From Local Chaos to Critical Slowing Down: A Theory of the Functional Connectivity of Small Neural Circuits

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Abstract

Functional connectivity is a fundamental property of neural networks that quantifies the segregation and integration of information between cortical areas. Due to mathematical complexity, a theory that could explain how the parameters of mesoscopic networks composed of a few tens of neurons affect the functional connectivity is still to be formulated. Yet, many interesting problems in neuroscience involve the study of networks composed of a small number of neurons. Based on a recent study of the dynamics of small neural circuits, we combine the analysis of local bifurcations of multi-population neural networks of arbitrary size with the analytical calculation of the functional connectivity. We study the functional connectivity in different regimes, showing that external stimuli cause the network to switch from asynchronous states characterized by weak correlation and low variability (local chaos), to synchronous states characterized by strong correlations and wide temporal fluctuations (critical slowing down). Local chaos typically occurs in large networks, but here we show that it can also be generated by strong stimuli in small neural circuits. On the other side, critical slowing down is expected to occur when the stimulus moves the network close to a local bifurcation. In particular, strongly positive correlations occur at the saddle-node and Andronov-Hopf bifurcations of the network, while strongly negative correlations occur when the network undergoes a spontaneous symmetry-breaking at the branching-point bifurcations. These results prove that the functional connectivity of firing-rate network models is strongly affected by the external stimuli even if the anatomical connections are fixed, and suggest an effective mechanism through which biological networks can dynamically modulate the encoding and integration of sensory information.

Author Summary

Functional connectivity is nowadays one of the most debated topics in neuroscience. It is a measure of the statistical dependencies among neurons, from which we can infer the segregation and integration of information in the nervous system. At the scale of cortical microcolumns, the neural tissue is composed of circuits containing only a few tens of neurons. However, and somewhat counter-intuitively, the functional connectivity of small neural networks can be much more difficult to study mathematically than that of large networks, because as is usual with small numbers, at this scale of spatial organization statistical procedures fail. For this reason, previous studies focused only on the analysis of the functional connectivity in large-scale descriptions of the neural tissue. In this work we introduce a new method for the analysis of the functional connectivity of multi-population neural networks of arbitrary size. In particular, we systematically quantify how the functional connectivity is affected by the external stimuli. Strong inputs drive the network toward asynchronous states where the neurons are functionally disconnected. On the other side, for special sets of stimuli the network becomes increasingly sensitive to external perturbations. There the neural activity becomes synchronous and the network is therefore characterized by strong...
functional integration. This result suggests a possible neurophysiologic mechanism through which sensory stimuli can dynamically modulate the information processing capability of afferent cortical networks.

1 Introduction

The brain is a complex organ with different scales of spatial organization [40]. At the macroscopic scale, its complexity is reflected by the high number of segregated sensory areas that accomplish specialized information processing tasks, such as vision and audition. Then, the sensory areas project to multimodal associative areas, where information is integrated to provide us a coherent representation of the world as we perceive it. This interplay of segregation and integration has been proposed as a possible explanation of human complex behavior [45] and is generally described by the term functional connectivity [36]. Therefore functional connectivity underlies an information flow between and within cortical areas, which at the macroscopic scale is measured by techniques such as fMRI [48]. More generally, Friston defines the functional connectivity as the set of statistical dependencies among neurons or neural populations [19]. According to this definition, the functional connectivity can be evaluated by means of different theoretical measures [14], one of the most used being cross-correlation. This allows us to define the functional connectivity also at the mesoscopic scale, and to record it by means of techniques such as EEG, MEG, ECoG and LFP [25]. The importance of the mesoscopic scale is underlined by its role in shaping human cognitive functions. For example, LFP oscillations are related to perceptual grouping and determine an exchange of information between neighboring and distant cortical columns through neural synchronization [39]. Another example is represented by the columnar mechanisms at the base of attention, which enhance the processing of the relevant information of a complex environment and suppress the unimportant one [24], resulting in a modulation of the functional connectivity of the brain [10].

Developing effective models of functional connectivity is nowadays a central problem in theoretical neuroscience. Recently, the quantification of the functional connectivity in terms of the cross-correlation structure has been considered under different theoretical frameworks [6,7,33,35,47]. An important aspect of the functional connectivity is to understand how it is modulated by the most relevant parameters of the system, in particular the stimulus and the strength of the synaptic connections, since they are the most likely to change over time. In their pioneer work [22], Ginzburg and Sompolinsky developed a theory of correlations for large neural networks described by Wilson-Cowan equations. They proved that neural activity can switch from asynchronous states characterized by weak correlation and low variability to synchronous states characterized by strong correlations and wide temporal fluctuations.

Neurons are said to be in an asynchronous regime when they show uncorrelated activity [15,35,44], while in mathematics a regime characterized by independent (though interacting) units is called local chaos \(^1\). For the sake of clarity, it is important to observe that asynchrony is a weaker condition than local chaos. Indeed, for a general probability distribution, independence does not imply decorrelation. However, the two conditions are equivalent if the neurons are jointly normally distributed, as in the case of the theory we propose in this work. In [22] the authors proved that asynchronous states occur in large networks since the correlations between neurons vanish as \(\frac{1}{N}\), where \(N\) is the size of the network. In a similar way, the emergence of local chaos in large neural networks was proven in [2,3,37,46]. Indeed, independence between interacting units is usually the hypothesis invoked to justify the mean-field approximation of large systems. On the other side, a synchronous regime typically occurs when the network undergoes critical slowing down. Generally this phenomenon happens when a system becomes

\(^1\)Local chaos is known in the kinetic theory of gases as molecular chaos. It was originally introduced by Boltzmann with the name *stosszahlansatz* (collision-number hypothesis) in his studies on the second law of thermodynamics [5]. According to this hypothesis, particles in a gas are statistically independent, even if they interact with each other. However, intuitively after a collision the particles should not be independent anymore since they exchange information. Indeed it can be proven that the inter-particle dependence never vanish during time evolution in a system composed of a finite number of particles and that the Boltzmann’s hypothesis is true only in the ideal limit of infinitely many particles (the so called thermodynamic limit).
increasingly sensitive to external perturbations [26]. In this situation the state variables undergo large and asymmetric fluctuations, with a strong increase of the cross- and auto-correlation functions [28,38]. Critical slowing down usually occurs at the bifurcation points of the system (but not all of them), where small variations of the parameters cause qualitative changes in its dynamics. For example, in [22] the authors showed the formation of critical slowing down in large networks when they approach a saddle-node or an Andronov-Hopf bifurcation, namely before catastrophic transitions or the emergence of oscillatory activity respectively.

Current theories of correlation can be typically applied to networks composed of few thousands of neurons or more, which represent the upper limit of the mesoscopic scale. However, and somewhat counter-intuitively, the cross-correlation structure of small neural networks containing only a few tens of neurons can be much more difficult to study mathematically than that of large networks. This is mainly due to the impossibility to apply the powerful methods of statistical analysis, such as the law of large numbers and the central limit theorem, to small neural circuits. Indeed, these statistical techniques can be typically applied in the limit of large populations of independent neurons. However, in [16] we recently introduced a method for studying the dynamics of neural circuits of arbitrary size, which does not rely on statistical methods. This approach proved effective in describing analytically the local bifurcations of small networks composed of a few tens of neurons such as cortical microcolumns [32], which represent the lower bound of the mesoscopic scale.

In this work we stochastically perturb a generalized version of the deterministic firing-rate network model introduced in [16]. This allows us to develop a theory of the functional connectivity of small neural circuits composed of several populations. Similarly to [22], we find that such networks display both synchronous and asynchronous regimes, with important qualitative and quantitative differences. As in [17], we prove that local chaos may occur also in small networks for strongly depolarizing or strongly hyperpolarizing stimuli. Then, as in [22], we prove the emergence of critical slowing down at the saddle-node and Andronov-Hopf bifurcations of the network, but we extend this result to the case of neural circuits of arbitrary size and with arbitrary correlations between the stochastic perturbations. Moreover, in [16] we found that small networks undergo also special bifurcations known as branching points or pitchfork bifurcations. Branching points correspond to a spontaneous symmetry-breaking of the neural activity. There we observe the spontaneous formation of heterogeneous activity from homogeneous inhibitory neurons without explicit asymmetries in the neural equations. In this work we prove that at these special bifurcation points the activity between inhibitory neurons undergoes critical slowing down characterized by strong anti-correlation: this is a consequence of the broken symmetry of the network, that was not considered in [22].

A systematic analysis of critical slowing down at bifurcations up to codimension two can be found in [28]. Kuehn’s analysis is based on the normal forms of bifurcations, namely simple dynamical systems which are locally equivalent to all systems exhibiting those bifurcations [29]. For this reason, the mathematical analysis in [28] is very general. However, the essential consequences of the theory have not been explicitly formulated in the specific case of neural networks. Therefore quantifying the relation between critical slowing down and the parameters of small neural networks is still an open problem, that we tackle in this work. Our approach is based on linear algebra, not on normal forms, thus it is accessible also to less mathematically minded readers.

The article is organized as follows. In Sec. (2) we introduce the firing-rate network model that we use for the calculation of the functional connectivity (SubSec. (2.1)), and different measures of functional connectivity in terms of the cross-correlation between neurons or neural populations (SubSec. (2.2)). In Sec. (3) we compare analytical and numerical calculations of the cross-correlation in the special case of networks composed of two populations. In more detail, in SubSec. (3.1) we prove the formation of local chaos for strong stimuli, while in SubSec. (3.2) we show the emergence of critical slowing down at the saddle-node, Andronov-Hopf and branching-point bifurcations of the network. To conclude, in Sec. (4) we discuss the importance and the biological implications of our results, while the extension of the theory to
the case of an arbitrary number of neural populations has been developed in the Supplementary Materials.

2 Materials and Methods

In this section we introduce the multi-population network that we study in the article (SubSec. (2.1)). Moreover, we propose different measures of functional connectivity that can be used to compare the theory with the numerical simulations (SubSec. (2.2)).

2.1 The Firing-Rate Network Model

Similarly to [16], we make some assumptions in order to make the network analytically tractable. In particular, we assume that the neurons in each population have homogeneous parameters, that the neurons are all-to-all connected to each other, and that the axonal delays are negligible. Moreover, we describe random fluctuations in the network by means of a white noise component in the external stimuli. Indeed, in humans and many other vertebrates, white noise may be interpreted as an ongoing flux of non-specific excitatory input from the reticular activating system within the brainstem [42].

In more detail, we describe the network by means of the following system of stochastic differential equations:

\[ \frac{dV_i(t)}{dt} = -\frac{1}{\tau_i} V_i(t) + \frac{1}{M_i} \sum_{j=0}^{N-1} J_{ij} \phi_j(V_j(t)) + I_i(t) + \sigma_i \frac{d\mathcal{B}_i(t)}{dt}, \quad i = 0, \ldots, N-1. \]  

Eq. (1) represents the stochastic perturbation to the firing-rate network model discussed in [16]. \( N \) is the number of neurons in the network, \( V_i(t) \) is the membrane potential of the \( i \)th neuron at the time instant \( t \), and \( \tau_i \) is its membrane time constant. The normalization factor \( M_i \) represents the number of incoming connections to the \( i \)th neuron, while \( J_{ij} \) is the weight of the synaptic connection from the \( j \)th (presynaptic) neuron to the \( i \)th (postsynaptic) neuron. \( \phi_j(\cdot) \) is an algebraic activation function which converts the membrane potential \( V \) into the corresponding firing rate \( \nu = \phi(V) \) according to the formula:

\[ \phi_j(V) = \frac{\nu_j^{\text{max}}}{2} \left[ 1 + \frac{\nu_j^{\text{max}} (V - V_j^T)}{\sqrt{1 + \nu_j^{\text{max}} (V - V_j^T)^2}} \right]. \]  

Here \( \nu_j^{\text{max}} \) is the maximum firing rate of the neuron, \( V_j^T \) is the threshold of the activation function, and \( \Lambda_j \) is its slope parameter. The latter represents the “speed” with which the neuron switches between low rates (\( \nu_j \approx 0 \)) and high rates (\( \nu_j \approx \nu_j^{\text{max}} \)). Moreover, in Eq. (1) \( I_i(t) \) is a deterministic external input (i.e. the stimulus) to the \( i \)th neuron, while \( \sigma_i \frac{d\mathcal{B}_i(t)}{dt} \) is a white noise input with normal distribution and standard deviation \( \sigma_i^{\mathcal{B}} \ll 1 \). The functions \( \mathcal{B}_i(t) \) are arbitrarily correlated Brownian motions, which represent the source of stochasticity of the model.

