Apparently similar neuronal dynamics may lead to different collective repertoire

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This report is concerned with the relevance of the microscopic rules, that implement individual neuronal activation, in determining the collective dynamics, under variations of the network topology. To fix ideas we study the dynamics of two cellular automaton models, commonly used, rather in-distinguishably, as the building blocks of large scale neuronal networks. One model, due to Greenberg & Hastings, (GH) can be described by evolution equations mimicking an integrate-and-fire process, while the other model, due to Kinouchi & Copelli, (KC) represents an abstract branching process, where a single active neuron activates a given number of postsynaptic neurons according to a prescribed “activity” branching ratio. Despite the apparent similarity between the local neuronal dynamics of the two models, it is shown that they exhibit very different collective dynamics as a function of the network topology. The GH model shows qualitatively different dynamical regimes as the network topology is varied, including transients to a ground (inactive) state, continuous and discontinuous dynamical phase transitions. In contrast, the KC model only exhibits a continuous phase transition, independently of the network topology. These results highlight the importance of paying attention to the microscopic rules chosen to model the inter-neuronal interactions in large scale numerical simulations, in particular when the network topology is far from a mean field description. One such case is the extensive work being done in the context of the Human Connectome, where a wide variety of types of models are being used to understand the brain collective dynamics.

I. INTRODUCTION

The animal brain is composed by billions of neurons, which interact with each other through thousands of synapses per neuron. The results of such interaction is the emergence of complex spatio-temporal patterns of neuronal activity supporting perception, action and behavior. A recent proposal considers the brain as a network of neurons poised near a dynamical transition \cite{1–4}, a view which is supported by experimental results gathered from animals both \textit{in vitro} \cite{5} and \textit{in vivo} \cite{6} as well as from whole brain neuroimaging human experiments \cite{7–9}.

The potential existence of critical phenomena in the brain motivated during the last decade the study of mathematical models to better explore the large-scale brain dynamics. A distinctive difference between the diversity of models is at the microscopic level. Some models consist of networks of simplified neurons, in which neurons themselves are represented by a wide variety of approaches, ranging from 2-state particles \cite{10} through discrete cellular automats \cite{11–14}, branching processes \cite{6, 15–18}, neural masses \cite{19}, coupled-maps \cite{20–23}, coupled Kuramoto oscillators \cite{24} up to detailed equations describing the evolution and spiking of the membrane potential \cite{25–27}. Thus, a natural question arises on how relevant may the microscopic process used to represent the individual neuronal dynamics be, and how they affect the dynamical collective repertoire exhibited by the network.

When focusing on collective properties, it is of course reasonable to seek minimal models which, even orphan of realistic microscopic rules, may reproduce relevant macroscopic behavior. However, it is not straightforward to determine in principle how general this assumption can be in the case of neuronal networks. Our point is that, even though the use of realistic microscopic dynamics is not necessarily a prerequisite to correctly describe universal macroscopic properties, \textit{microscopic rules do matter} and eventually can lead to different universal behavior. As a clarifying metaphor consider the Ising model. It is well known that algorithms with unrealistic non-local moves (so-called cluster algorithms \cite{28}) can correctly describe the static critical behavior. However, if
the dynamic rule did not follow detailed balance, then the modified dynamics would fail to reproduce equilibrium behavior, even if it could reproduce some sort of critical dynamics. And of course, even with detailed balance, the dynamical universality is altered by the non-local rule. A similar correspondence among microscopic rules and system’s dynamics appears when modeling brain dynamics, which is rarely considered, thus some extrapolations to real brain dynamics taken from numerical simulations in the current literature, may be hampered by the limitations of the microscopic details of the neuronal models employed. We are purposely not considering here a large chapter of models that include synaptic plasticity.

In this article, we illustrate the problem by studying the dynamics of two apparently similar neural network models: that of Greenberg & Hastings [29] as described in [11, 12] and that of Kinouchi & Copelli [15]. The main difference between these two models is related to the microscopic rule that propagates the activity: the first proposes (as many others of the same kind) a neuronal interaction rule that depends on the state of its presynaptic neighbors, while the second introduces a rule that, regardless of the number of connections, maintains a prescribed branching of activity on the target neurons. At first sight the differences seem innocent-looking, but as it will be shown, they lead to completely different behavior of the network: the first model exhibits continuous or discontinuous phase transitions depending on the network topology, while the second is completely insensitive to it.

We remark from the outset that the article’s aim is not to criticize any given model in particular, but to call the attention on the consequences of using them ignoring the limitations of the model’s original formulation, including possible misinterpretations. The article is organized as follows: in Sec. II we describe for both models the observables that will be used to characterize the dynamical regimes, in Sec. III we show the results of the numerical simulations, in Sec. IV we discuss present results in the context of recent research and we summarize the conclusions.

