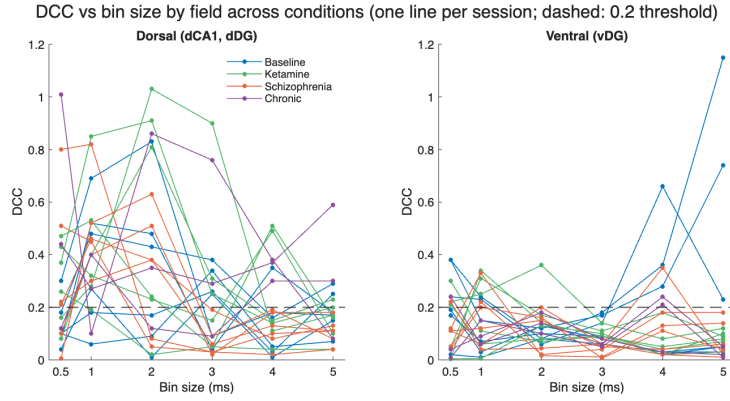


Figure 3.47: DCC as a function of temporal bin size by hippocampal field across conditions (one line per session, coloured by condition).

As emphasised for the whole hippocampus, the DCC is a scalar consistency check that cannot by itself distinguish critical from non-critical dynamics, therefore it has to be interpreted alongside the shape collapse. Under the ketamine condition, analysing Table 6.27, Fig. 3.48 and Fig. 3.49, avalanche shape collapse was observed with variable consistency across the dorsal and ventral hippocampal subregions. In Scz 1, the vDG showed consistently low CV values across all three tracks (0.042–0.057), with statistically significant differences from the shuffled null in all cases ($p < 0.003$). In dCA1, significant differences were observed in tracks 4 and 6 ($p = 2.09 \times 10^{-2}$ and $p = 6.26 \times 10^{-3}$, respectively), whereas track 5 did not show a significant difference ($p = 0.502$). The CV values were also higher in dCA1 (0.080–0.366) than in vDG.

In Scz 2, dDG showed significant differences from the shuffled null across all three tracks ($p < 0.003$), with CV values ranging from 0.073 to 0.112. For vCA3, a significant difference was observed only in track 5

($p = 5.12 \times 10^{-3}$), while tracks 4 and 6 did not reach significance. The vDG showed significant differences in tracks 4 and 6 ($p = 2.63 \times 10^{-3}$ and $p = 2.68 \times 10^{-3}$), but not in track 5 ($p = 0.425$). CV values in vCA3 and vDG remained relatively low across tracks, ranging from 0.064 to 0.106. Overall, these results suggest that avalanche shape collapse is more robust in some dentate gyrus recordings than in CA1 or CA3, with the ventral DG in Scz 1 providing the most consistent example. However, the regional variability indicates that shape collapse cannot be considered a uniform property of the hippocampus under the ketamine condition. This interpretation is further supported by the absence of a clear collapse in the dCA1 of SCZ1, suggesting that the observation of a good collapse in individual recordings should not be generalized to all dorsal hippocampal regions or pathological conditions.

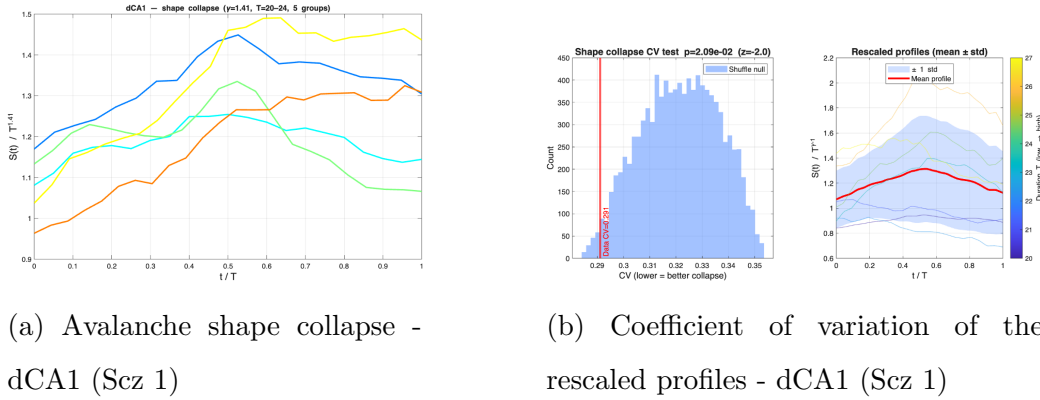
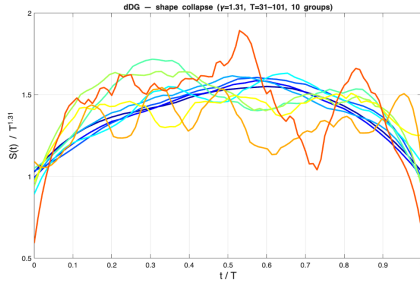
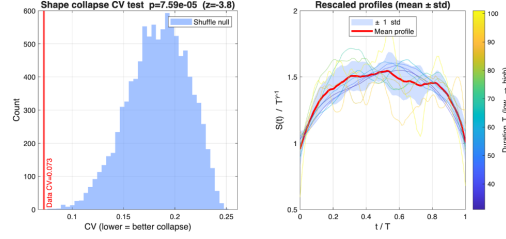


Figure 3.48: Avalanche shape collapse under the ketamine condition in the dorsal hippocampus, shown for a representative track from each animal.

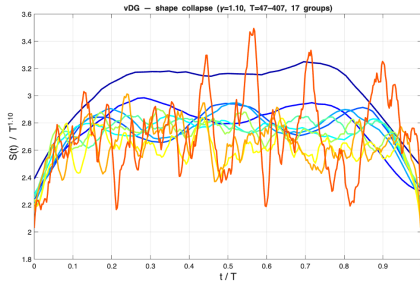


(c) Avalanche shape collapse - dDG (Scz 2)

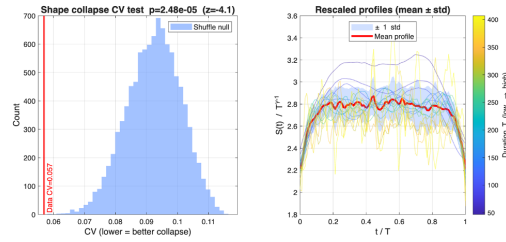


(d) Coefficient of variation of the rescaled profiles - dDG (Scz 2)

Figure 3.48: Avalanche shape collapse under the ketamine condition in the dorsal hippocampus, shown for a representative track from each animal.



(a) Avalanche shape collapse - vDG (Scz 1)



(b) Coefficient of variation of the rescaled profiles - vDG (Scz 1)

Figure 3.49: Avalanche shape collapse under the ketamine condition in the ventral hippocampus, shown for a representative track from each animal.

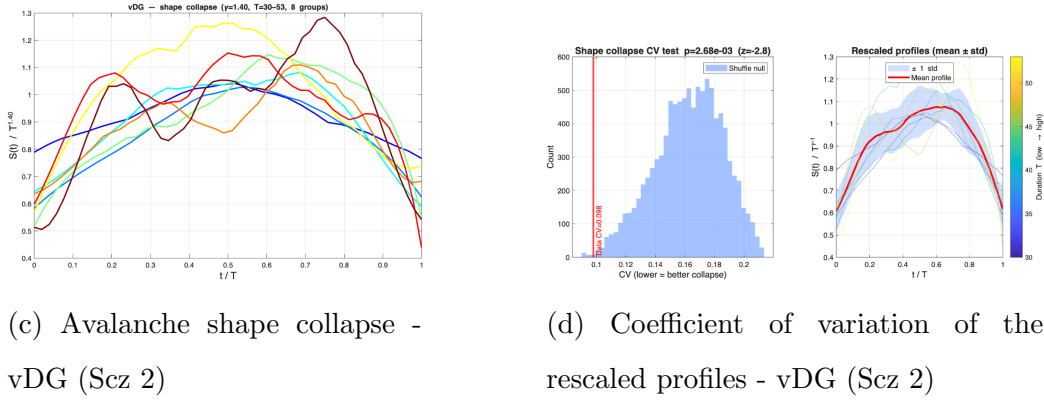


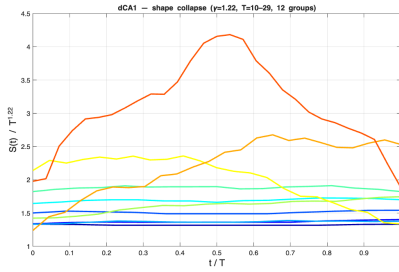
Figure 3.49: Avalanche shape collapse under the ketamine condition in the ventral hippocampus, shown for a representative track from each animal.

In the schizophrenia model, avalanche shape collapse showed variable consistency across the dorsal and ventral hippocampal subregions (Table 6.28, Fig. 3.50 and Fig. 3.51). In Scz 1, no clear shape collapse was observed in dCA1 across the three recordings. Although track 7 showed a statistically significant difference from the shuffled null ($p = 1.85 \times 10^{-11}$) and track 9 also reached significance ($p = 3.39 \times 10^{-3}$), track 8 did not ($p = 0.593$), and the relatively high CV in track 9 (0.314) indicates a less consistent collapse. This is also evident from the temporal profiles shown in Fig. 3.50, where the profiles do not clearly converge onto a common universal shape for dCA1. In contrast, vDG showed significant differences from the shuffled null in tracks 8 and 9 ($p = 2.34 \times 10^{-6}$ and $p = 8.67 \times 10^{-4}$), while track 7 did not ($p = 0.995$). The CV values in vDG remained low across all three recordings (0.043–0.096). However, as observed previously, track 7 represents an anomalous recording and should therefore be considered separately when assessing the overall collapse

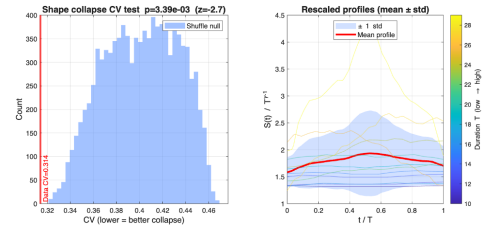
pattern.

In Scz 2, dDG showed significant differences from the shuffled null across all three recordings, with CV values ranging from 0.078 to 0.126 and $p < 0.004$. Similarly, vDG showed significant differences in all three tracks, with low CV values (0.060–0.104) and $p < 0.021$. In contrast, vCA3 did not reach significance in tracks 7 and 8 ($p = 0.108$ and $p = 0.727$), whereas a significant difference was observed in track 9 ($p = 0.0138$).

Overall, shape collapse was observed most consistently in the DG subregions, particularly in Scz 2. In Scz 1, vDG showed a more consistent pattern than dCA1, whereas dCA1 did not exhibit a clear shape collapse across the three recordings.

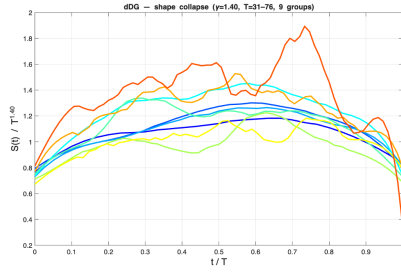


(a) Avalanche shape collapse - dCA1 (Scz 1)

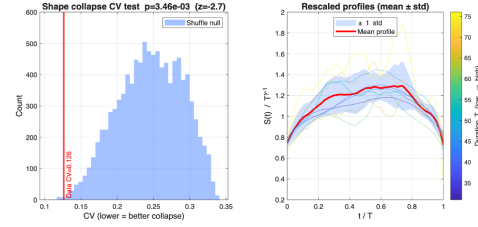


(b) Coefficient of variation of the rescaled profiles - dCA1 (Scz 1)

Figure 3.50: Avalanche shape collapse under the schizophrenia condition in the dorsal hippocampus, shown for a representative track from each animal.

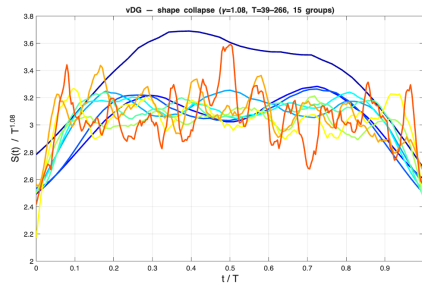


(c) Avalanche shape collapse - dDG (Scz 2)

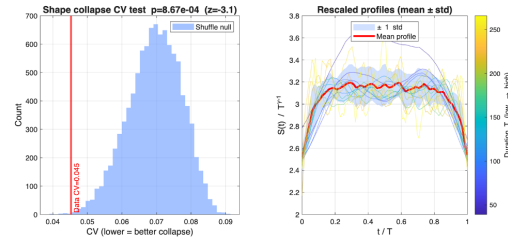


(d) Coefficient of variation of the rescaled profiles - dDG (Scz 2)

Figure 3.50: Avalanche shape collapse under the schizophrenia condition in the dorsal hippocampus, shown for a representative track from each animal.

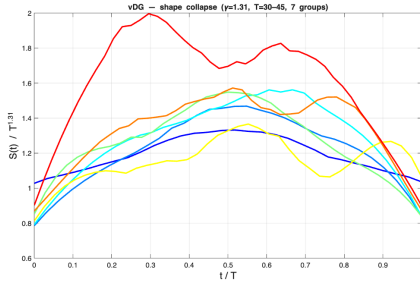


(a) Avalanche shape collapse - vDG (Scz 1)

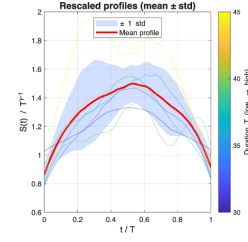
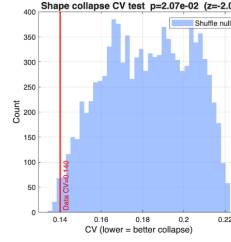


(b) Coefficient of variation of the rescaled profiles - vDG (Scz 1)

Figure 3.51: Avalanche shape collapse under the schizophrenia condition in the ventral hippocampus, shown for a representative track from each animal.



(c) Avalanche shape collapse - vDG (Scz 2)



(d) Coefficient of variation of the rescaled profiles - vDG (Scz 2)

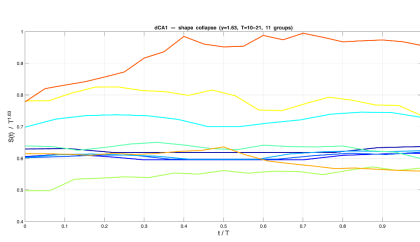
Figure 3.51: Avalanche shape collapse under the schizophrenia condition in the ventral hippocampus, shown for a representative track from each animal.

Under the chronic schizophrenia condition, only available for one animal, avalanche shape collapse showed variable consistency across the dorsal and ventral hippocampal subregions. In dCA1, no clear shape collapse was observed across the three recordings. Although all tracks showed statistically significant differences from the shuffled null ($p = 2.85 \times 10^{-3}$, 1.30×10^{-4} , and 4.70×10^{-3} for tracks 10, 11, and 12, respectively), the temporal profiles did not clearly converge onto a common universal shape. The CV values were relatively high (0.165–0.219), and the rescaling exponent varied considerably across recordings, particularly in track 11 ($\gamma = 2.16$) compared with tracks 10 and 12 ($\gamma = 1.12$ and 1.69 , respectively).

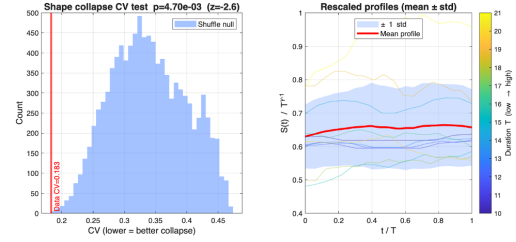
In contrast, vDG showed a consistent shape collapse across all three recordings, with low and stable CV values (0.038–0.057) and highly consistent rescaling exponents ($\gamma = 1.08$ – 1.10). All three recordings also showed significant differences from the shuffled null ($p = 3.74 \times 10^{-4}$,

1.42×10^{-6} , and 3.82×10^{-8} for tracks 10, 11, and 12, respectively).

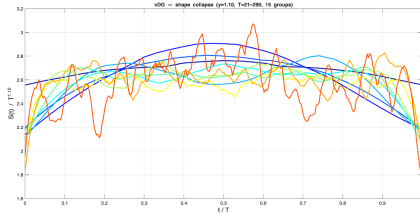
Overall, as observed in the other conditions, shape collapse was consistently observed in vDG, whereas dCA1 did not show a clear convergence of the avalanche profiles onto a common shape.



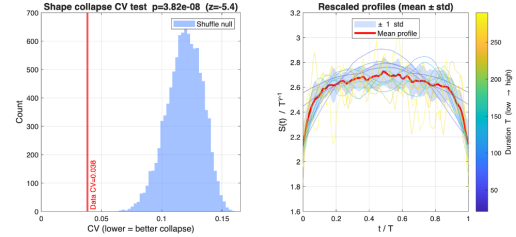
(a) Avalanche shape collapse - dCA1 (Scz 1)



(b) Coefficient of variation of the rescaled profiles - dCA1 (Scz 1)



(c) Avalanche shape collapse - vDG (Scz 1)



(d) Coefficient of variation of the rescaled profiles - vDG (Scz 1)

Figure 3.52: Avalanche shape collapse under the chronic schizophrenia condition (available only for one mouse) in the dorsal and ventral hippocampus, shown for a representative track.

Taken together, the dorsoventral analysis shows a consistent regional pattern of avalanche dynamics across all three conditions, summarised at the population level in Figure 3.53. The two criticality measures point to a clear difference between regions. The ventral dentate gyrus consistently

shows low DCC values and a strong shape collapse, with low and stable CV values and similar γ values across conditions. In contrast, dorsal CA1 shows higher DCC values, and its avalanche profiles do not clearly converge to a common shape. Ventral subregions show a strong collapse in all conditions ($CV \approx 0.04\text{--}0.1$, with z mostly below -2), while dorsal subregions show two to four times higher CV values and several non-significant collapses. Neither measure differs significantly between conditions (Kruskal–Wallis, all $p > 0.09$, session-wise).

Importantly, this difference is already present in the ketamine recordings and does not increase in the acute or chronic schizophrenia conditions. This suggests that the observed pattern is more likely related to intrinsic differences between hippocampal subregions than to disease progression. This interpretation is also consistent with the known anatomical and functional differences along the dorsoventral axis of the hippocampus [Fanselow and Dong, 2010]. Overall, the results suggest that ventral regions may show dynamics closer to criticality than dorsal regions.

However, these measures provide only partial evidence for criticality and are not sufficient on their own to demonstrate a critical state. In addition, the small number of animals and the variability between recordings, including the anomalous track 7, limit the strength of these conclusions. Therefore, the dorsoventral gradient observed here should be considered a possible regional pattern that needs to be confirmed in larger datasets.

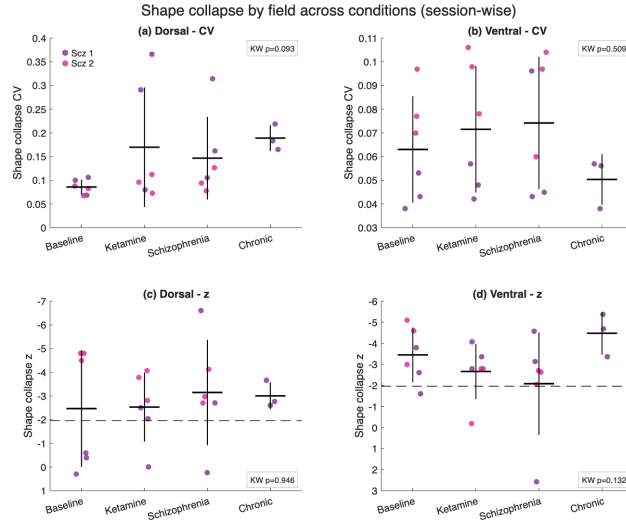


Figure 3.53: Avalanche shape collapse by hippocampal field across conditions (session-wise; one point per session, coloured by animal, bars mean $\pm$ SD). Top: collapse quality (CV, lower = tighter) for dorsal (a) and ventral (b) fields. Bottom: collapse significance (z against the shuffled null; more negative = better, dashed $z = -1.96$) for dorsal (c) and ventral (d). V

3.2.2 Epilepsy

Epilepsy provides an important test of the criticality framework. While the healthy brain is thought to operate close to a critical state, where activity is balanced between inhibition and excitation, epileptic activity is often associated with a shift towards supercriticality. Seizures are characterised by excessive, hypersynchronous, and self-amplifying activity, suggesting that the system is pushed beyond the critical regime [Meisel *et al.*, 2012, Zimmern, 2020]. In terms of neuronal avalanches, this shift may appear as a disruption of scale-free statistics. In simplified

experimental models, excessive excitation has been shown to alter the power-law distribution of avalanche sizes and increase the occurrence of large events, which is consistent with a supercritical state [Beggs and Plenz, 2004]. Similar deviations towards supercriticality have also been reported in the epileptic human brain [Arviv *et al.*, 2016]. These changes may therefore also be reflected in the DCC and in the avalanche shape collapse.

