Growth strategy determines network performance

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The interplay between structure and function is critical in determining the behavior of several systems. Here we propose an adaptive network model inspired in synaptic pruning that couples activity and topological dynamics. The coupling creates a discontinuous phase transition between an ordered memory phase and a disordered one as a function of the transient density. We prove that the existence of an initial transient period with relatively high density is critical in providing ordered stationary states that can be used to store stable memories. We also show that intermediate values of density are optimal in order to obtain these states with a minimum energy consumption, and that ultimately it is the transient heterogeneity in the network what determines the stationary state. Our results here could explain why the pruning curves observed in actual brain areas present their characteristic temporal profiles and, eventually, anomalies such as autism and schizophrenia associated, respectively, with a deficit or excess of pruning.

Complex networks are ubiquitous in nature: almost every biophysical, social or industrial system develops intricate relations among its components, resulting in a network configuration that is usually far from being homogeneous \cite{1}. Research on complex networks has received a tremendous amount of work over recent decades, in order both to understand and protect natural networks and to optimize technical designs. Most real networks have been shown to present non-trivial topological features, such as high clustering and short minimum paths (small-worldness), the existence of a modular structure, or cost-efficient wiring \cite{2,3}.

The vast majority of empirically-derived networks exhibit highly heterogeneous degree distributions (where the degree of a node is its number of neighbors), and, except in the case of social networks, negative degree-degree correlations – a property known as disassortativity. In other words, such networks include a small number of highly connected nodes, called hubs, which tend to be connected to low-degree nodes \cite{2}. Several studies have also discussed the effect that different network properties have on the dynamics of complex systems. For instance, it is known that both degree heterogeneity and degree-degree correlations strongly influence the signal to noise ratio in certain dynamical systems \cite{2,4,5}.

In order to understand how such non-trivial structures come about, much work has gone into investigating mechanisms of network evolution. Models in which networks are gradually formed, for instance by addition and/or deletion of nodes and edges, or by the rewiring of the latter, have been studied in various contexts \cite{6,11}. Typically, the probabilistic addition or deletion of elements at each time step is a function of the existing structure. For example, in the familiar Barabási-Albert model \cite{12}, a node’s probability of receiving a new edge at a given time is proportional to its current degree. Certain evolution rules have been shown to generate network topologies with particular properties, such as small-world, scale-free, or hierarchico-modular structures. These rules often give rise to phase transitions, such that different kind of network topologies can ensue depending on parameters. The idea behind all such evolving network models is that there should be general, relatively simple microscopic mechanisms which can give rise to these complex structures without the need for high levels of information or tuning \cite{3,4,5}.

In most real-world networks the evolution of the topology is invariably linked to the state of the network and vice versa \cite{13}. This has given rise to a novel field of study: co-evolving or adaptive networks \cite{14}. These couple the activity on the nodes of the network with the evolution of its topological structure, creating a feedback loop between dynamics and topology. Certain dynamic phenomena appear repeatedly in adaptive networks: the formation of complex topologies, robust dynamical self-organization, spontaneous emergence of different classes of nodes, and complex mutual dynamics in both activity and topology \cite{15,16,17}.

This framework can also be applied to brain development. The mammalian brain is formed by an initial rapid proliferation of synapses between neurons. Synaptic density reaches a peak during early infancy, and from then on it begins a steady decline down to about half this value later in life, in a process known as synaptic pruning \cite{18,19}. It is believed that the reason for reducing synaptic density is to become more energetically efficient \cite{20}. But then the question remains, why not begin life with the optimal synaptic density? There is also an increasing interest in studying the possible implications that synaptic pruning has on high-level brain
functions, and its relation with the emergence of some neurological disorders such as autism and schizophrenia [21, 22]. However, little is so far known about the influence of synaptic pruning the relatively high synaptic connectivity during early brain development on its performance.