II. NETWORK, MODELS AND OBSERVABLES

A. The interaction network

Both neuronal models are studied on an undirected Watts-Strogatz small-world network [30] with average connectivity $(k)$ and rewiring probability $\pi$. The network is constructed as usual [30] by starting from a ring of $N$ nodes (always $N = 20000$ in this report), each connected symmetrically to its $(k)/2$ nearest neighbors; then each link connecting a node to a clockwise neighbor is rewired to a random node with probability $\pi$, so that average connectivity is preserved. The rewiring probability is a measure of the disorder in the network: for $\pi = 0$ the network is circular and perfectly ordered, while for $\pi = 1$ it becomes completely random. In both models

\[ S_{i+1} = f(\{S_j, S_{kj}, \ldots, S_{ki}, W_{ki}, W_{kj}, \ldots, W_{jk}, T\}) \]

\[ S_{i+1} = f(\{S_j, S_{kj}, W_{kj}, \sigma_p\}) \quad \mu = 1, \ldots, k \]

FIG. 1. Rule for the propagation of activity in both models. Panel (a): In the GH model, a given neuron $I$ will become active at time $t+1$, if the contribution of all active presynaptic neurons (here $J_1$ and $J_2$), weighted by the interaction $W_{I,J}$ surpasses the threshold $T$. At each time step, this update is repeated for all quiescent neurons $I$. Panel (b): In the KC model a given active neuron $J$ will activate at time step $t+1$ a given number of $I$ neurons depending on the $W_{I,J}$ and $\sigma_p$ value. Note that $\sigma_p$ normalizes such probability by the number of interactions $(k)$. At each time step, this update is repeated for all active neurons $J$. In both panels, triangles represent neurons and lines synaptic interactions. Red filled triangles denotes active neurons and gray dashed ones inactive neurons, at times $t$ and $t+1$.

neurons are represented as nodes on a weighted undirected random graph with an associated discrete state variable, $S_i = 0, \ldots, n$, where $i = 1, \ldots, N$ identifies the node and $S_i = \{0, \ldots, n\}$. State 0 represents a quiescent (but excitable) neuron, 1 is the active state, and 2 ... $n$ are refractory states. The links of the graphs are represented by the $N \times N$ connectivity matrix $W$. Nonzero matrix elements indicate the presence of a link with a given weight. Weights are positive reals, so $W_{ij} \geq 0$, and the connectivity matrix is symmetric, $W_{ij} = W_{ji}$, since the graph is undirected. In this context symmetric connections need to be interpreted as two connections between any pair of nodes. Neither the connectivity nor the weights depend on time (i.e., we consider quenched disorder). The dynamical evolution is given by a discrete-time Markov process in which all sites are simultaneously updated, and with transition probabilities for each site given by the expressions below for each model.
1. GH model:

This model was introduced by Greenberg & Hastings [29] to mimic the excitable dynamics generically observed in neurons, forest fires, cardiac cells, chemical reactions and epidemic propagation. In the context of brain dynamics it was used recently by Haimovici et al. [11]. Here we follow closely the implementation of Zarepour et al. [12]. It is a cellular automaton endowed of the three states common to excitable dynamics: quiescent, active and refractory state, and the dynamics of site $i$ is updated by

$$P_{i,0 \rightarrow 1} = 1 - [1 - r_1] \left[ 1 - \Theta \left( \sum_{j=1}^{k_{in,i}} W_{ji} \delta_{S_j,1} - T \right) \right],$$

$$P_{i,1 \rightarrow 2} = 1,$$  

$$P_{i,2 \rightarrow 0} = r_2,$$  

where $P_{i,a \rightarrow b}$ is the probability that site $i$ will transition from state $a$ to state $b$, at time $t+1$, $S_i$ is computed at time $t$, and the sum is performed over all $j$ targeting $i$ and $k_{in,i}$ is the in-degree of node $i$. $\Theta(x)$ is Heaviside’s step function [$\Theta(x) = 1$ for $x \geq 0$ or 0 otherwise]. $\delta_{ij}$ is Kronecker’s delta, and $r_1$, $r_2$ and $T$ are control parameters which are set equal to all sites in the present work. Thus an active site always turns refractory in the next time step, and a refractory site becomes quiescent with probability $r_2$. The probability for a quiescent site to become active is written as 1 minus the product of the probabilities of not becoming active through the different mechanisms at work. In this model there are only two activation mechanisms: spontaneous activation, which occurs with a small probability $r_1$, or transmitted activation, which occurs deterministically whenever the sum of the weights of the links connecting $i$ to its active neighbors exceeds a threshold $T$ (see Panel (a) of Fig. 1). The non-null weights are drawn from an exponential distribution, $p(W_{ij} = w) = \lambda e^{-\lambda w}$, with $\lambda = 12.5$ chosen to mimic the weight distribution of the human connectome [12]. For the simulations described here, we use $r_1 = 0.001$, $r_2 = 0.3$ as in previous work [11, 12] which remain fixed in all simulations, while $T$ is used as control parameter.