In this section, the same analysis pipeline used for the ketamine and schizophrenia conditions is applied to the epileptic hippocampus. First, avalanche size and duration distributions are analysed to determine whether they retain a power-law form. The analysis then examines the two complementary criticality measures, DCC and avalanche shape collapse.. The aim is to determine whether epileptic activity shows a measurable deviation from the near-critical dynamics observed in the other conditions and whether this difference is also reflected along the dorsoventral axis of the hippocampus.

Power-law

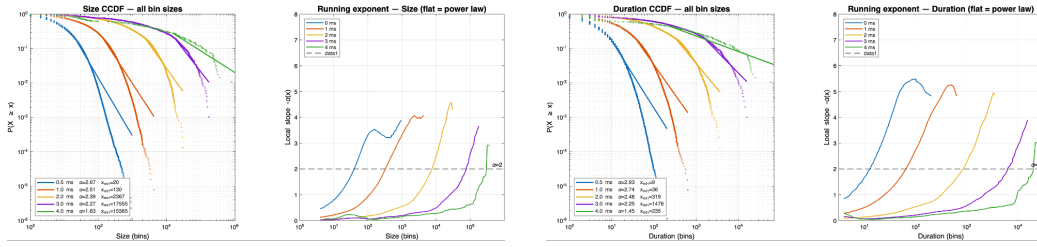
Whole hippocampus

Across both epilepsy phases (Table 6.30, Table 6.31 and Fig. 3.55), the whole-hippocampus size and duration distributions show an approximately power-law behaviour only over a limited range, and the fitted exponents depend strongly on the bin size. At the finest bin sizes (0.5–1 ms), the exponents are in a plausible near-critical range ($\tau \approx 2.4$ – 2.7 , $\alpha \approx 2.3$ – 2.9). However, as the bin size increases, both exponents decrease, while $x_{\min}$ increases by several orders of magnitude. This means that the fitted values

increasingly describe only the tail of the distribution rather than a clear scaling regime. This is particularly evident in the epileptogenesis recordings at 3–4 ms. In some cases, the fit starts only at very large values of $x_{\min}$ (track 6 and track 8, $x_{\min} \sim 10^4$), while in track 7 at 4 ms the fit covers only a very small range ($\tau = 1.20$, $x_{\min} = 6$). These cases should therefore be interpreted with caution.

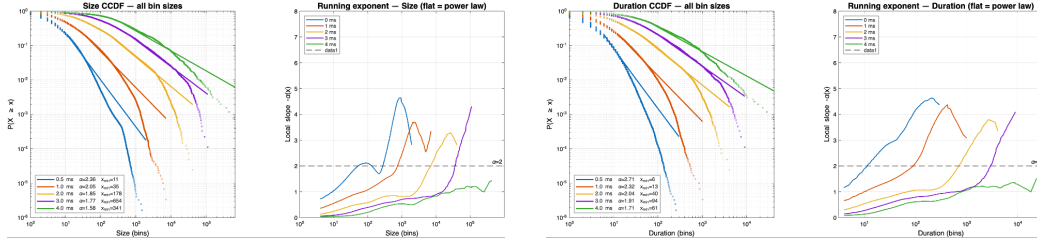
The main difference is observed between the two epilepsy phases rather than between different bin sizes. At matched bin sizes, the status epilepticus distributions are consistently shallower than those observed during epileptogenesis. At 0.5 ms, for example, $\tau = 2.36$ during status epilepticus compared with 2.66–2.70 during epileptogenesis, while $\alpha = 2.71$ compared with 2.91–2.93, respectively. This difference is also maintained at 1 and 2 ms. Lower exponents indicate heavier tails and a relative increase in large and long avalanches. This pattern is also visible in the running-exponent panels, where the local slope remains lower over a wider range of sizes and durations during status epilepticus. This is consistent with the expected shift from near-critical towards supercritical dynamics [Meisel *et al.*, 2012, Arviv *et al.*, 2016, Beggs and Plenz, 2004], in line with the hyperexcitable and self-amplifying activity associated with seizures.

However, these exponents alone are not sufficient to establish a departure from criticality. The observed difference is relatively modest and is based on recordings from a single animal. Therefore, the exponent relation and avalanche shape collapse are needed to determine whether this difference is associated with a genuine change in critical dynamics.



(a) Epileptogenesis - Avalanche size distribution

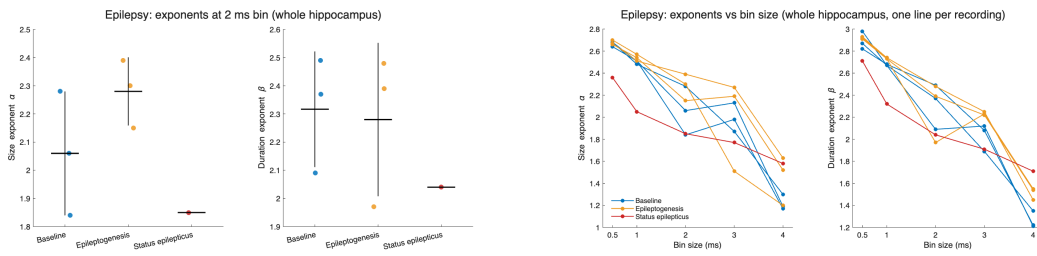
(b) Epileptogenesis - Avalanche duration distribution



(c) Status Epilepticus - Avalanche size distribution

(d) Status Epilepticus - Avalanche duration distribution

Figure 3.54: Avalanche size and duration distributions (CCDF) for the whole hippocampus under the two epilepsy phases, available for only one animal.



(a) Exponents at the 2 ms bin by condition (bars mean $\pm$ SD).

(b) Exponents as a function of bin size, one line per recording.

Figure 3.55: Avalanche size (α) and duration (β) exponents for the whole hippocampus in the single epilepsy animal, by condition.

Fig. 3.56 shows the model-selection analysis, in which each distribution is assigned to its best-fitting model (power law, truncated power law, lognormal, or exponential) rather than assuming a power-law model. For avalanche size, the results are the same across all three conditions, where all recordings are best described by a truncated power law in healthy, epileptogenesis, and status epilepticus, with no recording favouring a pure power law. For avalanche duration, all healthy recordings are best described by a truncated power law, as are the status epilepticus recordings. The only exception is epileptogenesis, where one of the three recordings is best described by a lognormal distribution. It should be noted that status epilepticus is based on a single recording, so this result should be considered only indicative.

It is evident that a pure power law is never selected in any condition. This is consistent with the limited scaling range and the curvature observed in the running-exponent panels, suggesting that the distributions are better described as truncated power laws. Second, the overall model composition remains similar across the three conditions. The only difference is the single lognormal duration distribution observed during epileptogenesis. This is consistent with the heavier tails and lower exponents described above, but the effect is limited, as it occurs in only one recording and only for avalanche duration. Moreover, this pattern is not observed during status epilepticus, where both size and duration remain best described by truncated power laws despite the lower exponents. Therefore, the possible shift towards supercritical dynamics suggested by the exponents is not accompanied by a clear change in the best-fitting distribution models. This

result should therefore be considered suggestive rather than conclusive, and the DCC and avalanche shape collapse are needed to further assess whether the observed distributions are consistent with near-critical dynamics.

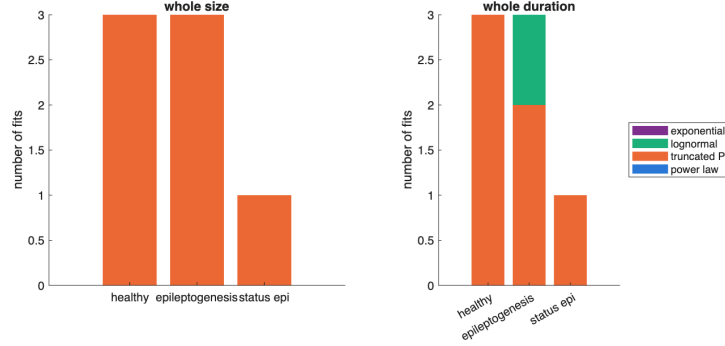


Figure 3.56: Comparison of the best-fitting models for avalanche size and duration distributions across healthy, epileptogenesis, and status epilepticus conditions.

Dorsal and Ventral Subregions

The subregional analysis (Tables 6.32 and 6.33, Figs. 3.57 and 3.59) shows the same dependence on bin size observed for the whole hippocampus, together with a clear difference between dorsal and ventral regions. At the finest bin sizes (0.5–1 ms), all subregions show exponents within a plausible near-critical range ($\tau \approx 2.5\text{--}3.0$, $\alpha \approx 2.8\text{--}3.3$). As the bin size increases, both exponents decrease and $x_{\min}$ increases. This effect is particularly strong in the dorsal regions, as already seen in the other animal models. For example, in dCA1 during epileptogenesis, $x_{\min}^S$ reaches values around 10^3 at 3–4 ms (e.g. track 8, $x_{\min}^S = 1001$). These large-bin fits are therefore based mainly on the extreme tail of the distribution and should be

interpreted with caution. This is also consistent with the running-exponent panels, where the local slope remains stable only over a limited range.

The ventral dentate gyrus shows a different pattern. Its $x_{\min}$ values remain relatively small, generally in the tens rather than thousands, while the exponents remain higher and more stable across bin sizes (τ and α mostly between 2.8 and 3.3 during epileptogenesis). Thus, vDG distributions are generally steeper than those of the dorsal regions. This dorsoventral difference is similar to the pattern observed in the healthy and schizophrenia conditions, still suggesting that it may reflect intrinsic differences between hippocampal subregions rather than an effect specific to epilepsy. However, this result should be interpreted with caution, as the higher ventral exponents may also be partly related to the smaller number of neurons recorded in vDG and the resulting finite-size effects.

The main difference between the two epilepsy phases is again the shift towards lower exponents during status epilepticus. At the same bin sizes, the dorsal regions generally show lower exponents than during epileptogenesis. For example, at 4 ms, dCA1 shows $\tau = 1.89$ and $\alpha = 2.14$ during status epilepticus, compared with $\tau = 2.30$ and $\alpha = 2.49$ during epileptogenesis. Similarly, dDG at 4 ms shows $\tau = 1.72$ and $\alpha = 2.18$ during status epilepticus, compared with $\tau \approx 2.8$ – 2.9 during epileptogenesis. The same tendency is also observed in vDG, where at 3–4 ms τ is approximately 2.0–2.1 during status epilepticus compared with approximately 2.3–3.0 during epileptogenesis. Lower exponents indicate heavier tails and a relative increase in large and long avalanches. This is consistent with the expected shift towards supercritical dynamics [Meisel *et al.*, 2012,

Arviv *et al.*, 2016,

Beggs and Plenz, 2004]. Importantly, this pattern is observed in both dorsal and ventral regions rather than being limited to one part of the hippocampal axis.

As for the whole hippocampus, however, the exponents alone are not sufficient to establish a change in criticality. The effect is relatively modest, the analysis is based on a single animal, and the fits at large bin sizes are often unreliable. The exponent relation and avalanche shape collapse are therefore needed to determine whether the observed changes reflect a genuine departure from near-critical dynamics.

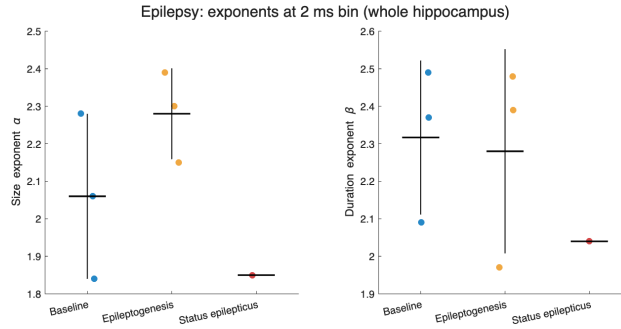
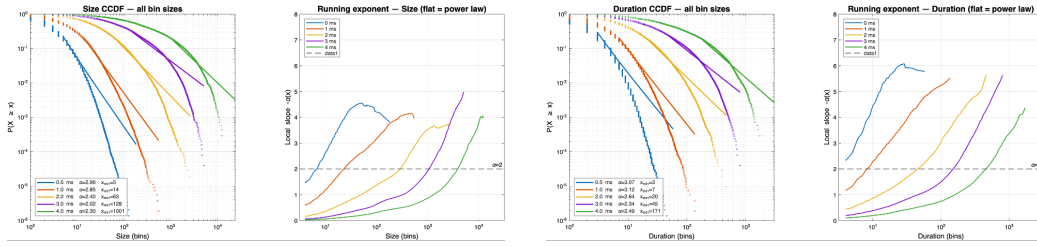
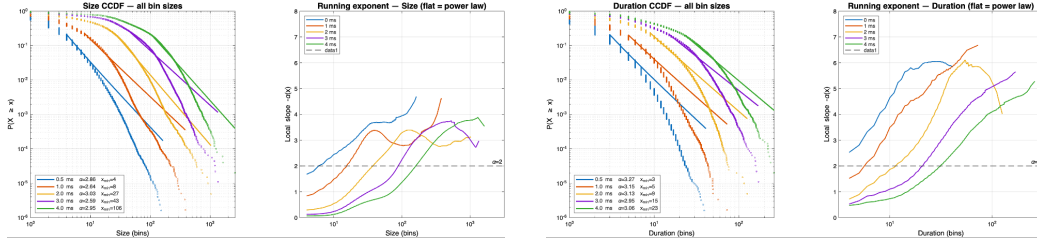


Figure 3.57: Avalanche size (α) and duration (β) exponents at the 2 ms bin by hippocampal field across epilepsy conditions, coloured by condition, bars mean $\pm$ SD).



(a) Epileptogenesis - Avalanche size distribution (dCA1)

(b) Epileptogenesis - Avalanche duration distribution (dCA1)



(c) Epileptogenesis - Avalanche size distribution (vDG)

(d) Epileptogenesis - Avalanche duration distribution (vDG)

Figure 3.58: Avalanche size and duration distributions during epileptogenesis in the dorsal CA1 and ventral dentate gyrus.

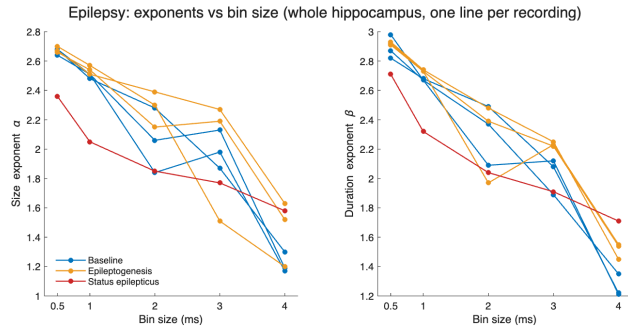
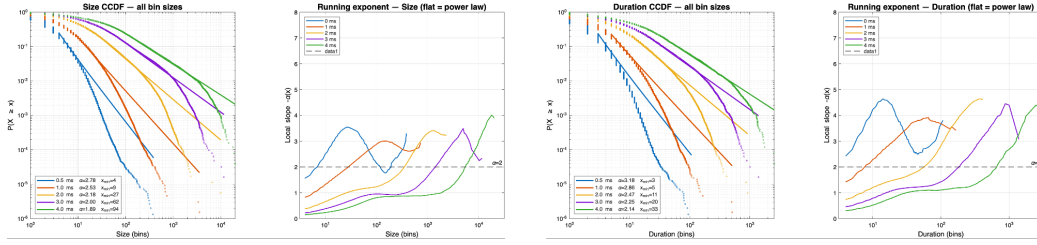
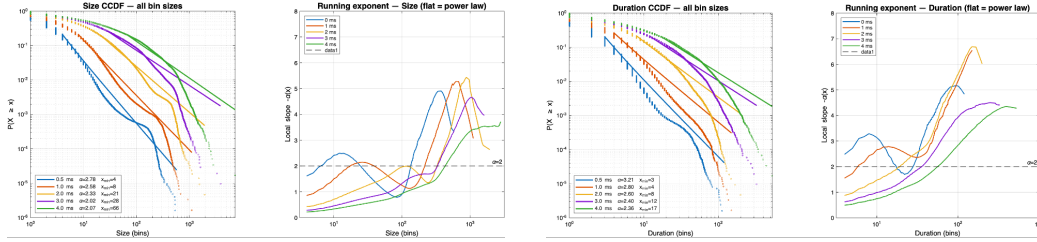


Figure 3.59: Avalanche size (α , top) and duration (β , bottom) exponents as a function of temporal bin size by hippocampal field across epilepsy conditions, one line per recording, coloured by condition.



(a) Status epilepticus - Avalanche size distribution (dCA1)

(b) Status Epilepticus - Avalanche duration distribution (dCA1)



(c) Status Epilepticus - Avalanche size distribution (vDG)

(d) Status Epilepticus - Avalanche duration distribution (vDG)

Figure 3.60: Avalanche size and duration distributions during status epilepticus in the dorsal CA1 and ventral dentate gyrus.

The model-selection analysis by subregion (Fig. 3.61) provides further information on the dorsoventral differences observed in the exponent analysis. The clearest difference is found for avalanche size. The ventral dentate gyrus is mainly described by lognormal distributions, instead the dorsal regions are mainly described by truncated power laws. This is consistent with the higher exponents and smaller $x_{\min}$ values observed in the ventral regions. The ventral size distributions therefore decrease more rapidly, making the lognormal model more suitable, while the broader tails of the dorsal distributions are better described by truncated power laws.

Since this pattern is already present in the healthy hippocampus, it is more likely to reflect differences between the subregions rather than an effect specific to epilepsy.

Across the different conditions, the overall distribution of the best-fitting models remains relatively similar, with only small changes. For avalanche size, a pure power law is selected in only a few cases: one dorsal and one ventral recording during epileptogenesis, and one dorsal recording during status epilepticus. It therefore never represents the main distribution type. For avalanche duration, the dorsal regions remain mainly described by truncated power laws. The main difference is observed in the ventral regions, where two of the three epileptogenesis recordings are best described by an exponential distribution, compared with a mixture of truncated power law and lognormal distributions in the healthy condition. Together with the lower exponents observed during status epilepticus, these results provide some indication of a possible departure from near-critical dynamics. However, the pattern is not consistent across size and duration or between the two epilepsy phases, and therefore does not show a clear supercritical signature.

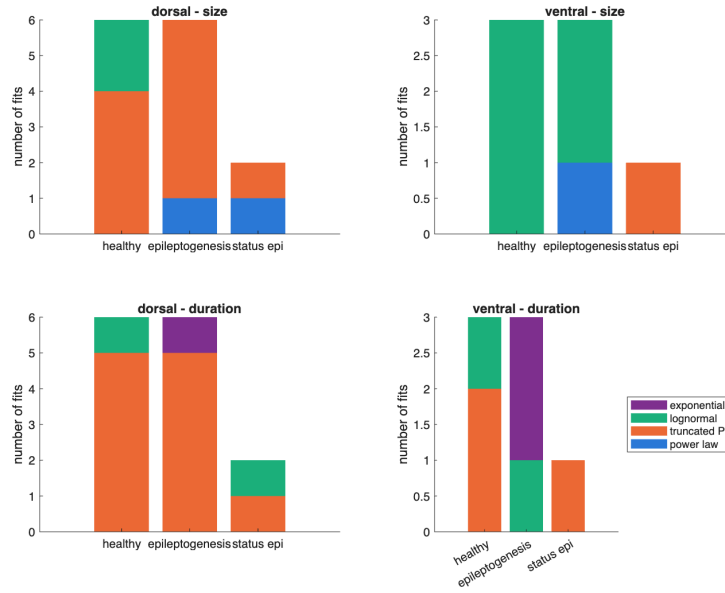


Figure 3.61: Comparison of the best-fitting models for avalanche size and duration distributions, for dorsal and ventral hippocampus, across healthy, epileptogenesis, and status epilepticus conditions.

