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Synchronization properties of coupled chaotic neurons: The role of random shared input

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Spike-time correlations of neighbouring neurons depend on their intrinsic firing properties as well as on the inputs they share. Studies have shown that periodically firing neurons, when subjected to random shared input, exhibit asynchrony. Here, we study the effect of random shared input on the synchronization of weakly coupled chaotic neurons. The cases of so-called electrical and chemical coupling are both considered, and we observe a wide range of synchronization behaviour. When subjected to identical shared random input, there is a decrease in the threshold coupling strength needed for chaotic neurons to synchronize in-phase. The system also supports lag–synchronous states, and for these, we find that shared input can cause desynchronization. We carry out a master stability function analysis for a network of such neurons and show agreement with the numerical simulations. The contrasting role of shared random input for complete and lag synchronized neurons is useful in understanding spike-time correlations observed in many areas of the brain. Published by AIP Publishing. [http://dx.doi.org/10.1063/1.4954377]

The manner in which rhythmic neuronal activity is generated in the brain is an issue of continuing interest in neuroscience. While giving insight into the regulation of behaviour, understanding these mechanisms is also important in diagnosis of pathophysiology. A fundamental requirement for generating collective rhythmic activity is that correlations should develop in the spike timings of groups of neurons. Many studies have addressed the synchronization of spiking activity when neurons are coupled physically via their synapses (for both electrical and chemical coupling), when connected on a network, or when subject to external noise. Given the high density, nearby neurons can overlap their dendritic fields and thereby share different inputs. This appears to be an efficient means of inducing correlations in neuronal networks. In this study, we systematically investigate the effects of random shared input on coupled chaotic neurons. The spiking of each individual neuron is intrinsically irregular, and their coupling results in chaotic synchronization. This may be contrasted with the effect of common random noise on regular neurons which are known to develop asynchrony. Neurons in a state of lag synchronization, however, become decorrelated when subjected to common random noise.

I. INTRODUCTION

Neuronal activity in the mammalian brain is characterized by rhythmic oscillations of a number of key quantities, with particular patterns of oscillation being associated with a given behaviour or of a pathological condition.1 Such rhythms can be generated by specific neurons which have sub-threshold resonance for a selective frequency or by specific synaptic connections.2–5 A key requirement for the emergence of these oscillations is correlation in neuronal spike-times.6

One source of rhythmic neuronal activity is from pacemaker neurons that synchronize their voltage fluctuations when coupled by either an electrical (gap junction)7,8 or through a chemical9 synapse. However, this property is exceptional: most of the neurons in the brain do not show pacemaking activity. The irregular (namely, non-pacemaker-like) firing of a neuron can arise from random temporal dynamics of its synaptic inputs10 as well as from the intrinsic properties of the cell.11 Such irregular dynamics has been extensively studied in a variety of theoretical models of neurons, and it has been seen that this latter behaviour can also lead to coherence. Chaotic neuronal models (e.g., the Hindmarsh-Rose (HR) model12) synchronize when coupled electrically and/or chemically. A rich spectrum of synchronization behavior emerges for these couplings.13,14

Here, we investigate the effect of common external inhibitory input on the synchronization properties of electrical and chemical coupled chaotic Hindmarsh-Rose neurons. The motivation for our study arises from the following consideration. Within the brain, neurons are organized in a compact fashion receiving many synaptic inputs on their dendritic fields. These fields necessarily overlap with the dendritic fields of other neurons, thus allowing neurons that are proximal to one another to share common input from individual afferents.15 namely, to experience the same ambient background. Experimental and theoretical studies have demonstrated synchronization when two neurons share identical stochastic inputs without requiring them to have any synaptic or electrical coupling.16,17 Earlier studies have examined synchronization of neurons coupled by gap junctions in the presence of random Gaussian noise,18,19 while shared common input has been shown to cause decorrelation in the
functions are given by
\[ x(t) = g(x(t)) - C_0 x(t), \]
where the superscripts \( a, b \) correspond to the synapse can be efficiently modeled as
\[ h(t) = \delta(t - T), \]
\[ s(t) = \langle x_1(t) x_2(t) \rangle - \langle x_1(t) \rangle \langle x_2(t) \rangle, \]
\[ D_m = \inf_{\tau} D^2(\tau, \varepsilon) \]
provides a suitable measure of the extent of synchrony: if the two signals are uncorrelated, \( D_m \approx 1 \), while if they are identical, then \( D_m \approx 0 \). When \( D_m \approx 0 \), if the corresponding value of \( \tau \) is zero, then one has complete synchronization while if \( \tau \) is nonzero, one has lag synchronization.

