Different scenarios of the transition to chaos in randomly connected neural networks were extensively studied over the last 30 years [1–7]. According to the prevailing assumption rooted in the central limit theorem, the total synaptic input current of each neuron can be modeled as a Gaussian random variable (Gaussian assumption). Here we argue that the Gaussian assumption cannot account for some of the experimentally observed features of neuronal circuits. We propose a novel, analytically tractable connectivity model with power-law distributed synaptic weights. When threshold neurons with biologically plausible many inputs are considered, our model, in contrast to the Gaussian counterpart, features a continuous transition to chaos and can reproduce biologically relevant low activity levels and associated scale-free avalanches, i.e., bursts of activity with power-law distributions of sizes and lifetimes.

Scale-free neuronal avalanches, commonly associated with criticality, have been observed in cortical networks in various settings, including cultured and acute slices from rat somatosensory cortex [8], eye-attached ex vivo preparation of turtle visual cortex [9], visual cortex in anesthetized rats [10], primary visual cortex in anesthetized monkeys [10], and premotor, motor, and somatosensory cortex in awake monkeys [11]. Criticality implies the existence of a continuous transition between two distinct collective phases. In the context of neuronal avalanches, most commonly studied transitions are between quiescent and active states [12, 13] or synchronous and asynchronous states [14]. In addition to providing a plausible generating mechanism for the neuronal avalanches, the existence of a continuous transition to chaos would have important functional implications, as it has been shown that computation is most efficient around the edge of chaos [13, 15–19]. However, the relation between neuronal avalanches and the edge of chaos is not well understood.

The continuous nature of the phase transition observed in conventional models of critically (either the edge of chaos or scale-free avalanches) is sensitive to theoretical assumptions that are not biologically grounded. Most works that study transition to chaos employ rate models with continuous non-threshold activation functions, often of a sigmoid-like shape. But real neurons spike only when driven by strong enough excitatory synaptic input above a threshold [20]. In contrast, works focusing on neuronal avalanches assume more biologically plausible single-neuron models, but rely on extremely sparse networks [15, 21]. However, many neurons in the vertebrate brain receive large number of inputs from other cells (∼ 10^3) [22]. We observed that the transition to chaos becomes discontinuous when densely connected threshold units are used in tandem with the Gaussian assumption (Fig. 4). This discontinuous character of the transition makes it hard for the network state to stay close to the edge of chaos or robustly generate scale-free avalanches.

To fix this issue, we draw on the experimental works reporting heavy-tailed distributions of synaptic weights in various areas of the brain [23–28]. Multiple theoretical mechanisms have been suggested to realize such distributions, e.g., modified spike-timing-dependent plasticity (STDP) rule [29] or STDP combined with homeostatic plasticity [30]. Notably, recent studies have suggested that experimentally observed
activity-independent intrinsic spine dynamics can straightforwardly explain the heavy-tailed distributions of synaptic weights \[31,35\].

Although extensively studied, the computational role of synaptic heavy tails is still not fully understood. A log-normal distribution is often assumed and the results are obtained by means of computer simulations \[27,36-39\]. However, in these models the effects of heavy tails are only visible through finite size effects because, as the number of incoming connections per neuron goes to infinity, i.e., in the thermodynamic limit, the model behavior becomes equivalent to that with Gaussian-distributed synapses due to the central limit theorem. This hinders theoretical analysis and limits our understanding. Therefore, a simple theoretical model that robustly predicts the effects of synaptic heavy tails is needed. We fill this gap by assuming random, power-law distributed synaptic weights.

Our aim is to inspect how the distribution of synaptic efficacies, modulated by the activation function, affects the transition to chaos and the associated avalanches. To this end, in our calculations we focus on the network effects and hence simplify the dynamics of individual neurons by considering the following discrete-time network dynamics

$$x_i(t+1) = \sum_{j=1}^{N} J_{ij} \phi(x_j(t)),$$  \hspace{1cm} (1)

where $\phi(x)$ is the activation function, assumed to be identical across the network, and $J$ is the connectivity matrix. The network is fully connected and the synaptic weights are independently drawn from the common Cauchy distribution

$$\rho(J_{ij}) = \frac{1}{\pi} \frac{g/N}{(g/N)^2 + J_{ij}^2},$$  \hspace{1cm} (2)

with the characteristic function

$$\Phi_J(k) = e^{-\gamma |k|},$$  \hspace{1cm} (3)

with $\gamma = \frac{g}{N}$. We refer to the model prescribed by (1) and (2) as the Cauchy network. Due to the generalized central limit theorem \[40\], in the thermodynamic limit of $N \to \infty$ results obtained for this model are applicable to networks with connections drawn independently from any symmetric distribution with $1/x^2$ tails that are scaled with the number of neurons
The randomness in the state of the network comes from two factors: random initial conditions and random weights. By averaging over both factors, the characteristic function of \( x_i(t+1) \) can be calculated as follows

\[
\langle e^{i k x_i(t+1)} \rangle = \exp \left( -g |k| N^{-1} \sum_{j=1}^{N} \phi(x_j(t)) \right) = \exp (-gm(t) |k|),
\]

where we have assumed that \( J_{ij} \) and \( \phi(x_j(t)) \) are independent, which is expected to hold in the thermodynamic limit. Clearly, with this assumption \( x_i(t) \) is a Cauchy random variable.