DCC and Shape Collapse

Whole Hippocampus

Considering the DCC together with the avalanche shape collapse (Tables 6.34, 6.35 and Figs. 3.64, 3.63). During epileptogenesis, the whole hippocampus DCC is low at the finer bin sizes, remaining below the 0.2 threshold up to 2 ms in all three recordings. However, it increases strongly at 3–4 ms, reaching values above 1 and up to 2.40–2.74 in track 7. This follows the same bin-size dependence observed for the size and duration exponents, where the fits at large bin sizes become less reliable. In contrast, during status epilepticus the DCC remains relatively low across the whole

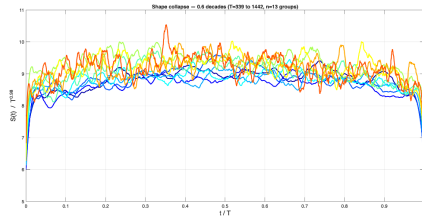
range, with a maximum of 0.37 at 0.5 ms and values ≤ 0.11 from 1 ms onwards. At first sight, this could indicate more consistent scaling during status epilepticus. However, since the DCC only measures the consistency between the fitted exponents, this result cannot be considered evidence of criticality on its own.

The shape collapse provides a more mixed picture. During epileptogenesis (Fig. 3.62, the rescaling exponents are close to 1 ($\gamma = 0.98$ – 1.11) and the CV values are low (0.037–0.050). However, the comparison with the shuffled null is not consistent across recordings. Only track 7 shows a significant difference ($p \approx 9.8 \times 10^{-4}$), while track 6 does not ($p = 0.21$) and track 8 shows no evidence of a better collapse than the null ($p = 0.99$, $z = +2.3$). Therefore, a low CV alone cannot be considered sufficient evidence of a genuine shape collapse, since simple or weakly structured profiles can also produce low CV values. During status epilepticus, the collapse covers a wider range of durations (1.6 decades) and is highly significant ($p \approx 1.8 \times 10^{-8}$). However, the CV is much higher (0.169), and the rescaled profiles do not clearly converge onto a common shape.

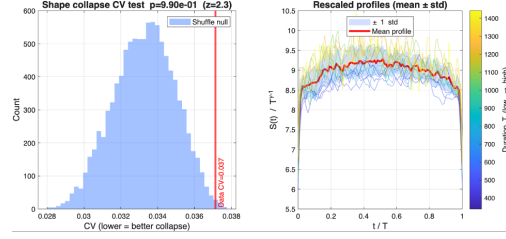
Therefore, the statistical difference from the null does not correspond to a strong collapse in absolute terms.

Overall, neither measure provides clear evidence of near-critical dynamics in the whole hippocampus during epilepsy. During epileptogenesis, the low DCC at fine bin sizes is not supported by a consistently significant shape collapse. During status epilepticus, a significant shape collapse is observed, but its quality is relatively poor, while the DCC remains low. These results are based on a single animal and are subject to the same limitations as the

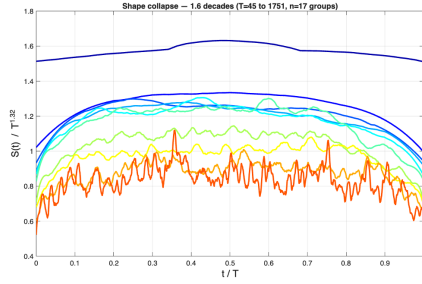
exponent analysis. The dorsal/ventral analysis is therefore considered next to determine whether the whole-hippocampus results may hide regional differences along the hippocampal axis.



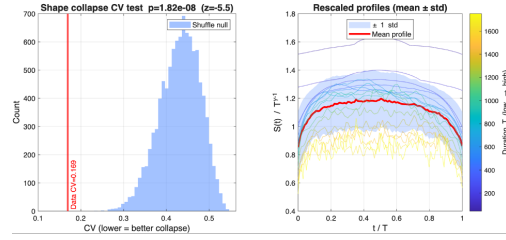
(a) Epileptogenesis - Avalanche shape collapse



(b) Epileptogenesis- Coefficient of variation of the rescaled profiles



(c) Status Epilepticus - Avalanche shape collapse



(d) Status Epilepticus - Coefficient of variation of the rescaled profiles.

Figure 3.62: Avalanche shape collapse and coefficient of variation of the rescaled avalanche profiles in the whole hippocampus during epileptogenesis and status epilepticus.

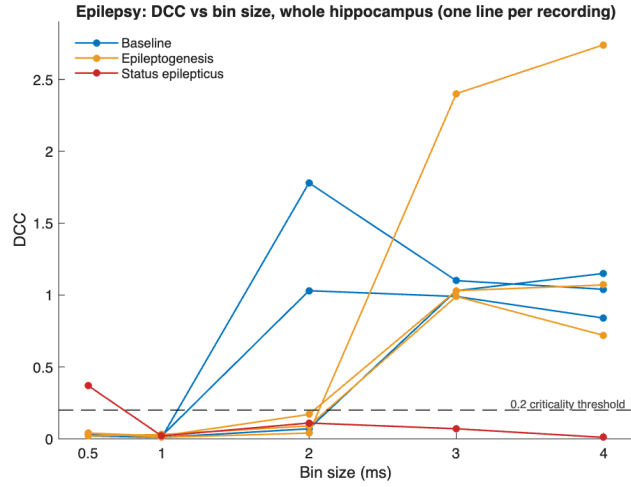


Figure 3.63: DCC as a function of temporal bin size for the whole hippocampus in the single epilepsy animal, one line per recording, coloured by phase.

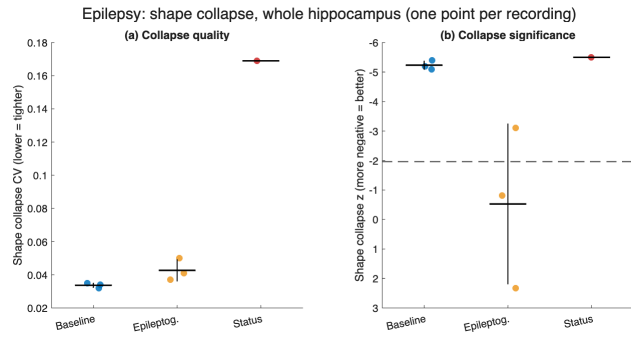


Figure 3.64: Avalanche shape collapse for the whole hippocampus in the single epilepsy animal, one point per recording. (a) Collapse quality (CV, lower = tighter). (b) Collapse significance (z against the shuffled null; more negative = better, dashed $z = -1.96$).

Dorsal and Ventral Subregions

As for the whole hippocampus, the DCC and the shape collapse should be

considered together, since neither measure is sufficient on its own (Tables 6.36, 6.37 and Figures 3.67, 3.68). The DCC shows the same dependence on bin size observed in the whole hippocampus, but this effect is mainly found in the dorsal regions. During epileptogenesis, dCA1 shows low DCC values at the finer bin sizes, but these increase strongly at 5 ms, reaching approximately 1 in all three recordings (0.96–1.13). dDG shows a similar but weaker pattern. In contrast, vDG remains mostly low, with an isolated increase at 3 ms (0.35–0.52). These increases at large bin sizes occur in the same range where the power-law fits become less reliable and should therefore not be interpreted as evidence of non-critical dynamics. During status epilepticus, the pattern is different across the hippocampal axis. dCA1 remains consistently low (≤ 0.05 at all bin sizes), while dDG shows higher and more variable values, reaching 1.11 at 0.5 ms. vDG also shows relatively high values at the finer bin sizes (0.39–0.59 up to 2 ms), which then decrease at larger bin sizes. Overall, the DCC does not identify a common near-critical range across the different subregions and epilepsy phases.

The shape collapse shows again a clearer regional difference. During epileptogenesis, dCA1 shows the most consistent collapse (Fig. 3.65). It combines low CV values (0.032–0.037) with highly significant differences from the shuffled null ($p \sim 10^{-11}$) and covers a relatively wide range of durations (~ 1.1 decades). The rescaled profiles also converge closely onto a common shape. dDG shows higher CV values (0.077–0.084) and a narrower range of durations (~ 0.4 – 0.5 decades). vDG has similarly low CV values (0.033–0.048), but the collapse covers a smaller range and the statistical

evidence is less consistent. In particular, track 7 does not differ significantly from the shuffled null ($p = 0.09$), and the ventral profiles are more scattered than those observed in dCA1.

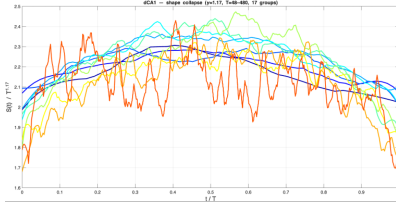
During status epilepticus, all three subregions show significant differences from the shuffled null, but the quality of the collapse is lower. dCA1 covers the widest range of durations (1.3 decades), but its CV increases to 0.064 and the profiles show a systematic separation with duration (Fig. 3.66).

vDG also shows a relatively high CV (0.087), and its profiles do not clearly converge onto a common shape. Therefore, statistical significance alone does not indicate a good shape collapse.

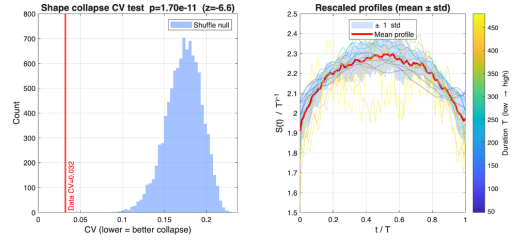
The dorsoventral pattern observed here differs from that found in the size and duration distributions. While the ventral regions showed steeper and more stable exponents, dCA1 shows the most consistent shape collapse.

However, this collapse occurs together with a strong increase in DCC at large bin sizes. The two measures therefore do not provide the same indication within dCA1, making it difficult to identify a clear and spatially consistent near-critical regime in the epileptic hippocampus.

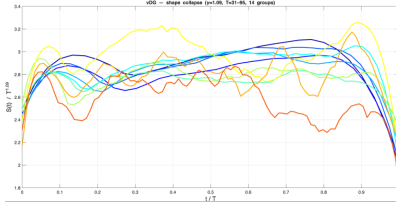
But again, these results are based on a single animal and the shape collapses cover at most about one decade of durations. Therefore, the apparently good shape collapse in dCA1 should be interpreted with caution and should not be considered sufficient evidence of criticality on its own.



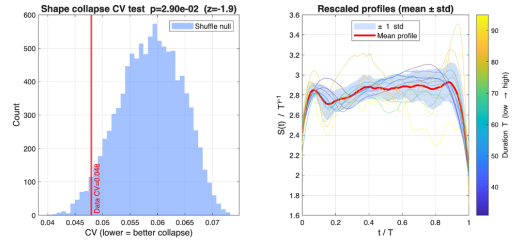
(a) Epileptogenesis - Avalanche shape collapse (dCA1)



(b) Epileptogenesis- Coefficient of variation of the rescaled profiles (dCA1)

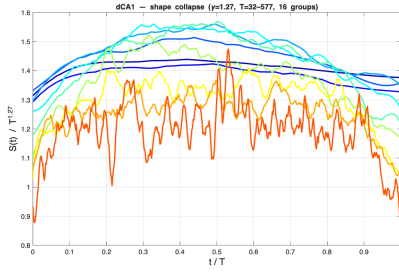


(c) Epileptogenesis - Avalanche shape collapse (vDG)

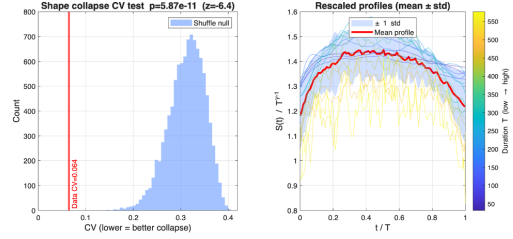


(d) Epileptogenesis - Coefficient of variation of the rescaled profiles (vDG)

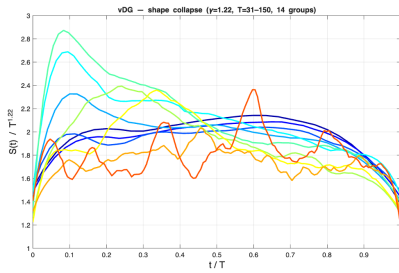
Figure 3.65: Avalanche shape collapse and coefficient of variation of the rescaled avalanche profiles in the dorsal and ventral hippocampus during epileptogenesis.



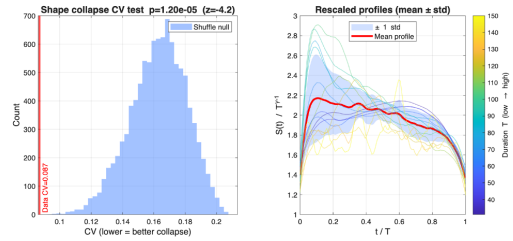
(a) Status Epilepticus - Shape collapse (dCA1)



(b) Status Epilepticus - CV of the rescaled profiles (dCA1)



(c) Status Epilepticus - Shape collapse (vDG)



(d) Status Epilepticus - CV of the rescaled profiles (vDG)

Figure 3.66: Avalanche shape collapse and coefficient of variation of the rescaled avalanche profiles in the dorsal and ventral hippocampus during status epilepticus.

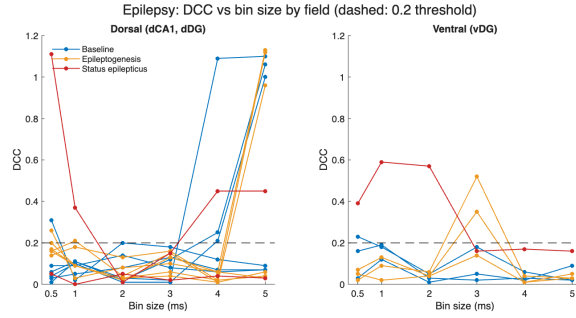


Figure 3.67: DCC as a function of temporal bin size by hippocampal field across epilepsy phases, one line per recording, coloured by phase.

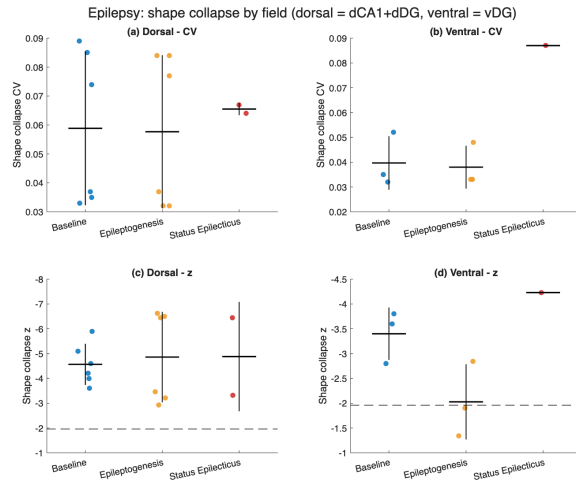


Figure 3.68: Avalanche shape collapse by hippocampal field across epilepsy phases(one point per recording): collapse quality (CV, lower = tighter) for dorsal (a) and ventral (b), and significance (z ; more negative = better, dashed $z = -1.96$) for dorsal (c) and ventral (d).

Chapter 4

Discussion

The aim of this work was to investigate whether the healthy mouse hippocampus operates close to a critical point, using in vivo Neuropixels 2.0 spike recordings, and whether this dynamic is altered in two pathological brain states: schizophrenia and epilepsy. The critical-brain hypothesis proposes that cortical and subcortical networks self-organise close to the critical point between an ordered, subcritical state and a disordered, supercritical state. This state is thought to maximise the network's dynamic range together with its ability to transmit and store information [Shew and Plenz, 2012]. If this hypothesis also applies to the hippocampus, healthy activity should show statistical features associated with criticality. Changes in the excitation/inhibition balance should then move the network away from this point. Schizophrenia and epilepsy are particularly useful to analyse because they are expected to move the network away from the critical point, in opposite directions. In particular, in a simplified view, schizophrenia is expected to move the network towards a subcritical state

[Nikulin *et al.*, 2012], while epilepsy is expected to move it towards a supercritical state [Arviv *et al.*, 2016]. However, these should be considered working hypotheses rather than established facts. In schizophrenia, human resting-state studies have not shown a clear shift towards a subcritical state. Instead, they have reported weaker scale-free and multifractal properties compared with healthy controls [Alamian *et al.*, 2022]. Avalanche measures have also been linked to cognitive function and possible inhibitory compensation, rather than simply to a loss of criticality [Zhao *et al.*, 2024]. The situation in epilepsy is less clear, and previous studies have reported different results. Since the healthy brain is already thought to operate close to criticality, epileptic activity may be better described as a change away from this state, however the direction of the change is still uncertain. Some studies have reported supercritical features, such as higher neural gain and larger avalanches during seizure activity [Arviv *et al.*, 2016]. Other studies have reported the opposite, with activity before and between seizures showing slightly subcritical dynamics [Meisel *et al.*, 2012]. In addition, single-neuron recordings from human epileptic cortex have found no clear transition, with the tissue remaining in a slightly subcritical state similar to healthy cortex, even close to seizure onset [Hagemann *et al.*, 2021]. Whole-brain studies also suggest that a strong increase in network coupling may be needed to drive the brain towards large-scale seizure dynamics [Burrows *et al.*, 2023].

Rather than relying on a single measure, criticality was assessed using several complementary features like avalanche size and duration distributions, the deviation from criticality coefficient (DCC), and

avalanche shape collapse. These measures were also compared with decorrelated null models. The branching ratio was also computed but, as explained previously, it was not informative because of the strong spatial subsampling of the recordings and was therefore not considered further. This multi-signature approach is important because no single measure is sufficient to demonstrate criticality on its own. A stronger conclusion can be reached only when several measures provide consistent evidence.

4.1 The healthy hippocampus operates near criticality

4.1.1 Reproducible near-critical signatures

Across the fifteen healthy recordings from four animals, the hippocampus reproduced several signatures consistent with near-critical dynamics like heavy-tailed, approximately power-law size and duration distributions, DCC values lower than the 0.2 threshold and a statistically significant avalanche shape collapse that was lost after neuronal correlations were removed by the decorrelation surrogate. The branching ratio was not considered one of these signatures. In fact it remained close to one not only in the actual recordings but also in both null models, so it could not distinguish genuine collective dynamics from surrogate activity. This is expected under strong spatial subsampling, which can push the branching-ratio estimate toward one regardless of the underlying state[Priesemann *et al.*, 2009]. For these reasons, the branching ratio was

considered unreliable for these populations.

Two complementary null models were used to test whether the observed structure could arise by chance. The autocorrelation null model randomly shuffles the sequence of population-activity bins in time. It preserves the distribution of instantaneous activity but removes its temporal order, and therefore tests whether the avalanche distributions depend on temporal structure rather than on the activity distribution alone [Ribeiro *et al.*, 2010]. The correlation null model instead applies an independent circular time shift to each neuron. It preserves each unit’s firing rate, inter-spike-interval distribution, and autocorrelation, while removing coordination between neurons. It therefore tests whether the avalanches depend on genuine interactions between neurons rather than on the combination of independently firing units [Louis *et al.*, 2010].

The two surrogates behave differently, which is itself informative. Removing cross-neuronal coordination with the correlation null significantly steepens the exponents and shifts the fits toward the lognormal, suggesting that the heavy tails depend on genuine spatial coordination. In contrast, removing only the temporal order with the autocorrelation null leaves the exponents close to those of the recorded data. Notably, the DCC criterion is satisfied by both surrogates, showing that it cannot by itself distinguish critical from non-critical activity in these recordings. It is therefore better treated as a necessary rather than a sufficient condition.

The shape collapse provides a more informative signature. The variability of the rescaled avalanche profiles in the real data is much lower than in the shuffled null, where profiles and durations were paired randomly (CV

≈ 0.05 ; $p \sim 10^{-6}$ to 10^{-8}). These features were also consistent across repeated tracks and animals, rather than being limited to a single recording. They therefore provide a useful benchmark for comparison with the pathological recordings [Sethna *et al.*, 2001].

4.1.2 Interpreting the exponent values

The fitted exponents did not match the canonical values first reported by Beggs and Plenz (2003) ($\alpha \approx 1.5$ for size and $\beta \approx 2$ for duration). Here, the size exponents ranged from approximately 2 to 2.8, while the duration exponents ranged from about 2.3 to 3. At first sight, these values may seem inconsistent with critical branching dynamics. However, we consider the possibility that the greater exponent magnitudes observed may be due to differences in preparation and recording methods.

