Self-organization toward criticality by synaptic plasticity

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Abstract

Self-organized criticality has been proposed to be a universal mechanism for the emergence of scale-free dynamics in many complex systems, and possibly in the brain. While such scalefree patterns were identified experimentally in many different types of neural recordings, the biological principles behind their emergence remained unknown. Utilizing different network models and motivated by experimental observations, synaptic plasticity was proposed as a possible mechanism to self-organize brain dynamics towards a critical point. In this review, we discuss how various biologically plausible plasticity rules operating across multiple timescales are implemented in the models and how they alter the network's dynamical state through modification of number and strength of the connections between the neurons. Some of these rules help to stabilize criticality, some need additional mechanisms to prevent divergence from the critical state. We propose that rules that are capable of bringing the network to criticality can be classified by how long the near-critical dynamics persists after their disabling. Finally, we discuss the role of self-organization and criticality in computation. Overall, the concept of criticality helps to shed light on brain function and self-organization, yet the overall dynamics of living neural networks seem to harnesses not only criticality for computation, but also deviations thereof.

1 Introduction

More than thirty years ago, Per Bak, Chao Tang, and Kurt Wiesenfeld [10] discovered a strikingly simple way to generate scale-free relaxation dynamics and pattern statistic, that had been observed in systems as different as earthquakes [58, 59], snow avalanches [18], forest fires [94], or river networks [137, 154]. Thereafter, hopes were expressed that this self-organization mechanism for scale-free emergent phenomena would explain how any complex system in nature worked, and hence it did not take long until the hypothesis sparked that brains should be self-organized critical as well [12].

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The idea that potentially the most complex object we know, the human brain, self-organizes to a critical state was explored early on by theoretical studies [28, 31, 46, 68], but it took more than 15 years until the first scale-free "neuronal avalanches" were discovered [12]. Since then, we have seen a continuous, and very active interaction between experiment and theory. The initial, simple and optimistic idea that the brain is self-organized critical similar to a sandpile has been refined and diversified. Now we have a multitude of neuroscience-inspired models, some showing classical self-organized critical dynamics, but many employing a set of crucial parameters to switch between critical and non-critical states [34, 46, 62, 183]. – Likewise the views on neural activity have been extended: We now have the means to quantify the distance to criticality even from the very few neurons we can record in parallel [172]. Overall, we have observed in experiments, how developing networks self-organize to a critical state [81, 155, 176], how states may change from wakefulness to deep sleep [4, 19, 89, 92, 123], under drugs [99] or in a disease like epilepsy [6, 61, 101, 136]. These results show how criticality and the deviations thereof can be harnessed for computation, but can also reflect cases where self-organization fails.

Parallel to the rapid accumulation of experimental data, models describing the complex brain dynamics were developed to draw a richer picture. It is worthwhile noting that the seminal sandpile model [9] already bears a striking similarity with the brain: The distribution of heights at each site of the system beautifully corresponds to the membrane potential of neurons, and in both systems, small perturbations can lead to scale-free distributed avalanches. However, whereas in the sandpile the number of grains naturally obeys a conservation law, the number of spikes or the summed potential in a neural network does not.

This points to a significant difference between classical SOC models and the brain: While in the SOC model the conservation law fixes the interaction between sites, in neuroscience connections strengths are ever-changing. Incorporating biologically plausible interactions is one of the largest challenges, but also the greatest opportunity for building the neuronal equivalent of a SOC model. Synaptic plasticity rules governing changes in the connections strengths often couple the interactions to the activity on different timescales. Thus, they can serve as the perfect mechanism for the self-organization and tuning the network's activity to the desired regime.

Here we systematically review biologically plausible models of avalanche-related criticality with plastic connections. We discuss the degree to which they can be considered SOC proper, quasicritical, or hovering around a critical state. We examine how they can be tuned towards and away from the classical critical state, and in particular, what are the biological control mechanisms that determine self-organization. Our main focus is on models that exhibit signatures of criticality captured by the avalanche size distribution.

2 Modeling neural networks with plastic synapses

Let us briefly introduce the very basics of neural networks, modeling neural circuits and synaptic plasticity. Most of these knowledge can be found in larger details in neuroscience text-books [38, 52, 72]. The human brain contains about 80 billion neurons. Each neuron is connected to thousands of other neurons. The connections between the neurons are located on fine and long trees of "cables". Each neuron has one such tree to collect signals from other neurons (dendritic tree), and a different tree to send out signals to another set of neurons (axonal tree). Biophysically, the connections between two neurons are realized by synapses. These synapses

are special: Only if a synapse is present between a dendrite and an axon can one neuron activate the other (but not necessarily conversely). The strength or weight w_{ij} of a synapse determines how strongly neuron j contributes to activating neuron i. If the summed input to a neuron exceeds a certain threshold within a short time window, the receiving neuron gets activated and fires a spike (a binary signal). If a synapse w_{ij} allows neuron j to send signals to neuron i, it does not mean that the reverse synapse, w_{ji} is also present. Thus, unlike classical physics systems, interactions between units are not symmetric but determined by a sparse, non-symmetric weight matrix W. Moreover, interactions are not continuous but pulse-like (spike), and they are time-delayed by a few milliseconds: It takes a few milliseconds for a spike to travel along an axon, cross the synapse, and reach the cell body of the receiving neuron. Most interestingly, the synaptic weights w_{ji} change over time. This is termed synaptic plasticity and is the core mechanism behind learning.

Before we turn to studying synaptic plasticity in a model, the complexity of a living brain has to be reduced into a simplified model. Typically, neural networks are modelled with a few hundred or thousand of neurons. These neurons are either spiking, or approximated by "rate neurons" which represent the joint activity of an ensemble of neurons. Such rate neurons also exist in vivo, e.g., in small animals, releasing graded potentials instead of spikes. Of all neurons in the human cortex, 80% are often modelled as excitatory neurons; when active, excitatory neurons contribute to activating their post-synaptic neurons (i.e., the neurons to whom they send their signal). The other 20% of neurons are inhibitory, bringing their post-synaptic neurons further away from their firing threshold. Effectively, an inhibitory neuron is modelled as having negative outgoing synaptic weights w_{ij} , whereas excitatory neurons have positive outgoing weights. In many simplified models, only one excitatory population is considered, and inhibition is implicitly assumed to be contributing to activity propagation probability that is already included in the excitatory connections. The connectivity matrix W between the neurons is typically sparse, since most of the possible synapses are not realized. In models, the connectivity and initial strength of synapses are often drawn from some random distribution. In some studies, however, the impact of specific choices for connectivity and topology is explicitly explored, as outlined in this review (section 2.3.4). Finally, the model neurons often receive some external activation or input in addition to the input generated from the network connections to keep the network going and avoid an absorbing (quiescent) state.

Numerous types of plasticity mechanisms shape the activity propagation in neuronal systems. One type of plasticity acts at the synapses regulating their creation and deletion, and determining changes in their weights w_{ij} . Thereby, regulating postsynaptic potentials, which govern the ability of the sending neuron to contribute to the activation of the receiving neuron and thus to activity propagation in the network. The other types of plasticity mechanisms regulate the overall excitability of the neuron, for example, by changing the spiking (activation) threshold or by adaptation currents.

The reasons and mechanisms of changing synaptic strength and neural excitability differ broadly. Changes of the synaptic strengths and excitability in the brain occur at different timescales that is particularly important for maintaining the critical dynamics. Some are very rapid acting within tens of milliseconds, or associated with every spike; others only make changes on the order of hours or even slower. For this review we simplified the classification in three temporally and functionally distinct classes, Figure 1.

The timescale of a plasticity rule influences how it contributes to the state and collective dynamics of brain networks. At the first level, we separate short-term plasticity acting on the

time (ms)

time (h)

post

time (ms)

Figure 1: Schematic examples of synaptic plasticity. (A) short-term synaptic depression acts on the timescale of spiking activity, and does not generate long-lasting changes. (B) For spike-timing dependent plasticity (STDP), a synapse is potentiated upon causal pairing of pre- and postsynaptic activity (framed orange) and depressed upon anti-causal pairing (framed green), forming long-lasting changes after multiple repetitions of pairing. (C) Homeostatic plasticity adjusts presynaptic weights (or excitability) to maintain a stable firing rate. After reduction of a neuron's firing rate (e.g. after a lesion and reduction of input), the strengths of incoming excitatory synapses are increased to reestablish the neuron's target firing rate. In contrast, if the actual firing rate is higher than the target rate, then synapses are weakened, and the neuron returns to its firing rate – on the timescales of hours or days.

timescale of dozens milliseconds, from the long-term plasticity acting with a time constant of minutes to days. As an illustration for short-term plasticity, we present prominent examples of short-term depression (see section 2.2). Among the long-term plasticity rules, we separate two distinct classes. First, plasticity rules that are explicitly associated with learning structures for specific activity propagation such as Hebbian and spike-timing-dependent plasticity (STDP, Figure 1, middle). Second, homeostatic plasticity that maintains stable firing rate by up or down regulating neuronal excitability or synaptic strength to achieve a stable target firing rate over long time. This plasticity rule is particularly active after sudden or gradual changes in input to a neuron or neural network, and aims at re-establishing the neuron's firing rate (Figure 1, right).

2.1 Criticality in network models

Studying the distributions of avalanches is a common way to characterize critical dynamics in network models. Depending on the model, avalanches can be defined in different ways. When it is meaningful to impose the separation of timescales (STS), an avalanche is measured as the entire cascade of events following a small perturbation (e.g., activation of a single neuron) - until the activity dies out. However, a STS cannot be completely mapped to living neural systems due to the presence of spontaneous activity or external input. The external input impedes the pauses between avalanches and makes an unambigous separation difficult [124]. In models, such external input can be incorporated to make them more realistic. To extract avalanches from living networks or from models with input, a pragmatic approach is chosen. The binary events (spikes or thresholded continuous signals) of all channels or neurons are binned in time. An avalanche is then defined as a sequence of active bins between two silent bins. In case the level of activity is so high that virtually no empty bins are observed, then the entire activity is thresholded, and only activity exceeding some threshold is considered as part of an avalanche. While both binning and thresholding methods are widely used, concerns were raised that depending on the bin size [12, 122–124], the value of the threshold [168], or the

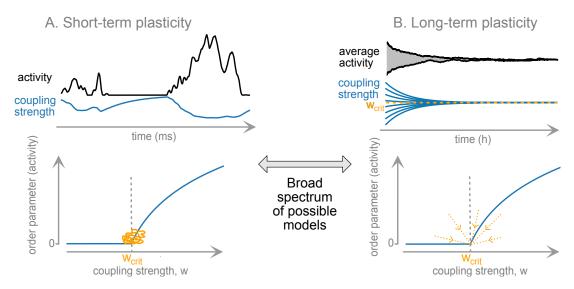


Figure 2: Classical plasticity rules and set-points of network activity. (A) Short-term plasticity serves as immediate feedback (top). The resulting long-term behavior of the network hovers near the critical point (orange trace, bottom panel). (B) Long-term plasticity results in slow (timescale of hours or longer) convergence to the fixpoint of global coupling strength. In some settings, this fixpoint may correspond to the second-order phase-transition point (bottom), rendering the critical point a global attractor of dynamics.

intensity of input [35] distribution of observed avalanches and estimated power-law exponents might be altered. Therefore, to characterize critical dynamics using avalanches it is important to investigate the fundamental scaling relations between the exponents of avalanche size, duration and shapes to avoid misleading results [50, 109], or instead use approaches to assess criticality that do not require the definition of avalanches [87, 172]. We elaborate on these challenges and bias-free solutions in a different book chapter [125]; for the remainder of this review, we assume that avalanches can be assessed unambiguously.

The timescale of a plasticity rule is crucial for the plasticity's ability in reaching and maintaining closeness to criticality. While short-term plasticity acts very quickly, it does not generate long-lasting, stable modifications of the network; and it can clearly serve as a feedback between activity and connection strength. Long-term plasticity, on the other side, takes longer to act, but can result in a stable convergence to critical dynamics, Figure 2. To summarize their properties:

- Long-term plasticity is timescale-separated from activity propagation, whereas short-term plasticity evolves at similar timescales.
- Long-term plasticity can self-organize a network to a critical state.
- Short-term plasticity constitutes an inseparable part of the network dynamics. It generates critical statistics in the data, working as a negative feedback.
- The core difference: long-term plasticity, after convergence, can be switched off and the system will remain at criticality. Switching off short-term plasticity will almost surely destroy apparent critical dynamics.
- There is a continuum of mechanisms on different timescales between these two extremes. Rules from this continuum can generate critical states that persist for varying time after rule-disabling, potentially even infinitely.

2.2 Short-term synaptic plasticity

The short-term plasticity (STP) captures activity-related changes in connection strength at a timescale close to the timescale of activity propagation, typically on the order of hundreds to thousands of milliseconds. There are two dominant contributors to the short-term synaptic plasticity: the depletion of synaptic resources used for synaptic transmission and the transient accumulation of the Ca²⁺ ions that are entering the cell after each spike [186].

Synaptic resources are used for each spike, depleting a ready-releasable pool and leading to synaptic depression, i.e., decreasing the coupling strength after each spike, Figure 1A. Synapses whose dynamics is dominated by depletion are called *depressing synapses* [96]. At the same time, for some synapses, recent firing increases the probability of release for the vesicles in a ready-releasable pool. This mechanism leads to the increase of the coupling strength for a range of firing frequencies. Synapses with measurable contributions from it are called *facilitating synapses* [159].

Although STP appears to be an inevitable consequence of synaptic physiology, multiple studies found that it can play an essential role in multiple brain functions. The most straightforward role is in the temporal filtering of inputs, i.e., short-term depression will result in low-pass filtering [3] that can be employed to reduce redundancy in the incoming signals [56]. Additionally, it was shown to explain well the working memory [107].

