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We have studied the effects of perturbations on the cat's cerebral cortex. According to the literature, this cortex structure can be described by a clustered network. This way, we construct a clustered network with the same number of areas as in the cat matrix, where each area is described as a sub-network with a small-world property. We focus on the suppression of neuronal phase synchronisation considering different kinds of perturbations. Among the various controlling interventions, we choose three methods: delayed feedback control, external time-periodic driving, and activation of selected neurons. We simulate these interventions to provide a procedure to suppress undesired and pathological abnormal rhythms that can be associated with many forms of synchronisation. In our simulations, we have verified that the efficiency of synchronisation suppression by delayed feedback control is higher than external time-periodic driving and activation of selected neurons of the cat's cerebral cortex with the same coupling strengths. © *2016 AIP Publishing LLC*. [http://dx.doi.org/10.1063/1.4945796]

The mammalian cerebral cortex has features of complex networks, as well as it is involved in cognitive functions and complex perceptual. In the literature, the cat's brain is one of the cortical networks for which information is widely available. The cat's corticocortical network is organised into the visual, auditory, somatosensory-motor, and frontolimbic regions, and can be separated into 65 cortical areas. The areas connected by fibres of different densities can be described through a connectivity matrix. By means of the cat connectivity matrix, we have studied the suppression of neuronal phase synchronisation. Synchronisation might be behind the way we perceive objects, but it is also responsible for abnormal behaviours. Clinical evidences pointed out that synchronisation of a small group of neurons plays a key role in some pathological conditions such as Parkinson's disease, tremor, and epilepsy. For this reason, the study of control of undesirable neuronal rhythms is relevant to restore normal spiking activity in a neuronal network. We focus, in particular, on three methods of intervention: delayed feedback control, external time-periodic driving, and activation of selected neurons. We have observed that these methods can suppress neuronal phase synchronisation and consequently can induce a mean field amplitude death (MFAD). Morever, with regard to suppression, we demonstrate that delayed feedback control has a better efficiency than external time-periodic driving and activation of selected neurons. We also show the importance of a time delay in the feedback control. Therefore, our results may help the understanding not only of suppression of synchronisation in mammalian cerebral cortex but also to develop methods of interventions for the treatment of severe neurological and psychiatric diseases.

I. INTRODUCTION

The cerebral cortex is an important part of the mammalian brain, and it is the outer covering of gray matter over the brain's hemispheres. It is responsible for cognitive tasks such as emotion, complex thought, memory, language comprehension, and consciousness.[1] In the literature, it is possible to find information about the structure of the cerebral cortex network for the macaque monkey,[2] *Caenorhabditis elegans*,[3] the cat,[4] etc. The cat's cerebral cortex connectivity data were first published by Scannell and Young.[5] The cat's corticocortical network can be separated into 65 areas, and the areas are connected by fibres of different densities that can be described through a connection matrix. The areas are separated into four clusters or cognitive regions named as visual, auditory, somatosensory-motor, and frontolimbic.

We study a model of neuronal network that contains a connectivity configuration in accordance with the cat matrix.[6] The clusters formed by cortical areas with common functional roles are responsible for the complexity of the cat's network.[7–9] With this in mind, we consider that each element of the matrix has a sub-network with a small-world property.[10] The small-world network is clustered like regular networks which has a path length comparable to random networks. The short path length is due to the existence of

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long-range connections. Stam *et al.*[11] presented studies about the presence of small-world characteristics in functional brain networks, and they also showed that Alzheimer's disease is characterised by a loss of small-world property. Epilepsy in a small-world network was investigated by Netoff *et al.*[12] They modelled activity in hippocampal slices by considering small-world networks of excitatory neurons that reproduce bursts and seizures.

In recent years, mathematical models to describe neuronal networks have been studied very intensively. Neuronal mathematical models can take many forms, e.g., differential equations such as the Hodgkin-Huxley model[13] and the Hindmarsh-Rose model,[14] as well as models with discrete time as the Rulkov map.[15,16] In this work, we consider a coupled Rulkov map network that presents not only discrete time but also discrete space. The Rulkov map presents two different time scales, where the variable with slow dynamics is responsible for the modulation of bursts in the fast variable.[17] Networks of coupled Rulkov maps have been used in studies on neuronal phase synchronisation,[18,19] suppression of bursting synchronisation,[20] and pattern formation.[21]

In the coupled map Rulkov network, the mean field exhibits large amplitude periodic oscillations when the neurons are bursting at approximately the same times, namely, when the network presents phase synchronisation, whereas the mean field exhibits small amplitude oscillations if the neurons are not bursting in phase.[18] This way, in this work, suppression of phase synchronisation means to get the amplitude of the mean field strongly reduced; in other words, the suppression induces a mean field amplitude death (MFAD). Oscillation quenching, e.g., oscillation (OD) and amplitude death (AD), is a fundamental emergent phenomenon in coupled oscillators.[22]