To proceed we assume self-averaging, i.e. the mean activity is assumed to behave the same way for each realization of the network. In the thermodynamic limit the mean activity can in this case be alternatively expressed as

\[
m(t) = \langle \phi(x_i(t)) \rangle_{J_i}.
\]

We plug (2) into (6) and arrive at the the evolution of the mean activity in a simple integral form

\[
m(t + 1) = \int_{-\infty}^{\infty} \mathcal{D}z \phi(gm(t)z),
\]

where \( \mathcal{D}z = \pi^{-1} dz/(1 + z^2) \) denotes that the integral is calculated with respect to the standard Cauchy measure. The steady-state mean activity can be obtained from (7) in a self-consistent manner.

We are now in the position to analyze the dependence of the dynamics of the Cauchy network on the activation function. For \( \phi(x) = x \), the integral on the right-hand side (RHS) of (7) diverges, suggesting that the network is unstable. Indeed, it is easy to understand why this is the case. For linear networks the dynamics is fully determined by the eigenvalues of the connectivity matrix \( J \). It is known that, in contrast to random matrices with Gaussian entries, a Cauchy random matrix features an unbounded support of the eigenvalues density, even in the limit of \( N \to \infty \) [41-43]. Thus, we can conclude that, regardless of the value of the \( g \), the dynamics of a Cauchy neural network is in this case divergent. For the same reason, any \( \phi(x) \) that is linear around \( x \approx 0 \) and grows sufficiently slow for large \( x \) leads to chaotic dynamics for any \( g \). However, in the biologically relevant regime neurons exhibit saturation and thresholding at, respectively, large and low values of total synaptic input. The corresponding Cauchy network generically exhibits two phases: quiescent and chaotic, and an associated transition between them [44].

To further simplify the calculations and to model the avalanches, in the following we focus our attention on the binary activation function

\[
\phi(x) = \begin{cases} 
1, & \text{for } x > \theta \\
0, & \text{for } x \leq \theta.
\end{cases}
\]

where \( \theta \) denotes the threshold. In this case the dynamical mean-field equation for \( m(t) \) simplifies to

\[
m(t + 1) = \frac{1}{\pi} \arctan (gm(t)g/\theta).
\]

The stability of the trivial fixed point in the binary case can be checked by expanding the RHS of (9) around \( m(t) = 0 \):

\[
m(t + 1) = \frac{g}{\pi \theta} m(t) + O(m(t)^2).
\]
The fixed point at $m(t) = 0$, corresponding to the quiescent phase, is unstable for $g > \pi \theta$. Since $\text{arctan}(x)/\pi$ is saturating and concave for all $x > 0$, another stable fixed point $m(t) = m^*$ close to 0 appears exactly when the trivial fixed point loses its stability ($m^* \approx \sqrt{3}(g/\theta)^{-3/2} \sqrt{\pi}/\theta - \pi$ near the transition point). Due to the quenched, asymmetric disorder of the connectivity matrix we can expect this fixed point to represent a chaotic attractor of the network [45], with a large sensitivity to small perturbations (i.e., the butterfly effect). Our computer simulations confirm this prediction (Fig. 2).

The transition from the quiescent to the chaotic phase (in the large $N$ limit) can be understood from the underlying structure of connections. Due to the power-law connectivity density, we can expect that only a small fraction of the connections contribute to the activity profile of the network. Indeed, as we show in the following, the transition to chaos is driven by the percolation transition of autocrat connections for which $J_{ij} > \theta$, i.e., an active pre-synaptic neuron will activate the post-synaptic neuron in the absence of other inputs. Around the critical point the mean activity of the network is infinitesimal and thus the higher order interaction events (e.g. two neurons activating another neuron) are negligible. In other words, to a good approximation, a neuron can only be activated by another single neuron through an autocrat connection, independently from other neurons. This suggests that the transition to chaos in the neural network model is related to the critical branching processes [46] (Fig. 1).