To model the changes in the connection strength associated with short-term synaptic plasticity it is sufficient to introduce two additional dynamic variables: J_i indicates the number of synaptic resources available in neuron i, and u_i fraction of these resources that will be used for spike. Coupling strength is captured by $w_i(t) = J_i(t)u_i(t)$ Each time when neuron i emits a spike at time $t_{\rm sp}^i$, J_i is reduced by $J_i(t_{\rm sp}^i)u_i(t_{\rm sp}^i)$. In between spikes, the resources recover and J_i approaches its resting value $J_{\rm rest}$ at a time scale τ_J .

$$\dot{J}_i = \frac{1}{\tau_J} \left(J_{\text{rest}} - J_i \right) - u_i J_i \delta \left(t - t_{\text{sp}}^i \right), \tag{1}$$

with δ denoting Dirac delta function. To add synaptic facilitation, we equip u_i with temporal dynamics, increasing it at each spike and decreasing between the spikes:

$$\dot{u}_i = \frac{1}{\tau_u} \left(u_{\text{rest}} - u_i \right) + (1 - u_i) u_{\text{rest}} \delta \left(t - t_{\text{sp}}^j \right). \tag{2}$$

Including depressing synapses (Eq. 1) in the integrate-and-fire neuronal network was shown to increase the range of coupling parameters leading to the power-law scaling of avalanche size distribution [84] as compared to the network without synaptic dynamics. If facilitation (Eq. 2) is included in the model, an additional first-order transition arises [85]. Both models have an analytical mean-field solution. In the limit of the infinite network size, the critical dynamics is obtained for any large enough coupling parameter. It was later suggested that the state reached by the system equipped with depressing synapses is not SOC, but self-organized quasi-criticality [22], as it is not locally energy preserving.

The mechanism of the near-critical region extension with depressing synapses is rather intuitive. If there is a large event propagating through the network, the massive usage of synaptic

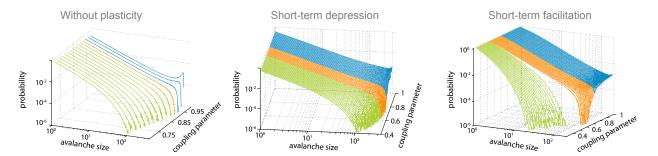


Figure 3: Short-term plasticity increases the range of the near critical regime. Left: model without plasticity reaches critical point only for single coupling parameter. Middle: short-term depression extends the range of parameters resulting in the critical dynamics. Right: short-term facilitation and depression together generate discontinuous transition.

resources effectively decouples the network. This in turn prevents the next large event for a while, until the resources are recovered. At the same time, series of small events allow to build up connection strength increasing the probability of large avalanche. Thus, for the coupling parameters above the critical values, the negative feedback generated by the synaptic depression allows to bring the system closer to the critical state. Complimentary, short-term facilitation can help to shift slightly subciritical systems to a critical state.

The system with STD is essentially a two-dimensional dynamical system (with one variable corresponding to activity, and other to momentary coupling strength). Critical behavior is observed in the activity-dimension, over a long period of time while the coupling is hovering around the mean value as response to the changing activity. If the plasticity is "switched off" only a single parameter is generating critical-like dynamics. For the large system size, where even the smallest parameter deviation results in the big difference in the distribution, the probability to switch off plasticity at the moment of critical coupling strength is 0. Short-term plasticity generates correlations between consequent avalanche sizes and inter-avalanche intervals, similar to what was observed in the neuronal data [91].

After the first publication [82], short-term depression was employed in multiple models discussing other mechanisms or different model for individual neurons. To name just few: in binary probabilistic networks [78], in networks with long-term plasticity [104, 178], in spatially pre-structured networks [170]. In one of the few studies using leaky integrate-and-fire neurons, short term depression was also found to result in critical dynamics under a specific way of defining neuronal avalanches [106]. Later, it was shown that this particular definition will lead to power-law statistics also in clearly non-critical systems [98]. In all cases the short-term plasticity contributes to the generation of a stable critical regime for a broad parameter range.

2.3 Long-term synaptic plasticity and network reorganization

Long-term modifications in neuronal networks are created by two mechanisms: long-term synaptic plasticity and network reorganization. With the long-term plasticity, synaptic weights change over long timescales, but the adjacency matrix of the network remains unchanged. However, with network reorganization, new synapses are created, and some synapses might be removed. Both of these mechanisms can potentially contribute to self-organizing the network dynamics towards or away from criticality.

Three types of long-term plasticity rules have been proposed as possible mechanisms for SOC: Hebbian plasticity, Spike-timing-dependent plasticity (STDP) and homeostatic plasticity. In Hebbian plasticity connections between near-synchronous neurons are strengthened, while in STDP a temporally asymmetric rule is applied where depending on the order of pre- and post-synaptic spike-timings, connections can be strengthened or weakened. On the other hand, homeostatic plasticity acts as a negative feedback that stabilizes the network's firing rate. In the following, we will discuss how each of these mechanisms can contribute to creating self-organized critical dynamics.

2.3.1 Hebbian-like plasticity

Hebbian plasticity is typically formulated in a slogan-like form: Neurons that fire together, wire together. This means that connections between neurons with similar spike-timing will be strengthened. This rule can imprint stable attractors into the network's dynamics, constituting the best candidate mechanism for memory formation. Hebbian plasticity in its standard form does not reduce coupling strength, thus without additional stabilization mechanisms Hebbian plasticity leads to runaway excitation. Additionally, presence of stable attractors makes it hard to maintain the scale-free distribution of avalanche sizes.

The first papers uniting Hebbian-like plasticity and criticality came from Lucilla de Arcangelis' and Hans J. Herrmann's labs. In a series of publications, they demonstrated that a network of non-leaky integrators, equipped with plasticity and stabilizing synaptic scaling develops both power-law scaling of avalanches (with exponent 1.2 or 1.5 depending on the external drive) and power-law scaling of spectral density [42, 117]. In the follow up paper, they realized multiple logical gates using additional supervised learning paradigm [5].

Using Hebbian-like plasticity to imprint patterns in the network and simultaneously maintain critical dynamics is a very non-trivial task. Uhlig et al. [164] achieved it by alternating Hebbian learning epochs with the epochs of normalizing synaptic strength to return to a critical state. The memory capacity of the trained network was close to the maximal possible capacity and remain close to criticality. However, the network without homeostatic regulation towards a critical state achieved better retrieval. This might point to the possibility that classical criticality is not an optimal substrate for storing simple memories as attractors. However, in the so-far unstudied setting of storing memories as dynamic attractors, the critical system's sensitivity might make it the best solution.

2.3.2 Spike-timing-dependent plasticity (STDP)

Spike-timing-dependent plasticity (STDP) is a form of activity-dependent plasticity in which synaptic strength is adjusted as a function of timing of spikes in pre- and post-synaptic neurons. It can appear both in the form of long-term potentiation (LTP) or long-term depression (LTD) [147]. Suppose the post-synaptic neuron fires shortly after the pre-synaptic neuron. In that case, the connection from pre- to the post-synaptic neuron is strengthened (LTP), but if the post-synaptic neuron fires after the pre-synaptic neuron, the connection is weakened (LTP), Figure 1 B. Millisecond temporal resolution measurements of pre- and postsynaptic spikes experimentally by Markram et al. [15, 95, 97] together with theoretical model proposed by Gerstner et al. [53] put forward STDP as a mechanism for sequence learning. Shortly after

that other theoretical studies [33, 47, 60, 74, 127] incorporated STDP in their models as a local learning rule.

Different functional forms of STDP are observed in different brain areas and across various species (for a review see [149]). For example, STDP in hippocampal excitatory synapses appear to have equal temporal windows for LTD and LTP [15, 112, 182], while in neocortical synapses it exhibits longer LTD temporal windows [48, 148]. Interestingly, an even broader variety of different STDP kernels were observed for inhibitory connections [66].

The classical STDP is often modeled by modifying the synaptic weight w_{ij} from pre-synptic neuron j to post-synaptic neuron i as

$$\Delta w_{ij} = \begin{cases} A_{+}(w_{ij}) \exp(\frac{t_{j} - t_{i}}{\tau_{+}}) & t_{j} < t_{i} \\ -A_{-}(w_{ij}) \exp(\frac{t_{j} - t_{i}}{\tau_{-}}) & t_{j} \ge t_{i} \end{cases}$$
(3)

where t_i and t_j are latest spikes of neurons i and j and τ_+ and τ_- are LTP and LTD time constants. Weight dependence functions $A_+(w_{ij})$ and $A_-(w_{ij})$ control the synaptic weights to stay between 0 and w_{max} , which is required from the biological point of view. Two families of weight dependence functions have been introduced: (i) soft weight bounds (multiplicative weights) [129], (ii) hard weight bounds (additive weights) [53]. Soft weight bounds are implemented as

$$A_{+}(w_{ij}) = (w_{max} - w_{ij})\eta_{+}, \quad A_{-}(w_{ij}) = w_{ij}\eta_{-}, \tag{4}$$

where $\eta_+ < 1$ and $\eta_- < 1$ are positive constants. Weight dependence functions with hard bounds are defined using a Heaviside step function H(x) as

$$A_{+}(w_{ij}) = H(w_{max} - w_{ij})\eta_{+}, \quad A_{-}(w_{ij}) = H(-w_{ij})\eta_{-}.$$
 (5)

There are two types of critical points that can be attained by networks with STDP. The first transition type is characterized by statistics of weights in the converged network. For instance, at this point synaptic coupling strengths [100] or the fluctuations in coupling strengths [143] follow a power-law distribution. The second transition type is related to network's dynamics, it is characterized by presence of scale-free avalanches [67, 75, 130]. In these models STDP is usually accompanied by fine-tuning of some parameters or properties of the network to create critical dynamics. This suggests that STDP alone might not be sufficient for SOC.

Rubinov et al. [130] developed a leaky integrate-and-fire (LIF) network model with modular connectivity (Figure 4A,B). In their model, STDP only gives rise to power-law distributions of avalanches when the ratio of connection between and within modules is tuned to a particular value. Their results were unchanged for STDP rules with both soft and hard bounds. However, they reported that switching off the STDP dynamics leads to the deterioration of the critical state, which disappears completely after a while. This property places the model in-between truly long-term and short-term mechanisms. Additionally, avalanches were defined based on the activity of modules (simultaneous activation of a large number of neurons within a module). In this modular definition of activity, SOC is achieved by potentiating within-module synaptic weights during module activation and depression of weights in-between module activations. While the module-based definition of avalanches could be relevant to the dynamics of cell-assemblies in the brain or more coarse-grained activity such as local field potentials (LFP), further investigation of avalanches statistics based on individual neurons activity is required.

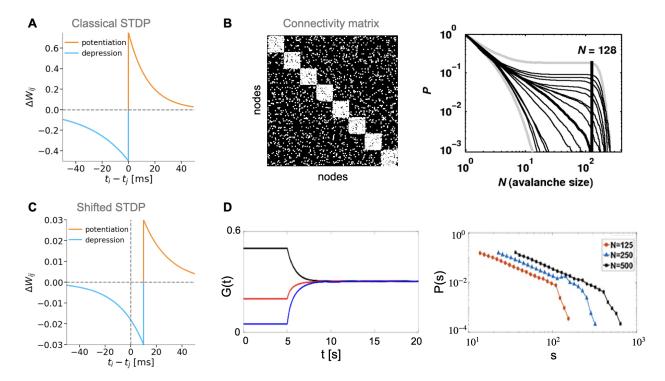


Figure 4: **Different STDP rules and their role in creating SOC.** (A) Classical STDP rule with asymmetric temporal windows. (B) A modular network that is rewired with a specific probability to create particular inter- and intra-modules connections (left) combined with classical STDP gives rise to dynamics characterized by power-law avalanche-size distribution (right, thick line). Non-power-law avalanche-size distributions correspond to other rewiring probabilities with the gray lines showing the two extremes of ordered and random networks (reproduced from [130] under CC BY license). (C) Shifted STDP rule. (D) (left) Average coupling strength G(t) in a network with shifted STDP will converge to a steady state value. (right) Setting the STDP time-shit to $\tau = 10$ ms (equal to axonal delay time-constant) leads to emergence of power-law avalanche-size distributions that scale with the system size (reproduced from [75] under CC BY license).

Observation of power-law avalanche distributions was later extended to a network of Izhikevich neurons with a temporally shifted soft-bound STDP rule [75] (Figure 4C,D). The shift in the boundary between potentiation and depression reduces the immediate synchronization between pre- and post-synaptic neurons that eventually stabilizes the synaptic weights and the post-synaptic firing rate similar to a homeostasis regulation [7]. In the model, the STDP time-shift is set to be equal to the axonal delay time constant that also acts as a control parameter for the state of dynamics in the network. The authors showed that for a physiologically plausible time constant ($\tau = 10$ ms) network dynamics self-organizes to the edge of synchronization transition point. At this transition point, distribution of size and duration of avalanches follow a power-law-like distribution. They showed that the power-law exponents can be approximately fitted in the standard scaling relation required for a critical system [50]. However, since they defined avalanches based on thresholding of the global network firing, estimated avalanche distributions and fitted exponents might be generated by the thresholding [168].