2. KC model:

This model was introduced by Kinouchi & Copelli [15] to show that a (Erdős–Renyi undirected) network of excitable elements has its sensitivity and dynamic range maximized at the critical point of a non-equilibrium phase transition. The model resembles a branching process [31, 32] in which the transition probabilities for neuron $i$ at time $t+1$ are:

$$P_{i,0 \rightarrow 1} = \left\{ \begin{array}{ll}
1 & \text{if } \sum_{j=1}^{k_{out,i}} [1 - pW_{ji} \delta_{S_j,1}], \\
0 & \text{otherwise}
\end{array} \right.$$  

where $S_j$ is evaluated at $t$, and the product is taken over all neurons $j$ pointing to $i$. The interaction rule in Eq. 2a contains two parameters: $r_1$ which (as in the GH model) determines the spontaneous activity of any inactive neuron and $\sigma_p$ which acts as a control parameter. (see Panel (b) of Fig. 1). This rule makes the main difference with the GH model: here an active site $j$ activates a *given number of neighbors $i$* with probability $\sigma_p W_{ji}$. If the variance of the chosen values for $k_{out}$ and $W$ are relatively small, (as in ref. [15]) and $\langle k \rangle$ is relatively large, each active neuron will excite, on average $\sigma_p \approx (\langle k \rangle - 1)p/2$ neurons. Thus $\sigma_p$ represents the desired branching ratio.

It is known that, for a wide variety of conditions, critical dynamics is expected for $\sigma_p \approx 1$ [31]. The interaction matrix is symmetric, $W_{ij} = W_{ji}$ following [15], and non-null elements are taken uniformly from $[0, 1]$. Similar to the GH model, an active site always becomes refractory, but instead of recovering randomly, here it becomes quiescent deterministically after $n - 1$ time steps. We note that this difference has no relevance for the present analysis.

For the simulations, values of $r_1 = 0.001$, and $n = 4$ (i.e., a fixed refractory period of 3 steps) are chosen, which remain fixed in all simulations. In passing, please notice that the interaction rule in the KC model is entirely stochastic and that neurons behave independently (as long as spontaneous activity is relatively low as dictated by the value of $r_1$ used here). Additional details can be found in Costa et al. [33] and Campos et al. [34]. The numerical implementation of the KC model admits a few variations which, nonetheless, does not change the present results (see Supplemental Material [35]).

B. Observables

To describe the state of the network, for both models, we define an order parameter $f_S(t)$ which corresponds to the fraction of active neurons at time $t$,

$$f_S(t) = \frac{1}{N} \sum_i \delta_{S_i(t),1}. \quad (3)$$

After any transient dies out, we also compute its variance, $\sigma^2_{f_S} = \langle f^2_S \rangle - \langle f_S \rangle^2$, where $\langle \ldots \rangle$ is a time average.
For the purposes of the present work, it is of particular interest the behavior of the (normalized) connected autocorrelation of the order parameter $f_S$,

$$AC(\Delta t) = \frac{1}{\sigma_{fS}} \left\langle \left( f_S(t) - \langle f_S \rangle \right) \times \left( f_S(t + \Delta t) - \langle f_S \rangle \right) \right\rangle,$$

which estimates the linear correlation between the network state at times $t$ and $t + \Delta t$, with $AC(\Delta t) \approx 1$ for highly correlated consecutive configurations and $AC(\Delta t) \approx 0$ when the configurations quickly decorrelate. It is known that the autocorrelation function is sensitive to the different dynamical regimes: close to a continuous phase transition, the dynamics undergoes critical slowing down, which implies that the autocorrelation function decays slower than in the supercritical or subcritical state [37]. For discontinuous phase transitions, a similar effect takes place at the spinodal points [38]. Here we focus on the autocorrelation at $\Delta t = 1$, $AC(1)$, also called first correlation coefficient, which has been shown to have a maximum at the transition point [39].

C. Parametric exploration

In this work we are interested in exploring the extent of the dynamical repertoire that each model is able to exhibit under a very wide range of: 1) neuronal dynamics and 2) topology of the underlying network. Thus, we proceed to scan the control parameter of the given neuron model for different network topologies (by varying $k$ and $\pi$). This implies to explore three parameters while classifying the dynamical regimes observed.

To identify and classify the dynamical regimes we track the behavior of $AC(1)$ as the control parameter ($T$ or $\sigma_p$) is increased and decreased. This is repeated for each combination of network parameters ($k$) and $\pi$). The simulations start at $T_0$ (or $\sigma_0$) (using a random initial condition for each neuron) and then it is increased by $\Delta T$ (or $\Delta \sigma$) after a given number of steps, up to a final value $T_F$ (or $\sigma_F$), without resetting the neuron states when changing the value of the control parameter. This parametric exploration allows us to determine the full repertoire of dynamical regimes which can emerge from the microscopic activity propagation rules acting on a given network topology.