Here we consider realistic pruning profiles that include an initial transient period of high synaptic density, and analyze who it affects pruning efficacy and the stationary state of the system, by adapting a biologically inspired evolving neural network model [23]. This couples a traditional associative memory model, the Amari-Hopfield model, with a preferential attachment model for network evolution [25], based on the fact that synaptic growth and death are related to neural activity [26]. This setup creates a feedback loop between structure and dynamics, leading to two qualitatively different behaviors. In one, the network structure becomes heterogeneous and disassortative and the system displays good memory performance. In the other, the structure remains homogeneous and incapable of pattern retrieval.

We show here that the inclusion of an initial, transient growth, even in a simple model, introduces a dependence on the initial synaptic density that increases network performance, in terms of memory retrieval, even in the presence of high levels of noise. The basic mechanism illustrated with our model here need not be restricted to neural networks, but may play a role in shaping the structures of other systems, such as protein interaction networks, which also change in evolutionary time scales in a way that is related to their physiological activity [27]. In fact, almost all biological networks change in time, so pruning may be a general mechanism for network optimization in order to minimize energy consumption in an environment of limited resources, without the need for detailed information specifying the initial topology.

**Synaptic pruning model**

We use a co-evolving model of synaptic pruning that couples a traditional associative memory model, the Amari-Hopfield model, with a preferential attachment model for network evolution [23]. The system consists of an undirected N-node network whose nodes represent neurons, whereas edges stand for synapses. Though the model may be easily generalized in this and other details, each neuron i is here a stochastic binary variable \( s_i(t) = \{0, 1\} \), indicating a silent or firing neuron, which follows an Amari-Hopfield dynamics [24]. The macroscopic state of the system is characterized by the overlap, \( m(t) \), of the network with a memorized pattern of activity, \( \{\xi_i\} \), which is stored by an appropriate definition of the synaptic weights \( w_{ij} \) [24]. In a fully connected network, \( m \) undergoes a continuous transition from a phase of memory recovery (\( m \to 1 \)) to one dominated by noise (\( m \approx 0 \)) as a function of the noise parameter or temperature \( T \), at the critical value \( T_c = 1 \).

Network structure is defined by the adjacency matrix \( e_{ij} = \{1, 0\} \), indicating the presence or absence of an edge, respectively. The topology changes in time according to the probabilities \( P^g \) and \( P^d \) that each node i has to gain of loose and edge at each time t. These are assumed to factorize in two terms, \( P^g = u(\kappa)\pi(I_i) \) and \( P^d = d(\kappa)\eta(I_i) \), where \( \kappa \) is the mean connectivity or degree of the nodes in the network and \( I_i \) stands for the incoming current at each neuron. Here \( u \) and \( d \) represent a global dependence to account for the fact that these processes rely in some way on diffusion of different molecules though large areas of tissue, for which \( \kappa \) is taken as a proxy. Provided that \( \pi(I_i) \) and \( \eta(I_i) \) are normalized functions, the evolution of \( \kappa(t) \) only depends on \( u(\kappa) \) and \( d(\kappa) \), and it is given by \( dx(\kappa)/dt = 2[u(\kappa(t)) - d(\kappa(t))] \) [24, 25]. On the other hand, \( \pi \) and \( \eta \) introduce a local dependence on the physiological state of the neurons, and account for local heterogeneity in the network. Following previous studies, we take \( \pi(I_i) \propto I_i^\alpha \), \( \eta(I_i) \propto I_i \), which corresponds to synapses being chosen at random for removal, which can be seen as a first order approximation of pruning dynamics [10]. Network structure is characterized by the homogeneity parameter \( g(t) \), which equals 1 if \( p(k) = \delta_{k_0,k} \) (homogeneous network) and tends to 0 for highly heterogeneous (bimodal) networks. In a topological limit of this limit, \( I_i \to k_i \), in \( \pi \) and \( \eta \), \( g(t) \) undergoes a continuous phase transition from homogeneous networks to heterogeneous ones, at \( \alpha_c = 1 \) [22].