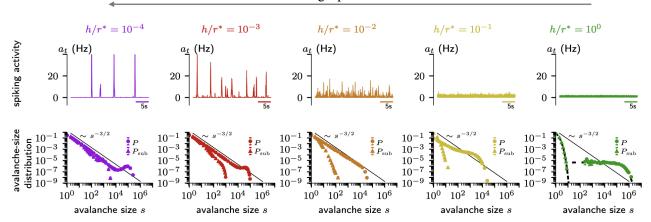


Figure 5: Homeostatic plasticity regulation can create different types of dynamics in the network depending on input strength h, target firing rate r^* and recurrent interactions. (top) example spiking activity traces. (bottom) full-sampled (circles) and subsampled (triangles) avalanche size distributions averaged over 12 independent simulations. From left to right: network generates bursting $(m > 1, h/r^* \le 10^{-3}$, purple-red), fluctuating $(m \approx 0.99, h/r^* \approx 10^{-2}$ and $m \approx 0.9, h/r^* \approx 10^{-1}$, orange-yellow) and irregular $(m \approx 0, h/r^* = 1, \text{ green})$. Solid lines shows the corresponding branching process avalanche-size distributions $P(s) \propto s^{-3/2}$ [63] and dashed line is the analytical avalanche-size distribution of a Poisson process [121] (reproduced from [183] by permission).

2.3.3 Homeostatic regulations

Homeostatic plasticity is a mechanism that regulates neural activity on a long timescale [37, 88, 114, 162, 163, 171]. In a nutshell, one assumes that every neuron has some intrinsic target activity rate. Homeostatic plasticity then presents a negative feedback loop that maintains that target rate and thereby stabilize network dynamics. In general, it reduces (increases) excitatory synaptic strength or neural excitability if the spike rate is above (below) a target rate, Figure 1C. This mechanism can stabilize a potentially unconstrained positive feedback loop through Hebbian-type plasticity [1, 17, 73, 105, 156, 161, 179, 180]. The physiological mechanisms of homeostatic plasticity are not fully disentangled yet. It can be implemented by a number of physiological candidate mechanisms, such as redistribution of synaptic efficacy [96, 160], synaptic scaling [49, 88, 114, 163], adaptation of membrane excitability [37, 120], or through interactions with glial cells [40, 169]. Recent results highlight the involvement of homeostatic plasticity in generating robust yet complex dynamics in recurrent networks [55, 65, 110].

In models, homeostatic plasticity was identified as one of the primary candidates to tune networks to criticality. The mechanism of it is straightforward: taking the analogy of the branching process, where one neuron (or unit) on average activates m neurons in the subsequent time step, the stable sustained activity that is the goal function of the homeostatic regulation requires $m=m_c=1$ which is precisely the critical value [63]. In 2007, Levina and colleagues made use of this principle. They devised a homeostasis-like rule, where all outgoing weights were normalized such that each neuron in the fully connected network activated on average m=1 neurons in the next time step [83]. Thereby, the network tuned itself to a critical state.

Similar ideas have been proposed and implemented first in simple models and later also in more detailed models. In the latter, homeostatic regulation tunes the ratio between excitatory and inhibitory synaptic strength [25, 32, 54, 65, 119]. It then turned out that due to the diverging

temporal correlations, which emerge at criticality, the time-scale of homeostasis would also have to diverge [25, 128]. If the time-scale of the homeostasis is faster than the timescale of the dynamics, then the network does not converge to a critical point, but hovers around it, potentially resembling supercritical dynamics [25, 128, 183]. It is now clear that a self-organization to a critical state (instead of hovering around a critical state) requires that the timescale of homeostasis is slower than that of the network dynamics [25, 128, 183].

Whether a system self-organizes to a critical state, or to a sub- or supercritical one is determined by a further parameter, which has been overlooked for a while: The rate of external input. This rate should be close to zero in critical systems to foster a separation of time scales [43, 124]. Hence, basically all models that studied criticality were implemented with a vanishing external input rate. In neural systems, however, sensory input and other brain areas provide continuous drive, and hence a separation of timescales is typically not realized [124]. As a consequence, avalanches merge, coalesce, and separate [36, 124, 184]. It turns out that under homeostatic plasticity, the external input strength can become a control parameter for the dynamics [183]: If the input strength is high, the system self-organizes to a subcritical state (Figure 5, right). With weaker input, the network approaches a critical state (Figure 5, middle). However when the input is too weak, pauses between bursts get so long that the timescale of the homeostasis again plays a role - and the network does not converge to a single state but hovers between sub- and supercritical dynamics (Figure 5, left). This study shows that under homeostasis the external input strength determines the collective dynamics of the network.

Assuming that *in vivo*, cortical activity is subject to some level of non-zero input, one expects a sightly subcritical state - which is indeed found consistently across different animals [61, 93, 172, 174, 183]. In vitro systems, however, which lack external input, are expected to show bursty avalanche dynamics, potentially hovering around a critical point with excursions to supercriticality [32, 183]. Such burst behavior is indeed characteristic for in vitro systems[12, 50, 155, 183].

Recently, Ma and colleagues characterized in experiments how homeostatic scaling might reestablish close-to-critical dynamics $in\ vivo$ after perturbing sensory input [93] (Figure 6). The past theoretical results would predict that after blocking sensory input in a living animal, the spike rate should diminish, and with the time-scale of homeostatic plasticity, a state close to critical or even super-critical would be obtained [32, 183]. In a recent experiment, however, the behavior is more intricate. Soon after blocking visual input, the network became subcritical (branching ratio m smaller than one [63, 172]) and not supercritical. It then recovered to a close-to-critical state again within two days, potentially compensating the lack of input by coupling stronger to other brain areas. The avalanche size distributions agree with the transient deviation to subcritical dynamics. This deviation to subcriticality is the opposite of what one might have expected under reduced input, and apparently cannot be attributed to concurrent rate changes (which otherwise can challenge the identification of avalanche distributions [121]): The firing rate only started to decrease one day after blocking visual input. The authors attribute this delay in rate decay to excitation and inhibition reacting with different time constants to the blocking of visual input [93].

Overall, although the exact implementation of the homeostatic plasticity on the pre- and post-synaptic sides of excitatory and inhibitory neurons remains a topic of current research, the general mechanism allows for the long-term convergence of the system to the critical point, Figure 1B. Homeostasis importantly contributes to many models including different learning mechanisms, stabilizing them.

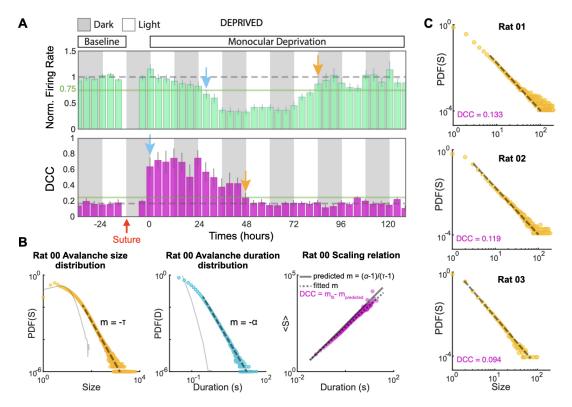


Figure 6: Homeostatic regulations in visual cortex of rats tune the network dynamics to near criticality. (A) (top) Firing rate of excitatory neurons during 7 days of recording exhibit a biphasic response to monocular deprivation (MD). After 37 h following MD firing rates were maximally suppressed (blue arrow) but came back to baseline by 84 h (orange arrow). Rates are normalized to 24 h of baseline recordings before MD. (bottom) Measuring the distance to criticality coefficient (DCC) in the same recordings. The mean DCC was significantly increased (blue arrow) upon MD, but was restored to baseline levels (near-critical regime) at 48 h (orange arrow). (B) An example of estimation of DDC (right) using the power-law exponents from the avalanche-size distribution (left) and the avalanche-duration distribution (middle). Solid gray traces show avalanche distributions in shuffled data. DCC is defined as the difference between the empirical scaling (dashed gray line) and the theoretical value (solid gray line) predicted from the exponents for a critical system as the displayed formula [50]. (C) Avalanche-size distributions and DCCs computed from 4 h of single-unit data in three example animals show the diversity of experimental observations (reproduced from the the bioRxiv version of [93] under CC-BY-NC-ND 4.0 international license).

2.3.4 Network rewiring and growth

Specific network structures such as small-world [39, 42, 84, 86] or scale-free [5, 16, 51, 70, 104, 115] networks were found to be beneficial for the emergence of critical dynamics. These network structures are in particular interesting since they have been also observed in both anatomical and functional brain networks [11, 26, 45, 64]. To create such topologies in neural networks long-term plasticity mechanisms have been used. For instance, scale-free and small-world structures emerge as a consequence of STDP between the neurons [143]. In addition, Hebbian plasticity can generate small-world networks [146].

Another prominent form of network structures are hierarchical modular networks (HMN) that can sustain critical regime for a broader range of control parameters [108, 130, 170]. Un-

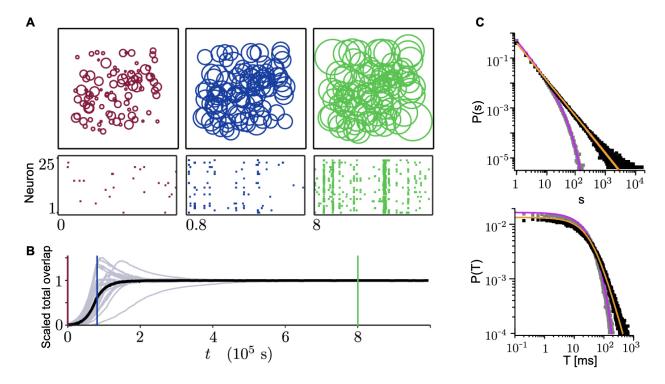


Figure 7: Growing connections based on the homeostatic structural plasticity in a network model leads to SOC. (A) Size of neurite fields (top) and spiking activity (bottom) change during the network growth process (from 25 sample neurons). From left to right: initial state (red), state with average growth (blue), stationary state reaching the homeostatic target rate (green). (B) Corresponding scaled total overlaps of 25 sample neurons (gray) and the population average (black) to the three different time points in (A). (C) Avalanche-size (top) and avalanche-duration (bottom) distributions. If the homeostatic target rate ($f_{target} = 2$ Hz) is significantly larger than the spontaneous rate ($f_0 = 0.01$ Hz) both distributions follow a power-law (black: simulation, orange: analytic). Small homeostatic target rate ($f_{target} = 0.04$ Hz) leads to subcritical dynamics (gray: simulation, pink: analytic) (reproduced from [71] with permission).

like a conventional critical point where control parameter at a single value leads to scale-free avalanches, in HMNs power-law scaling emerges for a wide range of parameters. This extended critical-like region can correspond to a Griffits phase in statistical mechanics [108]. Different rewiring algorithms have been proposed to generate HMN from an initially randomly connected [170] or a fully connected modular network [108, 130].

Experimental observations in developing neural cultures suggest that connections between neurons grow in a way such that the dynamics of the network eventually self-organizes to a critical point (i.e., observation of scale-free avalanches) [155, 176]. Motivated by this observation, different models have been developed to explain how neural networks can grow connections to achieve and maintain such critical dynamics using homeostatic structural plasticity [2, 44, 71, 79, 155, 166, 167] (for a review see [165]). In addition to homeostatic plasticity, other rewiring rules inspired by Hebbian learning were also proposed to bring the network dynamics towards criticality [23, 24, 131]. However, implementation of such network reorganizations seems to be less biologically plausible.

In most of the models with homeostatic structural plasticity, the growth of neuronal axons and dendrites is modeled as an expanding (or shrinking) circular neurite field. The growth of the neurite field for each neuron is defined based on the neuron's firing rate (or internal Ca^{2+} con-

centration). A firing rate below the homeostatic target rate (f_{target}) expands the neurite field, and a firing rate above the homeostatic target rate shrinks it. In addition, when neurite fields of two neurons overlap a connection between them will be created with a strength proportional to the overlapped area. Kossio et al. [71] showed analytically that if the homeostatic target rate is significantly larger than the spontaneous firing rate of the network, such growth mechanism would bring the network dynamics to a critical point with scale-free avalanches (Figure 7). However, for a small target rate subcritical dynamics will arise.

Tetzlaff et al. [155] proposed a slightly different mechanism where two neurites fields are assigned separately for axonal growth and dendritic growth to each neuron. While changes in the size of dendritic neurite fields follows the same rule as explained above, neurite fields of axons follow an exact opposite rule. The model simulations start with all excitatoryry neurons, but in the middle phase 20% of the neurons are changed into inhibitory ones. This switch is motivated by the transformation of GABA neurotransmitters from excitatory to inhibitory during development [13]. They showed that when the network dynamics converge to a steady-state regime, avalanche-size distributions follow a power-law.

3 Hybrid Mechanisms of Learning and Task Performance

In living neural networks, multiple plasticity mechanisms occur simultaneously. The joint contribution of diverse mechanisms has been studied in the context of criticality in a set of models [104, 118, 152]. A combination with homeostatic-like regulation is typically necessary to stabilize Hebbian or spike-timing-dependent plasticity (STDP), e.g., learning binary tasks such as an XOR rule with Hebbian plasticity [104] or sequence learning with STDP [90, 116, 133–135]. These classic plasticity rules have been paired with regulatory normalization of synaptic weights to avoid a self-amplified destabilization [73, 179, 180]. Additionally, short-term synaptic depression stabilizes the critical regime, and if it is augmented with meta-plasticity [118] the stability interval is increased even further, possibly allowing for stable learning.

In a series of studies, Scarpetta and colleagues investigated how sequences can be memorized by STDP, while criticality is maintained [133–135]. By controlling the excitability of the neurons, they achieved a balance between partial replays and noise resulting in power-law distributed avalanche sizes and durations [134]. They later reformulated the model and used the average connection strength as a control parameter, obtaining similar results [133, 135]. Whereas STDP fosters the formation of sequence memory, Hebbian plasticity is known to form assemblies (associations), and in the Hopfield network enables memory completion and recall [69]. A number of studies showed that the formation of such Hebbian ensembles is also possible while maintaining critical dynamics [132, 135, 164]. These studies show that critical dynamics can be maintained in networks, which are learning classical tasks.

