

Noise correlation length effects on a Morris-Lecar neural network

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The role of spatially correlated stochastic perturbations on a Morris-Lecar neural network subject to an aperiodic subthreshold signal is analyzed in detail. Our results suggest that optimum signal-to-noise ratios can be obtained for two critical noise intensities due to the interplay of the subthreshold Poisson process and the correlated Gaussian forcing. For the second peak, most of the cells are periodically excited, the information transfer is enhanced, and a collective behavior develops measured in terms of the averaged activity of the network. The maximum signal-to-noise ratio increases with the correlation length, although it saturates for global coupling. It was found that there is a range of mean frequencies of the subthreshold signal that increases the signal-to-noise ratio output.

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I. INTRODUCTION

In the last years one of the most important subjects of research has been the study of neuronal behavior [1]. The human brain is probably the most complex system we know. The number of its individual parts, the neurons, is estimated to be one hundred billion. A neuron can be connected with up to ten thousand other neurons. Other characteristic features of neurons have long been studied, and this field is still subject of intense investigations both experimental and theoretical. At any rate, it is well-established that neurons are highly nonlinear elements. At the same time it is evident from our daily experience that these neurons must cooperate in a well-coordinated fashion, as in the sequencing of movements or processes of pattern recognition, speech production, and so on. This high coordination becomes also macroscopically visible through magnetoencephalogram (MEG) and electroencephalogram (EEG) measurements of brain activity under different circumstances. To fully understand the mechanism needed to obtain this coordination is one of the most important subjects of study in neurobiology [1,2].

On the other hand, the noisy environment in which the brain works requires the introduction of stochastic perturbations in the deterministic models of neural systems to simulate the real behavior of the brain system [3,4]. Initially the noise was considered a destructive factor in the behavior of dynamical systems. However, several studies have shown that, in many complex systems, weak periodic signals can be strengthened by intermediate noise levels [5]. Most studies of this phenomenon, known as *stochastic resonance* (SR), have been on the enhanced system response to subthreshold periodic signals [6]. A close relative of SR (also known as “internal” or “autonomous” stochastic resonance) is the phenomenon of *coherence resonance* (CR) [7,8], wherein noise enhances the coherence of inherent modes of the system without the presence of periodic forcing as in SR. Coupling strength has been shown to enhance SR in arrays of nonlinear elements, which is known as *array enhanced SR* (AESR)

[9]. Finally, some studies have examined the effect of aperiodic subthreshold signals in systems forced by noise, and as in SR, there is an optimal noise level for information transfer in *aperiodic stochastic resonance* (ASR) [10,11]. All these phenomena can be summed up in the following sentence: *Noise sources, adequately coupled to a nonlinear system, may give rise to a rich new phenomenology that is not present in the deterministic system.* Further research in neuroscience has shown that random noise may improve the human brain’s ability to process information; these studies have shown that the visual-processing region of the human cortex responds better to an external periodic stimulus when external noise is also applied [12].

The neurons are subject to large numbers of random synaptic inputs and other endogenous noise. As a first approximation, and in order to simplify the modeling, these stochastic perturbations can be considered as Gaussian uncorrelated noises. However, the presence of correlation in response variation has been suggested to improve the behavior of a neuronal system, thus suggesting the presence of correlation in input noise [13,14]. Previous work reveals the importance of spatial correlation in the behavior of neural networks [15–17]. In general, the effect of correlated noise depends on how signals are combined, and although correlation may either aid or hinder noise removal, its impact on the amount of information transmitted by an ensemble of neurons may be profound [18]. In this paper, we will analyze the role of finite noise correlation lengths on the transmission of aperiodic subthreshold signals on a Morris-Lecar neural network.

The model developed by Morris and Lecar was first used to describe oscillations in the giant muscle fiber of barnacles [19]. Because it has biophysically meaningful and measurable parameters, the model became quite popular in the computational neuroscience community since it only needs two equations for the voltage-gated ionic currents, Ca^{2+} and K^+ . The model can exhibit various types of spiking, although tonic bursting occurs only when an additional equation is added [20]. Depending on the parameters used in the model, neurons can belong to types **I** or **II** of neural excitability [21]. For neurons of type **I** the frequency of tonic spiking depends on the strength of the input. The ability to fire low-frequency spikes when the input is weak is called type **I**

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excitability. Type **I** excitable neurons can encode the strength of the input into their firing rate and are difficult to synchronize. On the other hand, some neurons cannot fire low-frequency spike trains. That is, they are either quiescent or fire a train of spikes with a certain relatively large frequency. Such neurons are called type **II** excitable. Their firing rate is a poor predictor of the strength of stimulation [22]. The effect of random forcing on the Morris-Lecar model for both type **I** and **II** cases was recently considered [23].