In the Cauchy case the probability that a given connection is an autocrat reads

$$\text{Prob}(J_{ij} > \theta) = \frac{1}{\pi} \text{arctan} \left( \frac{g}{N \theta} \right).$$  \hspace{1cm} (11)

For a given neuron, the number of outgoing (or incoming) autocrat connections is a binomial random variable with $N$ trials and the probability of success given by (11). In the limit of $N \to \infty$ it converges to the Poisson random variable with intensity

$$\lambda = \lim_{N \to \infty} \frac{N}{\pi} \text{arctan} \left( \frac{g}{N \theta} \right) = \frac{g}{\theta \pi}.$$

Now, let the initial state of the network be such that only a single neuron (seed) is active. The number of active neurons (descendants) in the next step is given by the Poisson distribution and the mean number of active neurons is given by (12). The theory of branching processes predicts that the population will eventually die out almost surely for $\lambda \leq 1$ and has a finite survival probability for $\lambda > 1$. At $\lambda = 1$ the process is critical and features scale-free avalanches. The critical point predicted by the branching process formulation of the network dynamics,

$$g^* = \pi \theta,$$

is the same as the mean-field critical point predicted by (9).

The mapping to the branching process explains many features of the Cauchy neural network around the critical point. Below the critical point the steady state is quiescent and a bit-flip perturbation corresponds to a single neuron (seed) being activated. The local expansion rate of such perturbation is given by $\lambda$. Above the critical point ($g > \pi \theta$), each bit-flip contributes in the same manner as a single seed and, additionally, interacts with other active neurons to activate and deactivate other descendants. Thus, in the vicinity of the transition point $\lambda$ gives a lower bound on the local expansion rate of a perturbation in the steady state, and for $\lambda > 1$ the network is expected to be chaotic in the thermodynamic limit. Moreover, the transition to chaos belongs to the mean-field directed percolation universality class [47]. The propagation of the corresponding avalanches is characterized by [44] power-law distributed sizes $S$

$$\text{Prob}(S > s) \sim s^{-1/2}$$

and power-law distributed lifetimes $T$

$$\text{Prob}(T > t) \sim t^{-1}.$$ \hspace{1cm} (14)

These theoretical predictions were corroborated by our computer simulations of the Cauchy network, as shown in Fig. 3.

For a comparison, we have also studied Gaussian networks of threshold units with a fixed number of connections per neuron $K$ [44]. While extremely sparsely connected Gaussian networks ($K \ll 12$) behave qualitatively similar to the Cauchy network, the transition to chaos becomes discontinuous in the biologically relevant regime of $K \gtrsim 13$. With a biologically realistic $K$ and finite $N$, the network activity jumps between two metastable states near the transition point, and cannot be robustly posed at the edge of chaos. Importantly, although our theoretical predictions were derived assuming simplistic threshold neural units, they translate directly to networks of more biologically plausible leaky integrate-and-fire (LIF) neurons. The difference of continuous and discontinuous transition is confirmed by the presence or absence of a hysteresis loop in more realistic networks of LIF neurons (Fig. 4). Hence, unlike the Gaussian networks with realistic $K$, Cauchy networks demonstrate critical phenomena and can reproduce experimentally observed scale-free avalanches at the critical point. Moreover, a large Cauchy network can exhibit arbitrarily low, self-sustained activity levels. In contrast, the lowest possible activity level that can be achieved by the Gaussian network with realistic $K$ is about 11% in the binary case and 40 Hz in the LIF case (Fig. 4).

Power-law distributions of synaptic weights feature many very weak (or even silent) synapses, that do not directly contribute to the computation. Even though this may seem wasteful, we think that such architectures are not only biologically plausible [48] but may be beneficial. One possibility is that even weak connections can activate a neuron once contextual input from another part of the brain increases the baseline membrane potential close to its spiking threshold. Such contextual input can also raise the spiking probability of nearby neurons so that synchronous activation of weak connections is more likely. Silent synapses have also been reported to play a role in unsupervised features extraction [49]. More generally, the optimal degree of sparsity depends on the role of a given brain structure and the type of employed plasticity [50]. Power-law distributed synaptic weights may in this context provide a weakly informative [51] sparsity prior, with
weak and silent synapses providing a pool of potential connections that can be recruited when and if needed, as observed in the brain during development [53][54].

Our results have implications in theoretical neuroscience and machine learning. We showed that a heavy-tailed distribution of synaptic weights facilitates computations at the edge of chaos in binary and spiking neural networks. In this regime, information transmission is mediated by a sparse sub-set of chaotic synapses. Such sparse effective connectivity has been previously shown to maximize information storage capacity in symmetric [55] and asymmetric [56][57] networks. Moreover, deep neural networks are more expressive [58, 59] and can only be efficiently trained via error backpropagation [60][61] around the edge of stability analogous to the edge of chaos. Further, biologically inspired neuromorphic chips can be energy efficient, if the activity is sparse. Neurons should be inactive in the absence of inputs (θ > 0) to achieve this. We can therefore expect that both feedforward [62] and recurrent [63] spiking neural networks should benefit from the sparse effective connectivity.