The critical network can support not only memory but also real computations such as performing logical operations (OR, AND or even XOR) [5, 104]. To achieve this, the authors build upon the model with Hebbian-like plasticity that previously shown to bring the network to a critical point [42]. They added the central learning signal [8], resembling dopaminergic neuromodulation. Authors demonstrated both with [104] and without [5] short-term plasticity that the network can be trained to solve XOR-gate task.

These examples lead to the natural question of whether criticality is always optimal for learning. The criticality hypothesis attracted much attention, precisely because models at criticality show

properties supporting optimal task performance. A core properties of criticality is a maximization of the dynamic range [77, 185], the sensitivity to input, and diverging spatial and temporal correlation lengths [138, 151]. In recurrent network models and experiments, such boost of input sensitivity and memory have been demonstrated by tuning networks systematically towards and away from criticality [14, 20, 21, 76, 139–141].

When not explicitly incorporating a mechanism that drives the network to criticality, learning networks can be pushed away from criticality to a subcritical regime [34, 41, 80, 116]. This is in line with the results above that networks with homeostatic mechanisms become subcritical under increasing network input (Figure 5). Subcritical dynamics might indeed be favorable when reliable task performance is required, as the inherent variability of critical systems may corroborate performance variability [30, 57, 113, 124, 158, 173, 175].

Recently, the optimal working points of recurrent neural networks on a neuromorphic chip were demonstrated to depend on task complexity [34]. The neuromorphic chip implements spiking integrate-and-fire neurons with STDP-like depressive plasticity and slow homeostatic recovery of synaptic strength. It was found that complex tasks, which require integration of information over long time-windows, indeed profit from critical dynamics, whereas for simple tasks the optimal working point of the recurrent network was in the subcritical regime [34]. Criticality thus seems to be optimal particularly when a task makes use of this large variability, or explicitly requires the long-range correlation in time or space, e.g. for active memory storage.

4 Discussion

We summarized how different types of plasticity contribute to the convergence and maintenance of the critical state in neuronal models. The short-term plasticity rules were generally leading to hovering around the critical point, which extended the critical-like dynamics for an extensive range of parameters. The long-term homeostatic network growth and homeostatic plasticity, for some settings, could create a global attractor at the critical state. Long-term plasticity associated with learning sequences, patterns or tasks required additional mechanisms (i.e. homeostatic) to maintain criticality.

The first problem with finding the best recipe for criticality in the brain is our inability to identify the brain's state from the observations we can make. We are slowly learning how to deal with strong subsampling (under-observation) of the brain network [81, 122, 126, 150, 172, 181]. However, even if we obtained a perfectly resolved observation of all activity in the brain, we would face the problem of constant input and spontaneous activation that renders it impossible to find natural pauses between avalanches, and hence makes avalanche-based analyses ambiguous [124]. Hence, multiple avalanche-independent options were proposed as alternative assessments of criticality in the brain: (i.) detrended fluctuation analysis [87] allows to capture the scale-free behavior in long-range temporal correlations of EEG/MEG data, (ii.) critical slowing down [102] suggests a closeness to a bifurcation point, (iii.) divergence of susceptibility in the maximal entropy model fitted to the neural data [157], or the renormalization group approach [103] indicates a closeness to criticality in the sense of thermodynamic phase-transitions, and (vi.) estimating the branching parameter directly became feasible even from a small set of neurons; this estimate returns a quantification of the distance to criticality [172]. Finding the best way to unite these definitions, and select the most suitable ones for a given experiment remains largely an open problem.

Investigating the criticality hypothesis for brain dynamics has strongly evolved in the past

decades, but is far from being concluded. On the experimental side, sampling limits our access to collective neural dynamics [81, 111], and hence it is not perfectly clear yet how close to a critical point different brain areas operate. For cortex in awake animals, evidence points to a close-to-critical, but slightly subcritical state [61, 93, 174]. The precise working point might well depend on the specific brain area, cognitive state and task requirement [27, 29, 34, 140, 142, 144, 145, 153, 158, 175, 177]. Thus instead of self-organizing precisely to criticality, the brain could make use of the divergence of processing capabilities around the critical point. Thereby, each brain area might optimize its computational properties by tuning itself towards and away from criticality in a flexible, adaptive manner [175]. In the past decades, the community has revealed the local plasticity rules that would enable such a tuning and adaption of the working point. Unlike classical physics systems, which are constrained by conservation laws, the brain and the propagation of neural activity is more flexible and hence can adhere in principle a large repertoire of working points - depending on task requirements.

Criticality has been very inspiring to understand brain dynamics and function. We assume that being perfectly critical is not an optimal solution for many brain areas, during different task epochs. However, studying and modelling brain dynamics from a criticality point of view allows to make sense of the high-dimensional neural data, its large variability, and to formulate meaningful hypothesis about dynamics and computation, many of which still wait to be tested.

Conflict of interest statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author contributions

RZ, VP, and AL designed the research. RZ and AL prepared the figures. All authors contributed to writing and reviewing the manuscript.

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References

- [1] L. F. Abbott and S. B. Nelson. Synaptic plasticity: taming the beast. *Nature Neuroscience*, 3(11):1178–1183, Nov. 2000. ISSN 1546-1726. doi: 10.1038/81453. URL https://www.nature.com/articles/nn1100_1178. Number: 11 Publisher: Nature Publishing Group.
- [2] L. F. Abbott and R. Rohrkemper. A simple growth model constructs critical avalanche networks. *Prog. Brain. Res.*, 165:13–9, 2007.
- [3] L. F. Abbott, J. A. Varela, K. Sen, and S. B. Nelson. Synaptic depression and cortical gain control. *Science*, 275(5297):220–224, 1 1997. ISSN 00368075. doi: 10. 1126/science.275.5297.221. URL https://www.sciencemag.org/lookup/doi/10.1126/science.275.5297.221.
- [4] P. Allegrini, P. Paradisi, D. Menicucci, M. Laurino, A. Piarulli, and A. Gemignani. Self-organized dynamical complexity in human wakefulness and sleep: Different critical brain-activity feedback for conscious and unconscious states. *Physical Review E*, 92(3):032808, Sept. 2015. ISSN 1539-3755, 1550-2376. doi: 10.1103/PhysRevE.92.032808. URL https://link.aps.org/doi/10.1103/PhysRevE.92.032808.
- [5] L. d. Arcangelis and H. J. Herrmann. Learning as a phenomenon occurring in a critical state. *Proceedings of the National Academy of Sciences*, 107(9):3977–3981, Mar. 2010. ISSN 0027-8424, 1091-6490. doi: 10.1073/pnas.0912289107. URL https://www.pnas.org/content/107/9/3977. Publisher: National Academy of Sciences Section: Physical Sciences.
- [6] O. Arviv, M. Medvedovsky, L. Sheintuch, A. Goldstein, and O. Shriki. Deviations from Critical Dynamics in Interictal Epileptiform Activity. *Journal of Neuroscience*, 36(48): 12276–12292, Nov. 2016. ISSN 0270-6474, 1529-2401. doi: 10.1523/JNEUROSCI.0809-16. 2016. URL https://www.jneurosci.org/content/36/48/12276. Publisher: Society for Neuroscience Section: Research Articles.
- [7] B. Babadi and L. F. Abbott. Intrinsic Stability of Temporally Shifted Spike-Timing Dependent Plasticity. *PLOS Computational Biology*, 6(11):e1000961, Nov. 2010. ISSN 1553-7358. doi: 10.1371/journal.pcbi.1000961. URL https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1000961. Publisher: Public Library of Science.
- [8] P. Bak and D. R. Chialvo. Adaptive learning by extremal dynamics and negative feedback. *Physical Review E*, 63(3):031912, Feb. 2001. doi: 10.1103/PhysRevE.63.031912. URL https://link.aps.org/doi/10.1103/PhysRevE.63.031912. Publisher: American Physical Society.
- [9] P. Bak, C. Tang, and K. Wiesenfeld. Self-organized criticality: an explanation of 1/f noise. *Phys. Rev. Lett.*, 59:381–384, 1987.
- [10] P. Bak, C. Tang, and K. Wiesenfeld. Self-organized criticality. *Phys. Rev. A*, 38:364–374, 1988.
- [11] D. S. Bassett and E. Bullmore. Small-World Brain Networks. The Neuroscientist, 12 (6):512–523, Dec. 2006. ISSN 1073-8584. doi: 10.1177/1073858406293182. URL https://doi.org/10.1177/1073858406293182. Publisher: SAGE Publications Inc STM.

- [12] J. Beggs and D. Plenz. Neuronal avalanches in neocortical circuits. *J. Neurosci*, 23: 11167–11177, 2003.
- [13] Y. Ben-Ari, I. Khalilov, K. T. Kahle, and E. Cherubini. The GABA Excitatory/Inhibitory Shift in Brain Maturation and Neurological Disorders. *The Neuroscientist*, 18(5):467–486, Oct. 2012. ISSN 1073-8584. doi: 10.1177/1073858412438697. URL https://doi.org/ 10.1177/1073858412438697. Publisher: SAGE Publications Inc STM.
- [14] N. Bertschinger and T. Natschläger. Real-time computation at the edge of chaos in recurrent neural networks. *Neural Comput.*, 16(7):1413–1436, 2004. URL http://www.mitpressjournals.org/doi/abs/10.1162/089976604323057443.
- [15] G.-q. Bi and M.-m. Poo. Synaptic Modifications in Cultured Hippocampal Neurons: Dependence on Spike Timing, Synaptic Strength, and Postsynaptic Cell Type. *Journal of Neuroscience*, 18(24):10464–10472, Dec. 1998. ISSN 0270-6474, 1529-2401. doi: 10. 1523/JNEUROSCI.18-24-10464.1998. URL https://www.jneurosci.org/content/18/24/10464. Publisher: Society for Neuroscience Section: ARTICLE.
- [16] G. Bianconi and M. Marsili. Clogging and self-organized criticality in complex networks. Physical Review E, 70(3):035105, Sept. 2004. doi: 10.1103/PhysRevE.70.035105. URL https://link.aps.org/doi/10.1103/PhysRevE.70.035105. Publisher: American Physical Society.
- [17] E. L. Bienenstock, L. N. Cooper, and P. W. Munro. Theory for the development of neuron selectivity: orientation specificity and binocular interaction in visual cortex. *Journal of Neuroscience*, 2(1):32–48, Jan. 1982. ISSN 0270-6474, 1529-2401. doi: 10.1523/JNEUROSCI.02-01-00032.1982. URL https://www.jneurosci.org/content/2/1/32. Publisher: Society for Neuroscience Section: Articles.
- [18] K. W. Birkeland and C. C. Landry. Power-laws and snow avalanches. Geophysical Research Letters, 29(11):49-1-49-3, 2002. ISSN 1944-8007. doi: 10.1029/2001GL014623. URL https://agupubs.onlinelibrary.wiley.com/doi/abs/10.1029/2001GL014623.
- [19] H. Bocaccio, C. Pallavicini, M. N. Castro, S. M. Sánchez, G. De Pino, H. Laufs, M. F. Villarreal, and E. Tagliazucchi. The avalanche-like behaviour of large-scale haemodynamic activity from wakefulness to deep sleep. *Journal of The Royal Society Interface*, 16(158):20190262, Sept. 2019. doi: 10.1098/rsif.2019.0262. URL https://royalsocietypublishing.org/doi/10.1098/rsif.2019.0262. Publisher: Royal Society.
- [20] J. Boedecker, O. Obst, J. T. Lizier, N. M. Mayer, and M. Asada. Information processing in echo state networks at the edge of chaos. *Theory Biosci.*, 131(3):205–213, 2012. URL http://link.springer.com/article/10.1007/s12064-011-0146-8.
- [21] J. Boedecker, T. Lampe, and M. Riedmiller. Modeling effects of intrinsic and extrinsic rewards on the competition between striatal learning systems. Frontiers in Psychology, 4, 2013. ISSN 1664-1078. doi: 10.3389/fpsyg.2013.00739. URL https://www.frontiersin.org/articles/10.3389/fpsyg.2013.00739/full. Publisher: Frontiers.
- [22] J. A. Bonachela, S. De Franciscis, J. J. Torres, and M. A. Munoz. Self-organization without conservation: are neuronal avalanches generically critical? *Journal of Statistical Mechanics: Theory and Experiment*, 2010(02):P02015, 2010.