As in [16], in order to make our analysis analytically tractable, we suppose that all the parameters of the system are indexed only at the population level. This means that within a given population the parameters are homogeneous (see [16] for a discussion about the effects of heterogeneity). In other terms, this hypothesis allows us to define a modular network which is composed of an arbitrary number \( \Psi \) of homogeneous neural communities or populations. We define \( N_\alpha \) to be the size of population \( \alpha \) (namely
the number of neurons within that population), with \( \sum_{\alpha=0}^{\mathcal{P}-1} N_\alpha = N \), and we rearrange the neurons so that the structural connectivity of the network can be written as follows:

\[
J = \begin{bmatrix}
\delta_{00} & \delta_{01} & \cdots & \delta_{0,\mathcal{P}-1} \\
\delta_{10} & \delta_{11} & \cdots & \delta_{1,\mathcal{P}-1} \\
\vdots & \vdots & \ddots & \vdots \\
\delta_{\mathcal{P}-1,0} & \delta_{\mathcal{P}-1,1} & \cdots & \delta_{\mathcal{P}-1,\mathcal{P}-1}
\end{bmatrix},
\]

\[
\delta_{\alpha\beta} = \begin{cases} 
J_{\alpha\alpha} (\mathbb{I}_{N_\alpha} - \text{Id}_{N_\alpha}), & \text{for } \alpha = \beta \\
J_{\alpha\beta} \mathbb{I}_{N_\alpha,N_\beta}, & \text{for } \alpha \neq \beta 
\end{cases}
\]  

(3)

for \( \alpha, \beta = 0, \ldots, \mathcal{P} - 1 \). The real numbers \( J_{\alpha\beta} \) are free parameters that describe the strength of the synaptic connections from the population \( \beta \) to the population \( \alpha \). We have \( J_{\alpha\beta} \geq 0 \ \forall \alpha \) if the population \( \beta \) is excitatory, and \( J_{\alpha\beta} \leq 0 \ \forall \alpha \) if it is inhibitory. Moreover, \( \mathbb{I}_{N_\alpha,N_\beta} \) is the \( N_\alpha \times N_\beta \) all-ones matrix (here we use the simplified notation \( \mathbb{I}_{N_\alpha} \equiv \mathbb{I}_{N_\alpha,N_\alpha} \)), while \( \text{Id}_{N_\alpha} \) is the \( N_\alpha \times N_\alpha \) identity matrix. From our assumption on the indexes, we also obtain that the external input currents are organized into \( \mathcal{P} \) vectors \( I_\alpha \), one for each population, and such that:

\[
I_\alpha (t) = I_\alpha (t) \mathbf{1}_{N_\alpha},
\]

where \( \mathbf{1}_{N_\alpha} \equiv \mathbb{I}_{N_\alpha,1} \) is the \( N_\alpha \times 1 \) all-ones vector. The same subdivision between populations is performed for the parameters \( M, \tau, \nu^{\max}, \Lambda, V_T \).

We also suppose that the correlation structure of the white noise \( \frac{dR(t)}{dt} \) is given by the matrix

\[
\Sigma^R = \left[ \Sigma^R_{\alpha\beta} \right]_{\forall (\alpha, \beta)},
\]

where

\[
\Sigma^R_{\alpha\beta} = \begin{cases} 
(\sigma^R_{\alpha})^2 \left[ \text{Id}_{N_\alpha} + C^R_{\alpha\alpha} (\mathbb{I}_{N_\alpha} - \text{Id}_{N_\alpha}) \right], & \text{for } \alpha = \beta \\
\sigma^R_{\alpha} \sigma^R_{\beta} C^R_{\alpha\beta} \mathbb{I}_{N_\alpha,N_\beta}, & \text{for } \alpha \neq \beta 
\end{cases}
\]  

(4)

and \( C^R_{\alpha\beta} = C^R_{\beta\alpha} \) since \( \Sigma^R \) must be symmetric in order to be a true covariance matrix. \( \Sigma^R \) determines the correlation structure of the white noise since \( \text{Cov} \left( \frac{dR(t)}{dt}, \frac{dR(s)}{dt} \right) = \left[ \Sigma^R \right]_{ij} \delta (t - s) \).

A cortical column can be thought of as a network of neural masses distributed vertically across layers, and therefore it is composed of several populations of excitatory and inhibitory neurons (see for example [4]). Our theory can be used to study such cortical architectures, but the complexity of the resulting formulas increases considerably with the number of populations. Thus for the sake of example, we focus on the case \( \mathcal{P} = 2 \) with one excitatory (E) and one inhibitory (I) neural population, which is commonly considered a good approximation of a single neural mass [23]. The case of networks with an arbitrary number of populations is considered in the Supplementary Materials. From now on, it is convenient to change slightly the notation, and to consider \( \alpha, \beta = E, I \) rather than \( \alpha, \beta = 0, 1 \) (see Fig. (1)). Since we study the case of two neural populations, we can take advantage of the detailed bifurcation analysis performed in [16] (see also Fig. (2)), which we will use to determine where the functional connectivity undergoes the most interesting variations.
Figure 1: Example of neural network for $\Psi = 2$. The two populations, one excitatory ($E$) and one inhibitory ($I$), are composed of fully-connected neurons.

Table 1: Values of the parameters of the network for $\Psi = 2$. These parameters have been used to generate all the figures in the article, but Fig. (5), where the functional connectivity is evaluated for different values of $C_{\alpha\beta}$. The ratio $N_E/N_I = 4$ reflects the proportion between excitatory and inhibitory neurons in biological circuits (see [31]). Our theory can be applied to networks of arbitrary size, but in the article we consider the case of small networks ($N = 10$ in this example), see text.

2.2 Measures of Functional Connectivity

Cross-correlation is one of the most studied measures of functional connectivity [14]. For simplicity here we focus on pairwise correlations, which can be calculated through the Pearson coefficient formula:

$$
\text{Corr} (V_i (t), V_j (t)) \overset{\text{def}}{=} \frac{\text{Cov} (V_i (t), V_j (t))}{\sqrt{\text{Var} (V_i (t)) \text{Var} (V_j (t))}},
$$

where $\text{Var} (V_i (t)) = \text{Cov} (V_i (t), V_i (t))$. In [17] the authors derived the analytical expression of the covariance of the rate model (1) for a generic connectivity matrix $J$. This formula reads:

$$
\text{Cov} (V_i (t), V_j (t)) = \sum_{k=0}^{N-1} \left( \sigma_k^E \right)^2 \int_0^t \Phi_{ik} (t-s) \Phi_{jk} (t-s) ds,
$$

where $\Phi (t) = e^{Jt}$ is the fundamental matrix of the system at time $t$, while $J$ is its Jacobian matrix (which depends on $J$). However, when applied to our connectivity matrix (see Eq.(3) for $\Psi = 2$), Eqs. (5) + (6) provide a very cumbersome expression of the cross-correlation. Thus, for simplicity, in this article we consider only the limit $t \to +\infty$, even if correlations may be calculated at any finite $t$, if desired. Moreover, in Sec. (3) we will compare the resulting analytical expression with numerical evaluations of the correlation. In particular, the numerical results are obtained by integrating the neural equations (1) with the Euler-Maruyama method, for the values of the parameters reported in Tab. (1). The integration time step is $\Delta t = 0.001$, and the equations are integrated with a Monte Carlo method over $5,000$
Figure 2: Codimension two bifurcation diagram in the $I_E - I_I$ plane for $\Psi = 2$. This diagram was obtained in [16] for the values of the parameters reported in Tab. (1). The blue curves represent the saddle-node bifurcations (LP for short in Figs. (3), (5)) on the primary branch of stationary solutions of Eq. (1), with cusp bifurcations (CP). The red curves correspond to the Andronov-Hopf bifurcations (H for short in Figs. (4), (5)) on the primary branch, which in turn are divided into supercritical (plain) and subcritical (dashed) portions. The supercritical/subcritical portions are bounded by a generalized Hopf bifurcation (GH), and Bogdanov-Takens bifurcations (BT). The latter are the contact points among saddle-node, Andronov-Hopf and homoclinic bifurcation curves on the primary branch (hyperbolic-saddle/saddle-node homoclinic bifurcations are represented by plain/dashed orange curves). Saddle-node on invariant circle bifurcations (SNIC) correspond to the contact points between the saddle-node and the homoclinic curves. GH generates limit point of cycles curves, represented by dark green lines, that collapse into the homoclinic curves. The gray lines represent the torus bifurcations, while the light green dot-dashed curves correspond to the branching-point bifurcations (BP for short in Figs. (3), (4), (5)). The purple curves represent the Andronov-Hopf bifurcations that originate from the secondary branches, which meet the branching-point curves and the other Andronov-Hopf curves at the zero-Hopf bifurcations (ZH). The double-headed black arrows represent the ranges in which we varied the stimuli $I_E, I_I$ in order to study the behavior of the functional connectivity. In more detail, on the horizontal arrow the network switches from local chaos to critical slowing down near a saddle-node bifurcation (see also Fig. (3)). Moreover, on the vertical arrow the network switches from positively correlated activity at the Andronov-Hopf bifurcation curve, to anti-correlated activity in the inhibitory population at the branching-point curve (see also Fig. (4)). Adapted from [16] with permission of the authors.
repetitions of the network dynamics in the temporal interval $t = [0, 30]$. We assume that at $t = 30$ the transient regime of the correlation has already passed (so that correlation has already converged to its equilibrium solution), which is confirmed by the good agreement between the analytical and numerical results. According to [17], in order to compare analytical and numerical approximations of the functional connectivity, the membrane potentials have to stay as close as possible to a given equilibrium point. In order to avoid jumps of the potentials between different equilibria when the network is close to a saddle-node bifurcation, we consider Brownian motions with small standard deviation, namely $\sigma \ll 1, \sigma_I = 10^{-4}$. Moreover, this choice alleviates another numerical issue, as described hereafter. When the network is close to an Andronov-Hopf bifurcation, two eigenvalues are complex conjugate, therefore they give rise to a focus with damped oscillations in the phase portrait. This means that the random fluctuations of the noise move the state of the network from its equilibrium point, causing undesired sustained oscillations whose frequency corresponds to the imaginary part of the eigenvalues of the Jacobian matrix [49]. Only small $\sigma_{E,I}$ prevent the formation of wide oscillations around the equilibrium solution. On the other side, when the network is far from a bifurcation point, we obtain a good agreement between analytical and numerical results also for Brownian motions with larger standard deviations, namely $\sigma_{E,I} \sim 10^{-1}$ (results not shown). For even larger standard deviations, higher-order corrections to our perturbative approach must be considered, but this is beyond the purpose of the article.

Cross-correlation is related to the underlying information flow between neurons by the formula of the mutual information:

$$\mathcal{I}_{ij} (t) \overset{\text{def}}{=} \int_{\mathbb{R}^2} p(V_i, V_j, t) \log \left( \frac{p(V_i, V_j, t)}{p(V_i, t) p(V_j, t)} \right) dV_i dV_j = -\frac{1}{2} \log (1 - \text{Corr}^2 (V_i (t), V_j (t))) ,$$  \hspace{1cm} (7)

where $p(V_i, V_j, t)$ is the 2-neurons joint probability density at time $t$, while $p(V_i, t) = \int_{-\infty}^{+\infty} p(V_i, V_j, t) dV_j$ is the corresponding 1-neuron density. We observe that the last identity in Eq. (7) holds only for normal probability distributions. This is indeed our case, since we are going to adopt a linear approximation of Eq. (1), which is justified by our assumption $\sigma \ll 1$. Eq. (7) shows that the mutual information $\mathcal{I}_{ij} (t)$ depends trivially on the pairwise correlation between neurons. In particular, $\text{Corr} (V_i (t), V_j (t)) \to 0$ implies $\mathcal{I}_{ij} (t) \to 0$ (functional disconnection), while $\text{Corr} (V_i (t), V_j (t)) \to \pm 1$ implies $\mathcal{I}_{ij} (t) \to \infty$ (functional integration).

However, neuroscientists make use of measures of correlation between firing rates, rather than between membrane potentials. This is because only spiking events are transmitted to other neurons, while subthreshold membrane fluctuations are not. Since in our model the firing rates are given by the relation $\nu = \mathcal{A} (V)$, we get :

$$\text{Corr} (\nu_i (t), \nu_j (t)) \approx \text{Corr} (V_i (t), V_j (t)) ,$$  \hspace{1cm} (8)

under the assumption $\sigma \ll 1$, as proven in [17]. Still, the network is described by voltage-based equations (see (1)), thus it is more natural to study the correlation structure between the membrane potentials and to get that between the firing rates accordingly.

Furthermore, it is also possible to calculate the cross-correlation between mesoscopic quantities. For example, we introduce the neural activity of a group of neurons [21]:

$$a_g (t) \overset{\text{def}}{=} \frac{1}{N_g} \sum_{i \in g} \nu_i (t) ,$$
where the sum is over all the neurons in a given group $G$ of size $N_G$. The neural activity can be interpreted intuitively by observing that, in the limit of large $\Lambda$ (see Eq. (2)), $a_G(t)$ corresponds to the fraction of firing neurons in $G$. Indeed, $\lim_{\Lambda \to \infty} a_G(V) = \nu_{\text{max}} H(V - V^T)$, where $H(\cdot)$ is the Heaviside step function. Therefore $\lim_{\Lambda \to \infty} a_G(t) = \frac{\nu_{\text{max}}}{N_G} N_{\text{fire}}(t)$, where $N_{\text{fire}}(t) = \sum_{i \in G} H(V - V^T)$ is the number of firing neurons (i.e. such that $V > V^T$) in $G$ at the time instant $t$. The correlation between the activities of two neural groups $G$ and $H$ is:

$$\text{Corr}(a_G(t), a_H(t)) = \frac{\sum_{i \in G, j \in H} \text{Cov}(\nu_i(t), \nu_j(t))}{\sqrt{\left[\sum_{i \in G} \text{Cov}(\nu_i(t), \nu_i(t))\right] \left[\sum_{i \in H} \text{Cov}(\nu_i(t), \nu_j(t))\right]}}$$

(9)

where we defined $\text{Corr}(G, H) \equiv \text{Corr}(\nu_i(t), \nu_j(t))|_{i \in G, j \in H}$ and $\text{Corr}(G, G) \equiv \text{Corr}(\nu_i(t), \nu_j(t))|_{i, j \in G, i \neq j}$ (similarly for $H$). The last equality of Eq. (9) holds only if $G$ is a subset of a neural population where spontaneous symmetry-breaking did not occur, and the same for $H$ (so for example if the neurons in $G$ are excitatory while those in $H$ are inhibitory, in the case $\mathcal{P} = 2$ with weak inhibition). Therefore from Eqs. (8) + (9) we observe that also the correlation between population activities can be expressed in terms of the correlation between the corresponding membrane potentials.