For clarity we chose to limit our presentation to the Cauchy distribution of J[i,j]. As we have shown, this case is, to some extent, analytically tractable. However, our results naturally extend to other power-law distributions, where the mapping to the critical branching process in the case of the binary activation function [8] is still valid. To see this let the synaptic efficacy density asymptotically behave like a power-law $ρ(J_{ij}) ∼ Cα^{−N−1}|J_{ij}|^{−α}$ [64]. We then have $\text{Prob}(J_{ij} > θ) = CN^{−1}(g(θ)α)^{−N}$, which holds for large enough $N$. The branching parameter can be calculated as in [12] and reads $λ = C(g(θ)α)^{−N}$. A continuous transition takes place at $λ = 1$ and its features are, as before, described by the directed percolation universality class. This family of power-law models is loosely related to spin-glass models with power-law interactions [65][67]. Note, however, that due to the symmetric interaction matrix, spin-glass models behave qualitatively differently than neural models with asymmetric connectivity [68].

We have analytically characterized features of the transition to chaos in the Cauchy networks. More advanced calculations [69] suggest that an infinite spectrum of order parameters is required to fully specify the phase. The appearance of infinite spectrum of order parameters suggests an intriguing possibility that more phase transitions occur as the control parameters are varied. Although our numerical experiments did not uncover any additional transitions, more research is needed to clarify the exact shape of the phase diagram of the Cauchy and other power-law networks.

[1] H. Sompolinsky, A. Crisanti, and H.-J. Sommers, Physical review letters 61, 259 (1988).
[2] L. Molgedey, J. Schuchhardt, and H. G. Schuster, Physical review letters 69, 3717 (1992).
[3] K. Rajan, L. Abbott, and H. Sompolinsky, Physical Review E 82, 011903 (2010).
[4] M. Stern, H. Sompolinsky, and L. Abbott, Physical Review E 90, 062710 (2014).
[5] J. Almlof, M. Stern, and T. Sharpee, Physical review letters 114, 088101 (2015).
[6] J. Kadam and H. Sompolinsky, Physical Review X 5, 041030 (2015).
[7] A. Crisanti and H. Sompolinsky, Physical Review E 98, 062120 (2018).
[8] J. M. Beggs and D. Plenz, Journal of neuroscience 23, 11167 (2003).
[9] W. L. Shew, W. P. Clawson, J. Pobst, Y. Karimipanah, N. C. Wright, and R. Wessel, Nature Physics 11, 659 (2015).
[10] A. J. Fontenele, N. A. de Vasconcelos, T. Feliciano, L. A. Aguiar, C. Soares-Cunha, B. Coimbra, L. Dalla Porta, S. Ribiero, A. J. Rodrigues, N. Sousa, et al., Physical review letters 122, 208101 (2019).
[11] T. Petermann, T. C. Thiagarajan, M. A. Lebedev, M. A. Nicolelis, D. R. Chaval, and D. Plenz, Proceedings of the National Academy of Sciences 106, 15921 (2009).
[12] C. Haldeman and J. M. Beggs, Physical review letters 94, 058101 (2005).
[13] O. Knouche and M. Copelli, Nature physics 2, 348 (2006).
[14] S. di Santo, P. Villegas, R. Burioni, and M. A. Muñoz, Proceedings of the National Academy of Sciences 115, E1356 (2018).
[15] C. G. Langton, Physica D: Nonlinear Phenomena 42, 12 (1990).
[16] N. Bertschinger and T. Natschläger, Neural computation 16, 1413 (2004).
[17] R. Legenstein and W. Maass, New directions in statistical signal processing: From systems to brain , 127 (2007).
[18] T. Toyoizumi and L. Abbott, Physical Review E 84, 051908 (2011).
[19] J. Schuecker, S. Goedeke, and M. Helias, Phys. Rev. X 8, 041029 (2018).
[20] M. Hüsser and P. Monsinhas, Neuron 40, 449 (2003).
[21] D. Millman, S. Miralas, A. Kirkwood, and E. Niebur, Nature physics 6, 801 (2010).
[22] P. Dayan and L. F. Abbott, Theoretical Neuroscience: Computational and Mathematical Modeling of Neural Systems, The MIT Press, 2005.
[23] R. Sayer, M. Friedlander, and S. Redman, Journal of Neuroscience 10, 826 (1990).
[24] D. Feldmeyer, V. Egger, J. Lübke, and B. Sakmann, The Journal of physiology 521, 169 (1999).
[25] S. Song, P. J. Säätö, M. Reigl, S. Nelson, and D. B. Chklovskii, PLoS biology 3, e68 (2005).
[26] S. Lefort, C. Tomm, J.-C. F. Sarris, and C. C. Petersen, Neuron 61, 301 (2009).
[27] Y. Ikegaya, T. Sasaki, D. Ishikawa, N. Honma, K. Tao, N. Takahashi, G. Minamisawa, S. Ujita, and N. Matsuki, Cerebral Cortex 23, 293 (2012).
[28] Y. Loevenstein, A. Kurap, and S. Rumpel, Journal of Neuroscience 31, 9481 (2011).
[29] M. Gilson and T. Fukai, PLoS one 6, e25339 (2011).
[30] P. Zheng, C. Dimitriakakis, and J. Treisch, PLoS computational biology 9, e1002848 (2013).
[31] N. Yasumatsu, M. Matsuoka, T. Miyazaki, J. Noguchi, and H. Kasai, Journal of Neuroscience 28, 13592 (2008).
[32] A. Nagaoa, H. Takehara, A. Hayashi-Takagi, J. Noguchi,
In the binary case the system has a finite number of states for