Neuronal synchronisation can be found in neuronal activities due to coupling among neurons or by means of common inputs. Studies have demonstrated the importance of synchronisation of oscillatory phases between different brain regions in memory processes.[23] Roelfsema *et al.*[24] realised studies recording local field potentials from electrodes implanted in the cortex of cats. They verified large-scale synchronisation when cats were submitted to a sudden change in a visual pattern. Synchronisation in ensembles was also studied by Ivanchenko *et al.*,[25] who observed a second-order phase transition to synchronisation. Axmacher *et al.*[26] verified that specific forms of cellular plasticity during subsequent stages of memory formation are induced by synchronisation. Nevertheless, there are evidences that certain brain disorders are related to neuronal synchronisation, e.g., epilepsy that results from high and extended synchronisation.[27] Moreover, synchronisation of neuronal activity is a common finding in patients with Parkinson's disease.[28] Levy *et al.*[29] demonstrated that Parkinsonian patients are characterised by synchronised high-frequency activity in the subthalamic nucleus.

In face of neuronal synchrony in brain disorders, we study here three methods to suppress undesired synchronisation: delayed feedback control, external time-periodic driving, and activation of selected neurons. The delayed feedback control was proposed by Rosenblum and Pikowsky[30] to suppress synchronised pathological brain rhythms through a delayed feedback signal.[31] Batista *et al.*[32] observed that feedback control in networks of Hodgkin-Huxley-type neurons with chemical synapses can present more energy saving when compared to other suppression methods. Feedback control is a method that has been implemented in clinical applications by means of functional magnetic resonance imaging-based neurofeedback.[33] With regard to external time-periodic driving, it was verified that electrical stimulation of deep brain structures can reduce or completely suppress seizures.[34] In addition, the method about activation of selected neurons was used by Han and Boyden.[35] They used light pulses on genetically targeted neurons not only for activation but also for inhibition of neuronal activity.

All in all, we consider a clustered network that is composed of the matrix of corticocortical connections in the cat with small-world sub-networks using as local dynamics a two-dimensional map to describe the neuronal activity. We build small-world sub-networks according to the procedure proposed by Newman and Watts,[36] who inserted randomly chosen shortcuts in a regular network. With the objective of finding an effective way of suppression of neuronal synchronisation, we study 3 methods: external time-periodic signal, neuron control with light, and time-delayed feedback signal. One of our main numerical results is to show that the time-delayed feedback signal is more effective than the control with light and periodic signal in the cat's cerebral cortex. We also verify that a perturbation in the frontolimbic region affects the other regions.

This paper is organised as follows: In Section II, we present the mathematical model of the neuronal network. Section III shows the burst phase synchronisation. Section IV exhibits our numerical results with the different methods of suppression of neuronal phase synchronisation. In Section V, we draw our conclusions.

II. CLUSTERED NETWORK OF RULKOV NEURONS

We consider, as neuronal model, the Rulkov map[15] that reproduces neuronal bursting by means of two variables and is given by

$$\begin{aligned} x_{n+1} &= \frac{\alpha}{1+x_n^2} + y_n, \\ y_{n+1} &= y_n - \sigma(x_n - \rho), \end{aligned} \tag{1}$$

where x_n is the fast dynamical variable, y_n is the slow dynamical variable, α controls the duration of bursts, and σ and ρ describe the slow time-scale. Figure 1 exhibits an irregular sequence of bursts of the fast variable, where n_k denotes when the neuronal bursting starts, and k is an integer.

In accordance with the matrix that describes the corticocortical connectivity of the cat's brain, given by Scannell *et al.*,[4] we build a clustered neuronal network. Figure 2 shows the densities of connections by means of colours, where we can see white for no connections, sparse connections in red, intermediate connections in blue, and dense connections in green. Each one of the 65 areas is modelled by a small-world network with 100 neurons and 5% of shortcuts.

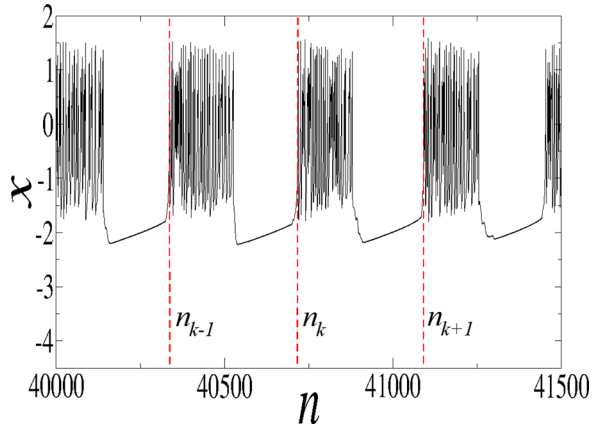

FIG. 1. Time evolution of the fast variable in the Rulkov map (Eqs. (1)), where n_k denotes when a new bursting starts, and k is an integer.