Previous work analyzed the effect of the coupling between the local structure of the network and the physiological dynamics. Three phases were shown to appear depending on \( \alpha \) and \( T \): a homogeneous memory phase when both \( \alpha \) and \( T \) are low, in which the network displays memory and its structure is homogeneous; a heterogeneous memory phase for high \( \alpha \), in which the network is bimodal (appearance of hubs); and a homogeneous noisy phase for high noise \( T \) [23]. Finally, a bistability region was also shown to appear between the heterogeneous memory and the homogeneous noisy phases (which corresponds to moderate \( \alpha \) values, \( 1 < \alpha < 2 \), and high temperature, \( T > 1 \)) as a consequence of the coupling introduced by the model. Due to the better memory performance in noisy environments of heterogeneous networks [4, 24], this gives rise to a feedback loop in which memory and heterogeneity promote each other for \( \alpha \geq 1 \). In this region the stationary state of the system depends on its initial configuration: networks that are initially heterogeneous display memory and enhanced heterogeneity during the whole evolution of the system, whereas homogeneous ones fall into the noisy state.

Pruning profiles have been simulated by considering an exponential decay from \( \kappa_0 \) to \( \kappa_\infty = \kappa(t \to \infty) \), \( \kappa_0 > \kappa_\infty \), by defining \( u(\kappa) = \frac{N}{2} \left(1 - \frac{\kappa}{\kappa_\infty}\right) \) and \( d(\kappa) = \frac{N}{2} \frac{\kappa}{\kappa_\infty} \), so that \( \kappa(t) = \kappa_\infty \left(1 - (1 - \kappa_0/\kappa_\infty) e^{-t/\tau_p}\right) \), where \( \tau_p = N \kappa_\infty/2n \). However, experimental evidence indicates a fast growth of the synaptic density following birth and preceding synaptic pruning, whose impact on brain development is yet to be fully clarified [11, 22].

Realistic
pruning profiles that include this feature have been reproduced by the introduction of a non-linear time dependent term in \( u(\kappa), c(t) = a \exp(-t/\tau_g) \), which gives rise to a long transient of high connectivity before pruning \[ \text{[23, 25]} \]. However, the effect of this non-linear evolution of the connectivity on the network performance has not been analyzed so far.

Here we analyze the effect of the non-trivial transient of high connectivity preceding synaptic pruning on the dynamics of the system. To do so we place ourselves on the bistability region (in particular, we choose dynamics of the system. To do so we place ourselves on the bistability region (in particular, we choose \( T = 1.3 \), where the system is more dependent on initial conditions. We consider a simple approximation to the realistic pruning profile: \( \kappa(t) \) is kept constant (and high) at the onset of the evolution, during a “frozen-density” time \( \Delta \), by imposing that the same number of edges are created and destroyed. Thereafter, the mean degree is allowed to vary following the pruning dynamics. Pruning profiles are thus fully characterized by two parameters that control the maximum of the initial density, \( \kappa_0 \), and its temporal duration, \( \Delta \) (time width). A full and comprehensive description of the model is detailed in the Methods.

**Transient state of high connectivity improves memory performance**

The initial “frozen-density” period, for \( 0 < t < \Delta \), provides a non-trivial transient of network evolution during which edges are added and removed but the mean degree \( \kappa(t) \) is kept constant, as depicted in the figure \( \text{[1]} \), where an exemplar evolution of the system is shown. If \( \kappa_0 \) is sufficiently large, the system can be capable of memory retrieval throughout this period even for \( T > 1 \), as indicated by an overlap \( m(t) \) significantly different from zero. In that case, and given that the topological dynamics is also taking place, there is meanwhile an underlying rewiring process which starts creating hubs and heterogeneity, provided that \( \alpha > 1 \), and thus decreasing the homogeneity parameter \( g(t) \). Therefore, due to the coupling between memory and heterogeneity in the model, if the network maintains memory it continues to heterogenize, creating a feedback between neural dynamics and topology.

Once \( t > \Delta \) and the pruning begins, the system can either fall into the noise state and lose its heterogeneity (green lines in figure \( \text{[1]} \)), or remain in the heterogeneous memory state (purple lines), showing multistability. If the system remains in the memory state, it continues to heterogenize \( g(t) \to 0 \), to the point that even as \( \kappa(t) \to \kappa_\infty \), the system maintains good memory performance (see purple lines in figure \( \text{[1]} \)). On the other hand, if the neural network falls into the noisy state \( m(t) \approx 0 \), neural activity – and hence synaptic growth and death – becomes uncorrelated with node degree, and the topology reverts gradually to a more homogeneous configuration \( g(t) \to 1 \), incapable of memory.