Beggs and Plenz measured local field potentials from a few tens of electrodes in in vitro cortical slice cultures [Beggs and Plenz, 2003]. LFPs are low resolution, low-pass-filtered signals that combine the activity of many neurons. Therefore, an event detected on one electrode already represents activity from a relatively large population. In contrast, the present analysis uses individual action potentials from more than 400 neurons recorded in vivo. These two definitions of an avalanche are therefore not directly equivalent. Combining many spikes into a broad population signal, as in LFP recordings, can make the size distribution look like a power law with a low exponent, even when the underlying dynamics are not critical. Spike recordings are less affected by this bias. Therefore, spike-based avalanches are expected to have steeper exponents than

LFP-based avalanches when they come from similar underlying dynamics [Neto *et al.*, 2022]. Consistent with this, several in vivo spike studies report steeper exponents and dynamics that are close to criticality but slightly subcritical [Priesemann *et al.*, 2014, Ma *et al.*, 2019]. Large-scale in vivo recordings in mice also show similar scaling behaviour, but with values shifted from those reported in vitro [Morales *et al.*, 2023].

An additional important effect that must be taken into account is spatial subsampling. Even a dense Neuropixels probe records only a small fraction of the neurons in the hippocampus. Both theoretical and experimental studies have shown that subsampling can distort avalanche size and duration distributions and affect the estimated exponents and branching ratio [Priesemann *et al.*, 2009,

Neto *et al.*, 2022]. The exponent values should therefore not be directly compared with those from other studies or experimental preparations. However, because all conditions in this study were analysed using the same pipeline and subsampling, differences between conditions can still be interpreted, especially when considered together with the other criticality measures. It is also crucial to evaluate the strong dependence of the exponents on the temporal bin size, which was evident in the present results. Because avalanches are defined by joining consecutive active bins, the bin width determines the timescale over which activity is grouped into cascades. Changing the bin size can therefore affect both the fitted exponents and the quality of the shape collapse. The exponents can also vary with the ongoing brain or network state [Fontenele *et al.*, 2019], so they should not be considered fixed properties of the tissue.

Considering the methodology used and previous studies based on in vivo spiking activity, these exponent values are consistent with the hippocampus operating in a near-critical regime. They should therefore not be interpreted as evidence of a departure from criticality when compared with the classic in vitro values of 1.5 and 2. The exponent values should therefore be interpreted in the context of the recording method, spatial sampling, and temporal binning rather than used alone to determine whether the system is critical.

4.1.3 Near-critical, not critical

A more careful interpretation of the healthy results is that the hippocampus operates near, but slightly below, the critical point, in a weakly subcritical regime. This is consistent with findings from several in vivo spike studies in the cortex, where continuous external and internal input keeps the network active and close to criticality without reaching the unstable regime of true criticality [Priesemann *et al.*, 2014, Wilting and Priesemann, 2019].

A slightly subcritical state may be useful for a behaving animal because it allows the network to remain close to criticality while limiting the spread of activity. This is relevant for the pathological comparisons because the healthy state can be considered a baseline from which changes toward either more subcritical or more supercritical dynamics can be evaluated. It is also relevant to note that the present data cannot distinguish true criticality from self-organised quasi-criticality, in which homeostatic and adaptive mechanisms keep the network close to, rather than exactly at, the critical point [Kinouchi and Copelli, 2006]. The present observations,

namely a reproducible near-critical state with consistent relationships between the exponents, are compatible with both possibilities. Distinguishing between them would require perturbation experiments that measure how the dynamics recover over time, as done in cortex [Ma *et al.*, 2019], rather than the observational approach used here. This limitation is not specific to the present study but is a general limitation of avalanche analysis based on spontaneous activity. It therefore limits how strongly criticality can be claimed, both for the healthy and pathological recordings.

4.1.4 Region-specific dynamics along the dorsoventral axis

A distinctive aspect of this work is that criticality was not assessed only at the level of the whole hippocampus, but also along the dorsoventral axis. This aspect is less often investigated in the criticality literature, where the recorded structure is often treated as a single population or studied at the whole-brain level using EEG or MEG. In the present data, the dorsal and ventral subregions showed different dynamics. In particular, dorsal regions, and dorsal CA1 more specifically, remained closer to a scale-free, power-law regime, whereas ventral regions showed more lognormal fits and a wider range of exponents.

This spatial difference is consistent with the known functional and anatomical organisation of the hippocampal longitudinal axis. The dorsal and ventral hippocampus differ in their cortical and subcortical connectivity, gene expression, and physiological properties. They also have partly different functional roles, with the dorsal hippocampus more

involved in spatial and cognitive processing and the ventral hippocampus more involved in emotional, motivational, and stress-related processes [Strange *et al.*, 2014]. One possible interpretation of the present results is that these differences in connectivity and excitability are associated with different levels of proximity to criticality, with the ventral hippocampus showing a greater departure from scale-free dynamics. The greater occurrence of lognormal fits in the ventral regions may indicate the presence of a characteristic scale in their activity, rather than the scale-free behaviour expected near criticality.

However there are still some factors that should be taken into account. For example, dividing the population by subregion reduces the number of neurons contributing to each estimate and therefore increases the effects of spatial subsampling discussed above. Some of the dorsoventral differences could therefore reflect differences in the number of recorded neurons rather than differences in the underlying dynamics, although the same general pattern was observed across recordings. Second, distinguishing between a truncated power law and a lognormal distribution is difficult over a limited range, and small changes in the fitted distributions can affect the model comparison.

With these limitations in mind, the results suggest that near-critical dynamics are not uniform along the hippocampal dorsoventral axis. Treating the hippocampus as a single homogeneous population may therefore hide differences between its subregions. The ability to examine these differences is an important contribution of the present work.

4.2 Deviations from criticality in pathological states

Having established a reproducible near-critical baseline, the same pipeline was applied to two pathologies that, according to the critical brain framework, are expected to shift away from criticality in different directions. A key factor in this framework is the balance between excitation and inhibition. Schizophrenia is associated with reduced input to inhibitory interneurons and impaired long-range coordination, which may lead to a more subcritical state [Lewis *et al.*, 2005, Alamian *et al.*, 2022]. Epilepsy instead is characterised by hyperexcitable and hypersynchronous networks, which would traditionally be expected to produce a shift toward a more supercritical state [Beggs and Plenz, 2004]. However, this assumption is not firmly confirmed. The healthy brain is itself near-critical, and epileptic activity may represent a departure from criticality in either direction. In addition, preseizure and interseizure states have sometimes been reported to show subcritical dynamics [Meisel *et al.*, 2012, Zimmern, 2020].

4.2.1 Schizophrenia

The critical-brain hypothesis predicts a shift toward a more subcritical state in schizophrenia. One proposed mechanism is NMDA-receptor hypofunction, which may impair parvalbumin-positive interneurons, reduce inhibition, disrupt gamma-band synchrony and affect long-range coordination [Lewis *et al.*, 2005]. However, the available evidence comes from experimental models and recording methods that differ substantially

from the present study. Nikulin *et al.* reported reduced long-range temporal correlations in scalp EEG from patients, providing a population-level, non-invasive measure based on oscillatory activity [Nikulin *et al.*, 2012]. Seshadri *et al.* used a developmental NMDAR hypofunction model based on neonatal phencyclidine administration in rats and two-photon calcium imaging of the motor cortex, providing cellular-resolution measurements but with a slower imaging-based signal [Seshadri *et al.*, 2018]. Reviews of criticality in psychiatric disorders also report a general shift away from criticality, based mainly on human M/EEG studies [Alamian *et al.*, 2022]. None of these studies uses spike-based recordings from the hippocampus, so the current study provides a different level of description and examines a different brain region. The hippocampus is also highly relevant to schizophrenia. Increased hippocampal activity during rest is one of the most consistent findings in the disorder and has been linked to psychotic symptoms. This hyperactivity is thought to be related to reduced inhibition from parvalbumin interneurons and a shift in the excitation-inhibition balance towards excitation [Heckers and Konradi, 2015]. This is also the type of change expected to move network dynamics away from criticality. Against this background, the fit statistics provide a more complex picture than a simple subcritical shift. The distribution of preferred models changed considerably under ketamine injection, but not uniformly. At the whole hippocampus level, both size and duration distributions shifted away from power-law and truncated power-law fits toward the lognormal. For avalanche size, five of six acute and two of three chronic schizophrenia

recordings were best fitted by a lognormal distribution. In healthy animals, the results were more mixed, with two power-law, three truncated power-law, and four lognormal fits out of nine recordings. Overall, lognormal fits were more common in schizophrenia (83% in the acute condition and 67% in the chronic condition) than at baseline (44%). However, this difference was not statistically significant, likely because of the small number of recordings (χ^2 test of proportions, healthy vs acute schizophrenia, $p = 0.13$). Whole-hippocampus duration fits showed a similar shift, with four of six acute recordings best fit by a lognormal, compared with two of nine in healthy animals. Therefore, the fit results do not indicate an unchanged scale-free regime. A shift toward the lognormal can indicate the emergence of a characteristic event size and therefore a departure from scale-free dynamics.

The shift was also regionally structured and differed between size and duration. For size, it was mainly driven by the ventral hippocampus, where five of six acute fields were best fit by a lognormal. In contrast, the dorsal hippocampus retained, and even increased, its fraction of power-law fits, with six of nine acute dorsal fields showing a power-law fit compared with four of twelve in healthy animals. Duration fits at the field level, by contrast, remained predominantly truncated power-law in both regions. The stronger effect in the ventral hippocampus is consistent with the dorsoventral gradient already observed in the healthy data and is also notable because the ventral hippocampus has been implicated in circuit alterations associated with schizophrenia [Schobel *et al.*, 2013]. Nevertheless, it should be considered that the number of fits is small,

ranging from three to twelve per condition, so these fractions should be considered trends rather than precise effect sizes. In addition, the distributional results differ from the other criticality measures. The DCC remained within the near-critical range, and the shape collapse remained significant relative to the shuffled null ($p \sim 10^{-6}$ to 10^{-7}), close to the healthy baseline. However, the DCC did not distinguish the real recordings from surrogate data in these experiments, so its value is limited for detecting this type of change.

Overall, the schizophrenia data provide qualified evidence for a departure from scale-free organisation, most clearly for avalanche size and in the ventral hippocampus. However, the main signatures of near-critical dynamics remain present, and the fitted model type alone cannot determine whether the change specifically reflects a more subcritical state.

4.2.2 Epilepsy

Epilepsy represents a different pathological state, with hyperexcitable and hypersynchronous activity expected to shift the network toward a more supercritical state. In this state, active neurons are more likely to recruit additional neurons, leading to larger and longer avalanches. However, this prediction is debated and applies most directly to sustained seizure activity rather than to epilepsy in general [Zimmern, 2020]. The present study is useful in this context because it compares epileptogenesis with status epilepticus, distinguishing between two pathological states of the disease. The prediction of supercriticality applies most directly to the latter, which represents a sustained ictal state, and it is also where the heavier-tailed

distributions were observed.

Beggs and Plenz observed supercritical avalanche statistics during pharmacological disinhibition *in vitro*, showing a link between reduced inhibition and deviations toward heavier-tailed activity [Beggs and Plenz, 2004]. Meisel *et al.* described a progressive departure from criticality in human intracranial recordings as activity approached seizure, suggesting that seizure development involves a transition away from the critical state [Meisel *et al.*, 2012]. Arviv *et al.* found higher neural gain and larger avalanches in interictal MEG recordings from patients, providing evidence for a supercritical state even between seizures [Arviv *et al.*, 2016]. Corsi *et al.* used source-reconstructed high-density EEG to show that avalanche features can distinguish patients with temporal-lobe epilepsy from controls, highlighting the potential of these measures as biomarkers [Corsi *et al.*, 2024]. Computational network models also reproduce seizure emergence through changes in network excitability [Terry *et al.*, 2012]. All these studies mainly operate at a macroscopic cortical scale, *in vitro*, or *in silico*, with none that examines hippocampal spikes *in vivo*.

In this study, the clearest effect was a difference between the two epilepsy phases. At matched bin sizes, the status epilepticus distributions had consistently lower exponents than those during epileptogenesis. For example, at 0.5 ms, $\tau \approx 2.36$ versus 2.66–2.70, and $\alpha \approx 2.71$ versus 2.91–2.93. Smaller exponents indicate heavier tails and a relative increase in large and long avalanches. This is consistent with the expected direction of a shift toward supercriticality and with the self-amplifying activity observed during seizures [Beggs and Plenz, 2004, Arviv *et al.*, 2016]. Therefore, the

difference between the two phases is in the direction expected from the supercritical prediction, with status epilepticus showing the stronger shift. This directional change in the exponents is, however, the only clear signal. The model-selection analysis does not provide additional evidence for supercriticality. A pure power law is almost never preferred, and the ventral dentate gyrus remains dominated by lognormal size fits, as in the healthy data. This suggests that the lognormal preference could be a regional feature rather than a specific effect of epilepsy. Although two of the three ventral epileptogenesis recordings shift to an exponential duration fit, the pattern is not consistent across size and duration or between the two epilepsy phases. On its own, it therefore does not indicate a clear departure from near-critical dynamics.

The two other measures show a more mixed picture. The DCC is strongly dependent on bin size. In particular, during epileptogenesis, it is low at fine bins but rises sharply above one at 3–4 ms, reaching 2.4–2.7 in one recording. This occurs in the same range in which the fits become unreliable. During status epilepticus, by contrast, the DCC remains low throughout, with a maximum of 0.37. Because the DCC measures only the internal consistency between the fitted exponents, neither pattern provides evidence of criticality on its own. The shape collapse also gives similar outcome. During epileptogenesis, the rescaling exponent is close to one and the CV is low (0.037–0.050), but comparison with the shuffled null is inconsistent across recordings. Only one of three recordings reaches significance ($p \approx 10^{-3}$), while the others do not. Thus, a low CV alone is not sufficient to establish a shape collapse. During status epilepticus, the

collapse covers a wider range and is highly significant ($p \approx 10^{-8}$), but its quality is lower ($CV \approx 0.17$), and, the rescaled profiles remain visibly scattered rather than overlapping onto a single curve (Fig.3.66). Overall, neither measure provides clear evidence of near-critical dynamics in the whole hippocampus during either phase.

Assessing these measures along the dorsoventral axis provides only a slightly clearer picture. Dorsal CA1 during epileptogenesis shows the strongest collapse in the dataset ($CV \approx 0.03$, $p \sim 10^{-11}$), with profiles converging over about one decade of durations. However, the same region shows the bin-size-dependent increase in DCC at larger bins, and during status epilepticus its collapse becomes weaker ($CV \approx 0.06$), with the profiles showing a systematic separation with duration. The ventral dentate gyrus also reaches low CV values, but over a narrower range and with inconsistent significance. In addition, the DCC does not identify a near-critical bin range shared across subregions and phases. No subregion therefore shows a spatially consistent near-critical signature.

Taken together, the epilepsy analysis supports only a modest conclusion based on a single animal. The heavier tails observed during status epilepticus relative to epileptogenesis are consistent with the expected direction of a supercritical shift, but the other criticality measures neither confirm near-criticality nor provide a clear supercritical signature. The disagreement between measures may partly reflect the dynamic nature of criticality in epilepsy. Network dynamics may move toward and away from the critical point during the seizure cycle [Meisel *et al.*, 2012], so an avalanche analysis over the entire recording may obscure changes that occur

over shorter timescales.

4.3 Methodological considerations and limitations

4.3.1 Bin size and the definition of an avalanche

One important methodological consideration is that avalanche statistics depends on several analysis choices rather than being determined by the data alone. The temporal bin width determines the timescale at which spikes are grouped into cascades, and all criticality measures depend on this choice. This dependence is systematic rather than random. When the bin is too small, genuine cascades can be divided into many short events, making the dynamics appear artificially subcritical. When the bin is too large, temporally separate cascades can be merged into a few very large avalanches, making the dynamics appear artificially supercritical. As a result, the fitted size exponent α generally increases with bin width [Beggs and Plenz, 2003].

There is no universal correct bin size. A common conventional approach, introduced with the first avalanche recordings, is to set the bin width to the mean inter-event interval [Beggs and Plenz, 2003]. However, this approach assumes a clear separation between the fast dynamics inside an avalanche and the slower periods between avalanches. This separation is often present in weakly driven in vitro cultures, which therefore show less bin-size dependence. In vivo activity, on the contrary, is continuously driven, so this

separation is less clear and avalanche statistics are more strongly dependent on bin size [Neto *et al.*, 2022]. The bin-size dependence observed throughout this study is therefore expected for in vivo spiking data and reflects the driven regime rather than simply a limitation of the analysis. A practical consequence is that the mean inter-event interval, and therefore the corresponding bin width, depends on firing rate, which can differ between brain states and pathological conditions [Fontenele *et al.*, 2019]. Comparing raw exponents between healthy, schizophrenia and epileptic recordings at the same bin size can therefore mix changes in network dynamics with changes in firing rate. A fairer comparison could be adjusting the bin size according to the event rate of each condition. For the same reason, absolute exponent values should be interpreted with caution. The definition of the type of event being analysed is a related and equally relevant choice. Using spikes rather than the local field potential changes what is defined as an avalanche and, as discussed above, leads to different exponent values compared with the classic in vitro LFP literature. LFP or multi-unit measurements can also be affected by measurement overlap, where neighbouring electrodes capture activity from overlapping regions and may therefore produce artificial shared events that increase avalanche sizes. Spike-based detection avoids this problem, but instead involves strong spatial subsampling [Neto *et al.*, 2022, Priesemann *et al.*, 2009]. Neither approach is free from at least a type of bias. The results should therefore be interpreted mainly through features that remain consistent across methodological choices rather than through the precise exponent values obtained with a particular method.

4.3.2 Spatial subsampling

Neuropixels 2.0 provides a high number of recorded units, but it still samples only a small and spatially biased fraction of the hippocampal population. Subsampling can affect avalanche size and duration distributions, including their tails, and can also bias the branching ratio [Wilting and Priesemann, 2019]. In this study, one way to reduce the impact of this limitation was to use the same analysis pipeline for all recordings. Because healthy and pathological recordings, as well as dorsal and ventral regions, were processed in the same way, the effects of subsampling should be at least partly shared. Relative differences can therefore be considered more reliable than absolute values. This limitation is particularly notable for the dorsoventral analysis, because dividing the hippocampus into smaller regions further reduces the number of recorded neurons available for the analysis.

4.3.3 Statistical power and generality

The healthy and schizophrenia conclusions are based on multiple recordings and animals, while the epilepsy analysis is based on a single animal. This limits the generality of the epilepsy results and prevents statistical inference across subjects for this condition. Comparisons with previous studies also involve different species, experimental preparations, signals and brain regions. Therefore, agreement with the literature should mainly be interpreted in terms of direction rather than as a direct quantitative comparison. The same nominal exponent can have different meanings when

estimated from human MEG, in vitro LFP, or mouse hippocampal spikes. These limitations do not affect the internal comparisons made using the same analysis pipeline, but they limit how broadly the absolute values can be generalised.

Chapter 5

Conclusions

This study aimed to test whether the mouse hippocampus operates near a critical point, using in vivo Neuropixels 2.0 spike recordings, and whether this organisation is altered under two pathological perturbations: a model of schizophrenia induced by ketamine, and an induced model of epilepsy.

Across the fifteen healthy recordings from four animals, the results support an affirmative but nuanced answer to the first question. The hippocampus displays reproducible near-critical dynamics, most plausibly a slightly subcritical regime rather than exactly at the critical point.

The strength of this conclusion is supported by the convergence of several signatures rather than on any single measure. The heavy-tailed, approximately power-law size and duration distributions were consistent with the empirical size-duration scaling, and a significant avalanche shape collapse was present in the recorded data but did not persist in the null models. Two null models helped to interpret these findings. Abolishing cross-neuronal coordination steepened the exponents and shifted the

preferred fits toward the lognormal, indicating that the heavy tails depend on neuronal coordination, while removing only the temporal ordering left them largely intact. One measure was deliberately excluded. The branching ratio remained close to one in both the data and the surrogates and was therefore set aside as uninformative under the strong spatial subsampling of these recordings [Levina and Priesemann, 2017].