### 3 Results

In this section we study the functional connectivity of the firing-rate network model introduced in Sub-Sec. (2.1). For simplicity we consider only the case of two neural populations, while the theory for an arbitrary number of populations is developed in the Supplementary Materials. According to Eq. (6), the functional connectivity depends on the fundamental matrix of the network, $\Phi(t) = e^{\mathcal{J}t}$. In the Supplementary Materials (see Eq. (S27)) we calculated $\Phi(t)$ in terms of the eigenvalues of the Jacobian matrix $\mathcal{J}$:

$$\lambda_E = -\left[\frac{1}{\tau_E} + \frac{J_{EE}^{ij} a_E^j(\mu_E)}{M_E}\right], \quad \lambda_I = -\left[\frac{1}{\tau_I} + \frac{J_{II}^{ij} a_I^j(\mu_I)}{M_I}\right], \quad \lambda_{0,1}^R = \frac{\gamma + Z \pm \sqrt{(Y - Z)^2 + 4X}}{2},$$

(10)

where:

$$X = \frac{N_E N_I}{M_E M_I} J_{EI} J_{EE} a_E^j(\mu_E) a_I^j(\mu_I), \quad Y = -\frac{1}{\tau_E} + \frac{N_E - 1}{M_E} J_{EE} a_E^j(\mu_E), \quad Z = -\frac{1}{\tau_I} + \frac{N_I - 1}{M_I} J_{II} a_I^j(\mu_I),$$

(11)

and in terms of the functions:

$$K_\alpha \equiv \frac{M_E \left(\lambda_{0,1}^R + \frac{1}{\tau_E}\right) - (N_E - 1) J_{EE} a_E^j(\mu_E)}{N_I J_{IE} a_I^j(\mu_I)} = \frac{N_E J_{IE} a_E^j(\mu_E)}{M_I \left(\lambda_{0,1}^R + \frac{1}{\tau_I}\right) - (N_I - 1) J_{II} a_I^j(\mu_I)}, \quad \alpha = 0, 1.$$ 

(12)
While we switched to the new notation \( \alpha = E, I \), we keep using \( \alpha = 0, 1 \) for the eigenvalues \( \lambda_{\alpha}^R \) (and the functions \( K_\alpha \)) because, differently from \( \lambda_{E,I} \), they depend on the parameters of both the populations. If the parameters of the network are such that \( \lambda_{E,I} \) and \( \lambda_{0,1}^R \) have negative real part, by applying Eqs. (6) + (S27) we end up with the following formula of the covariance matrix of the membrane potentials \( \Sigma^V \overset{\text{def}}{=} \lim_{t \to +\infty} \text{Cov}(V_i(t), V_j(t)) \)|_{\(\alpha i,j\)}:

\[
\Sigma^V = \begin{bmatrix} \Sigma^V_{EE} & \Sigma^V_{EI} \\ \Sigma^V_{IE} & \Sigma^V_{II} \end{bmatrix},
\]

where \( T \) denotes the transpose of a matrix, while the blocks \( \Sigma^V_{\alpha\beta} \) are given by the following formulas:

\[
\Sigma^V_{\alpha\alpha} = (\sigma^V_{\alpha})^2 \text{Id}_{N_\alpha} + \zeta^V_{\alpha\alpha} (t_{N_\alpha} - \text{Id}_{N_\alpha}), \quad \Sigma^V_{E,I} = \zeta^V_{E,I} \Sigma^V_{NE,NI}
\]

\[
(\sigma^V_{E})^2 = (\sigma^2_E)^2 \left\{ \Upsilon^E_{EE} \left[ \frac{1}{N_E} + C^R_{EE} \left( \frac{1}{N_E} \right) \right] - \frac{1}{2\lambda_E} \left( 1 - \frac{1}{N_E} \right) \left( 1 - C^R_{EE} \right) \right\}
\]

\[
+ (\sigma^2_I)^2 \Upsilon^I_{EI} \left[ \frac{1}{N_I} + C^R_{II} \left( \frac{1}{N_I} \right) \right] + 2\sigma^2_E \sigma^2_I \Upsilon^E_{EE} C^R_{EI}
\]

\[
(\sigma^V_{I})^2 = (\sigma^2_I)^2 \Upsilon^I_{II} \left[ \frac{1}{N_I} + C^R_{II} \left( \frac{1}{N_I} \right) \right]
\]

\[
+ (\sigma^2_I)^2 \left\{ \Upsilon^I_{EI} \left[ \frac{1}{N_I} + C^R_{II} \left( \frac{1}{N_I} \right) \right] - \frac{1}{2\lambda_I} \left( 1 - \frac{1}{N_I} \right) \left( 1 - C^R_{II} \right) \right\}
\]

\[
+ 2\sigma^2_E \sigma^2_I \Upsilon^E_{EE} C^R_{EI}
\]

\[
\zeta^V_{EE} = (\sigma^2_E)^2 \left\{ \Upsilon^E_{EE} \left[ \frac{1}{N_E} + C^R_{EE} \left( \frac{1}{N_E} \right) \right] + \frac{1}{2\lambda_E N_E} \left( 1 - C^R_{EE} \right) \right\}
\]

\[
+ (\sigma^2_I)^2 \Upsilon^I_{EE} \left[ \frac{1}{N_I} + C^R_{II} \left( \frac{1}{N_I} \right) \right] + 2\sigma^2_E \sigma^2_I \Upsilon^E_{EE} C^R_{EI}
\]

\[
\zeta^V_{II} = (\sigma^2_I)^2 \Upsilon^I_{II} \left[ \frac{1}{N_I} + C^R_{II} \left( \frac{1}{N_I} \right) \right]
\]

\[
+ (\sigma^2_I)^2 \left\{ \Upsilon^I_{EI} \left[ \frac{1}{N_I} + C^R_{II} \left( \frac{1}{N_I} \right) \right] + \frac{1}{2\lambda_I N_I} \left( 1 - C^R_{II} \right) \right\} + 2\sigma^2_E \sigma^2_I \Upsilon^E_{EE} C^R_{EI}
\]

\[
\zeta^V_{EI} = (\sigma^2_I)^2 \Upsilon^I_{EI} \left[ \frac{1}{N_I} + C^R_{II} \left( \frac{1}{N_I} \right) \right]
\]

\[
+ (\sigma^2_I)^2 \Upsilon^I_{EI} \left[ \frac{1}{N_I} + C^R_{II} \left( \frac{1}{N_I} \right) \right] + 2\sigma^2_E \sigma^2_I \Upsilon^E_{EE} C^R_{EI}
\]

The functions \( \Upsilon \) are defined as below:
\[
\begin{align*}
\gamma_{EE}^{EE} &= \frac{1}{(K_1 - K_0)^2} \left[ \frac{2K_0K_1}{\lambda_0^R + \lambda_1^R} - \frac{1}{2} \left( \frac{K_0^2}{\lambda_0^R} + \frac{K_1^2}{\lambda_1^R} \right) \right], \quad \gamma_{EE}^{II} &= \frac{1}{(K_1 - K_0)^2} \left[ \frac{2K_0K_1}{\lambda_0^R + \lambda_1^R} - \frac{1}{2} \left( \frac{1}{\lambda_0^R} + \frac{1}{\lambda_1^R} \right) \right], \\
\gamma_{II}^{EE} &= \frac{K_0^2K_1^2}{(K_1 - K_0)^2} \left[ \frac{2}{\lambda_0^R + \lambda_1^R} - \frac{1}{2} \left( \frac{1}{\lambda_0^R} + \frac{1}{\lambda_1^R} \right) \right], \quad \gamma_{II}^{II} = \frac{1}{(K_1 - K_0)^2} \left[ \frac{2K_0K_1}{\lambda_0^R + \lambda_1^R} - \frac{1}{2} \left( \frac{K_0^2}{\lambda_0^R} + \frac{K_1^2}{\lambda_1^R} \right) \right], \\
\gamma_{EE}^{EI} &= \frac{K_0K_1}{(K_1 - K_0)^2} \left[ \frac{K_0 + K_1}{\lambda_0^R + \lambda_1^R} - \frac{1}{2} \left( \frac{K_0}{\lambda_0^R} + \frac{K_1}{\lambda_1^R} \right) \right], \quad \gamma_{II}^{EI} = \frac{1}{(K_1 - K_0)^2} \left[ \frac{K_0 + K_1}{\lambda_0^R + \lambda_1^R} - \frac{1}{2} \left( \frac{K_0}{\lambda_0^R} + \frac{K_1}{\lambda_1^R} \right) \right], \\
\gamma_{EI}^{EE} &= \frac{1}{(K_1 - K_0)^2} \left[ K_0K_1 \left( \frac{1}{\lambda_0^R} + \frac{1}{\lambda_0^R} \right) - \frac{2}{\lambda_0^R + \lambda_1^R} \right] - \frac{K_0^2 + K_1^2}{\lambda_0^R + \lambda_1^R}. 
\end{align*}
\]

(15)

\(\sigma^V_\alpha\) is the standard deviation of the neurons in the population \(\alpha\), while \(\varsigma^V_{\alpha\beta}\) is the covariance between any pair of neurons in the same population \(\alpha\), and \(\varsigma^V_{EI}\) is the covariance between any pair of neurons in two different populations.

Finally, by replacing these results into the formula \(C^V_{\alpha\beta} \text{def} \frac{\varsigma^V_{\alpha\beta}}{\sigma^V_\alpha \sigma^V_\beta}\) obtained from Eq. (5), we get an expression of the functional connectivity \(C^V = \left[ C^V_{\alpha\beta} \right]_{\alpha\beta} \) of the network. From this formula we will prove that, depending on the values of the parameters, the network can switch from local chaos to critical slowing down. These special regimes have important and contrasting properties, which will be discussed in detail in SubSecs. (3.1) and (3.2).

### 3.1 Local Chaos

Local chaos is the condition that characterizes asynchronous neural states. Its most important features are small amplitude temporal fluctuations of the membrane potentials, as well as weak cross-correlations between them. Local chaos can be generated in two different ways. Probably the most known is the increase of the network’s size [2, 37, 46]. Indeed, for \(N = 2\), if \(C^E_{EE} = C^E_{II} = C^E_{EI} = 0\), from Eqs. (14) + (15) we observe that \(\varsigma^V_{\alpha\beta} \to 0\) and \((\sigma^V_\alpha)^2 \to \left( -\frac{1}{\lambda_0^R} \right) (\sigma^V_\alpha)^2 \approx \frac{1}{\lambda_0^R} (\sigma^V_\alpha)^2\) for \(\alpha, \beta \in \{E, I\}\) in the thermodynamic limit \(N_{E,I} \to \infty\). In other words, in infinite-size networks with independent Brownian motions, the membrane potentials are independent too, leading to local chaos. Local chaos is usually invoked to justify the mean-field description of large neural networks and is compatible with recent findings in visual cortex [15, 35, 44].

Interestingly, also finite-size networks can experience decorrelated activity. In [17] the authors showed that, for any \(N\), weak correlations occur for strongly depolarizing or strongly hyperpolarizing external inputs, if the Brownian motions are independent. This phenomenon can be proven for the two-populations case as a consequence of \(T_{\alpha\alpha}^{\alpha\alpha} \to \frac{-1}{\lambda_0^R}\), \(T_{\alpha\beta}^{\alpha\beta} \to 0\) (with \(\alpha \neq \beta\)) and \(T_{EI}^{\alpha\beta} \to 0\) for \(|I_{E,I}| \to \infty\), which in turn is due to the saturation of the activation function \(\mathcal{A}(V)\). For the same reason, the standard deviations \(\sigma^V_{E,I}\) of the neural activity in the two populations decrease with the input. Interestingly, the reduction of both the correlation and the variance of the neural responses is supported by experimental evidence [34, 43]. Fig. (3) shows an example of formation of local chaos in a finite-size network for \(N = 2\), which is obtained for the values of the parameters in Tab. (1) and for strong stimuli \((I_E > 13, I_I = -35)\).
Figure 3: Transition between local chaos and critical slowing down near a saddle-node bifurcation. The top panels show a good agreement between the numerical approximations of the standard deviation and correlation (left and right panel respectively), and the corresponding analytical formulas (see Eqs. (14) + (15)). The numerical approximations have been obtained by integrating the neural equations (1) with the Euler-Maruyama method, for the values of the parameters reported in Tab. (1) and $I_I = -35$. The integration time step is $\Delta t = 0.001$, and the equations are integrated with a Monte Carlo method over 5,000 repetitions of the network dynamics in the temporal interval $t = [0, 30]$. For large inputs ($I_E > 13$) we observe the formation of local chaos, which is characterized by weak correlation and low variability. On the other side, near a saddle-node bifurcation ($I_E \approx 11.86$, see the highlighted LP), we observe strong correlations and wide temporal fluctuations that characterize critical slowing down. The bottom panels show numerical simulations of the fluctuations of the membrane potentials in the excitatory and inhibitory population (left and right panel respectively), calculated at $t = 30$ for different values of $I_E$ and superposed to the codimension one bifurcation diagram of the network. The fluctuations are displayed at $3,000 \times$ actual size in the excitatory and inhibitory population, in order to make them visible on the bifurcation diagrams. The reader may verify the agreement between the standard deviations (top-left panel) and the envelope of the fluctuations of the membrane potentials.
We observe that local chaos always requires independent Brownian motions to occur. However, the theory developed in this article can also be applied to networks with correlated noise. In [17] the authors observed that if the Brownian motions are correlated, local chaos does not occur anymore, neither in large network nor for strong stimuli. The same result is obtained from Eqs. (14) + (15). In particular, since \( Y_{EI} \rightarrow 0 \) and \( Y_{EI} \rightarrow \frac{1}{2\sqrt{\lambda I}} \) for \( |I_{EI}| \rightarrow \infty \), we get \( \sigma^V_\alpha \rightarrow \sigma^B_\alpha \) and \( C^V_{\alpha\beta} \rightarrow C^B_{\alpha\beta} \) (see also Fig. (5), where we plot the functional connectivity for different values of \( C^B_{\alpha\beta} \)). In other words, for strong stimuli the correlation between the membrane potentials converges to that between the Brownian motions.