The cortical areas classified as sparse (red) have 50 randomly directed connections. The intermediate connectivities (blue) and the dense connectivities (green) have 100 and 150 randomly directed connections, respectively.

The areas are separated into 4 cognitive regions: visual, auditory, somatosensory-motor, and frontolimbic. The visual region is composed of 18 cortical areas and the auditory of 10 areas, while the somatosensory-motor and the frontolimbic present 18 and 19 cortical areas, respectively. The values of percentages, in Figure 2, describe the amount of connections between regions in relation to the total connectivity in the matrix. For instance, inside the visual region, there is 16.62% of the total connectivity, and the percentage of connections to the visual region from the auditory region is equal to 1.34%. The frontolimbic region has the largest amount of connections in the cat matrix.

In this work, the dynamics of the clustered network is based on the cat matrix and given by[19]

$$x_{n+1}^{(i,p)} = \frac{\alpha^{(i,p)}}{1+\left(x_n^{(i,p)}\right)^2} + y_n^{(i,p)} + \frac{g_e}{2}\left(x_n^{(i-1,p)} + x_n^{(i+1,p)} - 2x_n^{(i,p)}\right) - g_c \sum_{d=1}^{Q}\sum_{f=1}^{P}\left[A_{(d,f),(i,p)} H\left(x_n^{(d,f)} - \theta\right)\left(x_n^{(i,p)} - V_s\right)\right] + \Lambda_n, \tag{2}$$

$$y_{n+1}^{(i,p)} = y_n^{(i,p)} - \sigma\left(x_n^{(i,p)} - \rho\right), \tag{3}$$

where (i, p) denotes the neuron i ($i = 1, 2, \ldots, Q$) in the cortical area p ($p = 1, 2, \ldots, P$), $Q = 100$ and $P = 65$ are the quantity of neurons in each small-world subnetwork and the number of cortical areas, respectively. The third term of the first equation corresponds to the electrical coupling with strength g_e, and the fourth term is the chemical coupling with strength g_c. In the chemical coupling term, the chemical connection between one neuron (i, p) and another neuron (d, f) is given by the adjacency matrix $A_{(d,f),(i,p)}$. In addition, $H(x)$ is the Heaviside step function, where $\theta = -1.0$ is the presynaptic threshold for the chemical synapse, and V_s is the reversal potential. In our

