Diversity-induced antiresonance

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We analyze the effect of small-amplitude noise and heterogeneity in a network of coupled excitable oscillators with strong time scale separation. Using mean-field analysis, we uncover the mechanism of a new nontrivial effect — diversity-induced antiresonance (DIAR) — in which heterogeneity modulates the mechanism of self-induced stochastic resonance to inhibit the coherence of oscillations. We argue that DIAR may offer one possible mechanism via which, in excitable neural systems, generic heterogeneity and background noise can synergistically prevent unwanted resonances that may be related to hyperkinetic movement disorders.

I. INTRODUCTION

The role of disorder in the dynamics of complex networks has been extensively studied in terms of noise and diversity (i.e., heterogeneity) effects [1–6]. The relevance of heterogeneity in this context was first recognized by Cartwright, who observed the emergence of collective network oscillations in a cubic lattice of locally coupled and diverse FitzHugh-Nagumo (FHN) units, none of which were individually in an oscillatory state [7]. Subsequently, numerous authors studied the effects of diversity on complex networks dynamics. Tessone et al. demonstrated an amplification of the response of a coupled oscillator network to an external signal, driven by the heterogeneity of its elements, and named this effect diversity-induced resonance (DIR) [8–17]. Other authors showed that DIR can occur even in the absence of an external forcing [18, 19]. Some of these studies concluded that stochastic resonance (SR) and DIR are substantially analogous phenomena [8, 20], to the point that diversity may be viewed as a form of “quenched noise”.

Diversity in complex networks dynamics has been studied also in terms of its interaction with noise, by introducing in a system both types of disorder. Most of this research highlighted the possibility to amplify resonance effects caused by noise thanks to diversity optimization, and vice versa [21–24]. Recently, Scialla et al. [25] showed that the impact of diversity on network dynamics can be significantly different from that of noise and may result in an antagonistic effect, depending on the specific network configuration. At the same time, however, various regions of synergy between the two types of disorder, giving rise to strong resonance effects, were observed. Also, it has been shown that diversity in a network of FHN neurons can enhance coherence resonance (CR) [26], which is a regular response (i.e., a limit cycle behavior) to an optimal noise amplitude [27], occurring when the system is bounded near the bifurcation thresholds [28, 29].

Another form of noise-induced resonance is self-induced stochastic resonance (SISR), which has a different mechanism from CR for the emergence of regular oscillations [30, 31]. SISR occurs when a small-amplitude noise perturbing the fast variable of an excitable system with a strong time scale separation results in the onset of coherent oscillations [31, 32]. Due to the peculiarity of operating at relatively weak noise, SISR represents a particularly interesting case to study the effects of the interplay between noise and diversity. This is relevant to the potential role of SISR as a signal amplification mechanism in biological systems, given that diversity is inherent to networks of neurons or other cells.

In this Letter, we show that in contrast to CR [26] and stochastic synchronization [25], the effect of diversity on SISR can only be antagonistic, and consequently, we conclude that the enhancement or deterioration of a noise-induced resonance phenomenon by diversity strongly depends on the underlying mechanism.

We point out that not only constructive, but also destructive resonance effects may have significant biological consequences. For instance, an increasing number of studies on Parkinson’s disease [33] indicate that dopaminergic neurons are characterized by a relatively high degree of heterogeneity, and disease progression is associated with the death of only one or a few specific dopaminergic neuron subpopulations, leading to a loss of neuron diversity with respect to healthy brain tissues. Thus, the role of diversity in biological systems might be also to inhibit unwanted resonances through compensatory mechanisms between different neuron sub-types, which can result in pathological conditions, if missing.

There is still a very limited understanding of the named phenomena from a complex systems modeling viewpoint, as previous works have focused mostly on systems and conditions that favor constructive resonance effects. In this work, we uncover a mechanism that we term diversity-induced antiresonance (DIAR), where, in contrast to its effect on CR, diversity deteriorates the coherence of oscillations due to SISR.
II. NETWORK MODEL

As a paradigmatic model with well-known biological relevance, we study the effects of diversity in a network of globally coupled FHN units [34–36]:

\[
\begin{align*}
\frac{dv_i}{dt} &= v_i(a_i - v_i)(v_i - 1) - w_i + K \sum_{j=1}^{N} (v_j - v_i) + \eta_i(t), \\
\frac{dw_i}{dt} &= \varepsilon(bv_i - cw_i).
\end{align*}
\]

(1)

Here \((v_i, w_i) \in \mathbb{R}^2\) represent the fast membrane potential and slow recovery current variables of the elements, respectively; the index \(i = 1, ..., N\) stands for nodes; \(K > 0\) is the synaptic coupling strength; \(0 < \varepsilon \ll 1\) the time scale separation between \(v_i\) and \(w_i\) and \(b, c > 0\) are constant parameters. Diversity is introduced by assigning to each network element \(i\) a different value of \(a_i\), as specified below. The terms \(\eta_i\) \((i = 1, ..., N)\) are independent Gaussian noises with zero mean, standard deviation \(\sigma_a\), and correlation function \(\langle \eta_i(t), \eta_j(t') \rangle = \sigma_a^2 \delta_{ij}(t - t')\). The noise intensity applied to each neuron will be measured by \(\sigma_n\).