See the Supplementary Information for details.

and I. Z

References between asym-

Methods

Simulations

Computer simulations of binary networks were performed using custom-written code in Julia and Python. The results for spiking neural networks were obtained with NEST Simulator [70]. Details of the simulations: Fig. 2. The averaging was performed over all $N = 10^4$ neurons (i.e., including the unper-

turbated network, $N + 1$ replicas were simulated) and over 50 realizations of $J$. At time $T = 0$ the unperturbed network was prepared in the (a) steady state by evolving it for 500 steps before introducing perturbations or (b) quiescent state. $g = \pi$. Fig. 3. Networks had size $N = 10^4$ and results were averaged over 10 independent realizations of $J$. $g = \pi$. Fig. 4. Both Gaussian and Cauchy networks were fully connected with $N = 10^4$. The injected current changed between $-400 \text{ pA}$ and $400 \text{ pA}$ in small increments every 5 ms. All neurons were of type iaf_psc_alpha, which denotes a leaky integrate-and-fire neuron with alpha-shaped postsynaptic currents. Default par-

eters of the model neuron were used, i.e.: resting potential $E_L = -70 \text{ mV}$, capacity of the membrane $C_m = 250 \text{ pF}$, mem-


cicits, B. Z

Theory of branching processes, Courier Corporation, 2002.

M. A. Munoz, R. Dickman, A. Vespignani, and S. Zapperi, Physical Review E 59, 6175 (1999).

L. Consell, M. F. Iacaruso, D. R. Muir, R. Houlton, E. N. Sader, H. Ko, S. B. Hober, and T. D. Misco-Flogel, Nature 518, 399 (2015).

H. Huang, Journal of Physics A: Mathematical and Theoretical 51, 08LT01 (2018).

A. Litwin-Kumar, K. D. Harris, R. Axel, H. Sompolinsky, and L. Abbott, Neuron 93, 1153 (2017).

A. Gelman, A. Jakulin, M. G. Pittau, Y.-S. Su, et al., The Annals of Applied Statistics 2, 1360 (2008).

S. Van Dongen, Journal of Theoretical Biology 242, 90 (2006).

D. Liao, N. A. Hessler, and R. Malinow, Nature 375, 400 (1995).

G. A. Kerchner and R. A. Nicoll, Nature Reviews Neuroscience 9, 813 (2008).

G. Palm, Neural Networks 37, 165 (2013).

N. Brunel, V. Hákim, P. Isopé, J.-P. Nadal, and B. Barbour, Neuron 43, 745 (2004).

V. Folli, G. Gosti, M. Leonetti, and G. Ruocco, Neural Networks 104, 50 (2018).

M. Raghu, B. Poole, J. Kleinberg, S. Ganguli, and J. S. Dickstein, On the expressive power of deep neural networks, in Proceedings of the 34th International Conference on Machine Learning-Volume 70, pp. 2847–2854, JMLR. org, 2017.

B. Poole, S. Lahiri, M. Raghu, J. Sohn-Dickstein, and S. Ganguli, Exponential expressivity in deep neural networks through transient chaos, in Advances in neural information processing systems, pp. 3360–3368, 2016.

D. E. Rumelhart, G. E. Hinton, and R. J. Williams, Nature 323, 533 (1986).

S. S. Schoenholz, J. Gilmer, S. Ganguli, and J. Sohl-Dickstein, arXiv preprint arXiv:1611.01232 (2016).

F. Zenke and S. Ganguli, Neural computation 30, 1514 (2018).

G. Bellec, F. Scherr, E. Hajeck, D. Salai, R. ledgein, and W. Maass, arXiv preprint arXiv:1901.09049 (2019).

Note that we scale $J_\alpha$ with the number of neurons as $N^{-1/\alpha}$, which assures the existence of a non-trivial limit $N \to \infty$. For $\alpha > 2$ another choice is to scale the synaptic strengths as $N^{-1/2}$, which in the limit of $N \to \infty$ corresponds to the Gaussian network.