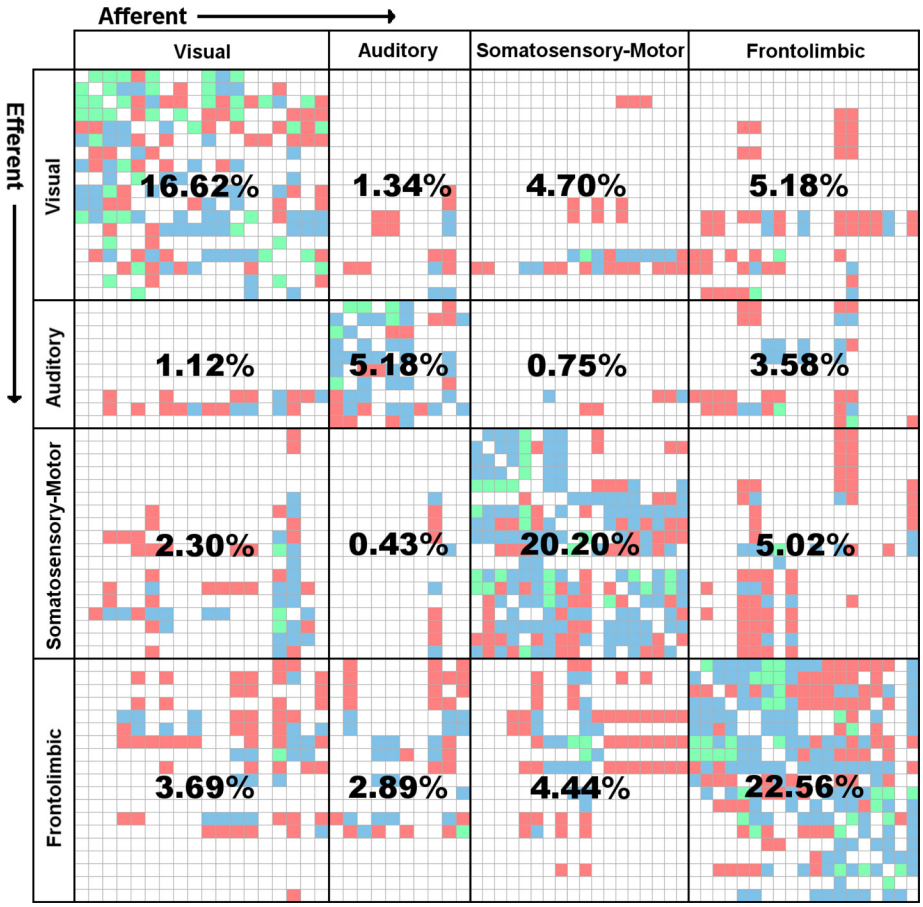

FIG. 2. Density of connections between cortical areas classified as absence of connection (white), sparse (red), intermediate (blue), and dense (green). The percentages correspond to the amount of intra- and interconnections in relation to the total connectivity matrix.

simulations, we have considered that the $\alpha^{(i,p)}$ are randomly distributed in the interval [4.1, 4.4], $\sigma = 0.001$, $\rho = -1.25$, $\theta = -1.0$, $V_s = 1.0$ for excitatory synapses, $V_s = -2.0$ for inhibitory, and 3 different methods for the perturbation Λ_n. With regard to the synapses, we consider that the electrical synapses are the connections between the nearest neighbours inside the small-world networks, while the chemical synapses are the shortcut connections inside the small-world and connections between areas. We have also considered 75% of excitatory and 25% of inhibitory chemical synapses in all the networks.

III. NEURONAL PHASE SYNCHRONISATION

In the following, we calculate the neuronal phase by means of the time evolution within each burst, varying from 0 to 2π as n evolves from n_k to n_{k+1} (Fig. 1):

$$\phi_n = 2\pi k + 2\pi \frac{n - n_k}{n_{k+1} - n_k}. \tag{4}$$

Through the phase, we compute the Kuramoto's order parameter R_n to check the synchronous behaviour,[37] which is given by

$$z_n^{(l)} = R_n^{(l)} \exp\left(\mathrm{i}\Phi_n^{(l)}\right) \equiv \frac{1}{N_l} \sum_{j \in I_l} \exp\left(\mathrm{i}\phi_n^{(j,I_l)}\right), \tag{5}$$

where R_n and Φ_n are the amplitude and the angle of a centroid phase vector, respectively. I_l denotes the cognitive areas, with $l = 1$ for visual, $l = 2$ for auditory, $l = 3$ for somatosensory-motor, and $l = 4$ for frontolimbic. N_l corresponds to the number of neurons of each area. The phase of the neurons j, in the cortical area I_l, is denoted by ϕ_n^{j,I_l}. The order parameter is equal to 1 for a completely synchronous behaviour and much less than 1 for uncorrelated phases.

The phase synchronisation as a function of the coupling strength can be analysed by means of the time average order parameter, given by

$$\bar{R} = \frac{1}{n_{\text{final}} - n_{\text{initial}}} \sum_{n_{\text{initial}}}^{n_{\text{final}}} R_n, \tag{6}$$

where $n_{\text{final}} - n_{\text{initial}}$ is the time window for measurements.

Figure 3 shows $\bar{R}$ as a function of the chemical coupling strength g_c for each cortical region, where the value of the electrical coupling $g_e = 0.05$. We see that the values of saturation of $\bar{R}$ for the visual and the somatosensory-motor regions are larger than those for the auditory and the frontolimbic regions. The auditory region presents a small percentage of intraconnections, and due to this fact, it is not possible to observe a strong synchronised state. Consequently, $\bar{R}$ saturates at a value smaller than 0.8. In addition, we also observe that all transitions are of second-order.

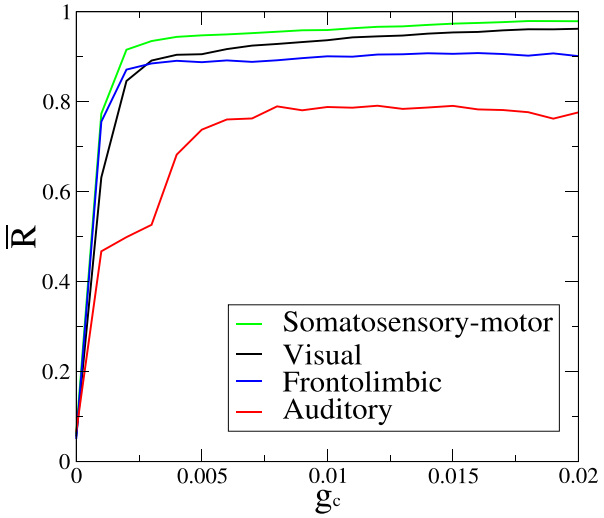

FIG. 3. Average order parameter $\bar{R}$ (Eq. (6)) of the cat's cognitive brain regions as a function of the chemical coupling strength g_c for a fixed electrical coupling $g_e = 0.05$. We consider 50 000 iterations where the first 20 000 were excluded as transient.