The coupled neuronal system is subjected to shared random input that is modeled as a Poisson process. The Poisson timings are generated at a rate \( \lambda \), and the synaptic variable corresponding to the synapse can be efficiently modeled as \( \alpha \)-function\(^{27}\) and is given by
\[ f_2(x_1', x_2') = 1 - 5(x_2')^2 - x_2', \]
\[ f_3(x_1', x_3') = -r x_3' + rs(x_1' - x_3). \]

The coupling function \( h(x_i, x_j) \) in Eq. (1) can represent either an electrical or a chemical synapse. The gap junction is modeled by a simple diffusive coupling of the form \( e(x_i - x_j) \) and the chemical synapse is modeled by fast threshold modulation of the form \( e(x_i - x_{sym})/\{1 + \exp(-2(x_i - \theta))\} \), where \( e \) is the coupling strength. We chose \( \lambda_0 = 0.5 \) and \( \theta = 0.85 \), \( e_{sym} \) is the reversal potential. The value of \( I_{app} \approx 3.281 \) is such that the dynamics of an individual neuron is chaotic (see Fig. 1(a) inset), and we take \( \tau = 0.0015, s = 4 \), and \( x_i = 1.6 \) in our simulations which are carried out using a fourth-order Runge–Kutta algorithm with time step 0.01.

Since our interest in the collective dynamics is in detecting both complete as well as lag synchrony, it is useful to compute the quantity\(^{13,26}\)
\[ D^2(\tau, \varepsilon) = \frac{(\langle x_1^2(t) \rangle - \langle x_1(t) \rangle^2)^2}{(\langle x_1^2(t) \rangle^2 - \langle x_1^2(t) \rangle^2)^2}, \]
where \( \tau \) is a time-shift and \( \langle \rangle \) represents the time average. This is a normalized distance between the two signals, and its minimum value
\[ D_m = \inf_{\tau} D^2(\tau, \varepsilon) \]
represents the time average.
\[ P(t, \tau_{inh}) = \frac{(t - t_k)}{\tau_{inh}} \exp \{1 - [(t - t_k)/\tau_{inh}]\}. \]  

(7)

This synaptic function ignores more complex dynamical transmission involved at the synapses but is useful in studying fast and slow synaptic modulations. Thus, the inhibitory input becomes

\[ I_p(x'_i, \lambda) = G_{inh} \sum_{t_k \leq t} P(t, \tau_{inh}) \Theta(t - t_k) (x'_i - E_{inh}), \]

(8)

where \( i \) labels the oscillators and the \( t_k \) are the random Poisson process arrival times, and \( \Theta(t - t_i) \) is the usual Heaviside step function. The variable \( P(t, \tau_{inh}) \) is essentially the convolution of \( t_k \) with an \( \alpha \)-synapse conductance, representing fast synaptic response for incoming presynaptic spikes. Both neurons are receiving the inputs at identical times \( \theta^i \) and conductance due to these pre-synaptic spikes depends on the nature of the synapse (Eq. (7)) and is multiplied with the voltage to cause a current change in the neuron. We take \( \tau_{inh} = 1 \) and \( E_{inh} = -1.25 \) in the numerical simulations. Both neurons are subject to the same random input, the equations of motion being modified as

\[ x'_i \rightarrow x'_i - I_p(x'_i, \lambda), \quad i = 1, 2. \]  