III. RESULTS

A. Characterizing the transitions

Now we proceed to describe how the dynamical repertoire of each model is determined from parametric exploration. An example is presented in Fig. 2 where panels on the left correspond to results obtained from the GH model and those on the right from the KC model. The figure shows that, as expected, the rate of activity changes as a function of its control parameter, but already demonstrating an important difference between the dynamical regimes exhibited by the two models. For this particular choice of topology, ($k$) = 30 and $\pi$ = 0.6, the GH model undergoes a discontinuous transition demonstrated by the abrupt change in $f_s$ (also noted in the appearance of the raster plot) and the presence of hysteresis. In contrast, in response to similar parametric scan, the KC model exhibits a continuous transition and does not show hysteresis. In addition, it is important to note that the GH model shows a large increase in the variability of the order parameter $f_s$ near the transition (see panel b), meanwhile the variance of the $f_s$ fluctuations shown by the KC model is relatively constant (see panel c), regardless of the value of the control parameter $\sigma_p$. These observations point to important dynamical differences between the two models, as will be expanded in the next sections.

FIG. 2. Examples of the evolution of the activity as the respective control parameters ($T$ or $\sigma_p$) is slowly varied. Left panels (a-c) correspond to the GH model and right panels (d-f) to the KC model. Top panels: value of the control parameters ($T$ in (a) and $\sigma_p$ in (d)) as a function of time $t$. Center panels: order parameter $f_S(t)$ for both models as a function of time. Bottom panels: raster plots of 300 selected neurons. Dashed lines in panels (a) and (b) mark the approximate values of $T_\pm$, and their respective times. In both cases ($k$) = 30, $\pi$ = 0.6. For the GH model, $\Delta T = \pm0.000025$, and for the KC model $\Delta \sigma = \pm0.00036$.

FIG. 3. Evolution of the order parameter $f_s$ and its autocorrelation $AC(1)$ as a function of the control parameter $T$ in the GH model. Panel (a): fraction of active neurons vs. threshold $T$. Panel (b): First autocorrelation coefficient $AC(1)$ vs. threshold $T$. In both panels the transition points, $T_-$ and $T_+$, are marked with vertical dashed lines. Simulations were performed in a network with ($k$) = 40 and $\pi$ = 0.6. Simulations started at $T_0 = 0$ and neurons in a random state. $T$ was slowly increased by $\Delta T = 0.005$ every $10^5$ time steps up to $T_F = 0.9$, then it was decreased back to $T_0$ in the same way.
The behavior of the autocorrelation function of the order parameter helps to identify the type of phase transition because it is known to peak near a transition. We compute \( AC(1) \) for each value of the control parameter, and define \( T_+ \) as the value that maximizes \( AC(1) \) in a run when \( T \) is being increased, and \( T_- \) as the value that maximizes \( AC(1) \) when decreasing \( T \). An example for the GH model is presented in Fig. 3. For the KC model we defined in the same way \( \sigma_p^+ \) and \( \sigma_p^- \), although we never observed dis-continuous transitions in that model.

Thus, according to the shape of the curves of \( AC(1) \) vs. control parameters, we can classify the dynamical behavior: if \( AC(1) \) is monotonic, then there is no phase transition, corresponding to the cases in which the network, after a transient goes quiescent. For network topologies in which \(|T_+ - T_-| \geq 2\Delta T\) (or \(|\sigma_p^+ - \sigma_p^-| \geq \Delta \sigma_p\)) the transition is considered discontinuous, and continuous otherwise. In other words, after exploring a reasonable range of values of the control parameter, the existence of a maximum in the \( AC(1) \) curve indicates (under the present context) a phase transition, which is considered continuous if there is no noticeable hysteresis or discontinuous otherwise.

An example of the behavior of \( AC(1) \) in the case of a continuous transition is shown in Fig. 4. This type of transition is observed in both models for a wide range of \( \langle k \rangle \) and \( \pi \) values, as will be described in the next section. It can be seen that a change of the control parameter on a range of values near the critical point is reflected on a non-monotonic change of the \( AC(1) \). The plots in the bottom panels illustrate the typical autocorrelation function of the order parameter \( f_{\pi} \). For control parameter values larger than \( T_c \) (or smaller than \( \sigma_c \)) the activity correlation vanishes quickly as indicated by the green triangle data points. In the other extreme, for control parameter values smaller than \( T_c \) (or larger than \( \sigma_c \), i.e., data points plotted as blue circles) the function shows an oscillatory pattern. The first zero crossing of the function is dictated by the duration of the refractory period of the neuronal models which is one of the determinants of the collective oscillation frequency. Finally, for values sufficiently close to \( T_c \) (or \( \sigma_c \)) the function \( AC(\Delta t) \) decays very slowly (as a power law) as shown in the figure by the data points plotted with red squares.