![FIG. 1.](image)

**Non-linear effect of the initial density**

The initial density \( \kappa_0 \) also has a major effect on the dynamics (as depicted in figure \( \text{[2]} \)), determining whether the system will be able to maintain memory retrieval and the minimum \( \Delta \) necessary for it. In the case \( \kappa_0 = \kappa_\infty \), \( \kappa(t) \) is trivially constant and, given that \( T > 1 \), the system falls into the noise state regardless of \( \Delta \) (see \( \kappa_0 = 20 \) in figure \( \text{[2]} \)). This continues up to slightly higher initial
In fact the opposite effect is obtained and the minimum $\kappa$ as a function of $\Delta$ even for very low densities ($\kappa_0 = 25$), where the memory state is reached even for very low $\Delta$. One might expect that the memory state would become easier to reach with higher $\kappa_0$, but in fact the opposite effect is obtained and the minimum time of initial frozen-density needed to reach memory, $\Delta_{\text{min}}$, increases almost linearly with $\kappa_0$ for $\kappa_0 \gg \kappa_\infty$ (see figure 2b). This apparent paradox is explained with a deeper look at the system. In fact, large $\kappa_0$ implies that networks are initially more homogeneous and take more time to become heterogeneous under the topology dynamics. Besides, more edges have to be pruned to make a significant change in the network, which slows down network evolution. In consequence, high $\kappa_0$ networks are more likely to fall into the noise state for a given $\Delta$.

One can explicitly measure the minimum value of $\Delta$ needed to achieve memory for a given density, as in figure 2. More generally, we define $\Delta_{\text{min}}^a$ as the minimum value of $\Delta$ needed to reach a stationary mean overlap equal to $m_a$ for a given $\kappa_0$, with $m_a = 0.1a \cdot \bar{m}$, $a = 0, 1, \ldots, 10$. This definition aims to measure how much time it takes for a given configuration to organize into the homogeneous state. Minimal values of $\Delta_{\text{min}}^a$ indicate an optimal initial configuration to reach memory with a minimal energy consumption. Our measures (fig. 3b) indicate a second order effect of $\kappa_0$, so that there is an optimal initial connectivity for memory retrieval and network evolution towards heterogeneity, $\kappa_0 \approx 27$. In fact, $\bar{m}(\kappa_0)$ reaches its maximum for this mean density value (fig. 3b), so that memory is not only reached faster but it is also stronger for this value of $\kappa_0$.

Finally, an integrated view of the effect of network dynamics and the emergent behavior of the system can be obtained by the phase diagram of the system, as shown in figure 4, for the control parameters $\Delta$ and $\kappa_0$. This has been obtained by the integrated analysis of $\bar{m}(\Delta, \kappa_0)$ and $g(\Delta, \kappa_0)$, which indicates the existence of a region of stationary memory and heterogeneous networks for both high $\Delta$ and high $\kappa_0$. The phase transition between noise and memory moves to higher $\Delta$ for higher $\kappa_0$ in an approximately linear manner, as previously discussed, leading to the contraction of the heterogeneous memory region. On the other hand, very small $\kappa_0$ never lead to memory, due to the high thermal noise.

Overall, our results show the benefit of intermediate densities with respect to very high ones in order to achieve memory in a noisy environment. This could also explain why for an evolving network such as the infant brain it is detrimental to initially grow a very high density of synapses, since this increases the energy costs during growth and also during the pruning process, and it does not improve memory retrieval or network structure. On the contrary, a neural network with intermediate values of transient synaptic density would perform more efficiently during pruning. Moreover, this would also be convenient in terms of energy consumption.