(9)

The master stability function (MSF) formalism proposed by Pecora and Carroll greatly facilitates further analysis of the effect of shared input on the synchronization of a network of HR neurons coupled by either electrical or chemical synapses. Generalizing to the case of a network of \( N \)-coupled neurons that share a random common input, the equations of motion can be written symbolically as

\[ \dot{x}^i_l = f_i(x', \mu) + I_p(x'_i, \lambda) + \sum_{j=1}^{N} C_{ij} H^i(x'_j), \]

\[ \dot{x}^i_2 = f_2(x', \mu), \]

\[ \dot{x}^i_3 = f_3(x', \mu), \]

(10)

where the superscript \( i \) labels the different nodes of the network, \( x' \) are the variables at the \( n \)th node, and \( \mu \) are the parameters that govern the flow. \( I_p(x', \lambda) \) is the common input arising from the Poisson process of rate \( \lambda \). The topology of the network is encoded in the connectivity matrix, \( C \), the elements \( C_{ij} \) being equal to 0 if nodes \( i \) and \( j \) are not connected, and if they are connected, we choose matrix elements differently for chemical and electrical couplings. For electrical coupling, the matrix \( C \) has Laplacian structure, i.e., \( \sum_j C_{ij} = 0 \). For chemical coupling, the elements corresponding to connected nodes are 1 such that \( \sum_j C_{ij} = k \) for \( k \) connections. The coupling functions \( H^1(x'), H^2(x') \) are simply 1 and \( x'_i \) for the case of electrical coupling. Due to the Laplacian structure of \( C \) in this case, the effective coupling becomes \( x'_i - x'_j \). For electrical synapse, \( H^1(x'_j) = x'_j - E_{syn} \) and

\[ H^2(x'_j) = 1/(1 + e^{\alpha(x'_j - \theta)}). \]  

(11)

The stability of the synchronization manifold, given by the condition \( (x'_1 = x'_2 = \ldots = x'_N = x') \), is determined through the variational equations

\[ \xi^1_1 = D_{1f_1}(x', \mu) \xi^1_1 + D_{2f_1}(x', \mu) \xi^1_2 + D_{3f_1}(x', \mu) \xi^1_3 + D_{1I_p}(x'_1, \xi_p) \xi^1_1 + \cH(x'_1) \xi^1_1, \]

\[ \xi^1_2 = D_{1f_2}(x', \mu) \xi^1_1 + D_{2f_2}(x', \mu) \xi^1_2 + D_{3f_2}(x', \mu) \xi^1_3, \]

\[ \xi^1_3 = D_{1f_3}(x', \mu) \xi^1_1 + D_{2f_3}(x', \mu) \xi^1_2 + D_{3f_3}(x', \mu) \xi^1_3, \]

(12)

where \( \xi^1_i \) are components of the eigenmode corresponding to the \( j \)th eigenvalue of the matrix \( C \). If the matrix \( C \) has Laplacian structure, as is the case for electrical coupling, then

\[ \Gamma(x'_1) = \gamma D_{1H^2}(x'_1), \]

(13)

where \( \gamma \) are the eigenvalues of the Laplacian. For the case of chemical coupling, it is convenient to first expand the connectivity matrix in Laplacian form, then

\[ \Gamma(x'_1) = k \tilde{\gamma} H^1(x'_1) D_{1H^2}(x'_1), \]

(14)

where \( k \) is the sum for each row of the matrix \( C \), and \( \{ \tilde{\gamma} \} \) are the eigenvalues of the Laplacian \( G \) associated with the matrix \( C \). For the symmetric matrix \( C (G) \) in Eq. (10), the eigenvalues \( \{ \gamma \} \) (resp. \( \{ \tilde{\gamma} \}) \) are all real.

The stability of the synchronization manifold is then governed by the largest Lyapunov exponent for each \( i \) in Eq. (12) as has been discussed earlier. If the Lyapunov exponent transverses to the synchronization manifold \( \lambda_s \), namely, the Master Stability Function is less than zero, the synchronization is stable.