3.2 Critical Slowing Down

Critical slowing down is the condition that characterizes synchronous neural states. Contrary to local chaos, its most important features are large temporal fluctuations of the membrane potentials and strong cross-correlations. Critical slowing down typically occurs at the bifurcation points of the system. In [16] we performed a detailed bifurcation analysis in the case \( \Psi = 2 \) and for the values of the parameters in Tab. (1), obtaining the entangled set of local and global bifurcations shown in Fig. (2). Local bifurcations occur when a parameter variation causes the stability of an equilibrium point to change, therefore they are studied through the eigenvalues of the Jacobian matrix. Local bifurcations can be of codimension one or two, depending on the number of parameters (i.e. \( I_{EI} \)) that must be changed for the bifurcation to occur. As shown in Fig. (2), the local bifurcations of codimension one the network undergoes are saddle-node, Andronov-Hopf and branching-point bifurcations, while those of codimension two are cusp, Bogdanov-Takens, generalized Hopf and zero-Hopf bifurcations. The remaining bifurcations are global, which means they cannot be studied through a local analysis in terms of the equilibrium points, but rather they require the analysis of (a part of) the phase portrait of the network. In particular, the homoclinic, limit point of cycles, and torus \(^2\) curves, are global bifurcations of codimension one, while saddle-node on invariant circle curves represent the only global bifurcations of codimension two. As discussed in [16], for \( N_I > 2 \) other kinds of local bifurcations of codimension two and global bifurcations may occur due to spontaneous-symmetry breaking. Nevertheless, for simplicity, in this article we restrict our discussion to the case \( N_I = 2 \) when \( \Psi = 2 \).

Similarly to [28], we study the behavior of the functional connectivity only at the local bifurcations of the network, and in particular we consider only those of codimension one. These bifurcations are studied in SubSecs. (3.2.1), (3.2.2), (3.2.3) for the case \( \Psi = 2 \) and in Sec. (S6) of the Supplementary Materials for the case of an arbitrary \( \Psi \). Our theory can also be used to study the behavior of the functional connectivity near local bifurcations of codimension two, but due to the high variety of the bifurcations the system exhibits, a complete study is beyond the purpose of this article.

Finally, to our knowledge no analytical method is known for studying the global bifurcations of Eq. (1). Currently the correlation at the global bifurcations can be studied only numerically, but this analysis again is beyond the purpose of the article.

3.2.1 Saddle-Node Bifurcations

Saddle-node bifurcations are tipping points at which tiny perturbations can cause an abrupt and discontinuous change of the equilibrium point of the system. These bifurcations have been proposed to occur in a set of dynamical systems such as ocean-climate systems, financial markets, ecosystems etc [38]. In neuroscience, phenomena compatible with the presence of saddle-node bifurcations in the cortex have been

\(^2\)More precisely, the torus bifurcation is a local bifurcation of the Poincaré map of a limit cycle of the network [29]. For this reason the torus bifurcation corresponds to a change of stability of the fixed points of the Poincaré map, and not to a change of stability of the equilibrium points of Eq. (1). For this reason the torus bifurcation cannot be studied through the eigenvalues of the Jacobian matrix of the network.

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observed for example during anesthetic administration at the edge between conscious and unconscious states [42].

In [16] we proved that, in the case $\mathcal{P} = 2$, the network undergoes a saddle-node bifurcation whenever one of the eigenvalues $\lambda^R_{0,1}$ in Eq. (10) tends to zero. The saddle-node bifurcations are described by the blue curves in Fig. (2). In [16] we also proved that a necessary condition for the formation of these bifurcations is:

$$\frac{N_E - 1}{N - 1} J_{EE} \frac{\nu_E^{\max} \Lambda_E}{4} \tau_E > 1,$$

(16)

or in other words sufficiently strong self-excitatory weights are required. From Eq. (15) we observe that for $\lambda^R_0 \to 0^-$ or $\lambda^R_1 \to 0^-$ the functions $\Upsilon$ diverge, therefore the terms proportional to $\frac{1}{\lambda^R_0}$ in Eq. (14) become negligible. This implies $\zeta^V_{\alpha} \sim (\sigma^V_{\alpha})^2 \to \infty$ and $\zeta^V_{EI} \sim \sigma^E \sigma^I$, therefore $C_{\alpha\beta}^V \sim 1$ between every population. Thus, when the network is close to a saddle-node bifurcation, we observe the emergence of critical slowing down. Moreover, we obtain a simple relation between the variances of the two neural populations, namely $\sigma^V_E \sim K \sigma^V_I$, where $K \equiv \lim_{\lambda^R_0 \to 0^-} K_0 = \lim_{\lambda^R_1 \to 0^-} K_1 = \frac{N_E J_{EE} \sigma^E_0 (\mu_E)}{\nu^2 - (N_I - 1) J_{II} \sigma^I_1 (\mu_I)}$.

The reader can also verify that $\zeta^V_{EI} > 0$ for $\lambda^R_0 \to 0$ as a consequence of $K > 0$, which in turn is due to $J_{EE} > 0$ and $J_{II} < 0$. An example of critical slowing down obtained for $I_E \approx 11.86, I_I = -35$ and $C_{EE}^I = C_{II}^I = C_{EI}^I = 0$ is reported in Fig. (3). We observe that this phenomenon occurs even if there is no correlation between the Brownian motions (i.e. $C_{\alpha\beta}^I = 0$), therefore it is entirely a consequence of the neural interactions mediated by the synaptic connections.

To conclude, from Eqs. (8) + (9) we observe that $\text{Corr} (V_i (t), V_j (t)) \to 1$ for every pair of neurons implies $\text{Corr} (a_G (t), a_H (t)) \to 1$. Therefore the populations become functionally connected also in terms of their neural activities.

### 3.2.2 Andronov-Hopf Bifurcations

Andronov-Hopf bifurcations correspond to the emergence of neural oscillations, which are thought to play a key role in many cognitive processes [50]. In [16] we proved that, in the case $\mathcal{P} = 2$, the network undergoes an Andronov-Hopf bifurcation whenever $\lambda^R_{0,1}$ in Eq. (10) are complex-conjugate purely imaginary. The Andronov-Hopf bifurcations are described by the red curves in Fig. (2). In [16] we also proved that a necessary condition for the formation of these bifurcations is:

$$\frac{\nu_E^{\max} \Lambda_E}{4i} > 1 \quad \text{and} \quad b^2 - 4ac > 0$$

(17)

where:
\[ j = \frac{-b - \sqrt{b^2 - 4ac}}{2a} \]

\[ a = \left( \frac{N_E - 1}{N - 1} J_{EE} \right)^2 - \frac{N_E N_I (N_E - 1) J_{EE} J_{EI} J_{IE}}{(N - 1)^2 (N_I - 1) J_{II}} \]

\[ b = -\frac{2}{\tau_E} \frac{N_E - 1}{N - 1} J_{EE} + \frac{N_E N_I}{(N - 1) (N_I - 1)} J_{EI} J_{IE} \left( \frac{1}{\tau_E} + \frac{1}{\tau_I} \right) \]

\[ c = \frac{1}{\tau_E} \]

Whenever the network approaches an Andronov-Hopf bifurcation, we get \( \lambda_0^I + \lambda_1^I \rightarrow 0^- \), which causes the terms \( \Upsilon \) to diverge (see Eq. (15)). For this reason the variance of the membrane potentials diverges as well, while the cross-correlation tends to one, similarly to the case of the saddle-node bifurcations. This proves that the network undergoes critical slowing down also at the Andronov-Hopf bifurcations. An example obtained for \( I_E = 1, I_I \approx -13.67 \) and \( C_{EE}^I = C_{II}^I = C_{EI}^I = 0 \) is shown in Fig. (4).

### 3.2.3 Branching-Point Bifurcations

In the deterministic model (i.e. for \( \sigma_{E,I}^\beta = 0 \)), according to the assumptions of Sec. (2.1), within each population the neurons are dynamically identical, namely the populations are homogeneous. This means that, in absence of noise, the network has the symmetry \( \times \sigma^\alpha \), where \( \sigma^\alpha \) is the permutation group on \( N_\alpha \) items (also known as symmetric group). When we turn on the noise (\( \sigma_{E,I}^\beta > 0 \)), we introduce a small explicit symmetry-breaking into Eq. (1). However, the behavior of a nearly symmetric dynamical system is more similar to that of an idealized symmetric system than that of a completely asymmetric one [41]. Therefore it is legitimate to study Eq. (1) as a perturbation of the corresponding deterministic system, if the degree of explicit heterogeneity introduced by the noise is not too strong. However, symmetry-breaking may occur also in the deterministic model. Indeed, at the branching-point bifurcations we observe the formation of a spontaneous symmetry-breaking [16], because some of the neurons within a given inhibitory population become dynamically distinct from the others. In other words, we observe the formation of an heterogeneous inhibitory population, even if the neural equations (1) for \( \sigma_{E,I}^\beta = 0 \) do not contain any term that breaks explicitly the symmetry. Interestingly, this phenomenon is a consequence of the finite size of the network, therefore it does not occur in the thermodynamic limit [16].

In [16] we also proved that, in the case \( \mathfrak{F} = 2 \), a branching-point bifurcation occurs whenever \( \lambda_I = 0 \) (see the light green dot-dashed curves in Fig. (2)) and that a necessary condition for their formation is:

\[ \frac{\tau_I |J_{II}| \nu_{\max}^I \lambda_I}{4(N-1)} > 1. \] (18)

This means that sufficiently strong self-inhibitory weights are required for the bifurcation to occur. According to Eq. (14), for \( C_{II}^\beta < 1 \) and \( \lambda_I \rightarrow 0^- \) only the variance of the inhibitory neurons diverges. As a consequence, in the case \( C_{II}^\beta < 1 \) we get \( (\sigma_I^V)^2 \sim (\sigma_I^\beta)^2 \left[ -\frac{1}{2N_I} \left( 1 - \frac{1}{N_I} \right) (1 - C_{II}^\beta) \right] \) and \( \nu_{II}^V \sim (\sigma_I^\beta)^2 \left[ \frac{1}{2N_I N_T} (1 - C_{II}^\beta) \right] \). From which we conclude that \( C_{II}^V \sim \frac{1}{1 - \nu_{II}^V} \). According to [17], this is the
Figure 4: Fluctuations and cross-correlations of the membrane potentials between Andronov-Hopf and branching-point bifurcations. The simulations are similar to those of Fig. (3), but now we set $I_E = 1$ and we vary the input to the inhibitory population, obtaining a transition between an Andronov-Hopf bifurcation ($I_I \approx -13.67$, see the highlighted H) and a branching-point bifurcation ($I_I \approx 1.165$, highlighted BP). We obtain a good agreement between numerical and analytical correlations for any current $I_I$ in the range, while the standard deviations display a good agreement only when $I_I$ is sufficiently far from the bifurcation points. At the Andronov-Hopf and branching-point bifurcations the standard deviations predicted by the analytical formulas are larger than those obtained numerically. This suggests that generally second-order corrections to Eqs. (14) + (15) play a stronger role when the network undergoes a local bifurcation. Nevertheless, the first-order approximation describes qualitatively the increase of the standard deviation that characterizes critical slowing down.
Figure 5: Fluctuations and cross-correlations of the membrane potentials as a function of the input and of the noise correlation. The top panels show the standard deviation (left) and the cross-correlation (right) of the membrane potentials when the network is close to a saddle-node bifurcation (similarly to Fig. (3)), for different values of the noise correlation. The curves have been obtained from Eqs. (14) + (15) for $C_{EE} = C_{II} = C_{EI} = 0, 0.2, 0.4, 0.6, 0.8, 0.97, 1$. The panels show that the noise correlation increases both the standard deviation and the cross-correlation, for $\sigma_{E,I}^2$ fixed (see Tab. (1)). The bottom panels show similar results for the neural states between Andronov-Hopf and branching-point bifurcations (compare with Fig. (4)). The only difference is observed close to the branching-point bifurcation, where $\sigma_{E,I}^2$ decrease with the noise correlation.
lower bound of the correlation between fully-connected neurons in a homogeneous population with size $N_I$. Since $\frac{1}{1-N_I} < 0$ for $N_I \geq 2$, at the branching-point bifurcations the inhibitory neurons are maximally anti-correlated. Moreover, according to this formula, correlation tends to $-1$ only for $N_I = 2$. Therefore we conclude that, contrary to the saddle-node and Andronov-Hopf bifurcations, at the branching points critical slowing down occurs only in the inhibitory population. This result is confirmed by Fig. (4), which shows an example obtained for $I_E = 1$, $I_I \approx 1.165$ and $C_{EE}^\theta = C_{EI}^\theta = C_{II}^\theta = 0$. Intuitively, the membrane potentials become anti-correlated because the inhibitory neurons follow different branches of stationary solutions beyond the branching-point (see the codimension one bifurcation diagram in the bottom-right panel of Fig. (4)). Therefore while the potential of one neuron increases due to noise fluctuations, the potential of the other neuron decreases and viceversa, resulting in a negative correlation.