IV. SUPPRESSION OF NEURONAL PHASE SYNCHRONISATION

Strong neuronal synchronisation can be associated with brain disorders, such as epilepsy and Parkinson's disease. Due to this fact, we have studied methods of suppression of neuronal phase synchronisation. As a diagnostic tool of suppression, we use the suppression factor S,[38] given by

$$S = \sqrt{\frac{\mathrm{Var}(M_n)}{\mathrm{Var}(M_n^p)}}, \tag{7}$$

where Var() is the variance, M_n and M_n^p are the mean fields of the fast dynamical variable x_n in the absence and presence of the control, respectively. The variance of M_n^p is small when synchronisation is suppressed; consequently, the suppression factor S is strongly increasing theirs. Nevertheless, S has a value approximately equal to one when the control is not efficient to suppress synchronisation.

We have considered three methods of suppression known in the literature: delayed feedback control, external time-periodic driving, and activation of selected neurons.

(i) In the delayed feedback control, the last term in Eq. (2) has the following form:

$$\Lambda_n = \frac{1}{N_l} \sum_{(i,p) \in I_l} x_{n-\tau}^{(i,p)}, \tag{8}$$

where N_l is the total number of neurons of the cognitive area l in that the mean field is calculated, τ represents the number of iterations before, and the control is only applied in one of the four cognitive areas to neurons randomly chosen at each iteration.

(ii) With regard to external time-periodic driving, we consider

$$\Lambda_n = I \sin(\omega n), \tag{9}$$

where $I = 1$ and $\omega = 1$ are the amplitude and frequency of the perturbation, respectively. The time-periodic driving is also applied on the 100 neurons randomly chosen in the cognitive area.

(iii) The third method is similar to light stimulation; in other words, a neuron receives a light pulse and goes

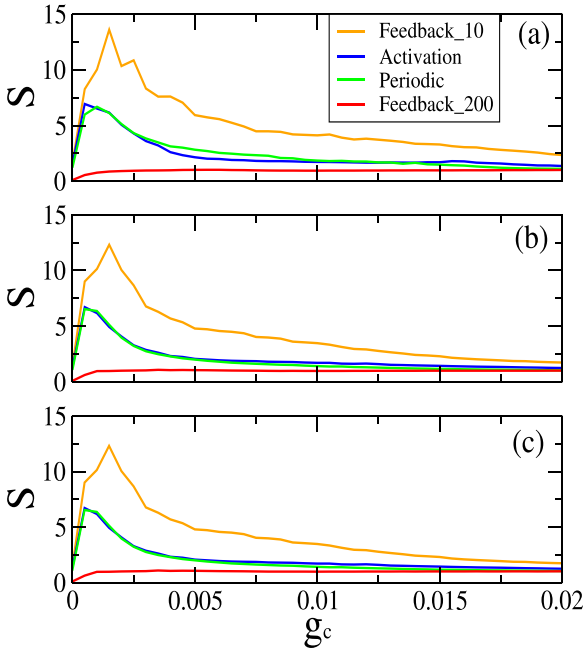

FIG. 4. Suppression factor S calculated for the area where the controls are applied as a function of the chemical coupling strength g_c for $g_e = 0.05$ and the different controls applied on (a) the visual, (b) the auditory, and (c) the somatosensory-motor areas.

to a state where it is forced to spike. In our simulations, 100 randomly chosen neurons are activated ($x_n = 1.2$) in a specific cognitive area when the perturbation is applied.

In Figure 4, the suppression factor S is calculated for the controls applied on (a) the visual, (b) the auditory, and (c) the somatosensory-motor regions by varying the chemical coupling strength g_c. The feedback is represented by orange and red lines for τ equal to 10 and 200 iterations, respectively. The external time-periodic driving is denoted by a green line, whereas the activation of the selected neurons is represented by a blue line. Our results show that the feedback control with τ equal to 200 does not produce a suppression of the synchronisation. Conversely, for $\tau = 10$, for activation, and for time-periodic driving, it is possible to get a suppression with $S > 2$ for $g_c < 0.01$. The activation and the time-periodic driving have approximately the same behaviour of the suppression factor. We also verify that the feedback with $\tau = 10$ presents the largest value of S. In addition, the other cognitive areas do not suffer a significant effect from these controlled areas (visual, auditory, and somatosensory-motor); in other words, the unperturbed areas remain in a synchronised state.