### III. RESULTS

We first consider the case of two coupled chaotic HR neurons with mutual coupling (electrical and chemical synapses) in the absence of any external input. We then subject the neurons to shared input and study the synchronization as a function of input characteristics. We also discussed the stability of synchronization manifold using master stability function.

Baptista et al. have shown that in the absence of noise, for a combination of large excitatory, small electrical, and large inhibitory, large electrical synapse strengthens the neurons to achieve complete synchronization. However, other studies have investigated the role of Poisson noise in a network of heterogeneous pacemaker neurons. The neurons show one-to-one phase locked state, in the absence of noise for sufficiently strong coupling (both excitation and inhibition), and the spike separation between locked spike pairs depends on the frequency heterogeneity. The synchrony is lost when the network is subjected to shared input.

### A. Electrical coupling

When the two neurons are coupled by electrical synapses (or gap junctions), the dynamics synchronizes as a function of increasing coupling. Shown in Fig. 1(a) is the distance measure \( D_m \) as a function of the coupling strength,
indicating synchrony for $\varepsilon$ above a threshold $\varepsilon_{th} \approx 0.55$ as well as a narrow region around $\varepsilon \sim 0.2$. The synchronization in this latter region has non-zero $s$, the two neurons being out of phase with one another, although with identical dynamics. Above $\varepsilon_{th}$, the synchrony is in-phase, namely, $s = 0$.

**Shared random input** is introduced in the network as discussed in Section II. The results as shown in Fig. 2 are for fixed $G_{inh} = 0.5$, and the most direct observation is that with increasing the rate of the Poisson input, $\lambda$, the threshold coupling needed for synchronization, $\varepsilon_{th}$, decreases linearly. Note that this only results in completely synchronization (see Fig. 3). Indeed, for $\lambda > 0.9$, $\varepsilon_{th}$ becomes zero, showing that the shared input is alone sufficient to synchronize the neurons even when they are not coupled.

The master stability function for electrical coupled neurons receiving shared input, shown in Fig. 4, confirms the validity of these general observations. The stability shifts towards lower values of $\varepsilon$ as the input frequency $\lambda$ is increased. One can also compute $\varepsilon_{th}$ as a function of $\lambda$ (Fig. 2 inset) for a fixed value of the input strength and as shown it decreases linearly with the increase in $\lambda$. We also examined the effect of $G_{inh}$ on the synchronizability of gap junction coupled neurons (Fig. 5). For small input frequency, we need large $G_{inh}$ for synchronization to occur. Fig. 5 suggests that both the coupling strength and the frequency of the shared input play an important role in the synchronization properties of gap junction coupled neurons. This result emphasizes the importance of characteristics of shared input in modulating synchronization properties of chaotic neurons and could act as an efficient mechanism underlying in the generation of fast oscillations observed in the olfactory bulb neurons.

With the increase in coupling strength $\varepsilon$, the HR model exhibits several periodic and chaotic attractors which can also coexist. These attractors correspond to neurons being synchronized with regular spiking and irregular spiking, respectively. When subject to random synaptic inputs generated through Poisson timing convolved with synaptic $\alpha$-function (Eq. (7)), the neurons achieve stochastic resonance with a preferred frequency that depends on the rate...
of input Poisson timings (Fig. 5 in Ref. 33). This appears to cause the synchronization manifold to become stable for smaller values of $e_{th}$ and hence the decrease in $e_{th}$ with increase in $\lambda$ and $G_{inh}$ as shown in Figs. 2 and 5.