### B. Models’ dynamical repertoire on parameter space

Here we describe the results of a systematic exploration of the collective dynamic as a function of network topology in each model. For each value of \( \langle k \rangle \) and \( \pi \), we computed 5 realizations of Watts-Strogatz graphs. In each case we classified the regimes as a function of the control parameter, according to the behavior of \( AC(1) \) as explained above. The dynamical regimes found include transients to no-activity, continuous phase transition or discontinuous phase transition (from no-activity to collective oscillations).

The results in Fig. 5 show the regions of parameters at which each regime was observed. In brief, both models exhibit no-transition for network topologies with \( \langle k \rangle = 2 \) and connectivity disorder \( \pi > 0 \) (red zone with squares in Fig. 5). For \( \pi = 0 \) the same regime extends to \( \langle k \rangle < 6 \) in both models.

For networks with relatively large values of \( \langle k \rangle \) both models exhibit a continuous phase transition as in the example featured already in Fig. 4. (black zone with circles in Fig. 5). The main difference between the models is found for relatively high values of degree and disorder. At this region of parameters the GH model shows a discontinuous phase transition (blue zone with triangles), while the KC model a continuous one.

The results in Fig. 6 are representative examples of the behavior of \( AC(1) \) as a function of the control parameter for selected values of \( \langle k \rangle \) and \( \pi \). For the GH model, the largest values of \( \langle k \rangle \) and \( \pi \) show clear hysteresis, with the peaks for the case of increasing \( T \) at a higher value than the peak found when is decreased. In most cases, the increasing and decreasing sweeps of control parameter yield the same curve, with a maximum value of \( AC(1) \) close to 1. For \( \pi = 0 \), the (single) peak tends to be rather broad. Finally, for \( \langle k \rangle = 2 \) and any value of \( \pi \), and for \( k = 4 \), \( \pi = 0 \), \( AC(1) \) behaves monotonically, which is indicative of no phase transition. The KC model shows less variation among the curves, with only a narrow range of monotonous curves, and most of the \( \langle k \rangle \), \( \pi \) plane yielding continuous transitions.

Finally, the results on Fig. 7 show examples of the spiking patterns observed as the control parameter is increased, for the same selected values of \( \langle k \rangle \) and \( \pi \) illus-
FIG. 5. Classes of collective dynamics emerging at different network topologies for both models (GH model in panel a, KC model in b). The graphs summarize the dynamical regimes observed for a wide range of network parameters (average connectivity $\langle k \rangle$ and degree of disorder $\pi$). Blue regions (triangles denote the values tested) indicate those values for which the networks exhibited discontinuous phase transitions to collective oscillations. Black regions (with circles) indicate continuous phase transitions, and red regions (with squares) transient dynamics to inactivity but no phase transition. The inset in (a) depicts a portion of the same data plot in the main panel in log-log scale.

FIG. 6. Representative examples of the typical behavior of $AC(1)$ as a function of the control parameter for diverse topologies in both models (GH model in panel a, KC model in b). Simulations were run for $5 \cdot 10^4$ time steps for each value of $T$ or $\sigma_p$, increasing (black circles) or decreasing (red lines) by $\Delta T = 0.005$ or $\Delta \sigma = 0.05$. For the GH model, $x$-axis range is $[0 : 0.3]$, for $\langle k \rangle = 2$, $4$ and $10$, and $[0.2 : 0.5]$ for other $\langle k \rangle$ values. For the KC model, $x$-axis range is $\sigma_p = 0.5$ to $\sigma_p = 2$. $y$-axis range is $AC(1) = 0.25$ to $AC(1) = 1$ for both models. A thick blue dash over the upper $x$-axis marks a $\pm 3\%$ range of the control parameter, about its critical value (or $T_+$ for discontinuous transitions). The boxes remarked with dashed lines correspond to the parameters used in Figs. 2-3-4.

IV. DISCUSSION

Summarizing, we have revisited two simplified models of neuronal activation to show that subtle differences may result in very different collective dynamics when embedded on networks. We found that the KC model dynamical repertoire includes, as a function of its control parameter, only a continuous phase transition being, by design, insensitive to the network topology. This is at odds with the GH model, in which each neuron outcome is influenced by its connectivity degree and therefore by the overall network topology.

We have used the first autocorrelation coefficient $AC(1)$ of the order parameter fluctuations and the presence or absence of hysteresis to identify whether a dynamical transition is present, and to distinguish continuous from discontinuous transitions. This observable is sensitive enough to even being able to tune a system towards criticality [36]. None of the present results depend on the use of the autocorrelation to track the dynamics.
The presence of phase transitions and hysteresis in these models has been studied with other observables, such as the fraction of active sites $f_S$, the variance of activity fluctuations $\sigma^2_f$, or cluster quantities such as the size of the largest or the second largest cluster ($S_1$, or $S_2$) as in [11, 12], yielding similar results. We used $AC(1)$ because its computation is straightforward and easy to replicate, it is almost parameter free, therefore very convenient for comparing the two models.