A second contribution is that this organisation is not spatially uniform. Resolving the analysis along the dorsoventral axis showed that dorsal fields, and dorsal CA1 in particular, more frequently exhibited power-law or truncated power-law fits, whereas the ventral fields were more often best described by lognormal fits. This gradient is consistent with the known functional and anatomical differentiation of the hippocampal longitudinal axis [Strange *et al.*, 2014], and indicates that studies treating the hippocampus as homogeneous, or measuring it only through whole-structure or whole-brain signals, may average over meaningful internal heterogeneity. The pathological perturbations gave weaker and more mixed results than expected under a simple opposite direction hypothesis, and the two disorders behaved differently. Under schizophrenia, the distribution of preferred fits shifted markedly toward the lognormal, most clearly for size and in the ventral hippocampus, while the dorsal hippocampus paradoxically retained or increased its power-law fraction. The other measures, DCC and shape collapse, meanwhile, remained close to the healthy baseline. These results therefore point in different directions: the regional, size-specific shift toward lognormal fits indicates a local departure from scale-free size distributions, yet it coexists with a shape collapse and

exponent consistency that remain essentially unchanged from the healthy state. The schizophrenia data are therefore best described as mixed rather than as evidence of a clear subcritical shift. They indicate, at most, a subtle and regionally confined departure from scale-free size distributions, and do not establish that the near-critical operating point of the hippocampus is genuinely shifted. The epilepsy analysis, limited to a single animal, produced smaller exponents, and the heavier tails observed during status epilepticus relative to epileptogenesis are consistent with a shift in the direction expected for supercritical dynamics. The DCC and shape collapse, however, were too sensitive by bin size and too inconsistent across recordings and subregions to establish the direction of any departure on their own. The supercritical interpretation is therefore based mainly on the exponent estimates and remains uncertain.

5.1 Implications

For basic neuroscience, these results extend the study of near-critical dynamics beyond the neocortex, where it has been mainly investigated, to a subcortical structure, using in vivo, spike-based recordings rather than the broader signals used in much of the literature. The dorsoventral gradient further indicates that internal anatomical organisation should be treated as a variable in criticality studies rather than averaged away. For translational neuroscience, the framework has already shown that avalanche metrics can discriminate patient populations from macroscopic recordings [Corsi *et al.*, 2024]. This motivates further investigation of avalanche statistics as

potential markers of altered network dynamics and excitation-inhibition balance.

This framework could also have implications for treatment. Instead of targeting symptoms directly, interventions could aim to restore the excitation-inhibition balance that helps keep the network close to its critical state. For example, this could involve increasing inhibitory control in schizophrenia or reducing excessive excitability in epileptic tissue. The distance from criticality could then be used as a functional measure of whether an intervention is moving the network towards a healthier state, alongside more traditional symptom-based measures. However, as discussed above, no single avalanche measure is sufficient on its own. Such an approach would therefore need to combine several measures and compare them with appropriate null models.

At the same time, the mixed results in the pathological conditions show that no single avalanche measure should be treated as a reliable biomarker. Even in the healthy recordings, the near-critical interpretation was supported only when several measures were considered together and compared with appropriate null models.

5.2 Future directions

Several directions follow directly from the limitations of this work. The epilepsy analysis should be repeated in additional animals and, because criticality-related dynamics in epilepsy are thought to vary over the seizure cycle [Meisel *et al.*, 2012], resolved in time rather than averaged over a

session, since a static analysis may blur genuinely dynamic departures. The strong dependence of the measures on temporal bin size, together with the bias introduced by spatial subsampling, motivates the use of subsampling-robust estimators of the branching parameter alongside distribution-based measures [Wilting and Priesemann, 2019], together with a more systematic treatment of bin-size dependence. Moving from the observational design used here to perturbation and recovery experiments would help distinguish true criticality from self-organised quasi-criticality [Kinouchi and Copelli, 2006, Ma *et al.*, 2019]. Finally, resolving avalanches by cell type or cortical layer, and relating the near-critical operating point and its dorsoventral gradient to behaviour and hippocampal functions such as memory, would connect the statistical signatures studied here to the computational advantages that motivate the critical-brain hypothesis in the first place [Shew *et al.*, 2011]. Together, these steps would move the largely descriptive picture established in this thesis toward a causal account of where, when, and why the hippocampus exhibits near-critical dynamics, and of how that dynamical state is altered in disease.

Chapter 6

List of Tables

Table 6.1: Size (τ) and duration (α) exponents for whole hippocampus, over five temporal bins. Animals. First three linear tracks for the three "disease model" mice

Animal	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	Animal	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$
Healthy Control1		0.5	2.56	11	2.80	5	Healthy Baseline 1(Scz)3		0.5	2.54	6	3.08	4
Healthy Control1		1	2.69	48	2.97	15	Healthy Baseline 1(Scz)3		1	2.36	18	2.75	8
Healthy Control1		2	2.50	347	2.68	61	Healthy Baseline 1(Scz)3		2	2.42	124	2.74	30
Healthy Control1		3	2.43	1486	2.46	162	Healthy Baseline 1(Scz)3		3	2.43	538	2.60	87
Healthy Control1		4	2.40	4668	2.43	415	Healthy Baseline 1(Scz)3		4	2.31	1852	2.44	226
Healthy Control2		0.5	2.44	11	2.88	6	Healthy Baseline(Epi) 1		0.5	2.68	34	2.87	13
Healthy Control2		1	2.66	51	2.89	15	Healthy Baseline(Epi) 1		1	2.51	321	2.67	70
Healthy Control2		2	2.46	320	2.62	54	Healthy Baseline(Epi) 1		2	2.06	5737	2.09	594
Healthy Control2		3	2.48	1337	2.59	156	Healthy Baseline(Epi) 1		3	2.13	78921	2.12	5171
Healthy Control2		4	2.37	3511	2.59	377	Healthy Baseline(Epi) 1		4	1.19	2	1.21	2
Healthy Control3		0.5	2.42	11	2.86	6	Healthy Baseline(Epi) 2		0.5	2.64	32	2.82	12
Healthy Control3		1	2.63	53	2.91	16	Healthy Baseline(Epi) 2		1	2.51	283	2.68	63
Healthy Control3		2	2.48	352	2.64	59	Healthy Baseline(Epi) 2		2	1.84	2231	2.49	816
Healthy Control3		3	2.45	1464	2.51	166	Healthy Baseline(Epi) 2		3	1.98	40578	2.08	3637

Table 6.1

Animal	TrackBin (ms) τ (S.) $x_{\min}^S$ α (D.) $x_{\min}^D$						Animal	TrackBin (ms) τ (S.) $x_{\min}^S$ α (D.) $x_{\min}^D$					
Healthy Control	3	4	2.33	3700	2.29	286	Healthy Baseline(Epi)	2	4	1.17	2	1.22	2
Healthy Control	4	0.5	2.48	9	2.96	5	Healthy Baseline(Epi)	3	0.5	2.68	28	2.98	11
Healthy Control	4	1	2.51	33	2.81	11	Healthy Baseline(Epi)	3	1	2.48	231	2.67	53
Healthy Control	4	2	2.38	196	2.59	38	Healthy Baseline(Epi)	3	2	2.28	4657	2.37	556
Healthy Control	4	3	2.33	718	2.44	91	Healthy Baseline(Epi)	3	3	1.87	20944	1.89	1593
Healthy Control	4	4	1.85	755	2.41	204	Healthy Baseline(Epi)	3	4	1.30	1687	1.35	228
Healthy Control	5	0.5	2.42	8	2.75	4	Healthy Baseline 2(Scz)	1	0.5	2.84	6	3.17	4
Healthy Control	5	1	2.50	32	2.87	11	Healthy Baseline 2(Scz)	1	1	2.68	23	2.95	10
Healthy Control	5	2	2.46	195	2.67	37	Healthy Baseline 2(Scz)	1	2	2.67	194	2.76	44
Healthy Control	5	3	2.44	733	2.58	96	Healthy Baseline 2(Scz)	1	3	2.74	1282	2.86	224
Healthy Control	5	4	2.48	2168	2.57	212	Healthy Baseline 2(Scz)	1	4	2.99	7305	2.94	889
Healthy Control	6	0.5	2.46	8	3.00	5	Healthy Baseline 2(Scz)	2	0.5	2.83	6	3.17	4
Healthy Control	6	1	2.60	33	2.90	11	Healthy Baseline 2(Scz)	2	1	2.63	22	2.84	9
Healthy Control	6	2	2.52	199	2.73	39	Healthy Baseline 2(Scz)	2	2	2.70	190	2.87	47
Healthy Control	6	3	2.51	789	2.64	106	Healthy Baseline 2(Scz)	2	3	2.61	1054	2.73	178
Healthy Control	6	4	2.21	1636	2.47	218	Healthy Baseline 2(Scz)	2	4	2.73	4778	2.80	621
Healthy Baseline 1(Scz)	1	0.5	2.61	6	3.16	4	Healthy Baseline 2(Scz)	3	0.5	2.83	6	3.20	4
Healthy Baseline 1(Scz)	1	1	2.41	17	2.84	8	Healthy Baseline 2(Scz)	3	1	2.59	20	2.88	9
Healthy Baseline 1(Scz)	1	2	2.44	119	2.77	30	Healthy Baseline 2(Scz)	3	2	2.67	166	2.89	42
Healthy Baseline 1(Scz)	1	3	2.51	567	2.64	93	Healthy Baseline 2(Scz)	3	3	2.45	735	2.65	146
Healthy Baseline 1(Scz)	1	4	2.44	2171	2.42	238	Healthy Baseline 2(Scz)	3	4	2.27	2267	2.56	432
Healthy Baseline 1(Scz)	2	0.5	2.62	6	3.17	4							
Healthy Baseline 1(Scz)	2	1	2.42	17	2.85	8							
Healthy Baseline 1(Scz)	2	2	2.43	117	2.73	29							
Healthy Baseline 1(Scz)	2	3	2.49	560	2.56	85							
Healthy Baseline 1(Scz)	2	4	2.14	1255	2.30	195							

Table 6.2: Size (τ) and duration (α) exponents for the dorsal hippocampal subregions, over five temporal bins. Control denotes the healthy control animal, Scz 1 and Scz 2 the two schizophrenia model animals, and Epi the epilepsy model mouse. Same tracks as Table 6.1.

Animal	Region	Track	Bin	(ms)	τ	(S.)	$x_{\min}^S$	α	(D.)	$x_{\min}^D$	Animal	Region	Track	Bin	(ms)	τ	(S.)	$x_{\min}^S$	α	(D.)	$x_{\min}^D$
ControldCA1	LT2			0.5	2.45	3	3.37	2	Epi	dCA1	LT1			0.5	2.88	6	3.18	4			
ControldCA1	LT2			1	2.37	4	2.79	2	Epi	dCA1	LT1			1	2.78	21	2.98	9			
ControldCA1	LT2			2	2.33	8	2.90	4	Epi	dCA1	LT1			2	2.44	152	2.68	40			
ControldCA1	LT2			3	2.30	13	2.78	5	Epi	dCA1	LT1			3	2.39	840	2.54	147			
ControldCA1	LT2			4	2.21	18	2.74	7	Epi	dCA1	LT1			4	2.54	4744	2.67	612			
ControldCA1	LT3			0.5	2.50	3	3.30	2	Epi	dCA1	LT2			0.5	2.90	6	3.21	4			
ControldCA1	LT3			1	2.37	4	3.14	3	Epi	dCA1	LT2			1	2.78	20	3.02	9			
ControldCA1	LT3			2	2.30	8	2.81	4	Epi	dCA1	LT2			2	2.37	122	2.66	37			
ControldCA1	LT3			3	2.33	14	2.95	6	Epi	dCA1	LT2			3	2.42	811	2.62	146			
ControldCA1	LT3			4	2.28	21	2.72	7	Epi	dCA1	LT2			4	2.51	4125	2.67	560			
ControldCA1	LT4			0.5	2.49	3	3.30	2	Epi	dCA1	LT3			0.5	2.95	6	3.27	4			
ControldCA1	LT4			1	2.36	4	3.16	3	Epi	dCA1	LT3			1	2.77	18	2.97	8			
ControldCA1	LT4			2	2.36	9	2.83	4	Epi	dCA1	LT3			2	2.29	86	2.61	29			
ControldCA1	LT4			3	2.38	15	2.96	6	Epi	dCA1	LT3			3	2.18	437	2.48	105			
ControldCA1	LT4			4	2.27	20	2.73	7	Epi	dCA1	LT3			4	2.33	2257	2.56	374			
ControldCA1	LT5			0.5	2.30	2	3.37	2	Epi	dDG	LT1			0.5	2.76	4	3.18	3			
ControldCA1	LT5			1	2.23	4	2.92	2	Epi	dDG	LT1			1	2.50	8	2.72	4			
ControldCA1	LT5			2	2.17	6	2.76	3	Epi	dDG	LT1			2	2.36	24	2.71	9			
ControldCA1	LT5			3	2.19	10	2.73	4	Epi	dDG	LT1			3	1.98	26	2.20	20			
ControldCA1	LT5			4	2.16	14	2.70	5	Epi	dDG	LT1			4	2.62	101	2.90	21			
ControldCA1	LT6			0.5	2.21	3	3.36	2	Epi	dDG	LT2			0.5	2.73	4	3.16	3			
ControldCA1	LT6			1	2.19	4	2.88	3	Epi	dDG	LT2			1	2.48	8	2.72	4			
ControldCA1	LT6			2	2.17	7	2.74	3	Epi	dDG	LT2			2	2.30	23	2.57	8			
ControldCA1	LT6			3	2.14	10	2.72	4	Epi	dDG	LT2			3	1.94	25	2.19	8			
ControldCA1	LT6			4	2.09	14	2.70	5	Epi	dDG	LT2			4	2.69	104	3.01	21			
ControldCA1	LT8			0.5	2.34	2	3.48	2	Epi	dDG	LT3			0.5	2.83	4	2.80	2			
ControldCA1	LT8			1	2.29	4	2.96	2	Epi	dDG	LT3			1	2.51	7	2.83	4			
ControldCA1	LT8			2	2.22	6	2.81	3	Epi	dDG	LT3			2	2.28	19	2.67	8			
ControldCA1	LT8			3	2.23	10	2.78	4	Epi	dDG	LT3			3	1.95	22	2.25	8			
ControldCA1	LT8			4	2.17	14	2.73	5	Epi	dDG	LT3			4	2.70	88	3.10	20			
Scz 2	dDG	LT1		0.5	2.71	3	3.14	2	Scz 1	dCA1	LT1			0.5	3.20	2	3.94	2			
Scz 2	dDG	LT1		1	2.43	5	2.84	5	Scz 1	dCA1	LT1			1	3.21	4	3.64	3			
Scz 2	dDG	LT1		2	2.32	13	2.73	6	Scz 1	dCA1	LT1			2	2.90	9	3.13	5			
Scz 2	dDG	LT1		3	2.29	28	2.65	10	Scz 1	dCA1	LT1			3	2.80	22	2.95	9			

Table 6.2

Animal	Region	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	Animal	Region	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$
Scz 2	dDG	LT1	4	2.36	57	2.70	16	Scz 1	dCA1	LT1	4	2.78	48	2.97	17
Scz 2	dDG	LT2	0.5	2.74	3	3.21	2	Scz 1	dCA1	LT2	0.5	3.17	2	3.89	2
Scz 2	dDG	LT2	1	2.49	5	2.92	3	Scz 1	dCA1	LT2	1	3.15	4	3.56	3
Scz 2	dDG	LT2	2	2.33	12	2.62	5	Scz 1	dCA1	LT2	2	2.89	10	3.02	5
Scz 2	dDG	LT2	3	2.29	25	2.63	9	Scz 1	dCA1	LT2	3	2.70	23	2.93	10
Scz 2	dDG	LT2	4	2.33	51	2.69	15	Scz 1	dCA1	LT2	4	2.66	51	2.87	18
Scz 2	dDG	LT3	0.5	2.68	3	3.11	2	Scz 1	dCA1	LT3	0.5	3.19	2	4.00	2
Scz 2	dDG	LT3	1	2.42	5	2.84	4	Scz 1	dCA1	LT3	1	2.89	3	3.02	2
Scz 2	dDG	LT3	2	2.26	12	2.58	5	Scz 1	dCA1	LT3	2	2.85	8	3.26	5
Scz 2	dDG	LT3	3	2.19	22	2.63	9	Scz 1	dCA1	LT3	3	2.71	17	3.00	8
Scz 2	dDG	LT3	4	2.37	48	2.82	14	Scz 1	dCA1	LT3	4	2.63	35	2.85	13

Table 6.3: Size (τ) and duration (α) exponents for the *ventral* hippocampal subregions, over five temporal bins. Control denotes the healthy control animal, Scz 1 and Scz 2 the two schizophrenia model animals, and Epi the epilepsy model mouse. Same tracks as Table 6.1.

Animal	Region	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	Animal	Region	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$
Control	vDG	LT2	0.5	2.68	6	3.07	4	Scz 2	vDG	LT1	0.5	2.74	2	3.49	2
Control	vDG	LT2	1	2.71	18	3.04	8	Scz 2	vDG	LT1	1	2.54	3	2.89	2
Control	vDG	LT2	2	2.26	40	2.33	11	Scz 2	vDG	LT1	2	2.37	6	2.90	4
Control	vDG	LT2	3	2.76	236	2.84	43	Scz 2	vDG	LT1	3	2.27	11	2.68	5
Control	vDG	LT2	4	2.60	515	2.52	62	Scz 2	vDG	LT1	4	2.16	15	2.75	8
Control	vDG	LT3	0.5	2.56	6	2.95	4	Scz 2	vDG	LT2	0.5	2.79	2	3.49	2
Control	vDG	LT3	1	2.63	19	2.96	8	Scz 2	vDG	LT2	1	2.56	3	2.88	2
Control	vDG	LT3	2	2.30	41	2.36	11	Scz 2	vDG	LT2	2	2.45	7	2.91	4
Control	vDG	LT3	3	2.73	196	2.83	35	Scz 2	vDG	LT2	3	2.33	12	2.82	6
Control	vDG	LT3	4	2.62	388	2.65	50	Scz 2	vDG	LT2	4	2.18	15	2.72	8
Control	vDG	LT4	0.5	2.53	6	2.92	4	Scz 2	vDG	LT3	0.5	2.75	2	3.43	2
Control	vDG	LT4	1	2.60	20	2.89	8	Scz 2	vDG	LT3	1	2.67	4	3.23	3
Control	vDG	LT4	2	2.25	43	2.30	11	Scz 2	vDG	LT3	2	2.44	8	2.78	4

Table 6.3

Animal	Region	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	Animal	Region	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$
Control	vDG	LT4	3	2.68	209	2.76	36	Scz 2	vDG	LT3	3	2.33	15	2.78	7
Control	vDG	LT4	4	2.67	468	2.68	60	Scz 2	vDG	LT3	4	2.07	15	2.81	11
Control	vDG	LT5	0.5	2.59	5	2.82	3	Epi	vDG	LT1	0.5	3.01	4	3.40	3
Control	vDG	LT5	1	2.66	16	2.96	7	Epi	vDG	LT1	1	2.95	9	3.25	5
Control	vDG	LT5	2	2.37	39	2.52	12	Epi	vDG	LT1	2	2.93	29	3.15	11
Control	vDG	LT5	3	2.64	166	2.78	32	Epi	vDG	LT1	3	2.99	79	3.11	21
Control	vDG	LT5	4	2.46	299	2.63	50	Epi	vDG	LT1	4	2.84	150	2.90	31
Control	vDG	LT6	0.5	2.64	5	2.88	3	Epi	vDG	LT2	0.5	2.88	4	3.26	3
Control	vDG	LT6	1	2.62	14	2.86	6	Epi	vDG	LT2	1	2.75	9	3.10	5
Control	vDG	LT6	2	2.47	41	2.68	13	Epi	vDG	LT2	2	2.96	30	3.06	10
Control	vDG	LT6	3	2.78	154	2.87	30	Epi	vDG	LT2	3	2.80	67	2.85	16
Control	vDG	LT6	4	2.69	313	2.63	42	Epi	vDG	LT2	4	2.92	131	3.02	27
Control	vDG	LT8	0.5	2.65	5	2.88	3	Epi	vDG	LT3	0.5	2.79	4	3.16	3
Control	vDG	LT8	1	2.65	14	2.88	6	Epi	vDG	LT3	1	2.75	10	2.98	5
Control	vDG	LT8	2	2.48	39	2.69	13	Epi	vDG	LT3	2	2.83	32	2.97	11
Control	vDG	LT8	3	2.80	156	2.95	32	Epi	vDG	LT3	3	2.26	43	2.54	14
Control	vDG	LT8	4	2.71	329	2.78	52	Epi	vDG	LT3	4	2.92	163	2.90	29
Scz 2	vCA3	LT1	0.5	3.19	2	2.98	2	Scz 1	vDG	LT1	0.5	3.12	3	3.13	2
Scz 2	vCA3	LT1	1	2.93	3	3.26	2	Scz 1	vDG	LT1	1	2.98	7	3.13	4
Scz 2	vCA3	LT1	2	2.59	4	3.07	3	Scz 1	vDG	LT1	2	2.63	24	2.85	10
Scz 2	vCA3	LT1	3	2.45	6	2.93	4	Scz 1	vDG	LT1	3	2.73	79	2.95	25
Scz 2	vCA3	LT1	4	2.37	9	2.82	5	Scz 1	vDG	LT1	4	2.74	221	2.87	54
Scz 2	vCA3	LT2	0.5	3.11	2	3.81	2	Scz 1	vDG	LT2	0.5	3.16	3	3.18	2
Scz 2	vCA3	LT2	1	2.85	3	3.20	2	Scz 1	vDG	LT2	1	2.87	6	3.19	4
Scz 2	vCA3	LT2	2	2.52	4	3.00	3	Scz 1	vDG	LT2	2	2.60	21	2.83	9
Scz 2	vCA3	LT2	3	2.36	6	2.84	4	Scz 1	vDG	LT2	3	2.72	68	2.94	22
Scz 2	vCA3	LT2	4	2.28	9	2.74	5	Scz 1	vDG	LT2	4	2.80	185	2.97	47
Scz 2	vCA3	LT3	0.5	3.02	2	3.75	2	Scz 1	vDG	LT3	0.5	3.06	3	3.10	2
Scz 2	vCA3	LT3	1	2.78	3	3.16	2	Scz 1	vDG	LT3	1	2.91	7	3.09	4
Scz 2	vCA3	LT3	2	2.47	4	2.96	3	Scz 1	vDG	LT3	2	2.59	24	2.84	10
Scz 2	vCA3	LT3	3	2.33	6	2.81	4	Scz 1	vDG	LT3	3	2.68	78	2.89	24
Scz 2	vCA3	LT3	4	2.26	10	2.71	5	Scz 1	vDG	LT3	4	2.71	212	2.74	46

Table 6.4: Size (τ) and duration (α) exponents recovered from the correlation null model (whole hippocampus), over five temporal bins. NaN denotes a failed or degenerate MLE fit. The correlation null strongly distorts the exponents relative to the intact data. Same tracks as Table 6.1.