In the same way that the controls are applied on the visual, the auditory, and the somatosensory-motor, we apply them on the frontolimbic area. Unlike the cases before, the uncontrolled cognitive areas show a small influence from the suppression in the frontolimbic area. S has its maximum value approximately equal to 2 in a small range of g_c, as shown in Figure 5 for the visual, the auditory, and the somatosensory-motor areas. In the frontolimbic area, the values of S have a behaviour similar to that obtained in Figure 4. In fact, the feedback with $\tau = 10$ exhibits the best efficiency compared with the activation, the time-periodic driving, and with $\tau = 200$.

In view of our results from the delayed feedback control better than the activation and time-periodic driving, we analyse how S is affected by the time delay τ and the percentage of perturbed neurons N_p. In Figure 6, we see S in the bar for the number of perturbed neurons versus τ, where the delayed feedback is applied on (a) the visual, (b) the auditory, (c) the somatosensory-motor, and (d) the frontolimbic areas. The black regions correspond to the case in which the cognitive area does not present a suppression of synchronisation ($S \leq 1$), the grey regions exhibit a small suppression of synchronisation with the suppression factor in the interval $1 < S \leq 3$, and the white regions show the values of N_p and τ where the feedback delayed control is more efficient ($S > 3$). As a result, we verify that the method by means of feedback delayed control is efficient not only for different N_p values but also for small time delays.

The time evolution of the mean field for the black region in Figure 6 exhibits large amplitude oscillations, as a result of the synchronised behaviour. In the white region, where there is no evidence of synchronisation, the mean field has small amplitude oscillations. Oscillation quenching has been investigated in systems of coupled nonlinear oscillators,[22] that are classified in: oscillation (OD) and amplitude death (AD). In our simulations, we have verified that the 3 methods of suppression can induce a mean field amplitude death (MFAD). This way, S can be used as a diagnostic tool of

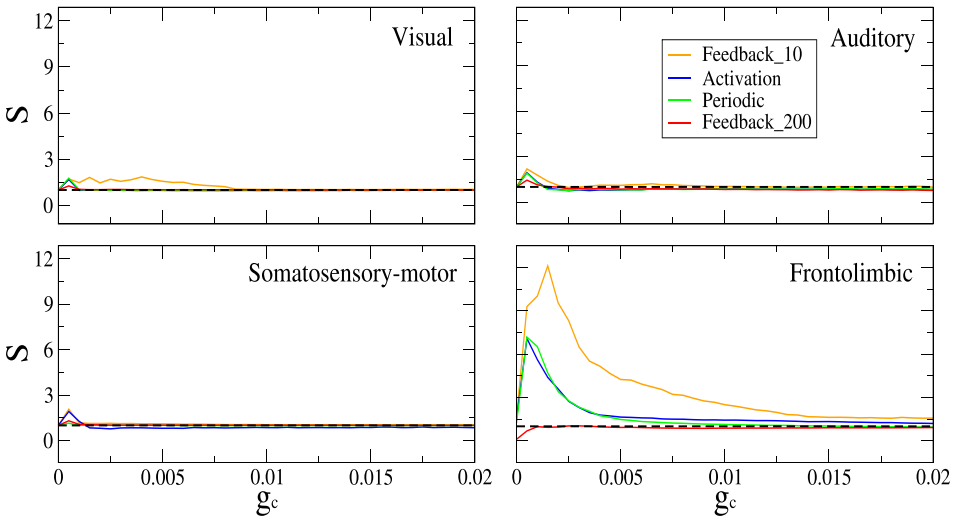

FIG. 5. Suppression factor as a function of the chemical coupling strength for (a) the visual, (b) the auditory, (c) the somatosensory-motor, and (d) the frontolimbic areas, where the controls are applied on the frontolimbic area and $g_e = 0.05$.

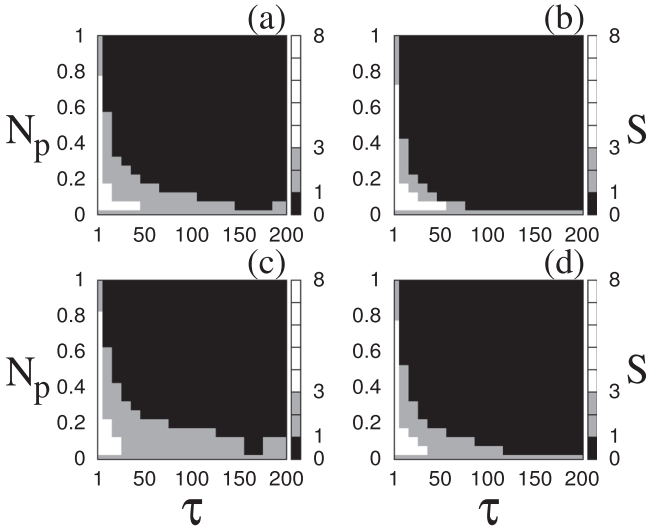

FIG. 6. Suppression factor in the bar for the percentage of perturbed neurons N_p versus the time delay τ, where we consider $g_e = 0.05$ and $g_c = 0.005$. Delayed feedback control applied on (a) the visual, (b) the auditory, (c) the somatosensory-motor, and (d) the frontolimbic areas.