On the other side, for $C_{II}^\theta = 1$ and $\lambda_I \to 0^-$, from Eq. (14) we get:

$$\left(\sigma_f^V\right)^2 = \sigma_f^V = \left(\sigma_f^E\right)^2 \gamma_{II}^E \left[1 - \frac{1}{N_E} \right] + \left(\sigma_f^E\right)^2 \gamma_{II}^I + 2\sigma_f^E \sigma_f^I \gamma_{II}^I C_{II}^\theta,$$

therefore now $\left(\sigma_f^V\right)^2$ does not diverge anymore and $C_{II}^\gamma = 1$ (see also Fig. (5)). To conclude, for $C_{II}^\theta = 1$ and $\lambda_I = 0$, Eq. (14) gives an indeterminate form $\frac{0}{0}$ for the variance $\left(\sigma_f^V\right)^2$, which is represented by the empty circles in the bottom panels of Fig. (5). This result can be intuitively interpreted as the consequence of the competition between the positive correlation introduced by the Brownian motions and the anti-correlation generated by the branching point.

4 Discussion

We developed a theory of the functional connectivity of a multi-population firing-rate network model of arbitrary size and with all-to-all topology. In particular, this theory can be used for studying the functional connectivity of small networks composed of a few tens of neurons, such as cortical microcolumns in mammals [32] and neural circuits in some invertebrates [52]. Our study relies on the methods introduced in [16, 17], which are not based on statistical averages. For this reason, our theory can be applied to networks of arbitrary size $N$, and is not restricted to large networks as in previous works on neural systems [6, 7, 22, 35].

The model we introduced is largely analytically tractable, and allowed us to derive explicit expressions for the functional connectivity of the network in terms of the cross-correlations between neurons or neural populations. Then we studied the behavior of the functional connectivity in terms of the stimuli $I_a$, and this analysis revealed the ability of the network to switch dynamically from asynchronous regimes characterized by weak correlation and low variability, to synchronous regimes characterized by strong correlations and wide temporal fluctuations of the state variables.

The asynchronous regime, known as local chaos in the mathematical literature, can be observed in large networks driven by independent sources of noise [2, 3, 37, 46]. In this article we proved that local chaos can be generated dynamically also by strong stimuli in small networks. The decrease of both the variance and the cross-correlation of the neural responses with the input occurs only in networks with saturating activation functions, and it is supported by experimental evidence [34, 43].

On the other side, the synchronous regime occurs near the bifurcation points of the network, which are analytically known [16]. In particular, in the present article we considered the local bifurcations of codimension one, namely the saddle-node, Andronov-Hopf and branching-point bifurcations. Contrary to the strongly positive correlations that occur at the saddle-node and Andronov-Hopf bifurcations, at the branching points we have observed the emergence of strong anti-correlations between inhibitory neurons.

The emergence of strong correlations at any of the local bifurcations of the network is a finite-size effect, and does not require correlated sources of noise. Indeed, for a network with independent Brownian
motions, in [17] the authors proved that the neurons are strongly synchronized at a time instant \( t_N \) that depends on the size of the network. \( t_N \to \infty \) in the limit \( N \to \infty \), therefore strong correlations are very unlikely to occur in large networks after short time intervals. However, exceptions may arise in sparsely-connected networks (see SubSec. (4.4)), or if the Brownian motions are correlated.

In SubSec. (4.1) we explain the importance of the synchronous and asynchronous regimes for the encoding and integration of stimulus information, while in SubSec. (4.2) we discuss how spontaneous symmetry-breaking is responsible for negative correlations. In SubSec. (4.3) we explain possible interactions between drugs and the functional connectivity of the network, and to conclude, in SubSec. (4.4) we discuss future extensions of this work.

4.1 The Role of Correlations in Encoding and Integrating Stimulus Information

Local chaos is very undesirable for the sake of functional integration since it is synonym of functional disconnection and of low information flow between neurons or populations. Notwithstanding, as explained in [1], local chaos proves very convenient in the population encoding of stimulus information. Intuitively, its role can be understood by observing that:

\[
\text{Var} (a_{G}(t)) = \frac{1}{N_G} \left[ \sum_{i \in G} \text{Var} (\nu_i (t)) + \sum_{i \neq j} \text{Cov} (\nu_i (t), \nu_j (t)) \right] = \frac{1}{N_G} \text{Var} (G) + \frac{N_G - 1}{N_G} \text{Cov} (G, G),
\]

(19)

where we defined \( \text{Var} (G) \) \( \equiv \text{Var} (\nu_i (t)) \) \( i \in G \) and \( \text{Cov} (G, G) \) \( \equiv \text{Cov} (\nu_i (t), \nu_j (t)) \) \( i, j \in G, i \neq j \). The last equality of Eq. (19) holds only if \( G \) is a subset of a neural population where spontaneous symmetry-breaking did not occur (so that all the neurons in \( G \) are homogeneous), and it shows that \( \lim_{N_G \to \infty} \text{Var} (a_{G}(t)) = 0 \) only if \( \lim_{N_G \to \infty} \text{Cov} (G, G) = 0 \) and \( \text{Var} (G) \sim N^\varphi \) with \( \varphi < 1 \). Therefore, the variability of the neural activity of a large population is much smaller than that of a single neuron only in the local chaos regime. This means that the population activity could be used to encode the stimulus information reliably. In synchronous states \( \text{Var} (a_{G}(t)) \) does not tend to zero for large \( N_G \), since \( \lim_{N_G \to \infty} \text{Cov} (G, G) \neq 0 \). This is the reason why strong correlations are commonly thought to degrade the performance of population encoding. In experiments with macaques, Ecker et al [15] showed that the information about the stimulus which is conveyed by intra-columnar neurons in the primary visual cortex is actually increased by local chaos. This confirms the role of small correlations in improving the encoding accuracy of large neural populations.

On the other side, for small networks local chaos improves again the encoding accuracy of the population, but the term \( \frac{1}{N_G} \text{Var} (G) \) in Eq. (19) may still be large for \( \text{Cov} (G, G) \to 0 \), depending on the variance of the noise \( (\sigma_{E,t}^2) \). For this reason small neural circuits may need to rely on other mechanisms for encoding information. In [1] Abbott and Dayan proved, somewhat contrary to intuition, that the information about the stimulus conveyed by the network increases in synchronous states with multiplicative noise (i.e. stimulus-dependent) statistics, if the neurons have heterogeneous responses. Due to the non-linearity of the activation function (2), the cross-correlation structure of our model depends on the stimulus, therefore the noise is multiplicative. Moreover, realistic networks have some degree of heterogeneity in the distribution of their parameters (for example see [17] for the extension of our results to networks with heterogeneous synaptic weights). We thus expect that when a biological network is close to a bifurcation point, despite the presence of correlations and the increase in the variability of the neural activity, critical slowing down strongly increases its encoding accuracy, even if the network is small-sized. This proves that at the bifurcation points both the encoding and the integration of stimulus
information are enhanced by correlations. In turn, this result suggests that the bifurcation points may be an ideal place for the brain to accomplish its functions. This intuition is supported by several experimental findings, that advance the hypothesis that self-organization naturally maintains the brain near criticality [9, 51].

4.2 Spontaneous Symmetry-Breaking as the Origin of Anti-Correlations

We proved that at the branching-point bifurcations the inhibitory neurons become strongly anti-correlated as a consequence of spontaneous symmetry-breaking. More generally, other kinds of spontaneous symmetry-breaking can occur in the network, depending on its symmetries. For example, in the case of two identical inhibitory populations, two different symmetries may be broken: the symmetry between neurons in a given population, and that between the two populations. In the latter case, the two populations would behave differently from each other, while keeping their corresponding neurons homogeneous. This phenomenon is also characterized by strongly positive intra-population correlations and strongly negative inter-population correlations (result not shown), reinforcing the idea of a general relationship between spontaneous symmetry-breaking and anti-correlations. In [16] we described possible extensions of our formalism to spatially extended networks with more complex symmetries, therefore spontaneous symmetry-breaking is likely to affect also the cross-correlation structure of large-scale neural models.

Negative correlations have been observed in resting-state fMRI experiments, for example during cognitive tasks performed by human subjects [18], and also in the frontolimbic circuit of awake rats [30], but their origin and functional role are still poorly understood. Our findings suggest branching-point bifurcations and spontaneous symmetry-breaking as a potential neurobiological basis of this phenomenon.

4.3 The Effect of Drugs on the Functional Connectivity

Our model may predict how information encoding and integration in cortical circuits are affected at the mesoscopic scale by drugs. This study can be performed through a bifurcation analysis in terms of the synaptic weights $J_{\alpha \beta}$, in order to simulate the effect of drugs on the excitability of the neural tissue. Glutamate is the major excitatory neurotransmitter in the adult brain, and some drugs such as memantine and lamotrigine inhibit its release [8, 12], resulting in a reduction of the excitatory weights. On the contrary, other compounds such as the ibotenic acid [27] are glutamate receptor agonists, therefore they result in an increase of the excitatory weights. Furthermore, the primary inhibitory neurotransmitter in the brain is the $\gamma$-Aminobutyric acid (GABA) and some drugs such as bicuculline and pentylentetrazol reduce its release [11, 13], while others such as propofol, thiopental and isoflurane enhance it [20]. Therefore their administration causes a reduction or an increase of the inhibitory synaptic weights respectively. This suggests that by modifying the parameters $J_{\alpha \beta}$ we may study how drugs affect the functional connectivity of the cortex at the mesoscopic scale. In particular, whenever for a set of synaptic weights the network does not satisfy the conditions (16), (17), (18), the corresponding bifurcations become forbidden for any pair of stimuli $(I_E, I_I)$. This means that the network cannot rely anymore on its local bifurcation points for integrating information. It is therefore natural to speculate that this phenomenon may provide a neurobiological basis for the strong cognitive impairments observed in drug users [53].

4.4 Future Directions

We studied the functional connectivity of multi-population networks near local bifurcations of codimension one. Furthermore, our theory can be easily extended to the analysis of local bifurcations of larger codimension, while the lack of analytical techniques restricts the study of global bifurcations to numerical methods only.

Another possible extension of our theory is the study of the functional connectivity of sparse networks. In [17] the authors showed that when the number of connections per neuron does not diverge for $N \rightarrow \infty$,
local chaos in general does not occur in the thermodynamic limit for weak stimuli. Therefore in sufficiently sparse networks asynchronous states can be generated only through strong stimuli. Moreover, in [16] we showed that in sparse networks the branching-point bifurcations are more likely to occur, even in excitatory populations, resulting in a considerable increase of the complexity of the bifurcation diagrams.

To conclude, it is possible to study the functional connectivity of small neural circuits with random synaptic weights, extending the results obtained in [22] for large random networks. In [17] the authors introduced explicit formulas for the calculation of the functional connectivity of networks with random weights, but their bifurcation structure is still unexplored. We started to tackle the problem in [16], where we showed that random synaptic weights cause an explicit symmetry-breaking between neurons and therefore the removal of the degeneracy of the eigenvalues. Depending on the degree of heterogeneity, random weights result in a further increase of the complexity of the bifurcation diagrams, which we will investigate in future work.

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From Local Chaos to Critical Slowing Down: A Theory of the Functional Connectivity of Small Neural Circuits

Supplementary Materials

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Structure of the Supplementary Materials

The Supplementary Materials are organized as follows. First, in Sec. (S1) we derive the formula of the Jacobian matrix \( \mathcal{J} \) of the multi-population network, whose eigenvalues and eigenvectors are calculated in Sec. (S2). Then, in Sec. (S3) we derive the fundamental matrix \( \Phi(t) = e^{\mathcal{J}t} \) of the system. In Sec. (S4) we show the constraints that must be satisfied by the covariance matrix of the noise \( \Sigma^\beta_B \) in order to be a genuine covariance matrix, while in Sec. (S5) we use all these results to calculate the functional connectivity of the network in terms of its cross-correlation structure. In particular, in SubSec. (S5.1) we use the formula of the functional connectivity to prove that the network undergoes a weak-correlation regime with small fluctuations (local chaos) when its size is increased to infinity or when the neurons are stimulated by strong inputs. On the other side, in SubSec. (S5.2) we prove that when the network approaches a bifurcation point, the neural activity undergoes a strong-correlation regime with large fluctuations (critical slowing down). In particular, we discuss only the local bifurcations of codimension one, namely saddle-node, Andronov-Hopf and branching-point bifurcations.