The key difference between the two models is in the rule that determines how the activity propagates from a given neuron to its connected neighbors. The GH model mimics a discrete integrate-and-fire process taking place in real neurons. There, the “decision” to fire is postsynaptic, based on the amount of total depolarization, on a small patch of membrane, produced by the contribution of hundreds to thousands of impinging neurons (notice the sum in Eq. (1a)). Disregarding the spontaneous activation term, the rule in the GH model is completely deterministic. In fact it is equivalent to a discretized partial differential equation, where the state $S_I(t+1)$ of a post-synaptic neuron $I$ at time $t+1$ depends only on local quantities: it is determined by the previous state of that neuron, $S_I(t)$, the contribution of all other presynaptic neurons $J_j$ to $I_k$ at time $t$, the weights $W_{ij}$ of the connections, and the excitation threshold, i.e.,

$$S_I(t+1) = f[S_I(t), S_J(t), \ldots, S_{J_k}(t), W_{J_i=1,k,t}, T].$$

In contrast, in the KC model the propagation rule is a probabilistic (notice the product Eq. (2a)) contagion-like process [31], where a single excited neuron determines, according to a prescribed value of $\sigma_p$ how many of all of the neurons that connects to will fire next time. Thus, here the decision of how many neurons will be activated is presynaptic: a spiking neuron $I$ excites on average $\sigma_p$ post-synaptic $I$ neurons, independently of the state of the other neurons connected to the same $I$ neuron. Since $\sigma_p$ accounts already for the average out degree of the network, the rule in Eq. (2a) determines, for each $I$ neuron independently the probability that such active neuron will have no-descendants, one or more than one descendants. It is then rather unsurprising that the KC model is insensitive to the network topology.

Although this is not the focus of the present report, it is worth to mention that the KC model rule is biologically implausible. As explained above, the actual activation mechanism of a given neuron involves a myriad of influences (thousands in mammalian brains) over a very small area. In addition, one has to consider that the output of an active neuron, after propagating through its axon from hundreds of microns to millimeters, will stimulate all its contacts roughly equally. Thus, a neuron in order to excite a given number of neighbors would actually have to have information on the number and state (active, refractory or silent) of the post-synaptic neurons and also for other presynaptic neurons attempting to excite the same neuron. This is biologically highly unrealistic, because the neurons involved may be centimeters away from each other, without direct connection among them. Moreover, such hypothetically very well informed $J$ neuron will have to selectively cancel its stimulation strength with certain $I$ neurons, as dictated by the value of $\sigma_p$, a requirement completely impossible for biological neurons.

Of course, the discussion above is not affecting the valuable points made in ref. [15] where the KC model was introduced to show that a network of identical excitable elements achieves maximum dynamic range at criticality. Since the simulations in Ref. [15] where made on fixed topology networks (Erdős-Rényi) the present results are not affecting any of its conclusions which, is worth noticing, were replicated in many other models as well as in experiments. Less clear are the results in other cases, when the KC model was used to study the dynamics of non random topologies. These include the deviations from mean field behavior found in scale free networks, as reported by Copelli & Campos [38] or Mosqueiro & Maia [39]. We note in passing that there are multiple instances in which the KC model was (mis)named as “Greenberg & Hastings stochastic model”, somewhat confusing according to the present results, as in the reports by Copelli & Campos [38], Wu et al., [40], Asis & Copelli [41], Mosqueiro & Maia [39].
& Maia [39], to name only a few.

It is worth to note that the crucial influence of topology on the type of dynamics exhibited by excitable models has been discussed earlier by Kuperman & Abramson [42] in the context of epidemics. They found that the network degree and disorder determines the conditions at which endemic or epidemic situations occur.

To conclude, the point here is not that one needs super-realistic microscopic rules to build a valid model, but that microscopic rules do matter, and may lead to different collective behavior. In particular, we have shown that the KC model exhibits only one type of transition, independent of a large variation in the network topology. The observation that topology has influence on the dynamics of certain models is fundamental at the present time, where several large scale international scientific collaborations are devoted to map and study the consequences of features of the human brain connectome [43–45]. Examples include the numerical simulations using simplified models over derived connectomes, in order to understand brain functioning [10]. However, not all neural models are the same, and special care should be taken on the biases and limitations introduced by the applied models, before drawing conclusions on real brains.