- [23] S. Bornholdt and T. Rohlf. Topological Evolution of Dynamical Networks: Global Criticality from Local Dynamics. *Physical Review Letters*, 84(26):6114–6117, June 2000. doi: 10.1103/PhysRevLett.84.6114. URL https://link.aps.org/doi/10.1103/PhysRevLett.84.6114. Publisher: American Physical Society.
- [24] S. Bornholdt and T. Röhl. Self-organized critical neural networks. *Physical Review E*, 67(6):066118, June 2003. doi: 10.1103/PhysRevE.67.066118. URL https://link.aps.org/doi/10.1103/PhysRevE.67.066118. Publisher: American Physical Society.
- [25] L. Brochini, A. de Andrade Costa, M. Abadi, A. C. Roque, J. Stolfi, and O. Kinouchi. Phase transitions and self-organized criticality in networks of stochastic spiking neurons. Scientific Reports, 6(1):35831, Nov. 2016. ISSN 2045-2322. doi: 10.1038/srep35831. URL https://www.nature.com/articles/srep35831. Number: 1 Publisher: Nature Publishing Group.
- [26] E. Bullmore and O. Sporns. Complex brain networks: graph theoretical analysis of structural and functional systems. Nature Reviews Neuroscience, 10(3):186–198, Mar. 2009. ISSN 1471-0048. doi: 10.1038/nrn2575. URL https://www.nature.com/articles/nrn2575. Number: 3 Publisher: Nature Publishing Group.
- [27] R. L. Carhart-Harris, S. Muthukumaraswamy, L. Roseman, M. Kaelen, W. Droog, K. Murphy, E. Tagliazucchi, E. E. Schenberg, T. Nest, C. Orban, R. Leech, L. T. Williams, T. M. Williams, M. Bolstridge, B. Sessa, J. McGonigle, M. I. Sereno, D. Nichols, P. J. Hellyer, P. Hobden, J. Evans, K. D. Singh, R. G. Wise, H. V. Curran, A. Feilding, and D. J. Nutt. Neural correlates of the LSD experience revealed by multimodal neuroimaging. *Proceedings of the National Academy of Sciences*, 113(17):4853–4858, Apr. 2016. ISSN 0027-8424, 1091-6490. doi: 10.1073/pnas.1518377113. URL https://www.pnas.org/content/113/17/4853. Publisher: National Academy of Sciences Section: Biological Sciences.
- [28] D.-M. Chen, S. Wu, A. Guo, and Z. R. Yang. Self-organized criticality in a cellular automaton model of pulse-coupled integrate-and-fire neurons. *Journal of Physics A: Mathematical and General*, 28(18):5177–5182, Sept. 1995. ISSN 0305-4470. doi: 10.1088/0305-4470/28/18/009. URL https://doi.org/10.1088%2F0305-4470%2F28%2F18%2F009. Publisher: IOP Publishing.
- [29] W. P. Clawson, N. C. Wright, R. Wessel, and W. L. Shew. Adaptation towards scale-free dynamics improves cortical stimulus discrimination at the cost of reduced detection. *PLOS Computational Biology*, 13(5):e1005574, May 2017. ISSN 1553-7358. doi: 10.1371/journal.pcbi.1005574. URL https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1005574. Publisher: Public Library of Science.
- [30] L. Cocchi, L. L. Gollo, A. Zalesky, and M. Breakspear. Criticality in the brain: A synthesis of neurobiology, models and cognition. *Progress in Neurobiology*, 158:132–152, Nov. 2017. ISSN 0301-0082. doi: 10.1016/j.pneurobio.2017.07.002. URL http://www.sciencedirect.com/science/article/pii/S0301008216301630.
- [31] Á. Corral, C. J. Pérez, A. Diaz-Guilera, and A. Arenas. Self-Organized Criticality and Synchronization in a Lattice Model of Integrate-and-Fire Oscillators. *Physical Review Letters*, 74(1):118–121, Jan. 1995. doi: 10.1103/PhysRevLett.74.118. URL https://link.aps.org/doi/10.1103/PhysRevLett.74.118. Publisher: American Physical Society.

- [32] A. A. Costa, L. Brochini, and O. Kinouchi. Self-Organized Supercriticality and Oscillations in Networks of Stochastic Spiking Neurons. *Entropy*, 19(8):399, Aug. 2017. doi: 10.3390/e19080399. URL https://www.mdpi.com/1099-4300/19/8/399. Number: 8 Publisher: Multidisciplinary Digital Publishing Institute.
- [33] R. P. Costa, R. C. Froemke, P. J. Sjöström, and M. C. van Rossum. Unified pre- and postsynaptic long-term plasticity enables reliable and flexible learning. *eLife*, 4:e09457, Aug. 2015. ISSN 2050-084X. doi: 10.7554/eLife.09457. URL https://doi.org/10.7554/eLife.09457. Publisher: eLife Sciences Publications, Ltd.
- [34] B. Cramer, D. Stöckel, M. Kreft, M. Wibral, J. Schemmel, K. Meier, and V. Priesemann. Control of criticality and computation in spiking neuromorphic networks with plasticity. *Nature Communications*, 11(1):2853, June 2020. ISSN 2041-1723. doi: 10.1038/s41467-020-16548-3. URL https://www.nature.com/articles/s41467-020-16548-3. Number: 1 Publisher: Nature Publishing Group.
- [35] A. Das and A. Levina. Critical neuronal models with relaxed timescales separation. arXiv preprint arXiv:1808.04196, 2018.
- [36] A. Das and A. Levina. Critical neuronal models with relaxed timescale separation. *Physical Review X*, 9(2):021062, 2019.
- [37] G. W. Davis. HOMEOSTATIC CONTROL OF NEURAL ACTIVITY: From Phenomenology to Molecular Design. *Annual Review of Neuroscience*, 29(1):307–323, June 2006. ISSN 0147-006X. doi: 10.1146/annurev.neuro.28.061604.135751. URL https://www.annualreviews.org/doi/10.1146/annurev.neuro.28.061604.135751. Publisher: Annual Reviews.
- [38] P. Dayan and L. Abbott. Theoretical Neuroscience. MIT Press, Cambridge, 2001.
- [39] L. de Arcangelis and H. J. Herrmann. Self-organized criticality on small world networks. *Physica A: Statistical Mechanics and its Applications*, 308(1):545–549, May 2002. ISSN 0378-4371. doi: 10.1016/S0378-4371(02)00549-6. URL http://www.sciencedirect.com/science/article/pii/S0378437102005496.
- [40] M. De Pittà, N. Brunel, and A. Volterra. Astrocytes: Orchestrating synaptic plasticity? Neuroscience, 323:43-61, May 2016. ISSN 0306-4522. doi: 10.1016/j.neuroscience.2015.04.001. URL http://www.sciencedirect.com/science/article/pii/S0306452215003188.
- [41] B. Del Papa, V. Priesemann, and J. Triesch. Fading Memory, Plasticity, and Criticality in Recurrent Networks. In N. Tomen, J. M. Herrmann, and U. Ernst, editors, *The Functional Role of Critical Dynamics in Neural Systems*, Springer Series on Bio- and Neurosystems, pages 95–115. Springer International Publishing, Cham, 2019. ISBN 978-3-030-20965-0. doi: 10.1007/978-3-030-20965-0_6. URL https://doi.org/10.1007/978-3-030-20965-0_6.
- [42] L. de Arcangelis, C. Perrone-Capano, and H. J. Herrmann. Self-Organized Criticality Model for Brain Plasticity. *Physical Review Letters*, 96(2):028107, Jan. 2006. doi: 10.1103/PhysRevLett.96.028107. URL https://link.aps.org/doi/10.1103/PhysRevLett.96.028107. Publisher: American Physical Society.
- [43] R. Dickman, M. A. Muñoz, A. Vespignani, and S. Zapperi. Paths to self-organized criticality. *Brazilian Journal of Physics*, 30(1):27–41, Mar. 2000. ISSN 0103-9733. doi:

- 10.1590/S0103-97332000000100004. URL http://www.scielo.br/scielo.php?script=sci_abstract&pid=S0103-97332000000100004&lng=en&nrm=iso&tlng=en. Publisher: Sociedade Brasileira de Física.
- [44] F. Droste, A.-L. Do, and T. Gross. Analytical investigation of self-organized criticality in neural networks. *Journal of The Royal Society Interface*, 10(78):20120558, 2013. doi: 10.1098/rsif.2012.0558. URL https://royalsocietypublishing.org/doi/full/10.1098/rsif.2012.0558.
- [45] V. M. Eguíluz, D. R. Chialvo, G. A. Cecchi, M. Baliki, and A. V. Apkarian. Scale-Free Brain Functional Networks. *Physical Review Letters*, 94(1):018102, Jan. 2005. doi: 10.1103/PhysRevLett.94.018102. URL https://link.aps.org/doi/10.1103/PhysRevLett.94.018102. Publisher: American Physical Society.
- [46] C. W. Eurich, J. M. Herrmann, and U. A. Ernst. Finite-size effects of avalanche dynamics. *Physical Review E*, 66(6):066137, Dec. 2002. doi: 10.1103/PhysRevE.66.066137. URL https://link.aps.org/doi/10.1103/PhysRevE.66.066137. Publisher: American Physical Society.
- [47] M. A. Farries and A. L. Fairhall. Reinforcement Learning With Modulated Spike Timing-Dependent Synaptic Plasticity. *Journal of Neurophysiology*, 98(6):3648-3665, Dec. 2007. ISSN 0022-3077. doi: 10.1152/jn.00364.2007. URL https://journals. physiology.org/doi/full/10.1152/jn.00364.2007. Publisher: American Physiological Society.
- [48] D. E. Feldman. Timing-Based LTP and LTD at Vertical Inputs to Layer II/III Pyramidal Cells in Rat Barrel Cortex. Neuron, 27(1):45-56, July 2000. ISSN 0896-6273. doi: 10.1016/S0896-6273(00)00008-8. URL http://www.sciencedirect.com/science/article/pii/S0896627300000088.
- [49] M.-f. Fong, J. P. Newman, S. M. Potter, and P. Wenner. Upward synaptic scaling is dependent on neurotransmission rather than spiking. *Nature Communications*, 6(1):6339, Mar. 2015. ISSN 2041-1723. doi: 10.1038/ncomms7339. URL https://www.nature.com/articles/ncomms7339/. Number: 1 Publisher: Nature Publishing Group.
- [50] N. Friedman, S. Ito, B. A. W. Brinkman, M. Shimono, R. E. L. DeVille, K. A. Dahmen, J. M. Beggs, and T. C. Butler. Universal Critical Dynamics in High Resolution Neuronal Avalanche Data. *Physical Review Letters*, 108(20):208102, May 2012. doi: 10.1103/PhysRevLett.108.208102. URL https://link.aps.org/doi/10.1103/PhysRevLett.108.208102. Publisher: American Physical Society.
- [51] P. Fronczak, A. Fronczak, and J. A. Hołyst. Self-organized criticality and coevolution of network structure and dynamics. *Physical Review E*, 73(4):046117, Apr. 2006. doi: 10.1103/PhysRevE.73.046117. URL https://link.aps.org/doi/10.1103/PhysRevE.73.046117. Publisher: American Physical Society.
- [52] W. Gerstner and W. M. Kistler. Spiking Neuron Models. Single Neurons, Populations, Plasticity. Cambridge University Press, 2002.
- [53] W. Gerstner, R. Kempter, J. L. van Hemmen, and H. Wagner. A neuronal learning rule for sub-millisecond temporal coding. *Nature*, 383(6595):76–78, Sept. 1996. ISSN 1476-4687. doi: 10.1038/383076a0. URL https://www.nature.com/articles/383076a0. Number: 6595 Publisher: Nature Publishing Group.

- [54] M. Girardi-Schappo, L. Brochini, A. A. Costa, T. T. A. Carvalho, and O. Kinouchi. Synaptic balance due to homeostatically self-organized quasicritical dynamics. *Physical Review Research*, 2(1):012042, Feb. 2020. ISSN 2643-1564. doi: 10.1103/PhysRevResearch.2.012042. URL https://link.aps.org/doi/10.1103/PhysRevResearch.2.012042.
- [55] J. Gjorgjieva, J. F. Evers, and S. J. Eglen. Homeostatic Activity-Dependent Tuning of Recurrent Networks for Robust Propagation of Activity. *Journal of Neuroscience*, 36(13): 3722-3734, Mar. 2016. ISSN 0270-6474, 1529-2401. doi: 10.1523/JNEUROSCI.2511-15. 2016. URL https://www.jneurosci.org/content/36/13/3722. Publisher: Society for Neuroscience Section: Articles.
- [56] M. S. Goldman, P. Maldonado, and L. Abbott. Redundancy reduction and sustained firing with stochastic depressing synapses. *Journal of Neuroscience*, 22(2):584–591, 2002.
- [57] L. L. Gollo. Coexistence of critical sensitivity and subcritical specificity can yield optimal population coding. Journal of The Royal Society Interface, 14(134):20170207, Sept. 2017. doi: 10.1098/rsif.2017.0207. URL https://royalsocietypublishing.org/doi/10.1098/rsif.2017.0207. Publisher: Royal Society.
- [58] B. Gutenberg and C. Richter. Seismicity of the Earth. Geological Society of America, 1941. ISBN 978-0-8137-2034-0.
- [59] B. Gutenberg and C. F. Richter. Earthquake magnitude, intensity, energy, and acceleration(Second paper). Bulletin of the Seismological Society of America, 46(2):105-145, Apr. 1956. ISSN 0037-1106. URL https://pubs.geoscienceworld.org/ssa/bssa/article/46/2/105/115777/Earthquake-magnitude-intensity-energy-and. Publisher: Geo-ScienceWorld.
- [60] R. Guyonneau, R. VanRullen, and S. J. Thorpe. Neurons Tune to the Earliest Spikes Through STDP. Neural Computation, 17(4):859–879, Apr. 2005. ISSN 0899-7667. doi: 10.1162/0899766053429390. URL https://doi.org/10.1162/0899766053429390. Publisher: MIT Press.
- [61] A. Hagemann, J. Wilting, B. Samimizad, F. Mormann, and V. Priesemann. No evidence that epilepsy impacts criticality in pre-seizure single-neuron activity of human cortex. arXiv:2004.10642 [physics, q-bio], Apr. 2020. URL http://arxiv.org/abs/2004.10642. arXiv: 2004.10642.
- [62] C. Haldeman and J. Beggs. Critical branching captures activity in living neural networks and maximizes the number of metastable states. *Phys. Rev. Lett.*, 94:058101, 2005.
- [63] T. E. Harris. The Theory of Branching Processes. Grundlehren der mathematischen Wissenschaften. Springer-Verlag, Berlin Heidelberg, 1963. ISBN 978-3-642-51868-3. URL https://www.springer.com/gp/book/9783642518683.
- [64] B. J. He. Scale-free brain activity: past, present, and future. Trends in Cognitive Sciences, 18(9):480-487, Sept. 2014. ISSN 1364-6613. doi: 10.1016/j.tics.2014.04.003. URL http://www.sciencedirect.com/science/article/pii/S1364661314000850.
- [65] P. J. Hellyer, B. Jachs, C. Clopath, and R. Leech. Local inhibitory plasticity tunes macroscopic brain dynamics and allows the emergence of functional brain networks. *NeuroImage*, 124:85–95, Jan. 2016. ISSN 1053-8119. doi: 10.1016/j.neuroimage.2015.08.069. URL http://www.sciencedirect.com/science/article/pii/S1053811915007909.