We also studied the role of shared input when the neurons are connected on a network. Fig. 6 shows the synchronization of a network of $n = 10$ neurons with all-to-all coupling and shared input. The parameters for each neuron are taken to be same as for the two coupled neurons in Fig. 2. We use average synchronization error $E_s$, given by $E_s = T^{-1} \sum_{t=0}^{T} e(t)$, where $e(t) = \frac{2}{N(N-1)} \sum_{i<j} |x_i^t - x_j^t|^2$ and $N$ is the total number of neurons in the network as a measure of network synchronization (Fig. 6). It should be noted that average synchronization error predicts same complete synchronization regimes as the distance measure for two coupled neurons. For the case of $n = 10$ coupled neurons, as described above, we find that the value of threshold coupling $e_{th}$ decreases with increase in the input frequency $\lambda$. The decrease in the value of $e_{th}$ is more rapid in a large network (cf. Figs. 2 and 6).

B. Chemical coupling

Chemical coupling is modeled by different coupling functions in comparison to the electrical case, and thus, the results are significantly different. Neurons coupled with chemical synapse display a variety of synchronization properties. Some studies suggest that a delicate balance between chemical and electrical synapse is needed to achieve complete synchronization. Here, we chose individual neurons exhibiting chaotic behavior, but the coupling leads to different regimes of the dynamics. We have therefore investigated the effect of shared random input on chaotic neurons for specific coupling corresponding to either electrical or chemical synapse, but not both forms of coupling simultaneously.

1. Complete synchronisation

Both excitatory and inhibitory chemical coupling can cause chaotic neuronal systems to display complete synchronization for chemical coupling. The effect of shared random input on a model chemical synapse is shown in Fig. 7.

In both excitatory and inhibitory synapses, increasing the frequency of the input induces synchronization for lower coupling $e$ as observed in the case of electrical coupling. The decrease in the threshold coupling $e_{th}$ is more rapid for inhibition in comparison to excitation, and a similar trend is observed for the dependence of the threshold on the input strength (data not shown here).

The role of shared input on coupled neurons using master stability function analysis is presented next. For fixed values of the input frequency $\lambda$ and strength $G_{inh}$, the master stability function, $\lambda_{msf}$, as a function of coupling strength $e$, is shown in Figs. 8(a) and 8(b) for excitation and inhibition coupled neurons, respectively. The $\lambda_{msf}$ fluctuates around

![FIG. 6. Network synchronization of all-to-all coupled (gap junction coupling) chaotic Hindmarsh-Rose neurons. The average synchronization error $E_s$ for shared input of different Poisson frequencies is shown. Here, we keep $G_{inh} = 0.5$ fixed for each neuron.]

![FIG. 7. The effect of shared random input $I_{inh}$ on excitatory and inhibitory coupled neurons. Distance measure $D_m$ as a function of coupling strength $e$ between the excitatory (a) and inhibitory (b) neurons. In both cases, complete synchronization occurs for lower values of $e$ by increasing $\lambda$. Parameters were fixed at $G_{inh} = 2$, $e_{syn} = 0.5$ for excitation and $e_{syn} = -0.5$ for inhibition.

![FIG. 8. The MSF for (a) excitatory (b) inhibitory coupling with fixed input strength $G_{inh} = 2$ and input rate $\lambda = 0.25$. (c) and (d) The threshold coupling as a function of Poisson input rate $\lambda$ when the shared input strength is $G_{inh} = 2$. Black curve is obtained by directly plotting the orbits, while red curve is obtained using the distance function $D_m$.]

zero, showing multiple regions of \( \varepsilon \) for which the neurons show stable synchronization, but these regions are small and for a particular threshold value \( \varepsilon_{th} \), the master stability function is negative. The threshold values calculated using both \( \lambda_{mf} \) (black curve) and \( D_m(\varepsilon, \tau) \) (red curve) are in close agreement (Figs. 8(c) and 8(d)).

2. Lag synchronisation

Physiological correlates of neuronal signals between different brain regions imply coordinated interaction that leads to coherent perception. These correlates include synchronous spiking, spike-to-spike phase locking between neurons and correlations among subthreshold oscillations, local field potential, and EEG. Phase synchronization of membrane potential does not necessarily mean spike synchronization and the reverse is true in the weakest sense. In lag-synchronization, the membrane potentials of two neurons, \( v_1(t) \) and \( v_2(t) \), track each other with a time-lag, i.e., \( v_1(t) = v_2(t-T) \) for some nonzero \( T \). This is also incorporated in the order parameter \( D_m \) described in Section II.