P. Cizeau and J. Bouchaud, Journal of Physics A: Mathematical and General 26, L187 (1993).

D. Parker, V. Dupuis, F. Ladieu, J.-P. Bouchaud, E. Dubois, R. Perzynski, and E. Vincent, Physical Review B 77, 104428 (2008).

K. Janzen, A. Engel, and M. Mézard, EPL (Europhysics Letters) 89, 67002 (2010).

Z. Li and P. Dayan, Computational differences between asymmetrical and symmetrical networks, in Advances in Neural Information Processing Systems, pp. 274–280, 1999.

To be published elsewhere.

C. Linssen, M. E. Lepperød, J. Mitchell, J. Pronold, J. M. Eppler, C. Kuep, A. Pesyer, S. Kunkel, P. Wedel, Y. Nodee, D. Terhorst, R. D. Drey, M. Deger, J. Haine, A. Sinha, A. Antonietti, M. Schmitz, L. Paz, J. Garrido, T. Iwen, L. Riquelme, A. Serenko, T. Kühn, I. Kitayama, H. Morik, S. Spreezer, J. Jordan, J. Krishnan, M. Senden, H. Hagen, A. Shusharian, S. B. Vennemo, D. Rodarrie, A. Morrison, S. Graber, J. Schuecker, S. Díaz, B. Zais, and H. E. Plesser, NEST 2.16.0, 2018.

P. Alström, Physical Review A 38, 4905 (1988).

G. Odor, Reviews of modern physics 76, 663 (2004).

B. Derrida, E. Gardner, and A. Zippelius, EPL (Europhysics Letters) 4, 167 (1987).

Methods

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eters of the model neuron were used, i.e.: resting potential $E_L = -70 \text{ mV}$, capacity of the membrane $C_m = 250 \text{ pF}$, mem-
brane time constant $\tau_m = 10$ ms, refractory period $\tau_{ref} = 2$ ms, spike threshold $V_{th} = -55$ mV, reset potential $V_{reset} = -70$ mV, rise time of the excitatory and inhibitory synaptic alpha function $\tau_{syn} = 2$ ms. Static synapses were used with the default delay of 1 ms, and weights were randomly drawn from symmetric (a) Gaussian distribution with $\sigma = 2.4 \times 10^3 / \sqrt{N}$ pA, and (b) Cauchy distribution with $\gamma = 1.92 \times 10^3 / N$ pA. Poisson noise was injected randomly into the network, activating each neuron approximately twice every second—without external input networks cannot recover from the quiescent state, since the model neurons are not spontaneously active.

Sub-sampling: For the sake of clarity and drawing efficiency, in the raster plots only 10% of spikes of 100 randomly chosen neurons were drawn. Activity histograms were created using all data.

**SUPPLEMENTARY INFORMATION**

### A. Avalanche statistics

The mapping to the branching process together with the general results known for critical branching processes provide the critical exponents [47, 71, 72]. Here, for completeness, we calculate two critical exponents in our specific case.

#### 1. Avalanche size distribution

The size of an avalanche is defined as the sum of a number of active neurons at each time step, from the beginning of the avalanche till its end. Let $S_m$ denote the size of an avalanche starting from $m$ seeds and $G_m(z)$ denote the corresponding generating function

$$
G_m(z) \equiv \langle z^{S_m} \rangle = \sum_{j=0}^{\infty} z^j \text{Prob}(S_m = j). \quad (16)
$$

Since the activity of network is assumed to be sparse, the avalanche that starts from $m$ seeds consists of $m$ independent avalanches starting from a single seed. Therefore we can write that $S_m = \sum_{i=1}^{m} S_1^{(i)}$, where $\{S_1^{(i)}\}$ is a set of i.i.d. random variables denoting sizes of single-seed generated avalanches. At the level of the generating functions, this assumption leads to a simple expression

$$
G_m(z) = \lfloor G_1(z) \rfloor^m. \quad (17)
$$

On the other hand, we know how a seed neuron propagates the activity through the network in a single step: it activates $m$ neurons with probability

$$
p_m = \frac{\lambda^m}{m!} e^{-\lambda}, \quad (18)
$$

where $\lambda$ is given by (12). This means that one seed generate an avalanche of size $1 + S_m$ with probability $p_m$, where 1 is from the first step (i.e., the seed) and $S_m$ is from the subsequent steps. Hence, the single-seed generating function can be calculated as follows

$$
G_1(z) = \sum_{m=0}^{\infty} p_m(z^{1+S_m}) = z \sum_{m=0}^{\infty} p_m G_m(z). \quad (19)
$$