S1 Jacobian Matrix

From now on we use the notation \([\mathcal{M}_{\alpha\beta}]_{\forall(\alpha,\beta)}\) to represent a block matrix containing \(\mathcal{M}_{\alpha\beta}\) blocks \(\mathcal{M}_{\alpha\beta}\) for \(\alpha, \beta = 0, \ldots, \mathcal{P} - 1\). Thus, for example the synaptic connectivity matrix (see Eq. (3) in the main text) can be written as \(\mathcal{J} = [\mathcal{J}_{\alpha\beta}]_{\forall(\alpha,\beta)}\). We use the same notation also for \(\mathcal{P} \times \mathcal{P}\) matrices. Thus, for example the reduced Jacobian matrix defined later in Eq. (S6) can be written as \(\mathcal{J}^R = [\mathcal{J}^R_{\alpha\beta}]_{\forall(\alpha,\beta)}\).

From Eq. (1) in the main text we get that the full Jacobian matrix of the network is \(\mathcal{J} = [\mathcal{J}_{\alpha\beta}]_{\forall(\alpha,\beta)}\), where:

\[
\mathcal{J}_{\alpha\beta} = \begin{cases} 
\frac{1}{\mu_\alpha} \text{Id}_{N_\alpha} + \mathcal{R}_\alpha (\mu_\alpha) (\text{Id}_{N_\alpha} - \text{Id}_{N_\alpha}) , & \text{for } \alpha = \beta \\
\frac{J^B_\alpha}{\mu_\alpha} \mathcal{R}_\beta (\mu_\beta) \text{Id}_{N_\alpha} , & \text{for } \alpha \neq \beta.
\end{cases}
\] (S1)

\(\mu_\alpha\) are the stationary membrane potentials obtained form Eq. (1) when the network has constant input \(I_\alpha\) and the noise is not present (\(\sigma^B_i = 0 \forall i\)). In other words, \(\mu_\alpha\) are the solutions of the following system of non-linear algebraic equations:
- \frac{1}{\tau_\alpha} \mu_\alpha + \frac{N_\alpha - 1}{M_\alpha} J_{\alpha\alpha} \phi_\alpha (\mu_\alpha) + \sum_{\beta=0}^{P-1} \frac{N_\beta}{M_\beta} J_{\alpha\beta} \phi_\beta (\mu_\beta) + I_\alpha = 0, \quad \alpha = 0, \ldots, P - 1. \quad \text{(S2)}

Eq. (S2) must be solved numerically, or by the asymptotic perturbative expansion that we introduced in the Supplementary Materials of [1].

**S2 Eigenvalues and Eigenvectors**

For every $\alpha = 0, \ldots, P - 1$, it is trivial to prove that the Jacobian matrix (S1) has $N_\alpha - 1$ eigenvalues of the form:

$$\lambda^P_\alpha = - \frac{1}{\tau_\alpha} + \frac{J_{\alpha\alpha}}{M_\alpha} \phi'_\alpha (\mu_\alpha),$$

with corresponding eigenvectors:

$$v^P_{\alpha;i} = \begin{bmatrix} 0 \\ \vdots \\ 1 \\ 1 \end{bmatrix}, \quad i = 0, \ldots, N_\alpha - 2. \quad \text{(S4)}$$

The term $1 - N_\alpha$ is the $(n_{\alpha-1} + i)$th entry of the vector $v^P_{\alpha;i}$, where:

$$n_{\alpha-1} \overset{\text{def}}{=} \sum_{p=0}^{\alpha-1} N_p$$

with $n_{-1} \overset{\text{def}}{=} 0$. In Eq. (S4), the $(n_{\alpha-1} + j)$th entries of $v^P_{\alpha;i}$ (with $j = 0, \ldots, N_\alpha - 1$ and $j \neq i$) are equal to 1, while all the remaining entries are equal to 0.

Now we prove that the remaining $P$ eigenvalues of $\mathcal{J}$, that we call $\lambda^R_\alpha$, are those of the $P \times P$ matrix $\mathcal{J}^R = \begin{bmatrix} \mathcal{J}^R_{\alpha\beta} \end{bmatrix}_{\alpha,\beta}$ (the “reduced” Jacobian matrix of the network), where:

$$\mathcal{J}^R_{\alpha\beta} = \begin{cases} - \frac{1}{\tau_\alpha} + \frac{N_{\alpha-1}}{M_\alpha} J_{\alpha\alpha} \phi'_\alpha (\mu_\alpha), & \text{for } \alpha = \beta \\ \frac{N_\beta}{M_\alpha} J_{\alpha\beta} \phi'_\beta (\mu_\beta), & \text{for } \alpha \neq \beta \end{cases} \quad \text{(S6)}$$
and the corresponding eigenvectors $v^R_\alpha$ are:

$$v^R_\alpha = \begin{bmatrix} 
\hat{v}^R_\alpha_0 \\
\vdots \\
\hat{v}^R_\alpha_{p-1} \\
\vdots \\
\hat{v}^R_\alpha_{N-1}
\end{bmatrix}.$$

(S7)

In (S7) the entry $\hat{v}^R_\alpha_\beta$ (namely the $\beta$th entry of the vector $\hat{v}^R_\alpha$) is repeated $N_\beta$ times, and $\hat{v}^R_\alpha = \begin{bmatrix} 
\hat{v}^R_\alpha_0, \\
\hat{v}^R_\alpha_1, \\
\vdots \\
\hat{v}^R_\alpha_{p-1}
\end{bmatrix}^T$ is the eigenvector of $J^R$ corresponding to the eigenvalue $\lambda^R_\alpha$. Indeed, by developing the product $J^R v^R_\alpha$ the equation $J^R v^R_\alpha = \lambda^R_\alpha v^R_\alpha$ can be rewritten as follows:

$$\begin{aligned}
\sum_{p=0}^{N_0-1} J^R_{kp} [v^R_\alpha]_p + \sum_{p=N_0}^{N_0+N_1-1} J^R_{kp} [v^R_\alpha]_p + \cdots = \lambda^R_\alpha [v^R_\alpha]_k, & \quad k = 0, \ldots, N_0 - 1 \\
\vdots \\
\sum_{p=0}^{N_0-1} J^R_{kp} [v^R_\alpha]_p + \sum_{p=N_0}^{N_0+N_1-1} J^R_{kp} [v^R_\alpha]_p + \cdots = \lambda^R_\alpha [v^R_\alpha]_k, & \quad k = N_0 + \cdots + N_{p-2}, \ldots, N - 1
\end{aligned}$$

(S8)

Therefore, if we define:

$$S_{\alpha\beta} \overset{\text{def}}{=} \sum_{k=n_\beta}^{n_\beta-1} [v^R_\alpha]_k$$

(S9)

for $\beta = 0, \ldots, p - 1$, and if we replace the terms $J^R_{kp}$ with the corresponding values given by Eq. (S1), the system (S8) can be rewritten as follows:

$$\sum_{\beta=0}^{\gamma-1} \frac{J^R_{\alpha\gamma}}{M^R_{\gamma}} \alpha^R_{\beta} (\mu_\beta) \Theta_{\alpha\beta} = \left( \frac{1}{r_\gamma} + \frac{J^R_{\gamma\gamma}}{M^R_{\gamma}} \alpha^R_{\gamma} (\mu_\gamma) + \lambda^R_\alpha \right) [v^R_\alpha]_k, \quad k_\gamma = n_{\gamma-1}, \ldots, n_\gamma - 1$$

(S10)

for $\gamma = 0, \ldots, p - 1$. Now, Eq. (S10) implies that $[v^R_\alpha]_{k_\gamma}$ does not depend on the index $k_\gamma$ for $\gamma$ fixed, therefore from Eq. (S9) we get:

$$\Theta_{\alpha\beta} = N_\beta \left[ v^R_\alpha \right]_{k_\beta}.$$
Therefore Eq. (S10) can be rewritten as follows:

\[
\sum_{\beta=0}^{P-1} \frac{J_{\gamma\beta}}{M_{\gamma}} \beta \left[ v^{R}_{\alpha} \right]_{k_{\beta}} = \left( \frac{1}{\tau_{\gamma}} + \frac{J_{\gamma\gamma}}{M_{\gamma}} \mu_{\gamma} + \lambda_{\alpha} \right) \left[ v^{R}_{\alpha} \right]_{k_{\gamma}}
\]  

(S11)

This is a homogeneous system in the unknowns \( \left[ v^{R}_{\alpha} \right]_{k_{\beta}} \), with coefficient matrix \( J^{R} - \lambda_{\alpha}^{R} \text{Id}_{P} \), where \( J^{R} \) is given by Eq. (S6). Therefore the system has a non-trivial solution if and only if \( \text{det} \left( J^{R} - \lambda_{\alpha}^{R} \text{Id}_{P} \right) = 0 \), which defines the characteristic polynomial of the reduced Jacobian matrix. The eigenvalues \( \lambda_{\alpha}^{R} \) are the roots of this polynomial, with corresponding eigenvectors \( \hat{v}^{R}_{\alpha} = \left[ [v^{R}_{\alpha}]_{k_{0}}, [v^{R}_{\alpha}]_{k_{1}}, \ldots, [v^{R}_{\alpha}]_{k_{P-1}} \right]^{T} \).

To conclude, we remind that the rank of the matrix \( J^{R} - \lambda_{\alpha}^{R} \text{Id}_{P} \) determines the number of free components \( f \) of \( v^{R}_{\alpha} \), through the relation \( f = p - \text{rank} \left( J^{R} - \lambda_{\alpha}^{R} \text{Id}_{P} \right) \) (rank-nullity theorem), with \( 0 < \text{rank} \left( J^{R} - \lambda_{\alpha}^{R} \text{Id}_{P} \right) < P \).

**Example for \( P = 2 \)**

For simplicity, in the case of one excitatory and one inhibitory population, we call the eigenvalues \( \lambda_{E}^{P} \) as \( \lambda_{E,I} \), and the corresponding eigenvectors \( v_{0,1}^{P} \) as \( v_{E,I}^{P} \). Then, according to Eq. (S3), we get:

\[
\lambda_{E} = -\left[ \frac{1}{\tau_{E}} + \frac{J_{EE}}{M_{E}} \mu_{E} \right] , \quad \lambda_{I} = -\left[ \frac{1}{\tau_{I}} + \frac{J_{II}}{M_{I}} \mu_{I} \right].
\]  

(S12)

with multiplicity \( N_{E} - 1 \) and \( N_{I} - 1 \) respectively, while from Eq. (S4) we obtain:

\[
v_{E,0} = \begin{bmatrix} 1 - N_{E} \\ 1 \\ \vdots \\ 1 \\ 0 \\ 0 \end{bmatrix}, \quad v_{E,1} = \begin{bmatrix} 1 \\ 1 \\ \vdots \\ 1 \\ 0 \\ 0 \end{bmatrix}, \quad \ldots, \quad v_{E,N_{E}-2} = \begin{bmatrix} 1 - N_{E} \\ 1 \\ \vdots \\ 1 \\ 0 \\ 0 \end{bmatrix},
\]  

(S13)

\[
v_{I,0} = \begin{bmatrix} 0 \\ 0 \\ \vdots \\ 0 \\ 1 - N_{I} \\ 1 \end{bmatrix}, \quad v_{I,1} = \begin{bmatrix} 0 \\ 0 \\ \vdots \\ 0 \\ 1 - N_{I} \\ 1 \end{bmatrix}, \quad \ldots, \quad v_{I,N_{I}-2} = \begin{bmatrix} 0 \\ 0 \\ \vdots \\ 0 \\ 1 - N_{I} \\ 1 \end{bmatrix},
\]

(S14)

The remaining eigenvalues of \( \mathcal{J} \) are those of the following reduced Jacobian matrix:
\[ J^R = \begin{bmatrix}
-\frac{1}{\tau_E} + \frac{N_E - 1}{M_E} J_{EE} \phi_E' (\mu_E) & \frac{N_I}{M_I} J_{EI} \phi_I' (\mu_I) \\
\frac{N_E}{M_E} J_{EE} \phi_E' (\mu_E) & -\frac{1}{\tau_I} + \frac{N_I - 1}{M_I} J_{II} \phi_I' (\mu_I)
\end{bmatrix},
\]

namely:

\[ \lambda_{0,1}^R = \frac{Y + Z \pm \sqrt{(Y - Z)^2 + 4X}}{2}, \quad (S14) \]

where:

\[ X = N_E N_I J_{EI} J_{II} \phi_E' (\mu_E) \phi_I' (\mu_I), \quad Y = -\frac{1}{\tau_E} + \frac{N_E - 1}{M_E} J_{EE} \phi_E' (\mu_E), \quad Z = -\frac{1}{\tau_I} + \frac{N_I - 1}{M_I} J_{II} \phi_I' (\mu_I). \quad (S15) \]

We observe that \( \text{rank} (J^R - \lambda_0^R \text{Id}_P) = 1 \), therefore the eigenvectors of \( J^R \) corresponding to the eigenvalues \( \lambda_{0,1}^R \) are:

\[ \hat{v}_0^R = x \begin{bmatrix} 1 \\ K_0 \end{bmatrix}, \quad \hat{v}_1^R = y \begin{bmatrix} 1 \\ K_1 \end{bmatrix}, \quad (S16) \]

where:

\[ K_\alpha = \frac{M_E \left( \lambda_\alpha^R + \frac{1}{\tau_E} \right) - (N_E - 1) J_{EE} \phi_E' (\mu_E)}{N_I J_{EI} \phi_I' (\mu_I)} = \frac{N_E J_{IE} \phi_I' (\mu_E)}{M_I \left( \lambda_\alpha^R + \frac{1}{\tau_I} \right) - (N_I - 1) J_{II} \phi_I' (\mu_I)} \quad (S17) \]

(the last equality is a consequence of \( \text{det} (J^R - \lambda_0^R \text{Id}_P) = 0 \)). Moreover, \( x, y \) are two free parameters that represent the free components of \( \hat{v}_{0,1}^R \) (one for each eigenvector, according to the relation \( f = \Psi - \text{rank} (J^R - \lambda_0^R \text{Id}_P) = 1 \)). Therefore the corresponding eigenvectors of \( J \) are:

\[ v_0^R = x \begin{bmatrix} 1 \\ \vdots \\ 1 \\ K_0 \\ K_0 \end{bmatrix}, \quad v_1^R = y \begin{bmatrix} 1 \\ \vdots \\ 1 \\ K_1 \\ K_1 \end{bmatrix}, \quad (S18) \]

where the first \( N_E \) entries of \( v_{0,1}^R \) in Eq. (S18) are equal to 1 and thus \( K_{0,1} \) are the remaining \( N_I \) entries.
S3  Fundamental Matrix

When $J$ is diagonalizable, its powers can be written as $J^n = PD^nP^{-1}$, where:

$$D = \text{diag} \left( \lambda_0^P, \ldots, \lambda_{P-1}^P, \lambda_0^R, \ldots, \lambda_{P-1}^R \right)$$

$$D^n = \text{diag} \left( \left( \lambda_0^P \right)^n, \ldots, \left( \lambda_{P-1}^P \right)^n, \left( \lambda_0^R \right)^n, \ldots, \left( \lambda_{P-1}^R \right)^n \right),$$

while $P$ is an $N \times N$ matrix whose columns are composed of the eigenvectors of $J$ calculated in Sec. (S2).