Coupled chaotic neurons can exhibit lag-synchronization. The mechanism has been extensively explored and it is known that both electrical and chemical coupling can lead to lag synchrony (Fig. 9): the distance measure \( D_m(\tau, \varepsilon) \) is zero (see the figure inset) for \( \tau_{min} \neq 0 \). For chemically coupled neurons, we used different set of parameters (\( \tau \approx 0.0021 \) and \( \varepsilon_{syn} = 0 \) for excitation and \( \varepsilon_{syn} = -1.25 \) for inhibition); mutual excitation gives complete synchronization and mutual inhibition gives lag synchronization (Figs. 10(a) and 10(c) for the voltage traces).

In contrast to complete synchronization, the shared random input plays different roles in the case of lag-synchronization as can be seen in Fig. 10. The coupled excitatory neurons show complete synchronization for no input (Fig. 10(a)), but the introduction of random input retains the complete synchronization (Fig. 10(b); note \( G_{inh}=0.5 \)). The effect of increasing input frequency \( \lambda \) follows the same trend as that shown for electrical synapse: \( \varepsilon_{th} \) decreases with increase in \( \lambda \) (Fig. 11(c)). On the contrary, mutually inhibitory neurons, which show lag-synchronization in the absence of input (Fig. 10(c)), become desynchronized for the same shared input (Fig. 10(d)). Unsynchronized neurons at some fixed \( \lambda \) and \( G_{inh} \) (Fig. 11(b)) attain in-phase synchronization (Fig. 11(d)) with large values of input frequency \( \lambda \). Similar results can be obtained for gap junction coupled neurons in lag-synchrony (around \( \varepsilon \approx 0.2 \) in Fig. 2).

IV. CONCLUSION

In this work, we have investigated the role of shared random input on the synchronization of chaotic HR neurons when coupled by either electrical or chemical synapse. In particular, we study the effect on complete and lag-synchronization shown by both coupling schemes. For gap junction coupled neurons, complete synchronization occurs in the chaotic regime while for chemical coupling synchrony

FIG. 9. The distance measure for coupled chaotic HR neurons. For mutual inhibition (\( \varepsilon_{syn} = -1.25 \)) (black dots) and electrical synapse (red dots) at \( \varepsilon \approx 0.2 \), the neurons show lag synchronization. \( \varepsilon \) as a function of \( \varepsilon \) is shown in the inset. The voltage traces for \( \varepsilon = 1 \) for chemical and \( \varepsilon = 0.2 \) for gap junction are also shown.

FIG. 10. The role of shared input on mutually excitatory and inhibitory neurons. (a) Complete synchronization of two mutually excitatory neurons \( (G_{inh} = 0) \), (b) the shared inputs retain the complete synchronization for mutual excitation \( (G_{inh} = 0.5) \), (c) lag synchronization shown by mutually inhibitory neurons without input \( (G_{inh} = 0) \), and (d) shared input causing desynchronization in case of mutual inhibition \( (G_{inh} = 0.5) \).

FIG. 11. The effect of shared random input on chemically coupled chaotic HR neurons. Mutually excitatory (a) and inhibitory (b) neurons for with and without shared input. (c) \( D_m(\varepsilon) \) as a function of coupling strength \( \varepsilon \) for two different \( \lambda \) values. (d) The variation of \( D_m(\varepsilon) \) with \( \lambda \) for a fixed value of \( G_{inh} \) and \( \varepsilon \). Here, we used \( G_{inh} = 0.5 \), \( \lambda = 0.2 \) for (a) and (b). In this case, for excitation, we used \( \varepsilon_{syn} = 0 \) and for inhibition \( \varepsilon_{syn} = -1.25 \).
it is known to occur in the non-chaotic regime. The shared random input induces irregular firing patterns in both cases and plays a critical role in synchronization. Our study found that the shared inhibitory input decreases the threshold coupling for complete synchronization and induces asynchronous behavior for lag synchronization.