MFAD and enables, in future works, studies about MFAD in networks with other topologies and local dynamic models.

V. CONCLUSIONS

In this work, we have studied suppression of burst phase synchronisation in a neuronal network with a structure according to the corticocortical connections of the cat's brain. We have considered the cat matrix, where each cortical area has a sub-network with small-world properties.

Considering an initial configuration in that the neuronal network presents a synchronous behaviour, we have applied and compared three different suppression methods: delayed feedback control, external time-periodic driving, and activation of neurons. As a result, we verify that it is possible to obtain suppression by means of the three methods. The methods produce suppression only in the cortical areas where they are applied, except when they are applied on the frontolimbic area. We observe a small suppression in the other areas when the perturbations are applied on the frontolimbic area. This occurs due to the fact that the frontolimbic area has a larger external connectivity than the other areas.

In our simulations, using the suppression factor as a diagnostic tool, the delayed feedback control has shown the best efficiency compared with the external time-periodic driving and the activation of neurons. In addition, we have verified that the delayed feedback is better for small values of the time delay in a large range of the number of controlled neurons. The delayed feedback control does not damage the neurons due to the fact that it uses a signal amplitude coming from by neuronal activity.

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[1]G. Buzsaki, *Rhythms of the Brain* (Oxford University Press, Oxford, 2006).
[2]J. S. Lund, T. Yoshioka, and J. B. Levitt, "Comparison of intrinsic connectivity in different areas of macaque monkey cerebral," Cereb. Cortex **3**, 148–162 (1993).
[3]E. J. Izquierdo and R. D. Beer, "Connecting a connectome to behavior: An ensemble of neuroanatomical models of C. elegans klinotaxis," PLoS One Comput. Biol. **9**, e1002890 (2013).
[4]J. W. Scannell, C. Blakemore, and M. P. Young, "Analysis of connectivity in the cat cerebral cortex," J. Neurosci. **15**, 1463–1483 (1995).
[5]J. W. Scannell and M. P. Young, "The connectional organization of neural systems in the cat cerebral cortex," Curr. Biol. **3**, 191–200 (1993).
[6]J. Gómez-Gardeñes, G. Zamora-López, Y. Moreno, and A. Arenas, "From modular to centralized organization of synchronization in functional areas of the cat cerebral cortex," PLoS One **5**, e12313 (2010).
[7]C. C. Hilgetag, G. A. Burns, M. Oneill, J. W. Scannell, and M. P. Young, "Anatomical connectivity defines the organization of clusters of cortical areas in the macaque monkey and the cat," Philos. Trans. R. Soc. London B **355**, 91–100 (2000).
[8]C. Zhou, L. Zemanová, G. Zamora, C. C. Hilgetag, and J. Kurths, "Hierarchical organization unveiled by functional connectivity in complex brain networks," Phys. Rev. Lett. **97**, 238103 (2006).
[9]L. Zemanová, C. Zhou, and J. Kurths, "Structural and functional clusters of complex brain networks," Physica D **224**, 202–212 (2006).
[10]D. Watts, J. Duncan, and S. H. Strogatz, "Collective dynamics of small-world networks," Nature **393**, 440–442 (1998).
[11]C. J. Stam, B. F. Jones, G. Nolte, M. Breakspear, and Ph. Scheltens, "Small-world networks and functional connectivity in Alzheimer's disease," Cereb. Cortex **17**, 92–99 (2007).
[12]T. I. Netoff, R. Clewley, S. Arno, T. Keck, and J. A. White, "Epilepsy in small-world networks," J. Neurosci. **24**, 8075–8083 (2004).
[13]A. L. Hodgkin and A. F. Huxley, "A quantitative description of membrane current and its application to conduction and excitation in nerve," J. Physiol. **117**, 500–544 (1952).
[14]J. L. Hindmarsh and R. M. Rose, "A model of neuronal bursting using three coupled first order differential equations," Proc. R. Soc. London B **221**, 87–102 (1984).
[15]N. F. Rulkov, "Regularization of synchronized chaotic bursts," Phys. Rev. Lett. **86**, 183–186 (2001).
[16]B. Ibarz, J. M. Casado, and M. A. F. Sanjuán, "Map-based models in neuronal dynamics," Phys. Rep. **501**, 1–74 (2011).
[17]M. Dhamala, V. K. Jirsa, and M. Ding, "Transitions to synchrony in coupled bursting neurons," Phys. Rev. Lett. **92**, 028101 (2004).
[18]C. A. S. Batista, E. L. Lameu, A. M. Batista, S. R. Lopes, T. Pereira, G. Zamora-López, J. Kurths, and R. L. Viana, "Phase synchronization of bursting neurons in clustered small-world networks," Phys. Rev. E **86**, 016211 (2012).
[19]E. L. Lameu, F. S. Borges, R. R. Borges, A. M. Batista, M. S. Baptista, and R. L. Viana, "Network induces burst synchronisation in cat cerebral cortex," Commun. Nonlinear Sci. Numer. Simul. **34**, 45–54 (2016).
[20]E. L. Lameu, C. A. S. Batista, A. M. Batista, K. Iarosz, R. L. Viana, S. R. Lopes, and J. Kurths, "Suppression of bursting synchronization in clustered scale-free (rich-club) neuronal networks," Chaos **22**, 043149 (2012).
[21]Q. Y. Wang, Z. S. Duan, L. Huang, G. R. Chen, and Q. S. Lu, "Pattern formation and firing synchronization in networks of map neurons," New J. Phys. **9**, 383 (2007).
[22]A. Koseska, E. Volkov, and J. Kurths, "Oscillation quenching mechanisms: Amplitude vs. oscillation death," Phys. Rep. **531**, 173–199 (1999).
[23]J. Fell and N. Axmacher, "The role of phase synchronization in memory processes," Nat. Rev. Neurosci. **12**, 105–118 (2011).
[24]P. R. Roelfsema, A. K. Engel, P. König, and W. Singer, "Visuomotor integration is associated with zero time-lag synchronization among cortical areas," Nature **385**, 157–161 (1997).
[25]M. V. Ivanchenko, G. V. Osipov, V. D. Shalfeev, and J. Kurths, "Phase synchronization in ensembles of bursting oscillators," Phys. Rev. Lett. **93**, 134101 (2004).
[26]N. Axmacher, F. Mormann, G. Fernández, C. E. Elger, and J. Fell, "Memory formation by neuronal synchronization," Brain Res. Rev. **52**, 170–182 (2006).
[27]P. J. Uhlhaas and W. Singer, "Neural synchrony in brain disorders: Relevance for cognitive dysfunctions and pathophysiology," Neuron **52**, 155–168 (2006).
[28]C. C. Chen, V. Litvak, T. Gilbertson, A. Kühn, C. S. Lu, S. T. Lee, C. H. Tsai, S. Tisch, P. Limousin, M. Hariz, and P. Brown, "Excessive synchronization of