- [66] G. Hennequin, E. J. Agnes, and T. P. Vogels. Inhibitory plasticity: balance, control, and codependence. *Annual review of neuroscience*, 40:557–579, 2017.
- [67] V. Hernandez-Urbina and J. M. Herrmann. Self-organized Criticality via Retro-Synaptic Signals. Frontiers in Physics, 4, 2017. ISSN 2296-424X. doi: 10.3389/fphy.2016. 00054. URL https://www.frontiersin.org/articles/10.3389/fphy.2016.00054/full. Publisher: Frontiers.
- [68] A. V. M. Herz and J. J. Hopfield. Earthquake Cycles and Neural Reverberations: Collective Oscillations in Systems with Pulse-Coupled Threshold Elements. *Physical Review Letters*, 75(6):1222–1225, Aug. 1995. doi: 10.1103/PhysRevLett.75.1222. URL https://link.aps.org/doi/10.1103/PhysRevLett.75.1222. Publisher: American Physical Society.
- [69] J. J. Hopfield. Neural networks and physical systems with emergent collective computational abilities. *Proc. Natl. Acad. Sci. USA*, 79(8):2554–2558, 1982.
- [70] D. Hughes, M. Paczuski, R. O. Dendy, P. Helander, and K. G. McClements. Solar Flares as Cascades of Reconnecting Magnetic Loops. *Physical Review Letters*, 90(13):131101, Mar. 2003. doi: 10.1103/PhysRevLett.90.131101. URL https://link.aps.org/doi/10.1103/PhysRevLett.90.131101. Publisher: American Physical Society.
- [71] F. Y. Kalle Kossio, S. Goedeke, B. van den Akker, B. Ibarz, and R.-M. Memmesheimer. Growing Critical: Self-Organized Criticality in a Developing Neural System. *Physical Review Letters*, 121(5):058301, Aug. 2018. doi: 10.1103/PhysRevLett.121.058301. URL https://link.aps.org/doi/10.1103/PhysRevLett.121.058301. Publisher: American Physical Society.
- [72] E. R. Kandel, J. H. Schwartz, and T. M. Jessell. *Principles of Neural Science*. Elsevier Science Publishing Co Inc, 3 edition, 1991.
- [73] T. Keck, T. Toyoizumi, L. Chen, B. Doiron, D. E. Feldman, K. Fox, W. Gerstner, P. G. Haydon, M. Hübener, H.-K. Lee, J. E. Lisman, T. Rose, F. Sengpiel, D. Stellwagen, M. P. Stryker, G. G. Turrigiano, and M. C. van Rossum. Integrating Hebbian and homeostatic plasticity: the current state of the field and future research directions. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 372(1715):20160158, Mar. 2017. doi: 10.1098/rstb.2016.0158. URL https://royalsocietypublishing.org/doi/10.1098/rstb.2016.0158. Publisher: Royal Society.
- [74] R. Kempter, W. Gerstner, and J. L. van Hemmen. Hebbian learning and spiking neurons. *Physical Review E*, 59(4):4498–4514, Apr. 1999. doi: 10.1103/PhysRevE.59.4498. URL https://link.aps.org/doi/10.1103/PhysRevE.59.4498. Publisher: American Physical Society.
- [75] M. Khoshkhou and A. Montakhab. Spike-Timing-Dependent Plasticity With Axonal Delay Tunes Networks of Izhikevich Neurons to the Edge of Synchronization Transition With Scale-Free Avalanches. Frontiers in Systems Neuroscience, 13, Dec. 2019. ISSN 1662-5137. doi: 10.3389/fnsys.2019.00073. URL https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6904334/.
- [76] O. Kinouchi and M. Copelli. Optimal dynamical range of excitable networks at criticality. *Nat. Phys.*, 2:348–352, 2006.

- [77] O. Kinouchi and M. Copelli. Optimal dynamical range of excitable networks at criticality. *Nat. Phys.*, 2:348–352, 2006.
- [78] O. Kinouchi, L. Brochini, A. A. Costa, J. G. F. Campos, and M. Copelli. Stochastic oscillations and dragon king avalanches in self-organized quasi-critical systems. *Scientific reports*, 9(1):1–12, 2019.
- [79] S. Landmann, L. Baumgarten, and S. Bornholdt. Self-organized criticality in neural networks from activity-based rewiring. arXiv:2009.11781 [cond-mat, physics:nlin, q-bio], Sept. 2020. URL http://arxiv.org/abs/2009.11781. arXiv: 2009.11781.
- [80] A. Lazar, G. Pipa, and J. Triesch. SORN: a self-organizing recurrent neural network. Frontiers in Computational Neuroscience, 3, 2009. ISSN 1662-5188. doi: 10.3389/neuro. 10.023.2009. URL https://www.frontiersin.org/articles/10.3389/neuro.10.023. 2009/full. Publisher: Frontiers.
- [81] A. Levina and V. Priesemann. Subsampling scaling. *Nature communications*, 8(1):1–9, 2017.
- [82] A. Levina, J. M. Herrmann, and T. Geisel. Dynamical synapses give rise to a power-law distribution of neuronal avalanches. In Y. Weiss, B. Schölkopf, and J. Platt, editors, Advances in Neural Information Processing Systems 18, pages 771–778. MIT Press, Cambridge, MA, 2006.
- [83] A. Levina, U. Ernst, and J. M. Herrmann. Criticality of avalanche dynamics in adaptive recurrent networks. *Neurocomputing*, 70(10-12):1877–1881, 2007.
- [84] A. Levina, J. M. Herrmann, and T. Geisel. Dynamical synapses causing self-organized criticality in neural networks. *Nat. Phys.*, 3:857–860, 2007. doi: http://dx.doi.org/10.1038/nphys758.
- [85] A. Levina, J. M. Herrmann, and T. Geisel. Phase transitions towards criticality in a neural system with adaptive interactions. *Phys. Rev. Lett.*, 102(11):118110, 2009.
- [86] M. Lin and T. Chen. Self-organized criticality in a simple model of neurons based on small-world networks. *Physical Review E*, 71(1):016133, Jan. 2005. doi: 10.1103/PhysRevE.71. 016133. URL https://link.aps.org/doi/10.1103/PhysRevE.71.016133. Publisher: American Physical Society.
- [87] K. Linkenkaer-Hansen, V. V. Nikouline, J. M. Palva, and R. J. Ilmoniemi. Long-range temporal correlations and scaling behavior in human brain oscillations. The Journal of neuroscience: the official journal of the Society for Neuroscience, 21(4):1370-7, 2001. ISSN 1529-2401. doi: 10.1002/anie.201106423. URL http://www.ncbi.nlm.nih.gov/pubmed/11160408.
- [88] D. V. Lissin, S. N. Gomperts, R. C. Carroll, C. W. Christine, D. Kalman, M. Kitamura, S. Hardy, R. A. Nicoll, R. C. Malenka, and M. v. Zastrow. Activity differentially regulates the surface expression of synaptic AMPA and NMDA glutamate receptors. *Proceedings of the National Academy of Sciences*, 95(12):7097–7102, June 1998. ISSN 0027-8424, 1091-6490. doi: 10.1073/pnas.95.12.7097. URL https://www.pnas.org/content/95/12/7097. Publisher: National Academy of Sciences Section: Biological Sciences.
- [89] C.-C. Lo, T. Chou, T. Penzel, T. E. Scammell, R. E. Strecker, H. E. Stanley, and P. C. Ivanov. Common scale-invariant patterns of sleep—wake transitions across mammalian

- species. Proceedings of the National Academy of Sciences, 101(50):17545–17548, Dec. 2004. ISSN 0027-8424, 1091-6490. doi: 10.1073/pnas.0408242101. URL https://www.pnas.org/content/101/50/17545. Publisher: National Academy of Sciences Section: Biological Sciences.
- [90] M. Loidolt, L. Rudelt, and V. Priesemann. Sequence memory in recurrent neuronal network can develop without structured input. bioRxiv, page 2020.09.15.297580, Sept. 2020. doi: 10.1101/2020.09.15.297580. URL https://www.biorxiv.org/content/10. 1101/2020.09.15.297580v1.
- [91] F. Lombardi, H. J. Herrmann, D. Plenz, and L. de Arcangelis. Temporal correlations in neuronal avalanche occurrence. *Scientific Reports*, 6(November 2015):1–12, 2016. ISSN 20452322. doi: 10.1038/srep24690. URL http://dx.doi.org/10.1038/srep24690.
- [92] F. Lombardi, M. Gómez-Extremera, P. Bernaola-Galván, R. Vetrivelan, C. B. Saper, T. E. Scammell, and P. C. Ivanov. Critical Dynamics and Coupling in Bursts of Cortical Rhythms Indicate Non-Homeostatic Mechanism for Sleep-Stage Transitions and Dual Role of VLPO Neurons in Both Sleep and Wake. *Journal of Neuroscience*, 40(1):171–190, Jan. 2020. ISSN 0270-6474, 1529-2401. doi: 10.1523/JNEUROSCI.1278-19.2019. URL https://www.jneurosci.org/content/40/1/171. Publisher: Society for Neuroscience Section: Research Articles.
- [93] Z. Ma, G. G. Turrigiano, R. Wessel, and K. B. Hengen. Cortical Circuit Dynamics Are Homeostatically Tuned to Criticality In Vivo. Neuron, 104(4):655-664.e4, Nov. 2019. ISSN 0896-6273. doi: 10.1016/j.neuron.2019.08.031. URL http://www.sciencedirect. com/science/article/pii/S0896627319307378.
- [94] B. D. Malamud, G. Morein, and D. L. Turcotte. Forest Fires: An Example of Self-Organized Critical Behavior. Science, 281(5384):1840–1842, Sept. 1998. ISSN 0036-8075, 1095-9203. doi: 10.1126/science.281.5384.1840. URL https://science.sciencemag.org/content/281/5384/1840. Publisher: American Association for the Advancement of Science Section: Report.
- [95] H. Markram. Regulation of Synaptic Efficacy by Coincidence of Postsynaptic APs and EPSPs. Science, 275(5297):213-215, Jan. 1997. ISSN 00368075, 10959203. doi: 10. 1126/science.275.5297.213. URL https://www.sciencemag.org/lookup/doi/10.1126/science.275.5297.213.
- [96] H. Markram and M. Tsodyks. Redistribution of synaptic efficacy between pyramidal neurons. *Nature*, 382:807–810, 1996.
- [97] H. Markram, P. J. Helm, and B. Sakmann. Dendritic calcium transients evoked by single back-propagating action potentials in rat neocortical pyramidal neurons. *The Journal of Physiology*, 485(1):1–20, 1995. ISSN 1469-7793. doi: 10.1113/jphysiol.1995.sp020708. URL https://physoc.onlinelibrary.wiley.com/doi/abs/10.1113/jphysiol.1995.sp020708.
- [98] M. Martinello, J. Hidalgo, A. Maritan, S. Di Santo, D. Plenz, and M. A. Muñoz. Neutral theory and scale-free neural dynamics. *Physical Review X*, 7(4):041071, 2017.
- [99] C. Meisel. Antiepileptic drugs induce subcritical dynamics in human cortical networks. Proceedings of the National Academy of Sciences, 117(20):11118–11125, May 2020. ISSN 0027-8424, 1091-6490. doi: 10.1073/pnas.1911461117. URL https://www.pnas.org/