The form of shared random input considered in this work was previously investigated in the case of conductance based models showing regular firing where it was seen that the shared input in chemically coupled spike-to-spike phase locked neurons induced desynchronization. Regularly firing pacemaker neurons coupled by chemical synapses were considered; a more realistic neuronal model would include their chaotic irregular firing as well when the oscillators are coupled both by electrical and chemical synapses.

In contrast in our system, the shared random input facilitates synchronization in both cases. We also show that in the case of complete synchronization, this facilitation occurs by shifting the stability of synchronization manifold to lower values of the coupling strength between the neurons. The effect of Poisson noise on the collective properties of neuronal systems with regular firing has been investigated in a population of identical uncoupled neurons. The neurons synchronize with the increase in noise strength and the inter-impulse interval. In these studies, the system receives inputs only at Poisson timings. In contrast, the ambient input considered in this study is a cumulative sum of Poisson timings convolved with a-synapse and multiplied with membrane potential to cause a current change. Chaotic synchronization observed in our study also shows a similar trend with that of regularly firing; however, the mechanism may differ.

In real neural networks, the input experienced by adjacent neurons in real neural networks can be anywhere from identical to completely uncorrelated. The spiking correlations of recurrent networks have been investigated in Refs. and where they show that in such networks, the spiking correlations are low although a substantial portion of the input is shared. The simultaneous activation of excitatory and inhibitory inputs cancels each other and thus leads to low spiking output. Here, we are concerned with identical inhibitory shared input and its effect on the synchronization properties of coupled chaotic neurons. Our results indicate that identical shared input can accelerate synchronization if the synaptic coupling induces strong output correlations, and it causes de-synchronization in case the synaptic coupling induces anti-phase synchronization.

There is contrasting behaviour for lag-synchronization. In electrically coupled neurons, the lag-synchronization manifold loses stability with shared input, and the neurons become desynchronized. In a network with fast inhibition, the lag-synchronization is a result of competition between the two neurons: activation of one cell inhibits the other and vice-versa. Any small perturbation such as shared input in this case removes the competition, thus leading to desynchronization. Many mechanisms for anti-phase synchronization of inhibitory neurons have been proposed. Belykh and Shilnikov have shown that coupled inhibitory neurons achieve complete synchronization in a network that is driven by weak common inhibition. The lag-synchronization presented in this paper is similar to anti-phase solution of Ref. the shared input disrupts the escape mechanism, thereby leading to desynchronization.

The manifestation of this contrasting role of shared input has also been illustrated in a small network with all-to-all coupling. The same qualitative behavior obtains in general: random shared input accelerates the synchronization in case of complete synchronization while for lag-synchronization, spike-time decorrelation occurs. The present results give useful insight in understanding the in-phase and lag-synchronization by inhibition that is observed in a number of model systems, good examples being the central pattern generators of lobster stomatogastric ganglion cells and the active decorrelation of basal ganglia neurons. The shifting of the threshold coupling for synchrony to smaller coupling is found to be valid for shared random input as generated by a Poisson process convolved with the a synapse.

Although our results are for HR model neurons, we believe that they will hold more generally. For example, the basic behaviour of HR neurons is oscillatory with chaotic bursts as a perturbation. Chaotic systems such as the Rössler oscillator and similarly derived neuronal models show similar behaviour vis-a-vis synchronization. It should be noted that the MSF of the Rössler equations and HR neurons fall in the same category. Clearly though, some features will be specific to this model, as, for example, transitions in Figs. (c) and (d).

Other forms of noise (uniform, 1/f or Gaussian) need to be studied in detail; the results of a forthcoming study will be reported elsewhere. It will also be of interest to study the role of such common input to a heterogeneous network of population having both electrical and chemical synapse, and to examine the role of network topology.

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