We combine (19) with (17) and (18) and arrive at an implicit expression for the one-seed avalanche size generating function

$$
G_1(z) = z \left\{ p_0 e^{S_0} + \sum_{m=1}^{\infty} p_m \lfloor G_1(z) \rfloor^m \right\} = z \exp(\lambda G_1(z) - \lambda). \quad (20)
$$

Note that we used $S_0 = 0$ (no avalanche without a seed). In order to inspect the tail of the distribution of $S_1$ we introduce an auxiliary function $g(\epsilon) = 1 - G_1(1-\epsilon)$ and expand the RHS of (20) assuming that $\epsilon \ll 1$ and $g(\epsilon) \ll 1$ (valid for $\lambda \leq 1$):

$$
1 - g(\epsilon) = (1 - \epsilon) \left( 1 - \lambda g(\epsilon) + \frac{\lambda^2 g(\epsilon)^2}{2} + O(g(\epsilon)^3) \right), \quad (21)
$$

which to the lowest order can be rewritten as

$$
g(\epsilon) = \begin{cases} 
\frac{\epsilon^2}{1 - \lambda}, & \text{for } \lambda \neq 1 \\
\sqrt{2\epsilon}, & \text{for } \lambda = 1.
\end{cases} \quad (22)
$$
The small $\epsilon$ behavior of $g(\epsilon) \sim \epsilon^{1/2}$ in the $\lambda = 1$ case translates into the tail behavior of the avalanche size density as

$$\text{Prob}(S_1 = s) \sim s^{-3/2} \quad (23)$$

for large $s$.

2. Avalanche lifetime distribution

Let $T_m$ be the lifetime (number of steps with nonzero activity) of an avalanche that starts from $m$ seeds. By definition $T_0 = 0$ and $T_m \geq 1$ for $m > 0$. As before, we treat an avalanche from different seeds as independent, and thus the following identity linking the survival probabilities holds

$$Q_m(t) = 1 - \text{Prob}(T_m \leq t) = 1 - [\text{Prob}(T_1 \leq t)]^m = 1 - [1 - Q_1(t)]^m. \quad (24)$$

As in the case of the size distribution, we can unwrap the first step of the dynamics starting from a single seed, which gives

$$Q_1(t + 1) = \sum_{m=1}^{\infty} P_m Q_m(t) = 1 - \sum_{m=0}^{\infty} P_m [1 - Q_m(t)]. \quad (25)$$

We plug (24) and (18) into (25) and arrive at the following recursive relation

$$Q_1(t + 1) = 1 - \exp[-\lambda Q_1(t)]. \quad (26)$$

If $\lambda > 1$ there exists a non-zero fixed point corresponding to the non-zero probability of survival at $t \to \infty$. In contrast, the activity eventually dies out almost surely for $\lambda \leq 1$. Assuming $\lambda Q_1(t) \ll 1$ the recursive relation simplifies to

$$Q_1(t + 1) = \lambda Q_1(t) - \frac{\lambda^2}{2} Q_1(t)^2, \quad (27)$$

which predicts an exponential decay for $\lambda < 1$. At the critical point $\lambda = 1$ and the decay is a power law. In that case the recursion can be solved with an ansatz $Q_1(t) = C/t^\delta$, leading to $\delta = 1$, as expected.

B. Existence of the transition for neurons with a positive threshold

Let $\phi(x)$ be an activation function such that $\phi(x) = 0$ for $x$ below a positive threshold $\theta$, and $\phi(x) \approx C$ for sufficiently large $x$ (i.e. $x > m_1$). Without much loss of generality, we additionally assume that $\phi(x) \geq 0$ and $\int_{-\theta}^{\theta} \phi(x) dx < \infty$. The integral in the mean-field equation can be then decomposed as follows:

$$m(t + 1) = \frac{1}{\pi} \int_0^{\infty} \frac{dz}{1 + z^2} = \frac{1}{\pi} \int_{m_1}^{m_1} \frac{dz}{1 + z^2} + \frac{C}{\pi} \int_{m_1}^{\infty} \frac{dz}{1 + z^2} = \frac{m(t)g}{\pi} \int_{-\theta}^{\theta} dy \frac{\phi(y)}{y^2} + \frac{C}{\pi} \arctan \left( \frac{m(t)g}{m_1} \right). \quad (28)$$