basal ganglia neurons at 20 Hz slows movement in Parkinson's disease," Exp. Neurol. **205**, 214–221 (2007).
[29]R. Levy, W. D. Hutchison, A. M. Lozano, and J. O. Dostrovsky, "High-frequency synchronization of neuronal activity in the subthalamic nucleus of Parkinsonian patients with limb tremor," J. Neurosci. **20**, 7766–7775 (2000).
[30]M. G. Rosenblum and A. S. Pikowsky, "Delayed feed back control of collective synchrony: An approach to suppression of pathological brain rhythms," Phys. Rev. E **70**, 041904 (2004).
[31]C. A. S. Batista, S. R. Lopes, R. L. Viana, and A. M. Batista, "Delayed feedback control of bursting synchronization in a scale-free neuronal network," Neural Networks **23**, 114–124 (2010).
[32]C. A. S. Batista, R. L. Viana, F. A. S. Ferrari, S. R. Lopes, A. M. Batista, and J. C. P. Coninck, "Control of bursting synchronization in networks of Hodgkin-Huxley-type neurons with chemical synapses," Phys. Rev. E **87**, 042713 (2013).
[33]D. E. J. Linden, I. Habes, S. J. Johnston, S. Linden, R. Tatineni, L. Subramanian, B. Sorger, D. Healy, and R. Goebel, "Real-time self-regulation of emotion networks in patients with depression," PLoS One **7**, e38115 (2012).
[34]R. P. Lesser, "Electrical stimulation depresses epileptiform activity," Epilepsy Curr. **3**, 137–138 (2003).
[35]X. Han and E. S. Boyden, "Multiple-color optical activation, silencing, and desynchronization of neural activity, with single-spike temporal resolution," PLoS One **2**, e299 (2007).
[36]M. E. Newman and D. J. Watts, "Renormalization group analysis of the small-world network model," Phys. Lett. A **263**, 341–346 (1999).
[37]Y. Kuramoto, *Chemical Oscillations, Waves, and Turbulence* (Springer Verlag, Berlin, 1984).
[38]M. G. Rosenblum and A. S. Pikovsky, "Controlling synchronization in an ensemble of globally coupled oscillators," Phys. Rev. Lett. **92**, 114102 (2004).