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VI. IMPLEMENTATION OF THE WATTS-STROGATZ NETWORK

In Fortran 90, the code Matrix.f90 is used to generate an undirected Watts-Strogatz network with parameters \( N, \pi, \) and \( \langle k \rangle \) which is saved as a two columns ascii file MyMatrix.txt of length \( N \cdot \langle k \rangle /2 \). Each row has two integer numbers, \( i \) and \( j \), which denote a symmetric edge between nodes \( i \) and \( j \).

For Python, the Watts-Strogatz network is generated using the available code from the networkx package.

VII. IMPLEMENTATION OF THE GREENBERG & HASTINGS (GH) MODEL

A. Fortran 90 code

The dynamics of the GH model, given by Eqs. 1 (a)-(c) of the main text, is implemented by the program GH_model.f90.

The main code reads the file MyMatrix.txt (generated as explained in the previous section), and assigns weights to each connection according to an exponential distribution as explained in the main text. For each value of the control parameter \( T \), it runs the ConstantStep subroutine for a given number of steps (typically 50,000), updating synchronously the network state using the Step subroutine. At the end of the ConstantStep subroutine, it computes the order parameter \( f_S \) and the autocorrelation coefficient \( AC(1) \). At each time step \( t \) the Step subroutine computes the state of all neurons at time \( t + 1 \), \( S(t+1) \), from \( S(t) \). For each neuron \( i \):

1. If the neuron \( i \) was quiescent (i.e., \( S_i(t) = 0 \)), it will become active \( S_i(t+1) = 1 \) with probability \( r_1 \). If that does not happen, a loop over all \( j \) connected to \( i \) is made. For each active \( j \) (i.e., \( S_j(t) = 1 \)), the weight \( W_{ij} \) is summed up. If the sum is larger than \( T \), then \( S_i(t+1) = 1 \).

2. If the neuron was active, (i.e., \( S_i(t) = 1 \)), it will become refractory (i.e., \( S_i(t+1) = 2 \)) always.

3. If the neuron was refractory, (i.e., \( S_i(t) = 2 \)), it will become quiescent (i.e., \( S_i(t+1) = 0 \)) with probability \( r_2 \).

All neurons are updated synchronously.

B. Python 3 code

The code GH_model.py in Python 3 follows the same steps and uses the same names for variables as in the Fortran 90 code. The code requires the following packages: numpy, random and networkx.

The neural activity as a function of time is saved on files named actN(N)K(K)PI(100*pi)T(1000*T).txt, where the value of \( \pi \) is multiplied by 100 and the value of \( T \) is multiplied by 1000, so that they are represented by integer numbers. For instance, for parameters \( N = 5000, \langle k \rangle = 10, \pi = 0.6 \) and \( T = 0.2 \) the output file will be named actN5000K10PI60T200.txt

VIII. IMPLEMENTATION OF THE KINOUCHI & COPELLI (KC) MODEL

In the following we describe the implementation of the code for the KC model used in the main manuscript. Two possible variations of the model are described later in section IX. The KC code follows the same structure as the GH counterpart, starting by reading the file MyMatrix.txt but changing a few relevant parts: the Step subroutine is obviously different and the program cycles trough values of \( \sigma_p \) instead of \( T \).

Here we call \( KC_m \) to the code implementation used for the results described in the main text, which follows Eqs. 2 (a)-(d). The codes are KC_m.f90 and KC_m.py for Fortran 90 and Python 3 respectively.

At each time step \( t \) the Step subroutine computes the state of all neurons at time \( t + 1 \), \( S(t+1) \), from \( S(t) \). For each neuron the Step subroutine updates the states as follows:

1. For each active neuron \( j \) (i.e., \( S_j(t) = 1 \)) a loop over all output connections is made. Each quiescent neuron \( i \) (i.e., \( S_i(t) = 0 \)), will become active with probability \( \sigma_p W_{ji} \).

2. If the neuron \( j \) was quiescent (i.e., \( S_j(t) = 0 \)), it will become active (\( S_j(t+1) = 1 \)) with probability \( r_1 \).

3. If the neuron \( j \) was active, (i.e., \( S_j(t) = 1 \)), it will become refractory (i.e., \( S_j(t+1) = 2 \)) always.

4. If the neuron \( j \) was refractory, in state \( m \), (i.e., \( S_j(t) = m > 1 \)), it will become refractory with state \( m+1 \) (i.e., \( S_j(t+1) = m+1 \)) unless \( m = n \). In that case, if becomes quiescent (i.e., \( S_j(t+1) = 0 \)).

All neurons are updated simultaneously. Within this implementation, it may happen that, in the same time step more than one active neuron activates the same quiescent neuron.
IX. TWO ALTERNATIVE IMPLEMENTATIONS OF THE KC MODEL.

As mentioned in the previous section, we explored some algorithmic alternatives for the implementation of the KC model. We will describe two of them here.