We expand (28) around $m(t) = 0$:

$$m(t + 1) = \frac{m(t)g}{\pi} \left( \int_{-\theta}^{\theta} dy \frac{\phi(y)}{y^2} + \frac{C}{m_1} \right) + O(m(t)^3), \quad (29)$$

and conclude that the transition between quiescent and active state occurs at the critical point described by the equation

$$\frac{g}{\pi} \left( \int_{-\theta}^{\theta} dy \frac{\phi(y)}{y^2} + \frac{C}{m_1} \right) = 1. \quad (30)$$

Hence, this guarantees the existence of positive and finite critical $g^*$ that solves the above equation. It is possible to extend these results to activation functions $\phi(x)$ that are non-zero around $x = 0$ and are non-saturating—the transition exists if $\phi(x)$ is sufficiently superlinear around $x = 0$ and sublinear for large $|x|$. 
FIG. 5. Mean field $m(t) \mapsto m(t + 1)$ mapping for fully connected (dense) Gaussian and Cauchy networks and sparse Gaussian network with a fixed number of incoming connections per neuron $K$. For large values of $K$, the MF equation looks similar to the fully connected Gaussian case. In particular, we observe a discontinuous transition at $g \approx 2.5$. (note that this similarity only holds if $g$ is not too large, since for any finite $K$ there is a continuous transition at which the trivial fixed point loses its stability). For an intermediate sparsity ($2 < K \leq 12$), only a second order transition is observed and the dynamics looks qualitatively similar to the dense Cauchy case. For $K \leq 2$ no transition to chaos is observed and the trivial fixed point is always stable (results not shown).

C. Gaussian threshold network

1. Fully connected network

For general activation function it is convenient to describe the behavior of the Gaussian networks in terms of another order parameter, $q_0(t) = (1/N) \sum_i \langle \phi(x_i(t))^2 \rangle$. However, in the binary case analyzed here it is equivalent to $m(t)$. The corresponding dynamical mean-field equation reads

$$m(t + 1) = \frac{1}{2} \left[ 1 - \text{erf} \left( \frac{\theta}{\sqrt{2}m(t)g} \right) \right].$$

(31)

Expanding the RHS of (31) around $m(t) = 0$ gives

$$m(t + 1) = \sqrt{\frac{m(t)g^2}{2\pi\theta^2}} \exp \left( -\frac{\theta^2}{2m(t)g^2} \right) \left( 1 + O(m(t)g^2) \right).$$

(32)

Due to the exponential factor $m(t + 1) < m(t)$ for small enough $m(t)$, which proves that the fixed point $m(t) = 0$ is locally stable under the evolution (31). Since the quiescent state is always stable, the transition to chaos, if present, must be discontinuous. This is confirmed by the graphical inspection of (31) (Fig. 5) and in computer simulations (Fig. 6).

The mapping to a branching process offers a simple way of understanding this result. The probability that a given synapse is
an autocrat reads

$$\text{Prob}(J_{ij} > \theta) = \frac{1}{2} \left( 1 - \text{erf} \left( -\sqrt{\frac{N}{2g}} \frac{\theta}{N} \right) \right)$$

In the thermodynamic limit the average number of autocrat connections per neuron can be calculated as before as

$$\lambda = \lim_{N \to \infty} P(J_{ij} > \theta),$$

which in our case leads to

$$\lambda = \lim_{N \to \infty} N g \sqrt{\frac{2}{\theta}} \exp \left( -\frac{N\theta^2}{2g^2} \right) \left( 1 + O(1/N^2) \right) = 0,$$

i.e. an activity starting from a single seed almost surely dies out.

2. Sparse network

Let each neuron receive exactly $K$ incoming connections, randomly chosen from the network, and let $J_{ij} \sim \mathcal{N}(0, g^2/K)$. If the mean activity of the network at time $t$ is $m(t)$, the probability that exactly $n$ incoming neurons are active is given by the binomial distribution

$$P(n; m(t)) = \binom{K}{n} m(t)^n (1 - m(t))^{K-n}.$$  

The membrane potential of a neuron $x$, conditioned on $n$ incoming connections being active, is a normal random variable with $\mu = 0$ and $\sigma^2 = ng^2/K$. The mean activity of the network in the next step is equal to the probability that the membrane potential
of any given neuron crosses the threshold, and so it reads

\[
m(t + 1) = 2^{-1} \sum_{n=1}^{K} \binom{K}{n} m(t)^n (1 - m(t))^{K-n} \left( 1 - \text{erf} \left( \frac{\sqrt{K}}{\sqrt{2ng}} \right) \right).
\] (36)

Our computer simulations corroborate the validity of (36), see Fig. 6. Note, additionally, that it is not difficult to show that (36) simplifies to the dense case (31) in the limit of \( K \to \infty \). Analogous calculations in the case of stochastic units were presented in [74], where the range of validity of the annealed approximation is also discussed.