If we introduce the matrices:

$$\Psi_{\alpha\beta; n} = \begin{cases} \Xi_{\alpha,n} \text{Id}_N + \Gamma_{\alpha,n} (I_N - \text{Id}_N), & \text{for } \alpha = \beta \\ \Omega_{\alpha\beta,n} \text{Id}_{N\beta}, & \text{for } \alpha \neq \beta \end{cases}$$

such that $J^n = [\Psi_{\alpha\beta; n}]_{\forall (\alpha, \beta)}$, and we replace Eq. (S19) and the matrix $P$ (expressed in terms of the eigenvectors of $J$) into the formula $J^nP = PD^n$, after some algebra we get the following set of equations:

$$\left\{ \begin{array}{c} N_\alpha \left[ \hat{v}_R^\gamma \right]_\alpha \Gamma_{\alpha,n} \sum_{\beta=0}^{P-1} N_\beta \left[ \hat{v}_R^\gamma \right]_\beta \Omega_{\alpha\beta,n} = \left[ \hat{v}_R^\gamma \right]_\alpha \left( \left( \lambda_\gamma^R \right)^n - \left( \lambda_\alpha^P \right)^n \right) \\ \Xi_{\alpha,n} - \Gamma_{\alpha,n} = \left( \lambda_\alpha^P \right)^n \end{array} \right. \quad \text{(S20)}$$

for $\gamma = 0, 1, \ldots, P - 1$. Now if we define:

$$\Omega_{\alpha\alpha; n} \overset{\text{def}}{=} \Gamma_{\alpha,n} + \frac{1}{N_\alpha} \left( \lambda_\alpha^P \right)^n,$$  \hspace{0.5cm} \text{(S21)}$$

the first equation of the system (S20) becomes:

$$\sum_{\beta=0}^{P-1} N_\beta \left[ \hat{v}_R^\gamma \right]_\beta \Omega_{\alpha\beta,n} = \left[ \hat{v}_R^\gamma \right]_\alpha \left( \lambda_\gamma^R \right)^n, \quad \gamma = 0, 1, \ldots, P - 1.$$  

In matrix form the system reads $\mathfrak{A} \mathbf{r}_{\alpha; n} = \mathbf{b}_{\alpha; n}$, where:

$$\mathfrak{A} = \left( P^R \right)^T \text{diag} \left( N_0, N_1, \ldots, N_{P-1} \right),$$  \hspace{0.5cm} \text{(S22)}$$

$$\mathbf{r}_{\alpha; n} = \begin{bmatrix} \Omega_{\alpha0,n} \\ \Omega_{\alpha1,n} \\ \vdots \\ \Omega_{\alpha,P-1,n} \end{bmatrix}, \quad \mathbf{b}_{\alpha; n} = \begin{bmatrix} \left[ \hat{v}_R^\gamma \right]_\alpha \left( \lambda_0^R \right)^n \\ \left[ \hat{v}_R^\gamma \right]_\alpha \left( \lambda_1^R \right)^n \\ \vdots \\ \left[ \hat{v}_R^{P-1} \right]_\alpha \left( \lambda_{P-1}^R \right)^n \end{bmatrix}, \quad \alpha = 0, 1, \ldots, P - 1.$$
In Eq. (S22), $P^R$ is a $\Psi \times \Psi$ matrix whose columns are composed of the eigenvectors of $J^R$, namely:

$$P^R = \begin{bmatrix}
\hat{v}_R^0 & \hat{v}_R^1 & \ldots & \hat{v}_R^{P-1} \\
\hat{v}_R^0 & \hat{v}_R^1 & \ldots & \hat{v}_R^{P-1} \\
\vdots & \vdots & \ddots & \vdots \\
\hat{v}_R^0 & \hat{v}_R^1 & \ldots & \hat{v}_R^{P-1}
\end{bmatrix}.$$ 

Therefore by inverting the system $A^\alpha x^{\alpha;n} = b^{\alpha;n}$, we obtain:

$$\Omega_{\alpha\beta;n} = \sum_{\gamma=0}^{P-1} C^{(\gamma)}_{\alpha\beta} \left( \lambda^{R\gamma}_\alpha \right)^n,$$

where:

$$C^{(\gamma)}_{\alpha\beta} = \left[ A^{-1} \right]_{\beta\gamma} \left[ \hat{v}_R^\gamma \right]_\alpha = \frac{1}{N_\beta} \left[ \left( P^R \right)^{-T} \right]_{\beta\gamma} \left[ \hat{v}_R^\gamma \right]_\alpha, \quad \left( P^R \right)^{-T} \equiv \left[ \left( P^R \right)^T \right]^{-1}. \quad (S23)$$

We observe that the matrix $C^{(\gamma)}_{\alpha\beta} \text{def} \left[ C^{(\gamma)}_{\alpha\beta} \right]_{\Psi(\alpha,\beta)}$ can be written as an outer product of vectors, namely:

$$C^{(\gamma)} = \begin{bmatrix}
\hat{v}_R^\gamma & \hat{v}_R^\gamma & \ldots & \hat{v}_R^\gamma \\
\hat{v}_R^\gamma & \hat{v}_R^\gamma & \ldots & \hat{v}_R^\gamma \\
\vdots & \vdots & \ddots & \vdots \\
\hat{v}_R^0 & \hat{v}_R^1 & \ldots & \hat{v}_R^{P-1}
\end{bmatrix} \left[ A^{-1} \right]_{0\gamma} \ldots \left[ A^{-1} \right]_{P-1,\gamma}, \quad (S24)$$

therefore it has rank one. This result will prove very important in demonstrating the formation of critical slowing down near the saddle-node bifurcations (see SubSec. (S5.2.1)).

Moreover, from the second equation of (S20) and from the definition (S21), we get:

$$\Xi_{\alpha;n} = \left( \lambda^{P}_\alpha \right)^n + \Gamma_{\alpha;n} = \left( 1 - \frac{1}{N_\alpha} \right) \left( \lambda^{P}_\alpha \right)^n + \Omega_{\alpha\alpha;n},$$

Therefore we can rewrite Eq. (S19) as follows:

$$\Psi_{\alpha\beta;n} = \begin{cases}
\sum_{\gamma=0}^{P-1} C^{(\gamma)}_{\alpha\beta} \left( \lambda^{R\gamma}_\alpha \right)^n \mathbb{I}_{N_\alpha} + \left( -\frac{1}{N_\alpha} \mathbb{I}_{N_\alpha} + \text{Id}_{N_\alpha} \right) \left( \lambda^{P}_\alpha \right)^n, & \text{for } \alpha = \beta \\
\sum_{\gamma=0}^{P-1} C^{(\gamma)}_{\alpha\beta} \left( \lambda^{R\gamma}_\alpha \right)^n \mathbb{I}_{N_\alpha, N_\beta}, & \text{for } \alpha \neq \beta.
\end{cases} \quad (S25)$$
To conclude, from the Taylor expansion \( e^{Jt} = \sum_{n=0}^{\infty} \frac{t^n}{n!} J^n \) and Eq. (S25) we get \( \Phi (t) = [\Phi_{\alpha\beta} (t)]_{\gamma(\alpha,\beta)} \), where:

\[
\Phi_{\alpha\beta} (t) = \begin{cases} 
\left( \sum_{\gamma=0}^{\nu_1-1} \rho_{\alpha\beta}^{(\gamma)} e^{\lambda_{\gamma} R t} \right) I_{N_\alpha} + \left( \frac{-1}{N_\alpha} I_{N_\alpha} + \text{Id}_{N_\alpha} \right) e^{\lambda_{\alpha} P t}, & \text{for } \alpha = \beta \\
\left( \sum_{\gamma=0}^{\nu_1-1} \rho_{\alpha\beta}^{(\gamma)} e^{\lambda_{\gamma} R t} \right) I_{N_{\alpha \beta}}, & \text{for } \alpha \neq \beta.
\end{cases}
\] (S26)

For the sake of clarity, we implemented this method in the supplemental Python script “Fundamental_Matrix.py” for an arbitrary number of populations. Furthermore, below we show the explicit calculation of the fundamental matrix in the case \( P = 2 \).

**Example for \( P = 2 \)**

In the case of two neural populations, from Eqs. (S16) we get \( P^R = \begin{bmatrix} 1 & 1 \\ K_0 & K_1 \end{bmatrix} \) and therefore \((P^R)^{-T} = \frac{1}{K_1 - K_0} \begin{bmatrix} K_1 & -K_0 \\ -1 & 1 \end{bmatrix} \). Thus, according to Eqs. (S23) + (S26), the blocks of the fundamental matrix \( \Phi (t) = \begin{bmatrix} \Phi_{EE} (t) & \Phi_{EI} (t) \\ \Phi_{IE} (t) & \Phi_{II} (t) \end{bmatrix} \) are given by the following formulas:

\[
\Phi_{EE} (t) = \frac{K_1 e^{\lambda_{\alpha} R t} - K_0 e^{\lambda_{\beta} R t}}{N_E (K_1 - K_0)} I_{N_E} + \left( \frac{-1}{N_E} I_{N_E} + \text{Id}_{N_E} \right) e^{\lambda_{\gamma} R t}, \\
\Phi_{EI} (t) = \frac{K_0 K_1 (e^{\lambda_{\alpha} R t} - e^{\lambda_{\beta} R t})}{N_I (K_1 - K_0)} I_{N_{I,E}}, \\
\Phi_{II} (t) = \frac{K_1 e^{\lambda_{\alpha} R t} - K_0 e^{\lambda_{\beta} R t}}{N_I (K_1 - K_0)} I_{N_I} + \left( \frac{-1}{N_I} I_{N_I} + \text{Id}_{N_I} \right) e^{\lambda_{\gamma} R t}, \\
\Phi_{IE} (t) = \frac{K_0 K_1 (e^{\lambda_{\alpha} R t} - e^{\lambda_{\beta} R t})}{N_E (K_1 - K_0)} I_{N_{I,E}}.
\] (S27)

### S4 Constraints for the Covariance Matrix of the Noise

In order to be a genuine covariance matrix, \( \Sigma^R \) (see Eq. (4) in the main text) must be positive-semidefinite. Being symmetric, \( \Sigma^R \) is positive-semidefinite if and only if its eigenvalues are non-negative. Since it has the same block structure of the Jacobian matrix, we can easily calculate its eigenvalues by adapting the results of Sec. (S2). For example, in the case \( P = 2 \) we get that the matrix \( \Sigma^R \) has to satisfy the following set of inequalities in order to be positive-semidefinite:

\[
\begin{align*}
\left( \sigma_{EE}^R \right)^2 \left[ 1 + (N_E - 1) C_{EE}^R \right] + \left( \sigma_{II}^R \right)^2 \left[ 1 + (N_I - 1) C_{II}^R \right] &\geq 0 \\
\left[ 1 + (N_E - 1) C_{EE}^R \right] \left[ 1 + (N_I - 1) C_{II}^R \right] &\geq N_E N_I \left( C_{EI}^R \right)^2.
\end{align*}
\]

The importance of this constraint is highlighted by the following relation:
\[
N^{-1} \sum_{i,j=0}^{N-1} x_i x_j \text{Cov} \left( \frac{dB_i(t)}{dt}, \frac{dB_j(t)}{dt} \right) = \text{Var} \left( \sum_{i=0}^{N-1} x_i \frac{dB_i(t)}{dt} \right).
\]

Indeed, from this equality we see that if \( \Sigma^B \) were not positive-semidefinite, the linear combination \( \sum_{i=0}^{N-1} x_i \frac{dB_i(t)}{dt} \) would have negative variance for some coefficients \( x_i \).