III. ANALYSIS

The excitable regime where the network of Eqs. (1) has a unique and stable fixed point is the required deterministic state for the occurrence of SISR [37–39]. When \(\eta_i = 0\), the point \((v, w) = (0, 0)\) becomes a fixed point of the network in Eqs. (1), and is unique if and only if

\[
\frac{(a_i - 1)^2}{4} < \frac{b}{c}.
\]

(2)

For the fixed point \((v_f, w_f) = (0, 0)\) to be stable, we must have \(\text{tr} J_{ij} < 0\) and \(\det J_{ij} > 0\), where \(J_{ij}\) is the Jacobian matrix of the linearized Eqs. (1). Since \(\varepsilon, c > 0\), we have \(\text{tr} J_{ij} < 0\) and \(\det J_{ij} > 0\) only if

\[
-3v_f^2 + 2(a_i + 1)v_f - a_i < 0.
\]

(3)

To ensure that the network of Eqs. (1) lies in the excitable regime required for SISR, in the following we set \(b = 1\) and \(c = 2\). We also set \(\varepsilon = 0.001\), \(K = 0.1\), and \(N = 100\). To introduce diversity, the values of \(a_i\) are drawn from a truncated Gaussian distribution in the interval \(a_i \in (0, 1 + \sqrt{2})\), and are randomly assigned to network elements. The standard deviation \(\sigma_d\) and mean \(a_m\) of the distribution measure diversity and how far the network is from the oscillatory regime (corresponding to \(a_i \leq 0\), respectively.

To study the effects of diversity \(\sigma_d\) on SISR analytically, we apply the mean field approach introducing the global variables \(V(t) = N^{-1} \sum_{i=1}^{N} v_i(t)\) and \(W(t) = N^{-1} \sum_{i=1}^{N} w_i(t)\). Adapting the method used in Refs. [8, 25, 40], we set \(v_i = V + \delta_i\) in Eqs. (1), alongside the assumptions that \(\sum_{i=1}^{N} \delta_i \approx 0\), \(\sum_{i=1}^{N} \delta_i^2 \approx 0\).

We further assume that the standard deviation \(\sigma_d\) of the \(a_i\) distribution is small, allowing the approximation

\[
\langle a_i((V + \delta_i)^2 - (V + \delta_i))\rangle \approx \langle a_i\rangle \langle ((V + \delta_i)^2 - (V + \delta_i)) \rangle,
\]

(4)

where \(\langle \ldots \rangle\) denotes an average over the \(N\) neurons.

Using these assumptions and averaging Eqs. (1) over the \(N\) neurons, we obtain the following dynamical equations for the global variables \(V\) and \(W\):

\[
\begin{align*}
\frac{dV}{dt} &= V[(A - V)(V - 1) - 3M] \\
&
+ M(A + 1) - W + \eta_c(t), \\
\frac{dW}{dt} &= \varepsilon(bV - cW),
\end{align*}
\]

(5)

where \(M = N^{-1} \sum_{i=1}^{N} \delta_i^2\) and \(A = N^{-1} \sum_{i=1}^{N} a_i\). \(M\) can be considered as a diversity parameter, in that it increases with diversity in the network and \(M = 0\) for a homogeneous system (\(\sigma_d = 0\)). Noise effects are represented by a global white noise term \(\eta_c = N^{-1} \sum_{i=1}^{N} \eta_i\) with zero mean and correlation function \(\langle \eta_c(t), \eta_c(t') \rangle = N^{-1} \sigma_n^2 \delta(t - t')\).

In the adiabatic limit \(\varepsilon \to 0\), the time scale separation between \(V\) and \(W\) becomes very large and Eqs. (5) reduce to the 1D Langevin equation

\[
\frac{dV}{dt} = -\frac{\partial U(V, W)}{\partial V} + \eta_c(t).
\]

(6)

In this limit, \(W\) is practically frozen and can be considered as a fixed parameter, its time variation providing only a \(O(\varepsilon)\) contribution to the dynamics governed by Eq. (6). The function \(U(V, W)\) in Eq. (6) is an effective double-well potential parametrically dependent on \(M\):

\[
U(V, W) = \frac{V^4}{4} - \frac{(1 + A) V^3}{3} + \frac{(3M + A) V^2}{2} - \frac{W}{W - M(1 + A)}. \quad \quad \quad (7)
\]