- content/117/20/11118. ISBN: 9781911461111 Publisher: National Academy of Sciences Section: Biological Sciences.
- [100] C. Meisel and T. Gross. Adaptive self-organization in a realistic neural network model. *Physical Review E*, 80(6):061917, Dec. 2009. doi: 10.1103/PhysRevE.80.061917. URL https://link.aps.org/doi/10.1103/PhysRevE.80.061917. Publisher: American Physical Society.
- [101] C. Meisel, A. Storch, S. Hallmeyer-Elgner, E. Bullmore, and T. Gross. Failure of Adaptive Self-Organized Criticality during Epileptic Seizure Attacks. *PLOS Computa*tional Biology, 8(1):e1002312, Jan. 2012. ISSN 1553-7358. doi: 10.1371/journal.pcbi. 1002312. URL https://journals.plos.org/ploscompbiol/article?id=10.1371/ journal.pcbi.1002312. Publisher: Public Library of Science.
- [102] C. Meisel, A. Klaus, C. Kuehn, and D. Plenz. Critical slowing down governs the transition to neuron spiking. *PLoS Comput Biol*, 11(2):e1004097, 2015.
- [103] L. Meshulam, J. L. Gauthier, C. D. Brody, D. W. Tank, and W. Bialek. Coarse graining, fixed points, and scaling in a large population of neurons. *Physical review letters*, 123 (17):178103, 2019.
- [104] L. Michiels van Kessenich, M. Luković, L. de Arcangelis, and H. J. Herrmann. Critical neural networks with short- and long-term plasticity. *Physical Review E*, 97(3):032312, Mar. 2018. doi: 10.1103/PhysRevE.97.032312. URL https://link.aps.org/doi/10.1103/PhysRevE.97.032312. Publisher: American Physical Society.
- [105] K. D. Miller and D. J. C. MacKay. The Role of Constraints in Hebbian Learning. Neural Computation, 6(1):100–126, Jan. 1994. ISSN 0899-7667. doi: 10.1162/neco.1994.6.1.100. URL https://doi.org/10.1162/neco.1994.6.1.100. Publisher: MIT Press.
- [106] D. Millman, S. Mihalas, A. Kirkwood, and E. Niebur. Self-organized criticality occurs in non-conservative neuronal networks during 'up' states. *Nature physics*, 6(10):801–805, 2010.
- [107] G. Mongillo, O. Barak, and M. Tsodyks. Synaptic Theory of Working Memory. *Science*, 319(5869):1543–1546, 2008. ISSN 0036-8075. doi: 10.1126/science.1150769. URL http://www.sciencemag.org/cgi/doi/10.1126/science.1150769.
- [108] P. Moretti and M. A. Muñoz. Griffiths phases and the stretching of criticality in brain networks. *Nature communications*, 4:2521, 2013. ISSN 2041-1723. doi: 10.1038/ncomms3521. URL http://www.ncbi.nlm.nih.gov/pubmed/24088740.
- [109] M. A. Muñoz, R. Dickman, A. Vespignani, and S. Zapperi. Avalanche and spreading exponents in systems with absorbing states. *Phys. Rev. E*, 59(5):6175, 1999. URL http://pre.aps.org/abstract/PRE/v59/i5/p6175_1.
- [110] J. Naudé, B. Cessac, H. Berry, and B. Delord. Effects of Cellular Homeostatic Intrinsic Plasticity on Dynamical and Computational Properties of Biological Recurrent Neural Networks. *Journal of Neuroscience*, 33(38):15032–15043, Sept. 2013. ISSN 0270-6474, 1529-2401. doi: 10.1523/JNEUROSCI.0870-13.2013. URL https://www.jneurosci.org/content/33/38/15032. Publisher: Society for Neuroscience Section: Articles.
- [111] J. P. Neto, F. P. Spitzner, and V. Priesemann. A unified picture of neuronal avalanches arises from the understanding of sampling effects. arXiv:1910.09984 [cond-

- mat, physics:nlin, physics:physics, q-bio], Mar. 2020. URL http://arxiv.org/abs/1910.09984. arXiv: 1910.09984.
- [112] M. Nishiyama, K. Hong, K. Mikoshiba, M.-m. Poo, and K. Kato. Calcium stores regulate the polarity and input specificity of synaptic modification. *Nature*, 408(6812):584–588, Nov. 2000. ISSN 1476-4687. doi: 10.1038/35046067. URL https://www.nature.com/articles/35046067/. Number: 6812 Publisher: Nature Publishing Group.
- [113] M. Nolte, M. W. Reimann, J. G. King, H. Markram, and E. B. Muller. Cortical reliability amid noise and chaos. *Nature Communications*, 10(1):3792, Aug. 2019. ISSN 2041-1723. doi: 10.1038/s41467-019-11633-8. URL https://www.nature.com/articles/s41467-019-11633-8. Number: 1 Publisher: Nature Publishing Group.
- [114] R. J. O'Brien, S. Kamboj, M. D. Ehlers, K. R. Rosen, G. D. Fischbach, and R. L. Huganir. Activity-Dependent Modulation of Synaptic AMPA Receptor Accumulation. *Neuron*, 21 (5):1067–1078, Nov. 1998. ISSN 0896-6273. doi: 10.1016/S0896-6273(00)80624-8. URL http://www.sciencedirect.com/science/article/pii/S0896627300806248.
- [115] M. Paczuski and D. Hughes. A heavenly example of scale-free networks and self-organized criticality. *Physica A: Statistical Mechanics and its Applications*, 342(1): 158–163, Oct. 2004. ISSN 0378-4371. doi: 10.1016/j.physa.2004.04.073. URL http://www.sciencedirect.com/science/article/pii/S0378437104004923.
- [116] B. D. Papa, V. Priesemann, and J. Triesch. Criticality meets learning: Criticality signatures in a self-organizing recurrent neural network. *PLOS ONE*, 12(5):e0178683, May 2017. ISSN 1932-6203. doi: 10.1371/journal.pone.0178683. URL https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0178683. Publisher: Public Library of Science.
- [117] G. L. Pellegrini, L. de Arcangelis, H. J. Herrmann, and C. Perrone-Capano. Activity-dependent neural network model on scale-free networks. *Phys. Rev. E*, 76(1):016107, 2007. doi: 10.1103/PhysRevE.76.016107. URL http://link.aps.org/abstract/PRE/v76/e016107.
- [118] J. Peng and J. M. Beggs. Attaining and maintaining criticality in a neuronal network model. *Physica A: Statistical Mechanics and its Applications*, 392(7):1611–1620, 2013.
- [119] S.-S. Poil, R. Hardstone, H. D. Mansvelder, and K. Linkenkaer-Hansen. Critical-State Dynamics of Avalanches and Oscillations Jointly Emerge from Balanced Excitation/Inhibition in Neuronal Networks. *Journal of Neuroscience*, 32(29):9817–9823, July 2012. ISSN 0270-6474, 1529-2401. doi: 10.1523/JNEUROSCI.5990-11.2012. URL https://www.jneurosci.org/content/32/29/9817. Publisher: Society for Neuroscience Section: Articles.
- [120] K. Pozo and Y. Goda. Unraveling Mechanisms of Homeostatic Synaptic Plasticity. *Neuron*, 66(3):337–351, May 2010. ISSN 0896-6273. doi: 10.1016/j.neuron.2010.04.028. URL http://www.sciencedirect.com/science/article/pii/S0896627310002990.
- [121] V. Priesemann and O. Shriki. Can a time varying external drive give rise to apparent criticality in neural systems? *PLOS Computational Biology*, 14(5):e1006081, May 2018. ISSN 1553-7358. doi: 10.1371/journal.pcbi.1006081. URL https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1006081. Publisher: Public Library of Science.

- [122] V. Priesemann, M. Munk, and M. Wibral. Subsampling effects in neuronal avalanche distributions recorded in vivo. *BMC neuroscience*, 10(1):40, 2009.
- [123] V. Priesemann, M. Valderrama, M. Wibral, and M. Le Van Quyen. Neuronal avalanches differ from wakefulness to deep sleep—evidence from intracranial depth recordings in humans. *PLoS Comput. Biol.*, 9(3):e1002985, 2013.
- [124] V. Priesemann, M. Wibral, M. Valderrama, R. Pröpper, M. Le Van Quyen, T. Geisel, J. Triesch, D. Nikolić, and M. H. J. Munk. Spike avalanches in vivo suggest a driven, slightly subcritical brain state. *Front. Syst. Neurosci.*, 8:108, 2014.
- [125] V. Priesemann, A. Levina, and J. Wilting. Assessing Criticality in Experiments. In N. Tomen, J. M. Herrmann, and U. Ernst, editors, *The Functional Role of Critical Dynamics in Neural Systems*, Springer Series on Bio- and Neurosystems, pages 199–232. Springer International Publishing, Cham, 2019. ISBN 978-3-030-20965-0. doi: 10.1007/978-3-030-20965-0_11. URL https://doi.org/10.1007/978-3-030-20965-0_11.
- [126] T. L. Ribeiro, M. Copelli, F. Caixeta, H. Belchior, D. R. Chialvo, M. A. Nicolelis, and S. Ribeiro. Spike avalanches exhibit universal dynamics across the sleep-wake cycle. *PLoS One*, 5(11):e14129, 2010. URL http://dx.plos.org/10.1371/journal.pone.0014129.
- [127] P. D. Roberts and C. C. Bell. Computational Consequences of Temporally Asymmetric Learning Rules: II. Sensory Image Cancellation. *Journal of Computational Neuroscience*, 9(1):67–83, July 2000. ISSN 1573-6873. doi: 10.1023/A:1008938428112. URL https://doi.org/10.1023/A:1008938428112.
- [128] R. P. Rocha, L. Koçillari, S. Suweis, M. Corbetta, and A. Maritan. Homeostatic plasticity and emergence of functional networks in a whole-brain model at criticality. *Scientific Reports*, 8(1):15682, Oct. 2018. ISSN 2045-2322. doi: 10.1038/s41598-018-33923-9. URL https://www.nature.com/articles/s41598-018-33923-9. Number: 1 Publisher: Nature Publishing Group.
- [129] M. C. W. v. Rossum, G. Q. Bi, and G. G. Turrigiano. Stable Hebbian Learning from Spike Timing-Dependent Plasticity. *Journal of Neuroscience*, 20(23):8812–8821, Dec. 2000. ISSN 0270-6474, 1529-2401. doi: 10.1523/JNEUROSCI.20-23-08812.2000. URL https://www.jneurosci.org/content/20/23/8812. Publisher: Society for Neuroscience Section: ARTICLE.
- [130] M. Rubinov, O. Sporns, J.-P. Thivierge, and M. Breakspear. Neurobiologically Realistic Determinants of Self-Organized Criticality in Networks of Spiking Neurons. *PLOS Computational Biology*, 7(6):e1002038, June 2011. ISSN 1553-7358. doi: 10.1371/journal.pcbi.1002038. URL https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1002038. Publisher: Public Library of Science.
- [131] M. Rybarsch and S. Bornholdt. Avalanches in self-organized critical neural networks: a minimal model for the neural soc universality class. *PloS one*, 9(4):e93090, 2014.
- [132] S. Scarpetta and A. d. Candia. Neural Avalanches at the Critical Point between Replay and Non-Replay of Spatiotemporal Patterns. *PLOS ONE*, 8(6):e64162, June 2013. ISSN 1932-6203. doi: 10.1371/journal.pone.0064162. URL https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0064162. Publisher: Public Library of Science.

- [133] S. Scarpetta and A. de Candia. Alternation of up and down states at a dynamical phase-transition of a neural network with spatiotemporal attractors. *Front. Syst. Neurosci.*, 8: 88, 2014.
- [134] S. Scarpetta, F. Giacco, F. Lombardi, and A. de Candia. Effects of Poisson noise in a IF model with STDP and spontaneous replay of periodic spatiotemporal patterns, in absence of cue stimulation. *Biosystems*, 112(3):258–264, June 2013. ISSN 0303-2647. doi: 10.1016/j.biosystems.2013.03.017. URL http://www.sciencedirect.com/science/article/pii/S0303264713000725.
- [135] S. Scarpetta, I. Apicella, L. Minati, and A. de Candia. Hysteresis, neural avalanches, and critical behavior near a first-order transition of a spiking neural network. *Physical Review E*, 97(6):062305, June 2018. doi: 10.1103/PhysRevE.97.062305. URL https://link.aps.org/doi/10.1103/PhysRevE.97.062305. Publisher: American Physical Society.
- [136] M. Scheffer, J. Bascompte, W. A. Brock, V. Brovkin, S. R. Carpenter, V. Dakos, H. Held, E. H. van Nes, M. Rietkerk, and G. Sugihara. Early-warning signals for critical transitions. Nature, 461(7260):53-59, Sept. 2009. ISSN 1476-4687. doi: 10.1038/nature08227. URL https://www.nature.com/articles/nature08227. Number: 7260 Publisher: Nature Publishing Group.
- [137] A. Scheidegger. Bull. Internat. Assoc. Sci. Hydrol, 12(1):15, 1967.
- [138] J. Sethna and L. o. A. a. S. S. P. J. P. Sethna. Statistical Mechanics: Entropy, Order Parameters, and Complexity. OUP Oxford, Apr. 2006. ISBN 978-0-19-856676-2.
- [139] W. L. Shew and D. Plenz. The Functional Benefits of Criticality in the Cortex, 2012. ISSN 1073-8584.
- [140] W. L. Shew and D. Plenz. The functional benefits of criticality in the cortex. *The Neuroscientist*, 19(1):88–100, 2013. URL http://nro.sagepub.com/content/19/1/88.short.
- [141] W. L. Shew, H. Yang, T. Petermann, R. Roy, and D. Plenz. Neuronal Avalanches Imply Maximum Dynamic Range in Cortical Networks at Criticality. *J. Neurosci.*, 29(49): 15595–15600, 2009. ISSN 0270-6474. doi: 10.1523/JNEUROSCI.3864-09.2009. URL http://www.jneurosci.org/cgi/content/abstract/29/49/15595.
- [142] W. L. Shew, W. P. Clawson, J. Pobst, Y. Karimipanah, N. C. Wright, and R. Wessel. Adaptation to sensory input tunes visual cortex to criticality. *Nature Physics*, 11(8):659–663, Aug. 2015. ISSN 1745-2481. doi: 10.1038/nphys3370. URL https://www.nature.com/articles/nphys3370. Number: 8 Publisher: Nature Publishing Group.
- [143] C.-W. Shin and S. Kim. Self-organized criticality and scale-free properties in emergent functional neural networks. *Physical Review E*, 74(4):045101, Oct. 2006. doi: 10.1103/PhysRevE.74.045101. URL https://link.aps.org/doi/10.1103/PhysRevE.74.045101. Publisher: American Physical Society.
- [144] O. Shriki, J. Alstott, F. Carver, T. Holroyd, R. N. Henson, M. L. Smith, R. Coppola, E. Bullmore, and D. Plenz. Neuronal avalanches in the resting meg of the human brain. J. Neurosci., 33(16):7079-7090, 2013.
- [145] J. Simola, A. Zhigalov, I. Morales-Muñoz, J. M. Palva, and S. Palva. Critical dynamics of endogenous fluctuations predict cognitive flexibility in the Go/NoGo task. *Scientific*