A. Backward update implementation

What we call the backward implementation (see codes KC_b.f90 and KC_b.py) is described also by Eqs. 2 (a)-(d) of the main text. The only subtle difference with $KC_m$ is in the activation loop within the Step subroutine: now it is performed not over the active neurons but over the quiescent neurons. Therefore in this implementation, the Step subroutine is:

1. If neuron $i$ was quiescent (i.e., $S_i(t) = 0$), a loop over all input connections $j$ is made. For each active neuron $j$ (i.e., $S_j(t) = 1$), an attempt to activate $i$ with probability $\sigma_p W_{ji}$ is made.

2. If the neuron $j$ was quiescent (i.e., $S_j(t) = 0$), it will become active ($S_j(t+1) = 1$) with probability $r_1$.

3. If the neuron $j$ was active, (i.e., $S_j(t) = 1$), it will become refractory (i.e., $S_j(t+1) = 2$) always.

4. If the neuron $j$ was refractory, in state $m$ (i.e., $S_j(t) = m > 1$), it will become refractory with state $m+1$ (i.e., $S_j(t+1) = m+1$) unless $m = n$. In that case, if becomes quiescent (i.e., $S_j(t+1) = 0$).

Similar to the $KC_m$ implementation, it may happen that neuron $i$ is simultaneously activated by several $j$'s.

An example of $f_S$ as a function of $\sigma_p$ computed with both $KC_b$ and $KC_m$ implementations in Fortran 90, is shown in Fig. 8 (a). The results are identical, nevertheless, one implementation may be considerably faster, due to the fact that, depending on the case, all or only the active neurons need to be updated. For instance, using gfortran, compiled with -03 flag, the $KC_m$ implementation takes about 2/3 of the time required by the $KC_b$ implementation to reproduce Fig. 8 (a).

B. Local out-degree implementation

We have also explored another implementation of the KC model, we called $KC_l$, by slightly modifying its activation rule. In this case, in each update the activation probabilities of each neuron depends on its out-degree (and the state of its target neurons). Specifically, a given active neuron $j$ counts how many connected quiescent neurons has at time $t$ (i.e., $\sum_{m=1}^{m_{out,j}} \delta_{S_m,0}$), and re-normalizes its contribution among them.

All the other transition rules and parameters are the same as in the $KC_m$ model. In this case the Step routine reads:

1. For each active neuron $j$ (i.e., $S_j(t) = 1$) a loop over all output connections is made. The number of $i$ neurons connected with $j$ that are quiescent is counted and defined as $NC$. Each quiescent neuron $i$, will become active with probability $2\sigma_p W_{i,j}/NC$.

2. If the neuron $j$ was quiescent (i.e., $S_j(t) = 0$), it will become active ($S_j(t+1) = 1$) with probability $r_1$.

3. If the neuron $j$ was active, (i.e., $S_j(t) = 1$), it will become refractory (i.e., $S_j(t+1) = 2$) always.

4. If the neuron $j$ was refractory, in state $m$ (i.e., $S_j(t) = m > 1$), it will become refractory with state $m+1$ (i.e., $S_j(t+1) = m+1$) unless $m = n$. In that case, if becomes quiescent (i.e., $S_j(t+1) = 0$).

$KC_l$ differs from $KC_{m/b}$ in that, in $KC_l$, the probability that a given neuron $j$ activates neuron $i$ depends of its degree (the number of connections of $j$), and the state of all other output connections. This introduces differences that deserve to be further explored. An example of $f_S$ as a function of $\sigma_p$, computed with Fortran 90 codes is shown in Fig. 8 (b). Further results for AC(1) as a function of $\sigma_p$ for $KC_l$ (similar to Fig. 6-(b) on main text), are shown in Fig. 9. Similar to $KC_m$, $KC_l$ model does not present discontinous transitions: for $\langle k \rangle = 2$, there are

![Fig. 8. Comparison of alternative implementations of the KC model.](image-url)
transients to no-activity, while for larger values of $\langle k \rangle$, there are continuous phase transitions. Notice that for this model the $AC(1)$ decays relatively faster for $\sigma_p$ values away from the critical value $\sigma_p = 1$ (compare with Fig. 6 on main text).

![Diagram](https://example.com/diagram.png)

**FIG. 9.** Results obtained with the alternative $KC_l$ described in section IVb. The data shows representative examples of the typical behavior of $AC(1)$ as a function of $\sigma_p$ for diverse topologies. Simulations were run increasing (black circles) or decreasing (red lines) $\sigma_p$ values by $\Delta \sigma = 0.05$ every $5 \cdot 10^4$ time steps. In each box, the $x$-axis range is $\sigma_p = 0.5$ to $\sigma_p = 2$ and the $y$-axis range is $AC(1) = 0.25$ to $AC(1) = 1$.

[1] The Fortran 90 and Python 3 codes are deposited in github (https://github.com/DanielAlejandroMartin/Apparently-Similar-Neuronal).