Based on large deviations theory [41, 42] and Kramers’ law [43], we write down for Eqs. (5) the generic conditions for the occurrence of SISR in slow-fast dynamical systems in the standard form [44, 45] as follows [32, 38, 46]

\[
\begin{align*}
\lim_{\langle \sigma_n, \varepsilon \rangle \to (0,0)} \left[ \frac{\sigma_n^2}{2} \ln(\varepsilon^{-1}) \right] \in \left( \Delta U^L(W_f, \Phi), \Phi \right), \\
\Delta U^L(W), \Delta U^R(W) &\to W \in [W_{min}, W_{max}].
\end{align*}
\]

(8)

Here, \((V_{min}, W_{min})\) and \((V_{max}, W_{max})\) are, respectively, the minimum and maximum points of the \(V\)-nullcline, and \((V_f, W_f)\) is the unique (and stable) fixed point of Eqs. (5), see Fig. 1. The left \((\Delta U^L(W) \geq 0)\) and right \((\Delta U^R(W) \geq 0)\) energy barriers of \(U(V, W)\) are:

\[
\begin{align*}
\Delta U^L(W) &= U(V_s^L(W), W) - U(V_s^R(W), W), \\
\Delta U^R(W) &= U(V_s^R(W), W) - U(V_s^R(W), W).
\end{align*}
\]

(9)
where $V_L^*(W) \leq V_S^*(W) \leq V_R^*(W)$ are the real roots of the equation $V[(A-V)(V-1)-3f] + M(A+1) - W = 0$, and are non-negative and monotonic functions of $W$, see Fig. 3(a). Figure 2 shows the landscape of $U(V,W)$ and how $\partial U^{L,R}(W)$ varies with $M$. We note that the asymmetry of $U(V,W)$ is governed by $W$ and the double-well tends to disappear upon increasing $M$, resulting in a loss of the bistability required for SISR occurrence.

In conditions (8), $\Phi$ represents the value of $\partial U^L(W_s)$ when $U(V_s,W_s)$ is symmetric at $W_s > W_f$, i.e.,

$$\Phi := \left\{ \partial U^L(W_s) : \partial U^L(W_s) = \partial U^R(W_s), W_s > W_f \right\}.$$  

(10)

Since the interval $(\partial U^L(W_f), \Phi)$ in the second condition in (8) is open, SISR deteriorates (i.e., the spiking becomes less coherent) and eventually disappears moving away from the center of the interval. Thus, for a given $\varepsilon \ll 1$, we use the boundaries of this interval to calculate the minimum ($\sigma_{\min}$) and maximum ($\sigma_{\max}$) noise intensity between which the highest degree of SISR can be achieved:

$$\sigma_{\min}^n = \sqrt{\frac{2\partial U^L(W_f)}{\ln(\varepsilon^{-1})}}, \quad \sigma_{\max}^n = \sqrt{\frac{2\Phi}{\ln(\varepsilon^{-1})}}.$$  

(11)

The quantities $\sigma_{\min}^n$ and $\sigma_{\max}^n$ have a dependence on the diversity parameter $M$ through $U(V,W)$ and $V_L^{*,S,R}(W)$. Thus, the length of the interval ($\sigma_{\min}^n$, $\sigma_{\max}^n$) can be controlled by $M$.

The occurrence of SISR depends on whether the parameter values of the system, including $M$, satisfy the three conditions (8) in the double limit $(\sigma, \varepsilon) \to (0,0)$. Hence, it suffices to study the variation of $\Phi$ versus $M$ to uncover the effect of diversity on the degree of SISR. This is done in Fig. 3, showing that $\Phi$ decreases upon increasing $M$. Thus, DIAR occurs when diversity in the network increases, leading to a deterioration and eventually destruction of the coherence of the spike train due to SISR, by shrinking the length of the interval ($\sigma_{\min}^n$, $\sigma_{\max}^n$) toward zero.

IV. NUMERICAL SIMULATIONS

We corroborate the theoretical analysis via numerical simulations. We numerically integrate Eqs.(1) for $N = 100$ neurons using the fourth-order Runge-Kutta algorithm for stochastic processes [47] and the Box-Muller algorithm [48]. The integration time step is $dt = 0.01$ and the total simulation time is $T = 1.5 \times 10^6$. For each realization, we choose for the $i$th neuron random initial conditions $[v_i(0), w_i(0)]$, with uniform probability in the ranges $v_i(0) \in (-1,1)$ and $w_i(0) \in (0,2,1)$. After an initial transient time $T_0 = 2.5 \times 10^5$, we start recording the neuron spiking times $t_i^s$ ($i \in N$ counts the spiking times). Averages are taken over 15 realizations, which warrant appropriate statistical accuracy.