**Transient heterogeneity determines network performance.**

We have shown a quadratic dependence of the stationary state on $\kappa_0$, and discussed the presence of multistability for intermediate values. However, what determines, on a given trial, the stationary state of the system? Based on the results shown above, we propose that it is the transient level of heterogeneity (that is, the heterogeneity at the onset of the pruning) which determines the probability that the network will maintain memory.

In order to explore this hypothesis, we first define $g_\Delta$ and $m_\Delta$ as the value of $g$ and $m$ at the beginning of the pruning, that is, $g_\Delta = g(t = \Delta)$ and $m_\Delta = m(t = \Delta)$.
These definitions allow us to explore how the stationary state depends on the transient evolution of the system. In particular, $\tilde{m}(g, \kappa)$ (fig. 4) shows two main findings. Firstly, a continuous transition from the heterogeneous memory state to the homogeneous noisy one as a function of $g$; and, secondly, a collapse of the curves for $\kappa_0 > 24$, that is, when memory is achieved. A more direct way to establish the determinant effect of $g$ on the stationary memory is to directly measure the probability of achieving stationary memory with a given transient heterogeneity, $P_{\text{mem}}$, as in figure 4. There is a transition from one to zero when increasing $g$ in which all the curves for every initial density $\kappa_0$ collapse. Analogous curves could be obtained for the stationary heterogeneity (data not shown). In consequence, $g$ determines whether the network will be able to maintain memory once the pruning begins: high onset heterogeneity (small $g$) implies stationary memory, whereas low heterogeneity (high $g$) implies a stationary noisy state. These results are independent of $\kappa_0$, indicating that $g$ is a strong indicator of stationary memory.

Finally, notice that $g$ depends both on $\kappa_0$ and $\Delta$, and also on $m$, since the rewiring process only improves heterogeneity when the network displays memory. This is shown in figure 4, where we display $\tilde{m}$ ($m$) and each curve corresponds to a $\Delta$. Since $m$ does not unequivocally determine the stationary state, the curves do not collapse in this case. What it is obtained is a quadratic dependence of $\tilde{m}$ on $m$, indicating an optimal value of transient memory of $m = 0.5$. This emerges because $m = 0.5$ strongly correlates with a minimum $\tilde{g}$, so that the value of $\Delta$ necessary for a stationary memory state is minimal for this value. In this sense, if $m = 0.5$, there is no transient memory because $\kappa_0$ is too small and, therefore, there is insufficient heterogeneity. On the other hand, if $m = 1$, then it is because $\kappa_0$ is large and the network is still very homogeneous when pruning begins. In both cases, the network evolves towards a homogeneous configuration.

**Discussion**

We have presented an adaptive network model inspired by synaptic pruning that creates a dependence of the final network structure and performance on the transient synaptic density, as expected in nature. This occurs because of an activity-topology feedback loop. In this model, the introduction of a high density transient allows for network heterogenization and the maintenance of memory in noisy conditions. We have analyzed in detail a point of the $(T, \alpha)$ phase space corresponding to a bistability area between heterogeneous memory and homogeneous noisy states. This has been done to place the system at a point where it is most sensitive to the details of the evolution and the initial conditions. This corresponds to high noise $T$ and also large $\alpha$, so that the system can heterogenize.

In these conditions, we have found that the model develops a first order phase transition with the length of the transient period of fixed density, $\Delta$, which also depends on $\kappa_0$. In fact, there is a quadratic effect of $\kappa_0$, such that medium values provide a faster and more stable evolution towards a memory stationary state. We have found an optimal $\kappa_0 = 27$ to optimize the evolution into such a state. Therefore, our results could explain why real world networks such as the brain do not create enormous numbers of synapses to begin with during early development. Moreover, being able to achieve eventual good performance with a limited density would also be preferable in terms of energy consumption. We have also shown that the transient heterogeneity determines the stationary state of the system. Given the feedback loop, this depends on the transient memory $m$ and $\kappa$, so that the stationary state of the system is ultimately determined by its physiological history.