Model	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	Model	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	
Control	2	0.5	1.85	2	3.72	6	Control	1	0.5	1.68	1	3.82	5	
Control	2	1	3.26	84	3.19	25	Control	1	1	3.29	55	3.40	20	
Control	2	2	3.25	2854	3.28	504	Control	1	2	3.07	1237	3.19	258	
Control	2	3	2.65	41859	2.65	4981	Control	1	3	2.39	11570	2.31	1421	
Control	2	4	NaN	NaN	NaN	NaN	Control	1	4	1.71	61971	1.72	6447	
Control	3	0.5	1.82	2	3.92	7	Scz	1	1	0.5	1.87	1	2.32	1
Control	3	1	3.26	95	3.37	31	Scz	1	1	1	3.19	24	3.58	12
Control	3	2	3.20	3336	2.97	470	Scz	1	1	2	3.25	382	3.32	95
Control	3	3	3.45	94056	3.43	10532	Scz	1	1	3	3.24	4367	3.25	728
Control	3	4	NaN	NaN	NaN	NaN	Scz	1	1	4	2.68	22913	3.32	4859
Control	4	0.5	3.50	13	3.82	7	Scz	1	2	0.5	1.88	1	2.34	1
Control	4	1	3.24	102	3.36	33	Scz	1	2	1	3.04	21	3.62	12
Control	4	2	3.09	3743	3.13	632	Scz	1	2	2	3.22	361	3.26	89
Control	4	3	3.37	129171	3.42	14995	Scz	1	2	3	3.19	4043	2.88	517
Control	4	4	NaN	NaN	NaN	NaN	Scz	1	2	4	2.88	26294	3.16	4496
Control	5	0.5	1.66	1	4.12	6	Scz	1	3	0.5	1.90	1	2.37	1
Control	5	1	3.24	59	3.34	21	Scz	1	3	1	2.98	19	3.05	8
Control	5	2	3.15	1482	3.22	291	Scz	1	3	2	3.20	325	3.24	82
Control	5	3	3.15	25338	2.98	3017	Scz	1	3	3	3.36	3521	3.35	592
Control	5	4	1.38	14594	1.38	1527	Scz	1	3	4	3.45	31662	3.46	4212
Control	6	0.5	1.67	1	3.82	5	Scz	2	1	0.5	3.52	7	3.38	4
Control	6	1	3.29	59	3.40	20	Scz	2	1	1	3.21	27	3.30	12
Control	6	2	3.22	1435	3.19	258	Scz	2	1	2	3.16	382	3.21	94
Control	6	3	3.33	27309	2.31	1421	Scz	2	1	3	2.77	3033	2.84	527
Control	6	4	1.84	70929	1.72	6447	Scz	2	1	4	2.96	33445	2.87	4166
Scz	2	2	0.5	3.26	6	3.40	4	Epi	1	0.5	3.40	16	3.34	39
Scz	2	2	1	3.26	27	3.34	12	Epi	1	1	3.27	162	3.24	746
Scz	2	2	2	3.15	362	3.22	90	Epi	1	2	3.21	12053	3.24	110233
Scz	2	2	3	3.11	4074	2.88	542	Epi	1	3	NaN	NaN	NaN	NaN

Table 6.4

Model	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	Model	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$
Scz 2	2	4	2.35	18995	2.28	2319	Epi	1	4	(3 bins only)			
Scz 2	3	0.5	3.38	7	3.27	4	Epi	2	0.5	3.34	15	3.36	38
Scz 2	3	1	3.26	31	3.30	13	Epi	2	1	3.23	148	3.23	683
Scz 2	3	2	3.21	457	3.18	103	Epi	2	2	3.26	15780	3.29	142449
Scz 2	3	3	3.06	4792	3.12	778	Epi	2	3	NaN	NaN	NaN	NaN
Scz 2	3	4	3.31	61991	3.25	7030	Epi	2	4	(3 bins only)			
							Epi	3	0.5	3.38	7	3.27	4
							Epi	3	1	3.26	31	3.30	13
							Epi	3	2	3.19	464	3.23	109
							Epi	3	3	2.72	4032	2.72	638

Table 6.5: Size (τ) and duration (α) exponents recovered from the autocorrelation null model, over five temporal bins. The autocorrelation null preserves the empirical marginals, so the exponents track those of the intact data. Same tracks as Table 6.1.

Model	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	Model	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$
Control 2	2	0.5	2.67	11	3.18	5	Control 1	1	0.5	2.67	11	3.18	5
Control 2	2	1	2.72	48	2.80	12	Control 1	1	1	2.72	48	2.80	12
Control 2	2	2	2.50	347	2.70	61	Control 1	1	2	2.50	347	2.70	61
Control 2	2	3	2.43	1486	2.46	159	Control 1	1	3	2.43	1486	2.46	159
Control 2	2	4	2.40	4668	2.43	415	Control 1	1	4	2.40	4668	2.43	415
Control 3	3	0.5	2.53	11	3.21	6	Scz 1	1	0.5	3.16	6	3.90	4
Control 3	3	1	2.69	51	2.72	12	Scz 1	1	1	2.74	23	3.16	10
Control 3	3	2	2.44	312	2.65	54	Scz 1	1	2	2.66	190	2.80	44
Control 3	3	3	2.48	1337	2.60	156	Scz 1	1	3	2.74	1282	2.84	215
Control 3	3	4	2.37	3511	2.60	377	Scz 1	1	4	2.99	7305	2.95	889
Control 4	4	0.5	2.52	11	3.19	6	Scz 1	2	0.5	3.15	6	3.90	4
Control 4	4	1	2.66	53	2.77	13	Scz 1	2	1	2.70	22	3.04	9
Control 4	4	2	2.48	352	2.66	59	Scz 1	2	2	2.71	190	2.82	43
Control 4	4	3	2.45	1467	2.52	166	Scz 1	2	3	2.62	1054	2.76	183

Table 6.5

Model	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$	Model	Track	Bin (ms)	τ (S.)	$x_{\min}^S$	α (D.)	$x_{\min}^D$
Control	4	4	2.33	3700	2.29	286	Scz	1	2	4	2.73	4778	2.80 621
Control	5	0.5	2.61	9	3.41	5	Scz	1	3	0.5	3.15	6	3.95 4
Control	5	1	2.55	33	2.97	11	Scz	1	3	1	2.66	20	3.10 9
Control	5	2	2.39	196	2.63	38	Scz	1	3	2	2.68	166	2.93 42
Control	5	3	2.33	718	2.47	95	Scz	1	3	3	2.44	715	2.60 134
Control	5	4	1.85	755	2.46	217	Scz	1	3	4	2.27	2267	2.56 432
Control	6	0.5	2.56	8	3.21	4	Scz	2	1	0.5	2.61	6	3.16 4
Control	6	1	2.54	32	3.05	11	Scz	2	1	1	2.41	17	2.84 8
Control	6	2	2.46	195	2.70	37	Scz	2	1	2	2.44	119	2.77 30
Control	6	3	2.45	733	2.59	96	Scz	2	1	3	2.51	567	2.64 93
Control	6	4	2.48	2168	2.57	212	Scz	2	1	4	2.44	2171	2.42 238
Scz	2	2	0.5	2.62	6	3.17	4	Epi	1	0.5	2.68	34	2.87 13
Scz	2	2	1	2.42	17	2.85	8	Epi	1	1	2.51	321	2.67 70
Scz	2	2	2	2.43	117	2.73	29	Epi	1	2	2.06	5737	2.09 594
Scz	2	2	3	2.49	560	2.56	85	Epi	1	3	2.13	78921	2.12 5171
Scz	2	2	4	2.14	1255	2.30	195	Epi	1	4	1.19	2	1.21 2
Scz	2	3	0.5	2.54	6	3.08	4	Epi	2	0.5	2.64	32	2.82 12
Scz	2	3	1	2.36	18	2.75	8	Epi	2	1	2.51	283	2.68 63
Scz	2	3	2	2.42	124	2.74	30	Epi	2	2	1.84	2231	2.49 816
Scz	2	3	3	2.43	538	2.60	87	Epi	2	3	1.98	40578	2.08 3637
Scz	2	3	4	2.31	1852	2.44	226	Epi	2	4	1.17	2	1.22 2
								Epi	3	0.5	2.54	6	3.08 4
								Epi	3	1	2.36	18	2.75 8
								Epi	3	2	2.42	124	2.74 30
								Epi	3	3	2.43	538	2.60 87
								Epi	3	4	2.31	1852	2.44 226

Table 6.6: Deviation from Criticality Coefficient (DCC) of the healthy recordings (whole hippocampus) as a function of temporal bin size. Lower values indicate closer consistency with the crackling-noise scaling relation.”-” denotes a bin size not evaluated for that track.

Model	Track	Bin size (ms)						
		0.5	1	1.5	2	3	4	5
Control	1	0.28	0.19	0.04	0.13	0.14	1.08	
Control	2	0.04	0.16	0.05	0.11	0.13	1.02	
Control	3	0.10	0.08	0.05	0.12	0.17	1.07	

Table 6.7: DCC of the two null models (correlation and autocorrelation) for the healthy recordings (whole hippocampus) as a function of temporal bin size. ”-” denotes a bin size not evaluated.

Model	Track	Correlation null – Bin size (ms)						Autocorrelation null – Bin size (ms)					
		0.5	1	2	3	4	5	0.5	1	2	3	4	5
Control	1	2.16	0.05	0.02	1.00	–	–	0.28	0.19	0.04	0.06	0.15	–
Control	2	2.54	0.03	0.13	0.99	–	–	0.05	0.16	0.06	0.03	0.15	–
Control	3	0.12	0.03	0.06	1.02	–	–	0.10	0.08	0.05	0.02	0.14	–
Control	4	3.08	0.10	0.02	1.06	–	–	0.26	0.10	0.07	0.01	0.66	–
Control	5	3.10	0.02	0.02	1.00	–	–	0.28	0.01	0.08	0.04	0.09	–
Control	6	3.12	0.03	0.05	0.94	–	–	0.15	0.07	0.08	0.01	0.18	–
Scz 1	1	0.50	0.14	0.00	0.26	–	–	0.06	0.10	0.02	0.01	0.98	–
Scz 1	2	0.53	0.25	0.00	0.86	–	–	0.11	0.07	0.01	0.02	1.04	–
Scz 1	3	0.50	0.00	0.01	0.54	–	–	0.11	0.07	0.04	0.04	0.36	–
Scz 2	1	0.11	0.01	0.01	0.05	–	–	0.16	0.05	0.12	0.02	0.11	–
Scz 2	2	0.03	0.00	0.00	0.89	–	–	0.07	0.01	0.11	0.04	0.02	–
Scz 2	3	0.18	0.01	0.03	0.02	–	–	0.00	0.02	0.09	0.01	0.09	–
Epi	1	0.00	0.00	0.99	–	–	–	0.02	0.00	1.03	0.99	2.83	–
Epi	2	0.03	0.01	0.98	–	–	–	0.03	0.00	1.78	1.10	0.41	–
Epi	3	0.16	0.02	0.03	–	–	–	0.00	0.02	0.09	0.01	0.09	–

Table 6.8: DCC of the healthy recordings by hippocampal subregion (dorsal: dCA1, dDG; ventral: vDG) as a function of temporal bin size. ”-” denotes a bin size not evaluated.

Model	Track	Region	Bin size (ms)					
			0.5	1	2	3	4	5
Control	1	dCA1	1.11	0.14	0.27	0.28	0.17	0.26
Control	1	vDG	0.07	0.03	0.49	0.02	0.01	0.03
Control	2	dCA1	0.75	0.22	0.31	0.34	0.03	0.15

Table 6.8

Model	Track	Region	Bin size (ms)					
			0.5	1	2	3	4	5
Control	2	vDG	0.17	0.15	0.34	0.01	0.03	0.03
Control	3	dCA1	0.83	0.47	0.27	0.38	0.21	0.26
Control	3	vDG	0.18	0.10	0.14	0.07	0.01	0.08
Control	4	dCA1	1.17	0.73	0.18	0.10	0.25	0.36
Control	4	vDG	0.09	0.05	0.29	0.06	0.12	0.12
Control	5	dCA1	2.09	1.15	0.10	0.03	0.37	0.42
Control	5	vDG	0.20	0.08	0.24	0.01	0.17	0.04
Control	6	dCA1	1.33	0.59	0.19	0.10	0.02	0.23
Control	6	vDG	0.11	0.06	0.30	0.05	0.01	0.06
Scz 1	1	dCA1	0.30	0.69	0.83	0.09	0.18	0.17
Scz 1	1	vDG	0.02	0.01	0.08	0.06	0.03	0.03
Scz 1	2	dCA1	0.18	0.48	0.43	0.38	0.16	0.29
Scz 1	2	vDG	0.17	0.07	0.10	0.09	0.02	0.05
Scz 1	3	dCA1	0.08	0.52	0.48	0.04	0.35	0.18
Scz 1	3	vDG	0.19	0.03	0.13	0.08	0.03	0.05
Scz 2	1	dDG	0.10	0.18	0.17	0.26	0.03	0.25
Scz 2	1	vDG	0.38	0.24	0.08	0.18	0.36	1.15
Scz 2	2	dDG	0.04	0.27	0.01	0.25	0.01	0.15
Scz 2	2	vDG	0.38	0.15	0.12	0.17	0.28	0.74
Scz 2	3	dDG	0.10	0.06	0.09	0.34	0.05	0.07
Scz 2	3	vDG	0.24	0.23	0.06	0.14	0.66	0.23
Epi	1	dCA1	0.06	0.11	0.03	0.02	1.09	1.10
Epi	1	dDG	0.09	0.09	0.14	0.08	0.06	0.07
Epi	1	vDG	0.16	0.19	0.03	0.02	0.03	0.02
Epi	2	dCA1	0.04	0.10	0.01	0.01	0.21	1.00
Epi	2	dDG	0.03	0.05	0.08	0.13	0.07	0.07
Epi	2	vDG	0.03	0.12	0.01	0.05	0.02	0.09
Epi	3	dCA1	0.01	0.11	0.02	0.12	0.25	1.06
Epi	3	dDG	0.31	0.02	0.20	0.18	0.12	0.09
Epi	3	vDG	0.23	0.18	0.05	0.18	0.06	0.02

Table 6.9: Avalanche shape collapse for the healthy hippocampus (whole hippocampus). For each recording: the size–duration scaling exponent γ , the collapse quality (CV, lower is better), the significance of the collapse against a shuffled null (p), and the dynamic range spanned (T range, in decades). All collapses are significantly better than chance.

Model	Track	γ	CV	p	Decades
Control	1	1.09	0.039	1.8×10^{-6}	0.8
Control	2	1.08	0.030	5.0×10^{-7}	0.8
Control	3	1.06	0.033	4.4×10^{-5}	0.9
Control	4	1.11	0.038	2.2×10^{-6}	0.8
Control	5	1.09	0.056	8.5×10^{-4}	0.9
Control	6	1.08	0.039	7.7×10^{-5}	0.6
Scz 1	1	1.06	0.029	2.5×10^{-6}	0.8
Scz 1	2	1.08	0.038	3.2×10^{-6}	0.8
Scz 1	3	1.11	0.043	9.4×10^{-7}	0.8
Scz 2	1	1.11	0.074	1.0×10^{-4}	0.7
Scz 2	2	1.09	0.053	7.9×10^{-4}	0.7
Scz 2	3	1.14	0.052	1.7×10^{-6}	0.7
Epi	1	1.10	0.032	1.3×10^{-7}	1.0
Epi	2	1.11	0.034	1.7×10^{-7}	0.9
Epi	3	1.12	0.035	4.0×10^{-8}	0.9

Table 6.10: Avalanche shape collapse under the two null models (whole hippocampus). For each recording and null model: the rescaling exponent γ , the collapse quality (CV), and the significance against a shuffled null (p , z).

Model	Track	Null	γ	CV	p	z
Control	2	corr	1.00	0.014	0.99	+2.5
Control	2	autocorr	1.09	0.049	0.90	+1.3
Control	3	corr	1.03	0.018	0.82	+0.9
Control	3	autocorr	1.08	0.047	0.95	+1.7
Control	4	corr	0.96	0.022	0.99	+2.4
Control	4	autocorr	1.07	0.040	0.98	+2.0
Control	5	corr	1.01	0.016	8×10^{-4}	-3.2
Control	5	autocorr	1.11	0.051	0.91	+1.3
Control	6	corr	1.01	0.012	0.16	-1.0
Control	6	autocorr	1.09	0.046	0.80	+0.9
Control	8	corr	1.01	0.011	2.5×10^{-4}	-3.5
Control	8	autocorr	1.08	0.048	0.99	+2.2
Scz 1	1	corr	1.03	0.017	2.0×10^{-3}	-2.9
Scz 1	1	autocorr	1.06	0.045	0.96	+1.8
Scz 1	2	corr	1.02	0.015	7.4×10^{-3}	-2.4
Scz 1	2	autocorr	1.08	0.061	0.98	+2.1
Scz 1	3	corr	1.02	0.015	1.6×10^{-3}	-3.0
Scz 1	3	autocorr	1.11	0.063	0.99	+2.3
Scz 2	1	corr	1.03	0.021	9.2×10^{-6}	-4.3
Scz 2	1	autocorr	1.11	0.046	0.94	+1.6
Scz 2	2	corr	1.03	0.015	2.0×10^{-4}	-3.5
Scz 2	2	autocorr	1.09	0.045	0.88	+1.2
Scz 2	3	corr	1.02	0.021	3.2×10^{-6}	-4.5
Scz 2	3	autocorr	1.14	0.057	0.73	+0.6
Epi	1	corr	1.00	0.214	3.0×10^{-2}	-1.9
Epi	1	autocorr	1.00	0.359	0.36	-0.3
Epi	2	corr	1.00	0.106	0.82	+0.9
Epi	2	autocorr	1.00	0.304	0.53	+0.1
Epi	3	corr	1.04	0.028	5.8×10^{-4}	-3.2

Table 6.10

Model	Track	Null	γ	CV	p	z
Epi	3	autocorr	1.14	0.057	0.30	-0.5

Table 6.11: Avalanche shape collapse by hippocampal subregion, healthy recordings. γ is the rescaling exponent from each panel, CV the collapse quality, p its significance against a shuffled null, and T the duration range (bins).