We illustrate the effect of diversity, synaptic noise, and
distance of the excitable network from the oscillatory regime, measured by $\sigma_d$, $\sigma_n$, and $a_m$, respectively, on the degree of coherence of the spikes induced by SISR. We use the coefficient of variation (cv) given by the normalized standard deviation of the mean interspike interval (ISI) [27]. For $N$ coupled neurons, cv is given by [49]

$$cv = \frac{\sqrt{\langle \tau^2 \rangle - \langle \tau \rangle^2}}{\langle \tau \rangle},$$

where $\langle \tau \rangle = \frac{1}{N} \sum_{i=1}^{N} \tau_i$ and $\langle \tau^2 \rangle = \frac{1}{N} \sum_{i=1}^{N} \tau_i^2$, with $\langle \tau_i \rangle$ and $\langle \tau_i^2 \rangle$ representing the mean and mean squared ISI (over time), $\tau_i = t_i^{f+1} - t_i^{f} > 0$, of neuron $i$.

We determine the spike occurrence times from the instant the membrane potential variable $v_i$ crosses the threshold $v_{th} = 0.3$. When $cv \ll 1$, the spiking becomes more coherent. If $cv = 1$, we get a Poissonian spike train (rare and incoherent spiking), and $cv > 1$ corresponds to a spiking process that is more variable than a Poisson process. The degree of coherence is illustrated in Fig. 4, which depicts cv against the synaptic noise $\sigma_n$ and diversity parameter $\sigma_d$ at two values of $a_m$.

In Fig. 4(a), the mean value $a_m = 0.05$ is close to the lower bound of the interval $(0.1 + \sqrt{2})$, i.e., close to the oscillatory regime. It can be observed that when $\sigma_n \in [10^{-4}, 10^{-3}]$ and $\sigma_d \in [0.0001, 0.7]$, we have a low cv $\in [0.107, 0.207]$, indicating a high degree of coherence due to SISR. For $\sigma_d > 0.7$, the $\sigma_n$ interval in which $cv < 0.207$ has shrunk to zero, i.e., $cv \geq 0.276$ for all $\sigma_n$ values, indicating a significant deterioration and eventual destruction of the coherence as $\sigma_d$ increases.

In Fig. 4(b), the mean of the diversity distribution is fixed at a higher value $a_m = 1.2$. In this case, the unique fixed point $(v_f, w_f) = (0, 0)$ becomes even more stable than in Fig. 4(a). Small diversities $\sigma_d \in [0.0001, 0.3]$ and weak synaptic noise intensities $\sigma_n < 6 \times 10^{-3}$ are not strong enough to induce spiking; thus the network remains inactive and the value of cv is undefined.

For $\sigma_n < 9 \times 10^{-4}$ and $\sigma_d > 2$, neurons respond differently to the synaptic noise due to the diverse strengths of the excitable regimes. Due to the all-to-all coupling in the network, the large diversity boosts the weak synaptic noise, leading to the production of spikes. However, because the diversity is large, the conditions required for SISR are violated and the spikes produced are incoherent — see in Fig. 4(b) the magenta region bounded by $\sigma_n < 9 \times 10^{-4}$ and $\sigma_d \in [1.7, 2.4]$, where $cv > 1.5$. At a relatively stronger synaptic noise intensity, i.e., $\sigma_n = 4 \times 10^{-3}$ and a very small diversity of $\sigma_d = 0.001$, the degree of coherence due to SISR is best and $cv = 0.14$. As $\sigma_d$ increases while the synaptic noise is fixed at $\sigma_n = 4 \times 10^{-3}$, the degree of SISR deteriorates and $cv \geq 1$.

The results in Fig. 4 were obtained for a specific value of the time scale parameter ($\varepsilon = 0.001$), which is a crucial parameter for SISR. Moreover, additional simulations performed for other values of $\varepsilon \ll 1$ and $K \in (0.025, 1.0)$ (not shown) lead to qualitatively similar results.
V. CONCLUSION

In conclusion, we have provided evidence that there are complex network configurations and parameter regimes where diversity can only cause a deterioration of well-known resonance phenomena, such as SISR. This is predicted by our mean field analysis and confirmed by numerical simulations. The anti-resonance effect appears as soon as there is a minimal degree of diversity in the system and rapidly grows up to a complete resonance muting as diversity increases. The basic mechanism of this effect is that diversity causes a partial or complete disappearance of the energy barrier in the mean field double-well potential, responsible for the coherent spiking corresponding to SISR.

We have illustrated the effect of DIAR in a prototypical excitable model network, which suggests that the effect may be common to other physical, chemical, and biological systems. Based on our analysis and on experimental evidence that a neuron diversity loss can be associated to hyperkinetic disorders characterized by involuntary movements, we hypothesize that diversity may be used in biological systems not only to amplify weak signals, as suggested by previous literature, but also as an efficient control mechanism to prevent undesired resonances.

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