The question this work set out to answer was why brain development should entail an initial growth of a great many synapses which are then gradually pruned. If the final density is optimal for energy consumption, why pass through a transient state of twice this density? We have considered a neural network with an evolving structure based on some simple biological considerations, and found that the memory performance of the eventual system does indeed depend on whether it passed through a transient period of relatively high synaptic density. The feedback loop which emerges between neural activity and network topology is such that, beginning with a random network, a transient state of high density can allow for
the subsequent pruning of synapses as the topology is optimized for memory performance. Why, though, should the brain not begin with both the low density and high heterogeneity needed for good memory performance? We conjecture that much less genetic information is needed for good memory performance? We have also seen that there is an optimal initial synaptic density for a high performance neural network to emerge. Our work here may serve as a starting point and a very suitable theoretical framework for studying the relationship that may exist between certain neurological disorders that appear during brain development, such as childhood autism and schizophrenia in young adults, and different synaptic pruning profiles, such as recent works have been reported.

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METHODS

The neural network model. Our system consists of an undirected $N$-node network whose edges change in discrete time. At time $t$, the adjacency matrix is $\{e_{ij}(t)\}$, for $i, j = 1, \ldots, N$, with elements 1 or 0 according to whether there exists or not an edge between the pair of nodes $(i, j)$, respectively. The degree of node $i$ at time $t$ is $k_i(t) = \sum_{j=1}^{N} e_{ij}(t)$, and the mean degree of the network is $\kappa(t) = N^{-1} \sum_{i=1}^{N} k_i(t)$.

Each node represents a neuron, and each edge a synapse. The neurons are stochastic binary units, $s_i(t) \in \{0, 1\}$, indicating a firing or silent neuron, respectively. Each edge $(i, j)$ is characterized by its synaptic weight $w_{ij}$, and the local field at neuron $i$ is $h_i(t) = \sum_{j=1}^{N} w_{ij} e_{ij}(t) s_j(t)$. The states of all neurons are updated in parallel at every time step following the Amari-Hopfield scheme, according to the transition probability $P \{s_i(t+1) = 1\} = \frac{1}{2} \left[ 1 + \tanh(\beta [h_i(t) - \theta_i(t)]) \right]$, where $\theta_i(t) = \frac{1}{2} \sum_{j=1}^{N} w_{ij} e_{ij}(t)$ is a neuron’s threshold for firing, and $\beta = T^{-1}$ is a noise parameter controlling stochasticity (analogous to the inverse temperature in statistical physics) [24, 29].

A set of $P$ patterns of activity, or memories, $\{\xi^\mu; \mu = 1, \ldots, P\}$, with mean $a_0 = \langle \xi^\mu \rangle$, is encoded through $w_{ij}$ via the Hebbian learning rule, $w_{ij} = \left[ \kappa_0 a_0 (1-a_0) \right]^{-1} \sum_{\mu=1}^{P} (\xi_i^\mu - a_0)(\xi_j^\mu - a_0)$, where $\kappa_0 = \kappa(t \rightarrow \infty)$. The macroscopic order parameter is the overlap of the state of the network with each of the memorized patterns of activity, $m^\mu(t) = (N a_0 (1-a_0))^{-1} \sum_{i=1}^{N} (\xi_i^\mu - a_0) s_i(t)$. Here we fix $P = 1$ and simplify the notation making $m^\mu = m$. The canonical Amari-Hopfield model, which is here a reference, is defined on a fully connected network ($e_{ij} = 1$, $\forall i \neq j$), and it exhibits a continuous phase transition at the critical value $T_c = 1$ [24].

The pruning model. Edge dynamics are modeled as follows. At each time $t$, each node has a probability $P_{ij}^d = u(\kappa) \pi(I_j)$ of being assigned a new edge to another node, randomly chosen. Likewise, each node has a probability $P_{ij}^l = d(\kappa) \eta(I_j)$ of losing one of its edges, randomly chosen. Here the time dependence has been dropped for clarity, and $I_j = |h_j - \theta_j|$ is a physiological variable that characterizes the local dependence.