Table 6.11

Model	Track	Region	γ	CV	p	T range
Table 6.11						
Model	Track	Region	γ	CV	p	T range
Control	2	dCA1	1.40	0.095	5.0×10^{-5}	20–71
Control	2	vDG	1.03	0.030	1.7×10^{-3}	79–562
Control	3	dCA1	1.46	0.078	2.7×10^{-6}	20–72
Control	3	vDG	1.05	0.036	8.9×10^{-3}	59–400
Control	4	dCA1	1.29	0.126	2.1×10^{-3}	20–75
Control	4	vDG	1.04	0.039	0.28	68–437
Control	5	dCA1	1.44	0.137	7.9×10^{-3}	20–59
Control	5	vDG	1.03	0.031	1.1×10^{-4}	54–347
Control	6	dCA1	1.42	0.124	2.8×10^{-2}	21–55
Control	6	vDG	1.04	0.182	0.98	35–387
Control	8	dCA1	1.68	0.135	5.7×10^{-5}	20–53
Control	8	vDG	1.05	0.043	7.8×10^{-3}	55–378
Scz 1	1	dCA1	1.17	0.069	0.28	20–26
Scz 1	1	vDG	1.08	0.038	7.0×10^{-5}	57–348
Scz 1	2	dCA1	1.18	0.106	0.34	20–25
Scz 1	2	vDG	1.09	0.053	5.3×10^{-3}	49–289
Scz 1	3	dCA1	1.11	0.100	0.62	20–28
Scz 1	3	vDG	1.07	0.043	6.0×10^{-2}	54–295
Scz 2	1	vCA3	1.37	0.062	2.0×10^{-4}	20–44

Table 6.11: Avalanche shape collapse by hippocampal subregion, healthy recordings. γ is the rescaling exponent from each panel, CV the collapse quality, p its significance against a shuffled null, and T the duration range (bins).

Table 6.11						
Model	Track	Region	γ	CV	p	T range
Table 6.11						
Model	Track	Region	γ	CV	p	T range
Scz 2	1	vDG	1.30	0.070	1.9×10^{-6}	20–74
Scz 2	1	dDG	1.23	0.083	3.9×10^{-6}	21–195
Scz 2	2	vCA3	1.36	0.070	1.9×10^{-5}	20–39
Scz 2	2	vDG	1.35	0.097	1.5×10^{-3}	20–58
Scz 2	2	dDG	1.19	0.068	9.9×10^{-7}	21–107
Scz 2	3	vCA3	1.33	0.064	1.0×10^{-5}	20–43
Scz 2	3	vDG	1.32	0.077	1.7×10^{-7}	20–75
Scz 2	3	dDG	1.29	0.088	9.0×10^{-7}	21–118
Epi	1	dCA1	1.13	0.037	1.6×10^{-7}	157–1020
Epi	1	dDG	1.15	0.074	1.1×10^{-5}	31–130
Epi	1	vDG	1.08	0.052	7.0×10^{-5}	31–137
Epi	2	dCA1	1.13	0.035	2.6×10^{-6}	157–908
Epi	2	dDG	1.13	0.085	1.5×10^{-4}	31–131
Epi	2	vDG	1.07	0.035	1.7×10^{-4}	31–119
Epi	3	dCA1	1.15	0.033	2.3×10^{-9}	112–847
Epi	3	dDG	1.13	0.089	3.0×10^{-5}	31–110
Epi	3	vDG	1.04	0.032	2.8×10^{-3}	31–125

Table 6.12: Size (α) and duration (β) exponents, whole hippocampus, ketamine condition (linear tracks 4–6). $x_{\min}$ marks the fitted lower cutoff.

Model	Track	Bin (ms)	α (S.)	$x_{\min}^S$	β (D.)	$x_{\min}^D$	Model	Track	Bin (ms)	α (S.)	$x_{\min}^S$	β (D.)	$x_{\min}^D$
Scz 1	4	0.5	2.98	6	3.31	4	Scz 2	4	0.5	2.65	6	2.86	3

Table 6.12

Model	Track	Bin (ms)	α (S.)	$x_{\min}^S$	β (D.)	$x_{\min}^D$	Model	Track	Bin (ms)	α (S.)	$x_{\min}^S$	β (D.)	$x_{\min}^D$
Scz 1	4	1	2.67	18	2.88	8	Scz 2	4	1	2.43	16	2.77	7
Scz 1	4	2	2.67	143	2.87	38	Scz 2	4	2	2.40	100	2.73	26
Scz 1	4	3	2.49	685	2.57	121	Scz 2	4	3	2.44	419	2.60	72
Scz 1	4	4	2.39	2515	2.15	217	Scz 2	4	4	2.38	1460	2.50	196
Scz 1	5	0.5	2.90	6	3.31	4	Scz 2	5	0.5	2.61	6	2.85	3
Scz 1	5	1	2.61	17	2.91	8	Scz 2	5	1	2.39	16	2.76	7
Scz 1	5	2	2.54	122	2.79	33	Scz 2	5	2	2.37	101	2.76	27
Scz 1	5	3	2.28	440	2.53	97	Scz 2	5	3	2.44	425	2.57	66
Scz 1	5	4	1.90	875	2.17	193	Scz 2	5	4	2.12	929	2.34	159
Scz 1	6	0.5	2.92	6	3.29	4	Scz 2	6	0.5	2.63	6	3.14	4
Scz 1	6	1	2.64	18	2.89	8	Scz 2	6	1	2.43	17	2.73	7
Scz 1	6	2	2.61	129	2.83	34	Scz 2	6	2	2.41	110	2.79	29
Scz 1	6	3	2.39	512	2.62	108	Scz 2	6	3	2.39	463	2.60	82
Scz 1	6	4	2.33	1961	2.25	203	Scz 2	6	4	1.99	849	2.19	148

Table 6.13: Size (α) and duration (β) exponents, whole hippocampus, schizophrenia condition (linear tracks 7–9).

Model	Track	Bin (ms)	α (S.)	$x_{\min}^S$	β (D.)	$x_{\min}^D$	Model	Track	Bin (ms)	α (S.)	$x_{\min}^S$	β (D.)	$x_{\min}^D$
Scz 1	7	0.5	2.17	24	2.40	10	Scz 2	7	0.5	2.61	6	3.14	4
Scz 1	7	1	1.88	58	2.07	23	Scz 2	7	1	2.44	18	2.82	8
Scz 1	7	2	1.94	469	2.01	76	Scz 2	7	2	2.47	125	2.80	31
Scz 1	7	3	1.30	6	1.41	6	Scz 2	7	3	2.39	521	2.58	91
Scz 1	7	4	1.24	4	1.33	4	Scz 2	7	4	2.40	2048	2.33	206
Scz 1	8	0.5	2.82	5	2.96	3	Scz 2	8	0.5	2.61	6	2.87	3
Scz 1	8	1	2.63	16	2.96	8	Scz 2	8	1	2.44	17	2.78	7
Scz 1	8	2	2.64	110	2.85	30	Scz 2	8	2	2.41	107	2.79	28
Scz 1	8	3	2.60	490	2.73	92	Scz 2	8	3	2.36	448	2.52	74
Scz 1	8	4	2.48	1638	2.59	246	Scz 2	8	4	2.34	1711	2.45	220
Scz 1	9	0.5	2.85	5	3.01	3	Scz 2	9	0.5	2.57	5	2.92	3
Scz 1	9	1	2.66	15	2.92	7	Scz 2	9	1	2.44	15	2.86	7
Scz 1	9	2	2.52	87	2.75	25	Scz 2	9	2	2.43	91	2.82	25
Scz 1	9	3	2.38	308	2.61	70	Scz 2	9	3	2.53	388	2.62	64
Scz 1	9	4	1.98	556	2.01	82	Scz 2	9	4	2.49	1328	2.38	139

Table 6.14: Size (α) and duration (β) exponents, whole hippocampus, chronic schizophrenia condition (linear tracks 10–12). Available for Scz 1 only.

Model	Track	Bin (ms)	α (S.)	$x_{\min}^S$	β (D.)	$x_{\min}^D$
Scz 1	10	0.5	2.95	5	3.11	3
Scz 1	10	1	2.66	13	2.85	6
Scz 1	10	2	2.01	31	2.74	22
Scz 1	10	3	2.48	275	2.67	61
Scz 1	10	4	2.42	865	2.52	140
Scz 1	11	0.5	2.91	5	3.13	3
Scz 1	11	1	2.60	12	2.89	6
Scz 1	11	2	2.12	35	2.43	14
Scz 1	11	3	2.50	242	2.64	51
Scz 1	11	4	2.28	612	2.18	75
Scz 1	12	0.5	2.93	5	3.14	3
Scz 1	12	1	2.61	12	2.89	6
Scz 1	12	2	2.13	35	2.54	16
Scz 1	12	3	2.45	229	2.63	51
Scz 1	12	4	2.26	592	2.41	109

Table 6.15: Size (α) and duration (β) exponents by subregion, ketamine condition (linear tracks 4–6). $x_{\min}$ is the fitted lower cutoff.

Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$	Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$
Scz1	4	dCA1	0.5	3.36	6	4.19	2	Scz1	4	vDG	0.5	3.20	3	3.18	2
Scz1	4	dCA1	1	3.05	3	3.13	2	Scz1	4	vDG	1	2.90	6	3.20	4
Scz1	4	dCA1	2	2.93	7	3.11	4	Scz1	4	vDG	2	2.63	21	2.83	9
Scz1	4	dCA1	3	2.74	14	2.98	7	Scz1	4	vDG	3	2.67	67	2.89	22
Scz1	4	dCA1	4	2.62	27	2.82	11	Scz1	4	vDG	4	2.65	175	2.80	45
Scz1	5	dCA1	0.5	3.16	2	4.05	2	Scz1	5	vDG	0.5	3.10	3	3.15	2
Scz1	5	dCA1	1	2.92	3	3.13	2	Scz1	5	vDG	1	2.83	6	3.15	4
Scz1	5	dCA1	2	2.85	7	3.15	4	Scz1	5	vDG	2	2.56	21	2.79	9
Scz1	5	dCA1	3	2.66	13	2.90	6	Scz1	5	vDG	3	2.57	65	2.81	21
Scz1	5	dCA1	4	2.56	24	2.85	10	Scz1	5	vDG	4	2.51	163	2.67	41
Scz1	6	dCA1	0.5	3.31	2	4.22	2	Scz1	6	vDG	0.5	3.06	3	3.09	2
Scz1	6	dCA1	1	3.05	3	3.22	2	Scz1	6	vDG	1	2.90	7	3.08	4

Table 6.15 (continued)

Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$	Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$
Scz1	6	dCA1	2	2.97	7	3.23	4	Scz1	6	vDG	2	2.55	23	2.81	10
Scz1	6	dCA1	3	2.71	12	2.95	6	Scz1	6	vDG	3	2.58	73	2.82	23
Scz1	6	dCA1	4	2.64	23	2.91	10	Scz1	6	vDG	4	2.52	180	2.69	45
Scz2	4	dDG	0.5	2.80	3	3.32	2	Scz2	4	vCA3	0.5	3.00	2	3.64	2
Scz2	4	dDG	1	2.50	4	3.09	3	Scz2	4	vCA3	1	2.73	3	3.05	2
Scz2	4	dDG	2	2.41	10	2.84	5	Scz2	4	vCA3	2	2.46	5	2.84	3
Scz2	4	dDG	3	2.30	18	2.67	7	Scz2	4	vCA3	3	2.28	7	2.68	4
Scz2	4	dDG	4	2.01	17	2.75	11	Scz2	4	vCA3	4	2.19	11	2.68	6
Scz2	4	vDG	0.5	2.93	2	3.63	2	Scz2	5	dDG	0.5	2.74	3	3.24	2
Scz2	4	vDG	1	2.68	3	2.93	2	Scz2	5	dDG	1	2.51	5	2.97	3
Scz2	4	vDG	2	2.55	7	2.94	4	Scz2	5	dDG	2	2.31	10	2.74	5
Scz2	4	vDG	3	2.40	12	2.80	6	Scz2	5	dDG	3	2.23	19	2.58	7
Scz2	4	vDG	4	2.40	23	2.78	9	Scz2	5	dDG	4	1.90	14	2.63	11
Scz2	5	vCA3	0.5	2.92	2	3.56	2	Scz2	5	vDG	0.5	2.96	2	3.69	2
Scz2	5	vCA3	1	2.65	3	2.98	2	Scz2	5	vDG	1	2.72	3	2.98	2
Scz2	5	vCA3	2	2.39	5	2.77	3	Scz2	5	vDG	2	2.55	6	3.03	4
Scz2	5	vCA3	3	2.23	8	2.61	4	Scz2	5	vDG	3	2.43	11	2.89	6
Scz2	5	vCA3	4	2.15	12	2.62	6	Scz2	5	vDG	4	2.34	18	2.75	8
Scz2	6	dDG	0.5	2.82	3	3.27	2	Scz2	6	vCA3	0.5	2.93	2	3.55	2
Scz2	6	dDG	1	2.47	4	3.02	3	Scz2	6	vCA3	1	2.67	3	3.00	2
Scz2	6	dDG	2	2.35	10	2.75	5	Scz2	6	vCA3	2	2.42	5	2.81	3
Scz2	6	dDG	3	2.24	19	2.56	7	Scz2	6	vCA3	3	2.26	8	2.64	4
Scz2	6	dDG	4	2.23	34	2.68	12	Scz2	6	vCA3	4	2.17	12	2.63	6
Scz2	6	vDG	0.5	2.95	2	3.66	2								
Scz2	6	vDG	1	2.70	3	2.96	2								
Scz2	6	vDG	2	2.61	7	3.01	4								
Scz2	6	vDG	3	2.45	12	2.87	6								
Scz2	6	vDG	4	2.36	19	2.86	9								

Table 6.16: Size (α) and duration (β) exponents by subregion, *schizophrenia* condition (linear tracks 7–9). $x_{\min}$ is the fitted lower cutoff.

Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$	Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$
Scz1	7	dCA1	0.5	2.61	6	2.90	4	Scz1	7	vDG	0.5	2.92	8	3.12	5
Scz1	7	dCA1	1	1.87	8	2.17	6	Scz1	7	vDG	1	2.39	31	2.54	13

Table 6.16 (continued)

Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$	Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$
Scz1	7	dCA1	2	1.96	13	2.17	7	Scz1	7	vDG	2	2.01	148	2.11	39
Scz1	7	dCA1	3	2.18	30	2.39	12	Scz1	7	vDG	3	1.94	345	2.03	67
Scz1	7	dCA1	4	2.28	66	2.49	21	Scz1	7	vDG	4	1.30	3	1.85	90
Scz1	8	dCA1	0.5	3.41	2	4.27	2	Scz1	8	vDG	0.5	3.09	3	3.11	2
Scz1	8	dCA1	1	3.14	3	3.26	2	Scz1	8	vDG	1	2.79	6	3.11	4
Scz1	8	dCA1	2	2.95	6	3.34	4	Scz1	8	vDG	2	2.51	21	2.75	9
Scz1	8	dCA1	3	2.89	12	3.11	6	Scz1	8	vDG	3	2.62	67	2.85	21
Scz1	8	dCA1	4	2.76	21	2.95	9	Scz1	8	vDG	4	2.62	165	2.78	41
Scz1	9	dCA1	0.5	3.31	2	4.31	2	Scz1	9	vDG	0.5	3.11	3	3.14	2
Scz1	9	dCA1	1	3.10	3	3.37	2	Scz1	9	vDG	1	2.85	6	3.17	4
Scz1	9	dCA1	2	2.85	5	3.09	3	Scz1	9	vDG	2	2.55	20	2.83	9
Scz1	9	dCA1	3	2.77	9	3.10	5	Scz1	9	vDG	3	2.57	60	2.77	19
Scz1	9	dCA1	4	2.71	16	2.93	7	Scz1	9	vDG	4	2.55	145	2.70	37
Scz2	7	dDG	0.5	2.79	3	3.31	2	Scz2	7	vCA3	0.5	2.99	2	3.61	2
Scz2	7	dDG	1	2.59	5	3.06	3	Scz2	7	vCA3	1	2.72	3	3.00	2
Scz2	7	dDG	2	2.36	10	2.77	5	Scz2	7	vCA3	2	2.44	5	2.79	3
Scz2	7	dDG	3	2.28	20	2.67	8	Scz2	7	vCA3	3	2.27	8	2.62	4
Scz2	7	dDG	4	2.26	36	2.69	12	Scz2	7	vCA3	4	2.18	12	2.62	6
Scz2	7	vDG	0.5	2.92	2	3.61	2	Scz2	8	dDG	0.5	2.69	3	3.35	2
Scz2	7	vDG	1	2.86	4	3.38	3	Scz2	8	dDG	1	2.45	4	3.14	3
Scz2	7	vDG	2	2.59	8	2.86	4	Scz2	8	dDG	2	2.35	9	2.88	5
Scz2	7	vDG	3	2.45	15	2.85	7	Scz2	8	dDG	3	2.27	17	2.68	7
Scz2	7	vDG	4	2.25	20	2.87	11	Scz2	8	dDG	4	2.22	30	2.60	10
Scz2	8	vCA3	0.5	3.00	2	3.67	2	Scz2	8	vDG	0.5	2.95	2	3.67	2
Scz2	8	vCA3	1	2.73	3	3.05	2	Scz2	8	vDG	1	2.68	3	2.92	2
Scz2	8	vCA3	2	2.46	5	2.84	3	Scz2	8	vDG	2	2.56	7	2.92	4
Scz2	8	vCA3	3	2.27	7	2.66	4	Scz2	8	vDG	3	2.45	14	2.76	6
Scz2	8	vCA3	4	2.18	11	2.67	6	Scz2	8	vDG	4	2.40	25	2.81	10
Scz2	9	dDG	0.5	2.61	2	3.39	2	Scz2	9	vCA3	0.5	3.00	2	3.68	2
Scz2	9	dDG	1	2.51	4	3.18	3	Scz2	9	vCA3	1	2.75	3	3.06	2
Scz2	9	dDG	2	2.40	9	2.73	4	Scz2	9	vCA3	2	2.48	5	2.85	3
Scz2	9	dDG	3	2.29	16	2.73	7	Scz2	9	vCA3	3	2.28	7	2.67	4
Scz2	9	dDG	4	2.21	26	2.66	10	Scz2	9	vCA3	4	2.18	11	2.54	5
Scz2	9	vDG	0.5	2.91	2	3.64	2								
Scz2	9	vDG	1	2.67	3	2.94	2								
Scz2	9	vDG	2	2.48	6	2.96	4								

Table 6.16 (continued)

Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$	Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$
Scz2	9	vDG	3	2.36	11	2.83	6								
Scz2	9	vDG	4	2.26	17	2.72	8								

Table 6.17: Size (α) and duration (β) exponents by subregion, *chronic schizophrenia* condition (linear tracks 10–12). Available for Scz 1 only. $x_{\min}$ is the fitted lower cutoff.

Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$	Mod.	Tr.	Reg.	Bin	α	$x_{\min}^S$	β	$x_{\min}^D$
Scz1	10	dCA1	0.5	3.31	2	3.04	2	Scz1	10	vDG	0.5	3.18	3	3.19	2
Scz1	10	dCA1	1	3.10	3	3.40	2	Scz1	10	vDG	1	2.92	6	3.22	4
Scz1	10	dCA1	2	2.84	5	3.14	3	Scz1	10	vDG	2	2.55	19	2.84	9
Scz1	10	dCA1	3	2.72	8	2.92	4	Scz1	10	vDG	3	2.62	59	2.81	19
Scz1	10	dCA1	4	2.65	13	2.91	6	Scz1	10	vDG	4	2.62	143	2.51	28
Scz1	11	dCA1	0.5	3.15	2	3.06	2	Scz1	11	vDG	0.5	3.14	3	3.19	2
Scz1	11	dCA1	1	2.99	3	3.44	2	Scz1	11	vDG	1	2.87	6	3.20	4
Scz1	11	dCA1	2	2.81	5	3.21	3	Scz1	11	vDG	2	2.51	18	2.76	8
Scz1	11	dCA1	3	2.73	8	3.02	4	Scz1	11	vDG	3	2.56	52	2.84	18
Scz1	11	dCA1	4	2.65	12	3.06	6	Scz1	11	vDG	4	2.39	95	2.78	33
Scz1	12	dCA1	0.5	3.22	2	3.05	2	Scz1	12	vDG	0.5	3.18	3	3.21	2
Scz1	12	dCA1	1	3.03	3	3.42	2	Scz1	12	vDG	1	2.90	6	3.24	4
Scz1	12	dCA1	2	2.82	5	3.18	3	Scz1	12	vDG	2	2.54	18	2.79	8
Scz1	12	dCA1	3	2.73	8	3.00	4	Scz1	12	vDG	3	2.53	50	2.78	17
Scz1	12	dCA1	4	2.64	12	3.01	6	Scz1	12	vDG	4	2.57	120	2.52	25

Table 6.18: DCC of the recordings under the ketamine condition (whole hippocampus) as a function of temporal bin size. Bold marks the per-track minimum. ”-” denotes a bin size not evaluated.