- Reports, 7(1):2909, June 2017. ISSN 2045-2322. doi: 10.1038/s41598-017-02750-9. URL https://www.nature.com/articles/s41598-017-02750-9. Number: 1 Publisher: Nature Publishing Group.
- [146] B. Siri, M. Quoy, B. Delord, B. Cessac, and H. Berry. Effects of Hebbian learning on the dynamics and structure of random networks with inhibitory and excitatory neurons. *Journal of Physiology-Paris*, 101(1):136–148, Jan. 2007. ISSN 0928-4257. doi: 10.1016/j. jphysparis.2007.10.003. URL http://www.sciencedirect.com/science/article/pii/S0928425707000356.
- [147] J. Sjöström and W. Gerstner. Spike-Timing Dependent Plasticity. Scholarpedia, 5(2):1362, Feb. 2010. ISSN 1941-6016. doi: 10.4249/scholarpedia.1362. URL /article/Spike-Timing_Dependent_Plasticity, http://www.scholarpedia.org/article/Spike-Timing_Dependent_Plasticity.
- [148] P. J. Sjöström, G. G. Turrigiano, and S. B. Nelson. Rate, Timing, and Cooperativity Jointly Determine Cortical Synaptic Plasticity. *Neuron*, 32(6):1149–1164, Dec. 2001. ISSN 0896-6273. doi: 10.1016/S0896-6273(01)00542-6. URL http://www.sciencedirect.com/science/article/pii/S0896627301005426.
- [149] P. J. Sjöström, E. A. Rancz, A. Roth, and M. Häusser. Dendritic Excitability and Synaptic Plasticity. *Physiological Reviews*, 88(2):769–840, Apr. 2008. ISSN 0031-9333. doi: 10.1152/physrev.00016.2007. URL https://journals.physiology.org/doi/full/10.1152/physrev.00016.2007. Publisher: American Physiological Society.
- [150] F. P. Spitzner, J. Dehning, J. Wilting, A. Hagemann, J. P. Neto, J. Zierenberg, and V. Priesemann. MR. Estimator, a toolbox to determine intrinsic timescales from subsampled spiking activity. arXiv:2007.03367 [physics, q-bio], July 2020. URL http://arxiv.org/abs/2007.03367. arXiv: 2007.03367.
- [151] H. E. Stanley. Introduction to Phase Transitions and Critical Phenomena. American Journal of Physics, 40(6):927–928, June 1972. ISSN 0002-9505. doi: 10.1119/1.1986710. URL https://aapt.scitation.org/doi/10.1119/1.1986710. Publisher: American Association of Physics Teachers.
- [152] N. Stepp, D. Plenz, and N. Srinivasa. Synaptic Plasticity Enables Adaptive Self-Tuning Critical Networks. *PLOS Computational Biology*, 11(1):e1004043, Jan. 2015. ISSN 1553-7358. doi: 10.1371/journal.pcbi.1004043. URL https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1004043. Publisher: Public Library of Science.
- [153] E. Tagliazucchi, F. v. Wegner, A. Morzelewski, V. Brodbeck, K. Jahnke, and H. Laufs. Breakdown of long-range temporal dependence in default mode and attention networks during deep sleep. *Proceedings of the National Academy of Sciences*, 110(38):15419–15424, Sept. 2013. ISSN 0027-8424, 1091-6490. doi: 10.1073/pnas.1312848110. URL https://www.pnas.org/content/110/38/15419. Publisher: National Academy of Sciences Section: Biological Sciences.
- [154] H. Takayasu and H. Inaoka. New type of self-organized criticality in a model of erosion. *Physical review letters*, 68(7):4, 1992.
- [155] C. Tetzlaff, S. Okujeni, U. Egert, F. Wörgötter, and M. Butz. Self-Organized Criticality in Developing Neuronal Networks. *PLOS Computational Biology*, 6(12):e1001013, Dec. 2010.

- ISSN 1553-7358. doi: 10.1371/journal.pcbi.1001013. URL https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1001013. Publisher: Public Library of Science.
- [156] C. Tetzlaff, C. Kolodziejski, M. Timme, and F. Wörgötter. Synaptic scaling in combination with many generic plasticity mechanisms stabilizes circuit connectivity. *Frontiers in computational neuroscience*, 5:47, 2011.
- [157] G. Tkacik, T. Mora, O. Marre, D. Amodei, S. E. Palmer, and W. Bialek. Thermodynamics and signatures of criticality in a network of neurons. *PNAS*, 112(37), 2015. doi: 10.1073/pnas.1514188112.
- [158] N. Tomen, D. Rotermund, and U. Ernst. Marginally subcritical dynamics explain enhanced stimulus discriminability under attention. Frontiers in Systems Neuroscience, 8(August):1–15, 2014. ISSN 1662-5137. doi: 10.3389/fnsys.2014.00151. URL http://journal.frontiersin.org/article/10.3389/fnsys.2014.00151/abstract.
- [159] M. Tsodyks, K. Pawelzik, and H. Markram. Neural networks with dynamic synapses. Neural Computation, 10:821–835, 1998.
- [160] M. V. Tsodyks and H. Markram. The neural code between neocortical pyramidal neurons depends on neurotransmitter release probability. *Proc. Natl. Acad. Sci. USA*, 94:719–723, 1997.
- [161] G. G. Turrigiano and S. B. Nelson. Hebb and homeostasis in neuronal plasticity. Current Opinion in Neurobiology, 10(3):358-364, June 2000. ISSN 0959-4388. doi: 10.1016/S0959-4388(00)00091-X. URL http://www.sciencedirect.com/science/article/pii/S095943880000091X.
- [162] G. G. Turrigiano and S. B. Nelson. Homeostatic plasticity in the developing nervous system. *Nature Reviews Neuroscience*, 5(2):97–107, Feb. 2004. ISSN 1471-0048. doi: 10.1038/nrn1327. URL https://www.nature.com/articles/nrn1327. Number: 2 Publisher: Nature Publishing Group.
- [163] G. G. Turrigiano, K. R. Leslie, N. S. Desai, L. C. Rutherford, and S. B. Nelson. Activity-dependent scaling of quantal amplitude in neocortical pyramidal neurons. *Nature*, 391: 892–896, 1998.
- [164] M. Uhlig, A. Levina, T. Geisel, and J. M. Herrmann. Critical dynamics in associative memory networks. *Front. Comp. Neurosci.*, 7, 2013.
- [165] A. van Ooyen and M. Butz-Ostendorf. Homeostatic Structural Plasticity Can Build Critical Networks. In N. Tomen, J. M. Herrmann, and U. Ernst, editors, *The Functional Role of Critical Dynamics in Neural Systems*, Springer Series on Bio- and Neurosystems, pages 117–137. Springer International Publishing, Cham, 2019. ISBN 978-3-030-20965-0. doi: 10.1007/978-3-030-20965-0_7. URL https://doi.org/10.1007/978-3-030-20965-0_7.
- [166] A. van Ooyen and J. van Pelt. Activity-dependent Outgrowth of Neurons and Overshoot Phenomena in Developing Neural Networks. *Journal of Theoretical Biology*, 167 (1):27–43, Mar. 1994. ISSN 0022-5193. doi: 10.1006/jtbi.1994.1047. URL http://www.sciencedirect.com/science/article/pii/S0022519384710472.
- [167] A. van Ooyen and J. van Pelt. Complex Periodic Behaviour in a Neural Network Model with Activity-Dependent Neurite Outgrowth. *Journal of Theoretical Biology*, 179(3):

- 229-242, Apr. 1996. ISSN 0022-5193. doi: 10.1006/jtbi.1996.0063. URL http://www.sciencedirect.com/science/article/pii/S0022519396900636.
- [168] P. Villegas, S. di Santo, R. Burioni, and M. A. Muñoz. Timeseries thresholding and the definition of avalanche size. arXiv preprint arXiv:1902.10465, 2019.
- [169] Y. S. Virkar, W. L. Shew, J. G. Restrepo, and E. Ott. Feedback control stabilization of critical dynamics via resource transport on multilayer networks: How glia enable learning dynamics in the brain. *Physical Review E*, 94(4):042310, Oct. 2016. doi: 10.1103/PhysRevE.94.042310. URL https://link.aps.org/doi/10.1103/PhysRevE.94.042310. Publisher: American Physical Society.
- [170] S.-J. Wang and C. Zhou. Hierarchical modular structure enhances the robustness of self-organized criticality in neural networks. *New Journal of Physics*, 14, 2012. ISSN 13672630. doi: 10.1088/1367-2630/14/2/023005.
- [171] A. H. Williams, T. O'Leary, and E. Marder. Homeostatic Regulation of Neuronal Excitability. Scholarpedia, 8(1):1656, Jan. 2013. ISSN 1941-6016. doi: 10.4249/scholarpedia. 1656. URL http://www.scholarpedia.org/article/Homeostatic_Regulation_of_Neuronal_Excitability.
- [172] J. Wilting and V. Priesemann. Inferring collective dynamical states from widely unobserved systems. *Nature Communications*, 9(1):2325, June 2018. ISSN 2041-1723. doi: 10.1038/s41467-018-04725-4. URL https://www.nature.com/articles/s41467-018-04725-4. Number: 1 Publisher: Nature Publishing Group.
- [173] J. Wilting and V. Priesemann. 25 years of criticality in neuroscience established results, open controversies, novel concepts. *Current Opinion in Neurobiology*, 58:105—111, Oct. 2019. ISSN 0959-4388. doi: 10.1016/j.conb.2019.08.002. URL http://www.sciencedirect.com/science/article/pii/S0959438819300248.
- [174] J. Wilting and V. Priesemann. Between Perfectly Critical and Fully Irregular: A Reverberating Model Captures and Predicts Cortical Spike Propagation. *Cerebral Cortex*, 29(6):2759–2770, June 2019. ISSN 1047-3211. doi: 10.1093/cercor/bhz049. URL https://academic.oup.com/cercor/article/29/6/2759/5476016. Publisher: Oxford Academic.
- [175] J. Wilting, J. Dehning, J. Pinheiro Neto, L. Rudelt, M. Wibral, J. Zierenberg, and V. Priesemann. Operating in a Reverberating Regime Enables Rapid Tuning of Network States to Task Requirements. Frontiers in Systems Neuroscience, 12, 2018. ISSN 1662-5137. doi: 10.3389/fnsys.2018.00055. URL https://www.frontiersin.org/articles/10.3389/fnsys.2018.00055/full. Publisher: Frontiers.
- [176] Y. Yada, T. Mita, A. Sanada, R. Yano, R. Kanzaki, D. J. Bakkum, A. Hierlemann, and H. Takahashi. Development of neural population activity toward self-organized criticality. *Neuroscience*, 343:55–65, Feb. 2017. ISSN 0306-4522. doi: 10.1016/j.neuroscience.2016.11.031. URL http://www.sciencedirect.com/science/article/pii/S0306452216306522.
- [177] S. Yu, T. L. Ribeiro, C. Meisel, S. Chou, A. Mitz, R. Saunders, and D. Plenz. Maintained avalanche dynamics during task-induced changes of neuronal activity in nonhuman primates. *eLife*, 6:e27119, 2017.

- [178] G. Zeng, X. Huang, T. Jiang, and S. Yu. Short-term synaptic plasticity expands the operational range of long-term synaptic changes in neural networks. *Neural Networks*, 118:140–147, Oct. 2019. ISSN 0893-6080. doi: 10.1016/j.neunet.2019.06.002. URL http://www.sciencedirect.com/science/article/pii/S0893608019301753.
- [179] F. Zenke and W. Gerstner. Hebbian plasticity requires compensatory processes on multiple timescales. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 372(1715):20160259, Mar. 2017. doi: 10.1098/rstb.2016.0259. URL https://royalsocietypublishing.org/doi/full/10.1098/rstb.2016.0259. Publisher: Royal Society.
- [180] F. Zenke, G. Hennequin, and W. Gerstner. Synaptic Plasticity in Neural Networks Needs Homeostasis with a Fast Rate Detector. *PLOS Computational Biology*, 9(11):e1003330, Nov. 2013. ISSN 1553-7358. doi: 10.1371/journal.pcbi. 1003330. URL https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1003330. Publisher: Public Library of Science.
- [181] R. Zeraati, T. A. Engel, and A. Levina. Estimation of autocorrelation timescales with Approximate Bayesian Computations. bioRxiv, page 2020.08.11.245944, Aug. 2020. doi: 10.1101/2020.08.11.245944. URL https://www.biorxiv.org/content/10.1101/2020.08.11.245944v1.
- [182] Y.-C. Zhang. Scaling theory of self-organized criticality. *Phys. Rev. Lett.*, 63(5):470–473, 7 1989. doi: 10.1103/PhysRevLett.63.470.
- [183] J. Zierenberg, J. Wilting, and V. Priesemann. Homeostatic Plasticity and External Input Shape Neural Network Dynamics. *Physical Review X*, 8(3):031018, July 2018. doi: 10.1103/PhysRevX.8.031018. URL https://link.aps.org/doi/10.1103/PhysRevX.8.031018. Publisher: American Physical Society.
- [184] J. Zierenberg, J. Wilting, V. Priesemann, and A. Levina. Description of spreading dynamics by microscopic network models and macroscopic branching processes can differ due to coalescence. *Physical Review E*, 101(2):022301, 2020.
- [185] J. Zierenberg, J. Wilting, V. Priesemann, and A. Levina. Tailored ensembles of neural networks optimize sensitivity to stimulus statistics. *Physical Review Research*, 2(1): 013115, 2020.
- [186] R. S. Zucker and W. G. Regehr. Short-term synaptic plasticity. *Annu. Rew. Physiol.*, 64: 355–405, 2002.