Initially, network topology is defined as in the configuration model [28]. Time evolution is then accomplished in practice via computer simulations as follows. First, the number of links to be created and destroyed is chosen according to two Poisson distributions with means $N u(\kappa)$ and $N d(\kappa)$, respectively. Then, as many times as needed according to this draw, we choose a node $i$ with probability $\pi(I_j)$ to be assigned a new edge, to another node randomly chosen; and similarly we choose a new node $j$ according to $\eta(I_j)$ to lose an edge from one of its neighbors, randomly chosen. This procedure uses the BKL algorithm to assure proper evolution towards stationarity [21]. Therefore, each node can then gain (or lose) an edge via two paths, and we define the effective values of $\pi$ and $\eta$ to account for this effect: $\tilde{\pi} = 1/2 (\pi(I_i) + 1/N)$ and $\tilde{\eta} = 1/2 (\eta(I_i) + k_i/(\kappa N))$, where the $1/2$ factor is included to assure normalization.

It is not at present known exactly how synaptic growth and death depend on neural activity. For the sake of simplicity, we shall consider $\tilde{\pi}$ and $\tilde{\eta}$ to be power-law distributed [23], which allows one to move smoothly from a sub-linear to a super-linear dependence with a single parameter, $\tilde{\pi} = I_i^{\gamma}/((I_i^\alpha + N) \tilde{\pi} = I_i^{\gamma}/((I_i^\beta + N) N)$. Previous studies have shown that the qualitative results do not depend independently on $\alpha$ and $\gamma$ but on the ratio between them, so that for simplicity we fix $\gamma = 1$, and leave $\alpha$ as a topological control parameter.

Network structure is characterized by its degree distribution at each time, $p(k, t)$, whose homogeneity may be measured via $g(t) = \exp\left(-\sigma^2(t)/\kappa^2(t)\right)$, where $\sigma^2(t)$ is the variance of the distribution and $\kappa$ its mean, as defined before. $g(t) = 1$ for highly homogeneous networks ($p(k) = \delta(k - k_0)$, and goes to zero for highly heterogeneous ones.

Time evolution of the mean connectivity. The time evolution of $\kappa(t)$ can be obtained in the topological limit in the model, defined by making the substitution $I_i \rightarrow k_i$, so that $\tilde{\eta} = \tilde{\eta}(k_i)$ and $\tilde{\pi} = \tilde{\pi}(k_i)$. This is possible since in the memory regime it follows that $I_i \propto k_i$, whereas in the noisy one both variables follow equivalent noisy dynamics [23]. In this way we can construct a master equation for $p(k, t)$ by considering network evolution as a one step process with transition rates $u(k)\tilde{\pi}(k)$ for degree increment and $d(k)\tilde{\eta}(k)$ for the decrement. Approximating the temporal derivative for the expected value of the difference in a given $p(k, t)$ at each time step we get: \[ \frac{dp(k,t)}{dt} = u(k)\tilde{\pi}(k-1) p(k-1,t) + d(k)\tilde{\eta}(k+1) p(k+1,t) - u(k)\tilde{\pi}(k) p(k,t) - d(k)\tilde{\eta}(k) p(k,t), \] which is exact in the limit of no degree-degree correlations between nodes. From here it follows that $\frac{d\kappa(t)}{dt} = 2 [u(\kappa(t)) - d(\kappa(t))]$.

The time scale for structure changes is set by the parameter $n$, whereas the time unit for activity changes, $h_a$, is the number of Monte Carlo Steps (MCS) that the states of all neurons are updated according to the Hopfield dynamics between each structural network update. Previous studies show a low dependence on this param-
eter in the cases of interest, so we report results here for $h_s = 10$ MCS \[23\].

Statistics and general methods In this work, we used systems sizes $N = 800, 1600$ and $3200$, as indicated in each section. Results for $N < 800$ presented strong finite size effects, so they were discarded (data not shown). The sample size for each result was chosen by convergence of the mean value. Measures of the global variables on the stationary state are obtained by averaging during a long window of time: $\bar{f} = \Delta t^{-1} \sum_{t=t_0}^{t_0+\Delta t} f(t)$.

Code availability Generated codes are available from the corresponding author upon reasonable request.

Data availability All data that support this study are available from the corresponding author upon reasonable request.