Model	Track	Bin size (ms)					
		0.5	1	2	3	4	5
Scz 1	4	0.01	0.03	0.03	0.02	0.33	1.04
Scz 1	5	0.22	0.01	0.08	0.09	0.13	0.26
Scz 1	6	0.11	0.06	0.05	0.11	0.09	1.03
Scz 2	4	0.21	0.07	0.03	0.01	0.12	–
Scz 2	5	0.19	0.08	0.15	0.05	0.11	–
Scz 2	6	0.06	0.09	0.08	0.01	0.19	–

Table 6.19: DCC of the recordings under the acute schizophrenia condition (whole hippocampus) as a function of temporal bin size. Bold marks the per-track minimum. '-' denotes a bin size not evaluated.

Model	Track	Bin size (ms)					
		0.5	1	2	3	4	5
Scz 1	7	0.51	0.16	0.04	0.06	0.03	1.03
Scz 1	8	0.17	0.02	0.04	0.01	0.06	–
Scz 1	9	0.03	0.05	0.06	0.06	0.05	2.76
Scz 2	7	0.004	0.03	0.07	0.01	0.15	–
Scz 2	8	0.18	0.08	0.04	0.01	0.21	–
Scz 2	9	0.11	0.08	0.04	0.02	0.03	–

Table 6.20: DCC of the recordings under the chronic schizophrenia condition (whole hippocampus). Available for Scz 1 only. Bold marks the per-track minimum.

Model	Track	Bin size (ms)					
		0.5	1	2	3	4	5
Scz 1	10	0.02	0.03	0.78	0.07	0.02	0.29
Scz 1	11	0.15	0.07	0.07	0.04	0.15	0.16
Scz 1	12	0.08	0.07	0.18	0.05	0.01	0.17

Table 6.21: Avalanche shape collapse under the ketamine condition (whole hippocampus). γ is the rescaling exponent, CV the collapse quality, p its significance against a shuffled null, and the dynamic range in decades.

Model	Track	γ	CV	p	Decades
Scz 1	4	1.11	0.036	1.8×10^{-6}	0.7
Scz 1	5	1.11	0.047	2.7×10^{-6}	0.7
Scz 1	6	1.11	0.046	6.6×10^{-6}	0.7
Scz 2	4	1.20	0.065	4.7×10^{-6}	0.7
Scz 2	5	1.14	0.051	2.0×10^{-6}	0.7
Scz 2	6	1.18	0.059	7.8×10^{-7}	0.7

Table 6.22: Avalanche shape collapse under the schizophrenia condition (whole hippocampus).

Model	Track	γ	CV	p	Decades
Scz 1	7	1.17	0.064	1.4×10^{-3}	1.1
Scz 1	8	1.09	0.050	2.5×10^{-5}	0.6
Scz 1	9	1.11	0.053	1.8×10^{-5}	0.6
Scz 2	7	1.15	0.045	1.6×10^{-6}	0.7
Scz 2	8	1.22	0.055	3.4×10^{-7}	0.8
Scz 2	9	1.23	0.048	1.4×10^{-6}	0.6

Table 6.23: Avalanche shape collapse under the *chronic schizophrenia* condition (whole hippocampus). Available for Scz 1 only.

Model	Track	γ	CV	p	Decades
Scz 1	10	1.09	0.038	1.4×10^{-3}	0.5
Scz 1	11	1.10	0.049	1.4×10^{-2}	0.5
Scz 1	12	1.12	0.048	6.0×10^{-4}	0.5

Table 6.24: DCC by hippocampal subregion, ketamine condition, as a function of bin size. Bold marks the per-recording minimum.

Model	Track	Region	0.5	1	2	3	4	5
Scz 1	4	dCA1	0.37	0.85	0.91	0.25	0.14	0.20
Scz 1	4	vDG	0.21	0.06	0.08	0.09	0.02	0.10
Scz 1	5	dCA1	0.16	0.28	1.03	0.90	0.11	0.17
Scz 1	5	vDG	0.30	0.11	0.07	0.09	0.04	0.07
Scz 1	6	dCA1	0.08	0.40	0.81	0.31	0.15	0.23
Scz 1	6	vDG	0.004	0.005	0.13	0.10	0.02	0.03
Scz 2	4	dDG	0.47	0.53	0.24	0.05	0.51	0.16
Scz 2	4	vCA3	0.08	0.14	0.07	0.01	0.10	0.57
Scz 2	4	vDG	0.22	0.25	0.36	0.14	0.08	0.12
Scz 2	5	dDG	0.43	0.32	0.23	0.15	0.49	0.10
Scz 2	5	vCA3	0.10	0.16	0.11	0.08	0.14	0.39
Scz 2	5	vDG	0.004	0.34	0.14	0.09	0.05	0.08
Scz 2	6	dDG	0.26	0.19	0.02	0.05	0.04	0.04
Scz 2	6	vCA3	0.06	0.13	0.10	0.06	0.14	0.57
Scz 2	6	vDG	0.05	0.31	0.17	0.11	0.18	0.09

Table 6.25: DCC by hippocampal subregion, schizophrenia condition, as a function of bin size. Bold marks the per-recording minimum.

Model	Track	Region	0.5	1	2	3	4	5
Scz 1	7	dCA1	0.10	0.40	0.51	0.02	0.18	0.18
Scz 1	7	vDG	0.12	0.33	0.016	0.011	0.13	0.14
Scz 1	8	dCA1	0.8	0.82	0.08	0.03	0.02	0.04
Scz 1	8	vDG	0.22	0.04	0.044	0.06	0.02	0.01
Scz 1	9	dCA1	0.005	0.52	0.63	0.09	0.19	0.08
Scz 1	9	vDG	0.11	0.12	0.15	0.05	0.04	0.06
Scz 2	7	dDG	0.51	0.45	0.05	0.03	0.10	0.11
Scz 2	7	vCA3	0.10	0.16	0.11	0.09	0.14	0.59
Scz 2	7	vDG	0.11	0.06	0.20	0.06	0.35	0.02
Scz 2	8	dDG	0.22	0.30	0.38	0.19	0.08	0.13
Scz 2	8	vCA3	0.18	0.12	0.07	0.05	0.18	0.58
Scz 2	8	vDG	0.04	0.20	0.16	0.007	0.11	0.04
Scz 2	9	dDG	0.21	0.46	0.38	0.06	0.13	0.11
Scz 2	9	vCA3	0.05	0.09	0.07	0.01	0.11	0.61
Scz 2	9	vDG	0.05	0.22	0.02	0.04	0.18	0.18

Table 6.26: DCC by hippocampal subregion, chronic schizophrenia condition. Available for Scz 1 only. Bold marks the per-recording minimum.

Model	Track	Region	0.5	1	2	3	4	5
Scz 1	10	dCA1	0.12	0.40	0.12	0.09	0.30	0.30
Scz 1	10	vDG	0.04	0.09	0.18	0.08	0.03	0.02
Scz 1	11	dCA1	1.01	0.10	0.86	0.76	0.38	0.08
Scz 1	11	vDG	0.24	0.06	0.14	0.06	0.24	0.05
Scz 1	12	dCA1	0.44	0.27	0.35	0.29	0.37	0.59
Scz 1	12	vDG	0.008	0.15	0.10	0.05	0.21	0.01

Table 6.27: Avalanche shape collapse under the ketamine condition for the dorsal and ventral hippocampal subregions. γ is the rescaling exponent, CV the collapse quality, and p its significance against a shuffled null.

Model	Region	Track	γ	CV	p
Scz 1	dCA1	4	1.36	0.291	2.09×10^{-2}
Scz 1	vDG	4	1.07	0.042	2.63×10^{-3}
Scz 1	dCA1	5	1.41	0.080	5.02×10^{-1}
Scz 1	vDG	5	1.08	0.048	3.75×10^{-4}
Scz 1	dCA1	6	1.43	0.366	6.26×10^{-3}
Scz 1	vDG	6	1.10	0.057	2.48×10^{-5}
Scz 2	dDG	4	1.23	0.112	2.55×10^{-3}
Scz 2	vCA3	4	1.30	0.064	2.26×10^{-1}
Scz 2	vDG	4	1.43	0.078	2.63×10^{-3}
Scz 2	dDG	5	1.43	0.096	2.50×10^{-5}
Scz 2	vCA3	5	1.39	0.070	5.12×10^{-3}
Scz 2	vDG	5	1.41	0.106	4.25×10^{-1}
Scz 2	dDG	6	1.31	0.073	7.59×10^{-5}
Scz 2	vCA3	6	1.36	0.091	2.32×10^{-1}
Scz 2	vDG	6	1.40	0.098	2.68×10^{-3}

Table 6.28: Avalanche shape collapse under the schizophrenia condition for the dorsal and ventral hippocampal subregions. γ is the rescaling exponent, CV the collapse quality, and p its significance against a shuffled null.

Scz 1					Scz 2				
Region	Track	γ	CV	p	Region	Track	γ	CV	p
dCA1	7	1.19	0.162	1.85×10^{-11}	dDG	7	1.28	0.094	1.48×10^{-3}
vDG	7	0.99	0.096	9.95×10^{-1}	vCA3	7	1.36	0.072	1.08×10^{-1}
dCA1	8	1.08	0.105	5.93×10^{-1}	vDG	7	1.22	0.097	4.22×10^{-3}
vDG	8	1.08	0.043	2.34×10^{-6}	dDG	8	1.52	0.078	1.80×10^{-5}
dCA1	9	1.22	0.314	3.39×10^{-3}	vCA3	8	1.27	0.084	7.27×10^{-1}

Table 6.28

Scz 1					Scz 2				
Region	Track	γ	CV	p	Region	Track	γ	CV	p
vDG	9	1.08	0.045	8.67×10^{-4}	vDG	8	1.22	0.060	3.53×10^{-3}
					dDG	9	1.40	0.126	3.46×10^{-3}
					vCA3	9	1.32	0.084	1.38×10^{-2}
					vDG	9	1.31	0.104	2.07×10^{-2}

Table 6.29: Avalanche shape collapse under the chronic schizophrenia condition for the dorsal and ventral hippocampal subregions. γ is the rescaling exponent, CV the collapse quality, and p its significance against a shuffled null.

Model	Region	Track	γ	CV	p
Ares	dCA1	10	1.12	0.219	2.85×10^{-3}
Ares	vDG	10	1.08	0.057	3.74×10^{-4}
Ares	dCA1	11	2.16	0.165	1.30×10^{-4}
Ares	vDG	11	1.09	0.056	1.42×10^{-6}
Ares	dCA1	12	1.69	0.183	4.70×10^{-3}
Ares	vDG	12	1.10	0.038	3.82×10^{-8}

Table 6.30: Avalanche size (τ) and duration (α) power-law exponents for the whole hippocampus during epileptogenesis (induced epilepsy), available only for one animal, as a function of bin size. $x_{\min}$ is the lower cutoff of the fitted range for size (S) and duration (D).

Track	Bin (ms)	τ (size)	$x_{\min}^S$	α (dur.)	$x_{\min}^D$
6	0.5	2.66	20	2.92	9
	1	2.54	136	2.73	36
	2	2.15	1763	1.97	134
	3	2.19	18084	2.23	1562
	4	1.52	8190	1.55	603
7	0.5	2.70	21	2.91	9
	1	2.57	145	2.73	37
	2	2.30	2261	2.39	302
	3	1.51	1411	2.22	1583
	4	1.20	6	1.54	829
8	0.5	2.67	20	2.93	9
	1	2.51	130	2.74	36
	2	2.39	2367	2.48	319
	3	2.27	17555	2.25	1476
	4	1.63	15365	1.45	235

Table 6.31: Avalanche size (τ) and duration (α) power-law exponents for the whole hippocampus during status epilepticus, available only for one animal, as a function of bin size. $x_{\min}$ is the lower cutoff of the fitted range for size (S) and duration (D).

Bin (ms)	τ (size)	$x_{\min}^S$	α (dur.)	$x_{\min}^D$
0.5	2.36	11	2.71	6
1	2.05	35	2.32	13

Table 6.31

Bin (ms)	τ (size)	$x_{\min}^S$	α (dur.)	$x_{\min}^D$
2	1.85	178	2.04	40
3	1.77	654	1.91	94
4	1.58	341	1.71	61

Table 6.32: Size (τ) and duration (α) exponents by subregion, *epileptogenesis* condition (induced epilepsy, linear tracks 6–8), available for only one animal. $x_{\min}$ is the fitted lower cutoff for size (S) and duration (D).

Reg.	Tr.	Bin	τ	$x_{\min}^S$	α	$x_{\min}^D$	Reg.	Tr.	Bin	τ	$x_{\min}^S$	α	$x_{\min}^D$
dCA1	6	0.5	2.94	5	3.04	3	dDG	6	0.5	2.89	3	3.07	2
dCA1	6	1	2.82	14	3.09	7	dDG	6	1	2.76	6	2.84	3
dCA1	6	2	2.45	72	2.68	22	dDG	6	2	2.41	14	2.66	6
dCA1	6	3	1.96	133	2.16	36	dDG	6	3	2.22	23	2.54	9
dCA1	6	4	2.35	1215	2.45	182	dDG	6	4	2.84	65	3.18	17
dCA1	7	0.5	2.99	5	3.09	3	dDG	7	0.5	2.85	3	3.02	2
dCA1	7	1	2.89	14	3.14	7	dDG	7	1	2.69	6	2.79	3
dCA1	7	2	2.46	66	2.70	21	dDG	7	2	2.39	15	2.75	7
dCA1	7	3	2.03	135	2.26	38	dDG	7	3	2.13	22	2.45	9
dCA1	7	4	2.35	1096	2.53	186	dDG	7	4	2.78	71	3.09	18
dCA1	8	0.5	2.96	5	3.07	3	dDG	8	0.5	2.80	3	2.97	2
dCA1	8	1	2.85	14	3.12	7	dDG	8	1	2.64	6	3.08	4
dCA1	8	2	2.40	63	2.64	20	dDG	8	2	2.43	17	2.71	7
dCA1	8	3	2.02	128	2.34	45	dDG	8	3	2.11	23	2.35	8
dCA1	8	4	2.30	1001	2.49	171	dDG	8	4	2.93	77	3.26	19
vDG	6	0.5	2.85	4	3.25	3	vDG	7	0.5	2.83	4	3.23	3
vDG	6	1	2.64	8	3.15	5	vDG	7	1	2.72	9	3.11	5
vDG	6	2	2.97	27	3.10	9	vDG	7	2	3.00	29	3.14	10
vDG	6	3	2.33	31	2.88	15	vDG	7	3	2.22	27	2.89	16
vDG	6	4	2.85	109	2.96	23	vDG	7	4	2.96	120	3.06	25
vDG	8	0.5	2.86	4	3.27	3							
vDG	8	1	2.64	8	3.15	5							
vDG	8	2	3.03	27	3.13	9							
vDG	8	3	2.59	43	2.95	15							
vDG	8	4	2.95	106	3.06	23							

Table 6.33: Size (τ) and duration (α) exponents by subregion, *status epilepticus* condition, available for only one animal. $x_{\min}$ is the fitted lower cutoff for size (S) and duration (D).

Reg.	Bin	τ	$x_{\min}^S$	α	$x_{\min}^D$	Reg.	Bin	τ	$x_{\min}^S$	α	$x_{\min}^D$
dCA1	0.5	2.78	4	3.18	3	dDG	0.5	2.86	3	3.11	2
dCA1	1	2.53	9	2.86	5	dDG	1	2.60	5	2.92	3
dCA1	2	2.18	27	2.47	11	dDG	2	2.22	9	2.47	4
dCA1	3	2.00	62	2.25	20	dDG	3	2.01	14	2.34	6
dCA1	4	1.89	94	2.14	33	dDG	4	1.72	8	2.18	7
vDG	0.5	2.78	4	3.21	3						
vDG	1	2.58	8	2.80	4						
vDG	2	2.33	21	2.60	8						
vDG	3	2.02	28	2.40	12						
vDG	4	2.07	66	2.36	17						

Table 6.34: DCC for the whole hippocampus under the two epilepsy phases (induced epilepsy), available for only one animal, as a function of bin size.

Phase	Track	0.5	1	2	3	4
Epileptogenesis	6	0.04	0.02	0.17	1.03	1.07
	7	0.04	0.01	0.04	2.40	2.74
	8	0.02	0.03	0.09	0.99	0.72
Status epilepticus	1	0.37	0.02	0.11	0.07	0.01

Table 6.35: Avalanche shape collapse for the whole hippocampus under the two epilepsy phases (induced epilepsy), animal Epi (Asclepius). γ is the rescaling exponent, CV the collapse quality, p its significance against a shuffled null, and “decades” the dynamic range of durations spanned by the collapse.

Phase	Track	γ	CV	p	Decades
Epileptogenesis	6	1.02	0.041	2.10×10^{-1}	0.9
	7	1.11	0.050	9.76×10^{-4}	0.7
	8	0.98	0.037	9.90×10^{-1}	0.6
Status epilepticus	1	1.32	0.169	1.82×10^{-8}	1.6

Table 6.36: DCC by hippocampal subregion under the two epilepsy phases (induced epilepsy), animal Epi (Asclepius), as a function of bin size. Values are read from the DCC-vs-bin-size curves (± 0.02 – 0.05). Bold marks the per-recording minimum.

Phase	Reg.	Tr.	0.5	1	2	3	4	5
Epileptog.	dCA1	6	0.16	0.09	0.03	0.04	0.01	0.96
		7	0.17	0.09	0.03	0.06	0.02	1.12
		8	0.20	0.09	0.02	0.13	0.06	1.13
	dDG	6	0.16	0.21	0.04	0.15	0.01	0.06
		7	0.14	0.18	0.13	0.16	0.02	0.04
		8	0.26	0.03	0.08	0.10	0.06	0.03
	vDG	6	0.05	0.02	0.04	0.35	0.01	0.03
		7	0.02	0.09	0.06	0.52	0.04	0.03

Table 6.36

Phase	Reg.	Tr.	0.5	1	2	3	4	5
		8	0.07	0.13	0.04	0.14	0.01	0.05
	dCA1	–	0.05	0.00	0.05	0.02	0.04	0.03
Status ep.	dDG	–	1.11	0.37	0.01	0.15	0.45	0.45
	vDG	–	0.39	0.59	0.57	0.16	0.17	0.16

Table 6.37: Avalanche shape collapse by hippocampal subregion under the two epilepsy phases (induced epilepsy), animal Epi (Asclepius). γ is the rescaling exponent, CV the collapse quality, p its significance against a shuffled null, and “decades” the dynamic range of durations ($\log_{10}(T_{\max}/T_{\min})$).

Phase	Reg.	Tr.	γ	CV	p	Decades
Epileptog.	dCA1	6	1.16	0.037	5.47×10^{-11}	1.1
		7	1.16	0.032	4.13×10^{-11}	1.1
		8	1.17	0.032	1.70×10^{-11}	1.0
	dDG	6	1.11	0.084	6.55×10^{-4}	0.5
		7	1.13	0.084	2.66×10^{-4}	0.5
		8	1.11	0.077	1.72×10^{-3}	0.4
	vDG	6	1.07	0.033	2.23×10^{-3}	0.5
		7	1.03	0.033	8.96×10^{-2}	0.5
		8	1.09	0.048	2.90×10^{-2}	0.5
Status ep.	dCA1	–	1.27	0.064	5.87×10^{-11}	1.3
	dDG	–	1.18	0.067	4.43×10^{-4}	0.5
	vDG	–	1.22	0.087	1.20×10^{-5}	0.7

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