**S5 Functional Connectivity**

Now we have all the ingredients for studying the phenomena that affects the functional connectivity of the network. In particular, in this section we prove that, depending on the parameters of the network, the neural activity spans from strong anti-correlation (i.e. \( \text{Corr} (V_i(t), V_j(t)) \rightarrow -1 \)) to strongly positive correlation (\( \text{Corr} (V_i(t), V_j(t)) \rightarrow 1 \)), passing through arbitrarily weak correlation (\( \text{Corr} (V_i(t), V_j(t)) \rightarrow 0 \)).

We consider the weak-correlation regime in SubSec. (S5.1), while we study the strong-correlation regime in SubSec. (S5.2) in relation to the local bifurcations of the network. For both the regimes, we analyze the variance and the cross-correlation for an arbitrary number of populations, extending the results of the case \( P = 2 \) that we developed in the main text (see Eqs. (14) + (15)) without the need of explicit formulas for the functional connectivity. Indeed, we show that it is possible to evaluate the qualitative behavior of the covariance matrix of the membrane potentials \( \Sigma^V \) even if the explicit expressions of the coefficients \( C_{\alpha \beta}^{(\gamma)} \) in Eq. (S26) are not known.

**S5.1 Weak-Correlation Regime (Local Chaos)**

According to the mean-field theory developed by McKean, Tanaka and Sznitman [3-10], the cross-correlation of the multi-population network in the thermodynamic limit (\( N_\alpha \rightarrow \infty \forall \alpha \)) tends to zero for every pair of neurons if the Brownian motions are independent (i.e. \( \Sigma^B_{\alpha \beta} = 0 \forall \alpha, \beta \)). This result was proved in [11] for an arbitrary number of neural populations with infinite size.

However, having an infinite size is not the only way a network can experience low levels of correlation. As explained in [2], for strongly depolarizing or strongly hyperpolarizing stimuli the activation function saturates to \( \nu^{\max} \) or 0 respectively (see Eq. (2) of the main text), therefore \( \mathcal{A}'(V) \rightarrow 0 \). Given a network with an arbitrary number of populations of arbitrary size, if the stimulus is strong enough for all the populations, the Jacobian matrix of the network (see Eq. (S1)) becomes diagonal. In other words, the neurons become effectively disconnected. For this reason the correlation structure of the membrane potentials is determined only by the Brownian motions, namely \( \text{Corr} (V_i(t), V_j(t)) \rightarrow [\Sigma^B]_{ij} \). Therefore, the necessary requirement to generate weak correlation between neurons is again the absence of noise correlations, namely \( \Sigma^B_{\alpha \beta} = 0 \forall \alpha, \beta \). This mechanism is different from that described in [11], since it occurs for \( |I_\alpha| \rightarrow \infty \) rather than \( N_\alpha \rightarrow \infty \), and it allows the network to create decorrelated activity even if its size is small.

**S5.2 Strong-Correlation Regime (Local Bifurcations)**

Interestingly, the network is able to show also high values of correlation, either positive or negative. This phenomenon occurs at the (local) bifurcation points of the network, and therefore for special combinations of the network’s parameters. As we explained in the main text, we consider only the codimension one bifurcations of the network, namely saddle-node, Andronov-Hopf and branching-point bifurcations. The relation between these bifurcations and the functional connectivity of the system is described in the next subsections.
S5.2.1 Saddle-Node Bifurcations

The multi-population neural network undergoes saddle-node bifurcations when one of the eigenvalues of the reduced Jacobian matrix tends to zero [1]. Therefore, if $\lambda_{ij}^{R} \to 0^{-}$ for a given $\gamma$, and all the other eigenvalues have negative real part, then for $t \gg \frac{1}{|\lambda_{ij}^{R}|}$ Eq. (S26) gives:

$$\Psi_{\alpha,\beta} (t) \approx e^{i \lambda_{ij}^{R} t} E_{N_{\alpha},N_{\beta}} \forall \alpha, \beta.$$ 

According to Eq. (6) of the main text, this implies:

$$\text{Cov} (V_{i} (t), V_{j} (t)) \approx \frac{1}{2|\lambda_{ij}^{R}|^{2}} \sum_{\beta=0}^{N-1} N_{\beta} \left( \sigma^{\beta}_{\alpha} \sigma^{\beta}_{\beta} \right) \left( \Psi^{(\gamma)}_{p(i),\beta} \Psi^{(\gamma)}_{p(j),\beta} - \Psi^{(\gamma)}_{p(i),\beta} \Psi^{(\gamma)}_{p(j),\beta} \right)^{2} = 0.$$ 

(S28)

where $p (k)$ represents the neural population the $k$th neuron belongs to. Now, since $\text{Var} (V_{i} (t)) = \text{Cov} (V_{i} (t), V_{i} (t)) \approx \frac{1}{2|\lambda_{ij}^{R}|}$, the amplitude of the fluctuations diverges for $\lambda_{ij}^{R} \to 0^{-}$. Moreover, if $p(i) = p(j)$, Eq. (S28) gives $\text{Cov} (V_{i} (t), V_{j} (t)) \approx \text{Var} (V_{i} (t)) = \text{Var} (V_{j} (t))$, therefore according to Pearson’s formula $\text{Cov} (V_{i} (t), V_{j} (t)) \approx 1$. On the other side, if $p(i) \neq p(j)$, by replacing Eq. (S28) within the condition $\text{Cov}^{2} (V_{i} (t), V_{j} (t)) = \text{Var} (V_{i} (t)) \text{Var} (V_{j} (t))$, after some algebra we get:

$$\sum_{\beta_{0},\beta_{1} = 0}^{N-1} N_{\beta_{0}} N_{\beta_{1}} \left( \sigma^{\beta_{0}}_{\beta_{0}} \sigma^{\beta_{1}}_{\beta_{1}} \right) \left( \Psi^{(\gamma)}_{p(i),\beta_{0}} \Psi^{(\gamma)}_{p(j),\beta_{1}} - \Psi^{(\gamma)}_{p(i),\beta_{1}} \Psi^{(\gamma)}_{p(j),\beta_{0}} \right)^{2} = 0.$$ 

This equality is satisfied if and only if:

$$\Psi^{(\gamma)}_{p(i),\beta_{0}} \Psi^{(\gamma)}_{p(j),\beta_{1}} - \Psi^{(\gamma)}_{p(i),\beta_{1}} \Psi^{(\gamma)}_{p(j),\beta_{0}} = \text{det} \left( \begin{bmatrix} \Psi^{(\gamma)}_{p(i),\beta_{0}} & \Psi^{(\gamma)}_{p(j),\beta_{0}} \\ \Psi^{(\gamma)}_{p(i),\beta_{1}} & \Psi^{(\gamma)}_{p(j),\beta_{1}} \end{bmatrix} \right) = 0 \forall \beta_{0}, \beta_{1},$$

namely if and only if the matrix $\Psi^{(\gamma)}$ has rank 1, which is indeed the case (see Sec. (S3)). This proves the emergence of strong correlations also between the neurons of different populations when the network is close to a saddle-node bifurcation.

Interestingly, critical slowing down occurs regardless of the strength of the correlation between the Brownian motions.

S5.2.2 Andronov-Hopf Bifurcations

The multi-population neural network undergoes an Andronov-Hopf bifurcation whenever the reduced Jacobian matrix has two complex-conjugate purely imaginary eigenvalues [1]. If the real part of a pair of complex-conjugate eigenvalues $\lambda_{ij}^{R,\delta}$ tends to zero (i.e. $\Re (\lambda_{ij}^{R,\delta}) \to 0^{-}$), and if all the other eigenvalues have negative real part, then for $t \gg \frac{1}{|\Re (\lambda_{ij}^{R,\delta})|}$ Eq. (S26) gives:
\[
\Phi_{\alpha \beta} (t) \approx \left( C^{(\gamma)}_\alpha e^{\lambda^R_\gamma t} + \overline{C^{(\gamma)}_\alpha} e^{-\lambda^R_\gamma t} \right) \mathbb{I}_{N_\alpha, N_\beta} \quad \forall \alpha, \beta
\]

where \(\lambda^R_\gamma = \lambda^R_\delta\) and the overline represents the complex conjugate operator. According to Eq. (6) of the main text, after some algebra we get:

\[
\text{Cov} (V_i (t), V_j (t)) \sim \frac{1 - e^{-2|\lambda^R_\gamma| t}}{|\Re (\lambda^R_\gamma)|} \sum_{\beta = 0}^{p-1} N_\beta \left( \sigma_{\beta}^2 \right) \Re \left( C^{(\gamma)}_{p(i), \beta} \overline{C^{(\gamma)}_{p(j), \beta}} \right), \quad \forall i, j
\]

(S29)

thus, similarly to the saddle-node bifurcations, for \(t \gg \frac{1}{|\Re (\lambda^R_\gamma)|}\) the amplitude of the fluctuations diverges for \(\Re (\lambda^R_\gamma) \to 0^-\). Moreover, if \(p (i) = p (j)\), Eq. (S29) gives \(\text{Cov} (V_i (t), V_j (t)) \approx \text{Var} (V_i (t)) = \text{Var} (V_j (t))\), therefore \(\text{Corr} (V_i (t), V_j (t)) \approx 1\). On the other side, if \(p (i) \neq p (j)\), from Eq. (S29) it is possible to prove that the condition \(\text{Cov}^2 (V_i (t), V_j (t)) = \text{Var} (V_i (t)) \text{Var} (V_j (t))\) is satisfied if and only if \(\Re \left( C^{(\gamma)}_{p(i), \beta} \right) = \Re \left( C^{(\gamma)}_{p(j), \beta} \right) = 0 \forall \beta\). However, these imaginary parts cannot be equal to zero at an Andronov-Hopf bifurcation, the latter being defined by a pair of purely imaginary eigenvalues. Therefore, contrary to the saddle-node bifurcations, the cross-correlation between neurons in different populations does not tend to one at an Andronov-Hopf bifurcation. This is also confirmed by the top-right panel of Fig. (4) in the main text, in the special case \(\mathfrak{F} = 2\).

S5.2.3 Branching-Point Bifurcations

Branching-point bifurcations occur whenever \(\lambda^P_\gamma \to 0^-\) for a given \(\gamma\) [1]. According to Eq. (S3), only sufficiently strong self-inhibitory synaptic weights \(J_{\gamma i}\) give rise to this kind of bifurcation. As we explained in [1], branching points do not occur in the mean-field approximation of the network, therefore the study of the functional connectivity near these bifurcations is one of the results of most interest of our article.

If \(\lambda^P_\gamma \to 0^-\) and all the other eigenvalues have negative real part, for \(t \gg \frac{1}{|\lambda^P_\gamma|}\) Eq. (S26) gives:

\[
\Phi_{\gamma \beta} (t) \approx \begin{cases} 
\left( -\frac{1}{N_\gamma} I_{N_\gamma} + \text{Id}_{N_\gamma} \right) e^{-|\lambda^P_\gamma| t}, & \text{for } \gamma = \beta \\
\Omega_{N_\gamma, N_\beta}, & \text{for } \gamma \neq \beta
\end{cases}
\]

(S30)

where \(\Omega_{N_\gamma, N_\beta}\) is the \(N_\gamma \times N_\beta\) null matrix. According to Eq. (6) of the main text, if \(p (i) = p (j) = \gamma\) we get:

\[
\text{Cov} (V_i (t), V_j (t)) \approx \frac{1 - e^{-2|\lambda^P_\gamma| t}}{2 |\lambda^P_\gamma|} \left( \sigma_{\gamma}^2 \right)^2 \left( -\frac{1}{N_\gamma} \right), \quad \text{for } i \neq j
\]

(S31)

\[
\text{Var} (V_i (t)) = \text{Var} (V_j (t)) \approx \frac{1 - e^{-2|\lambda^P_\gamma| t}}{2 |\lambda^P_\gamma|} \left( \sigma_{\gamma}^2 \right)^2 \left( 1 - \frac{1}{N_\gamma} \right)
\]

(S32)

thus for \(t \gg \frac{1}{|\lambda^P_\gamma|}\) the amplitude of the fluctuations diverges for \(\lambda^P_\gamma \to 0^-\), while \(\text{Corr} (V_i (t), V_j (t)) \approx \frac{1}{1 - \frac{1}{N_\gamma}}\). Interestingly, according to [2] this is the lower bound of the correlation between fully-connected
neurons in a homogeneous population with size $N_\gamma$. Since $1 - N_\gamma < 0$ for $N_\gamma \geq 2$, the neurons in population $\gamma$ are maximally anti-correlated at the branching-point bifurcations. Moreover, according to this formula, correlation tends to $-1$ only for $N_\gamma = 2$ (which is confirmed by the top-right panel of Fig. (4) in the main text, in the special case $\Psi = 2$).

To conclude, if $p(i) = p(j) \neq \gamma$, the matrix $\Phi_{p(i),p(j)}(t)$ is not dominated by the term $e^{-|\lambda_\gamma|^t}$, therefore we do not observe neither the divergence of the variance nor close-to-one correlations in population $p(i)$. This prevents also the formation of strong inter-population correlations between $\gamma$ and $p(i)$. In the case $\Psi = 2$ this phenomenon occurs for the excitatory neurons since $\gamma = I$ and $p(i) = E$, and indeed this result is confirmed by the top panels of Fig. (4) in the main text.

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