II. MODEL

Throughout this paper we have used a coupled neuronal network composed of Morris-Lecar units. The dynamic equations for the network are described as follows:

$$\begin{aligned} C \frac{dV_i}{dt} &= -I_{ionic}(V_i, W_i) + I_i^{ext}(t), \\ \frac{dW_i}{dt} &= \frac{w_\infty(V_i) - W_i}{\tau_w(V_i)}. \end{aligned} \quad (1)$$

Here V_i is the membrane potential (in millivolts), $C = 1 \mu\text{F}/\text{cm}^2$ the capacity of the membrane, W_i is the activation variable for potassium, and i runs from 1 to N (number of neurons). All currents are in units of microamperes per square centimeter ($\mu\text{A}/\text{cm}^2$) and time t is measured in milliseconds. The ionic current $I_{ionic}(V_i, W_i)$, has three components

$$\begin{aligned} I_{ionic}(V_i, W_i) &= g_{Ca} m_\infty(V_i)(V_i - V_{Ca}) + g_K W_i(V_i - V_K) \\ &+ g_L(V_i - V_L), \end{aligned} \quad (2)$$

and $m_\infty(V_i) = 0.5\{1 + \tanh[(V_i - V_1)/V_2]\}$, $w_\infty(V_i) = 0.5\{1 + \tanh[(V_i - V_3)/V_4]\}$, and $\tau_w(V_i) = \phi / \cosh[(V_i - V_3)/2V_4]$. Parameters are $V_1 = -0.01$, $V_2 = 0.15$, $V_3 = 0.1$, $V_4 = 0.145$, $g_{Ca} = 1.0$, $g_K = 2.0$, $g_L = 0.5$, $V_{Ca} = 1.0$, $V_K = -0.7$, $V_L = -0.5$, and $\phi = 5.0$ (all conductances are in units of milliseconds per square centimeter and reversal potentials in millivolts) [24].

Here, we assume that each neuron i is subject to a *different external current* $I_i^{ext}(t)$ consisting of a white Gaussian noise with zero mean correlated in space plus a train of square pulses firing at times t_i^j ($j = 1, \dots, M$) generated by a random Poisson process $P(t)$ with mean λ

$$I_i^{ext}(t) = I_{max} \sum_{j=1}^M [\Theta(s_j) - \Theta(s_j - t^*)] + \xi_i(t), \quad (3)$$

where $s_j = t - t_i^j$, the pulse width $t^* = 1$ ms, and $\Theta(s)$ is the Heaviside function. The trains of square pulses are supposed to be mutually independent for all neurons. The value of $I_{max} = 0.4 \mu\text{A}/\text{cm}^2$ was chosen such that the signal is subthreshold without noise.

In Eq. (3), the spatially correlated noise $\xi_i(t)$ is obtained as a linear combination of a more simple noise field. Here, we follow a similar numerical implementation as in Refs. [25,26]. Then, the noise in each lattice cell at time t is

$$\xi_i(t) = \frac{1}{\sqrt{2m+1}} \sum_{i-m < j < i+m} \bar{\xi}_j(t), \quad (4)$$

where the index j labels a domain of $2m$ cells around cell i , and a circular geometry is considered. $\bar{\xi}_i$ is a white Gaussian noise in lattice cell i , statistically independent of the other lattice points (white noise in space), whose correlation function is given by

$$\langle \bar{\xi}_m(t) \bar{\xi}_n(t') \rangle = 2D \delta_{m,n} \delta(t - t'), \quad (5)$$

and noise intensity D ($\mu\text{A}^2/\text{cm}^4$). The noise $\xi_i(t)$ consists of the mixing of the values of the original noise $\bar{\xi}_i(t)$ for a finite number $(2m+1)$ of neighboring cells, and the factor $(2m+1)^{-1/2}$ measures the weight with which each neighbor contributes.

The output of the network [14] is defined as

$$I^{out}(t) = \frac{1}{N} \sum_{i=1}^N \Theta[V_i(t) - V_{th}], \quad (6)$$

where V_{th} is the firing threshold taken as 0 mV. The asymptotic value $I^{out}(t \rightarrow \infty)$ is regarded as the averaged activity over the neurons. The signal-to-noise output (SNR) is defined as $10 \log_{10}(S/B)$, with S and B representing the maximum signal strength in the power spectrum of $I^{out}(t)$ and the mean amplitude of the background noise, respectively.

To estimate the correlation between the aperiodic input stimulus, the Poisson process $P(t)$, and the system response I^{out} on a dynamical basis, we use the cross-correlation coefficient (power norm) defined by [10,11]

$$C_0 = \langle (I^{out} - \langle I^{out} \rangle_t)(P - \langle P \rangle_t) \rangle_t, \quad (7)$$

where $\langle \rangle_t$ denotes time averaging and P is the contribution of the Poisson input signal to the external current, Eq. (3), of any cell in the array. Equation (7) is a measure of the coincidence fidelity between the subthreshold signal and the noise-induced system response.

In this paper, we study the effects of noise (4) on the firing capability of the neuronal network as the noise amplitude D and the correlation length m are varied. Equation (1) was numerically integrated using an explicit Euler method with a time step of $\Delta t = 10^{-4}$ ms. The number of neurons in the network was set to $N = 101$ and the mean period of the Poisson process to $\lambda = 50$ ms unless otherwise specified. Averages over 15 different noise realizations were performed to obtain the results that follow below.

III. RESULTS

The response of the neural network to noise intensity is shown in Figs. 1 and 2 for a network consisting of uncorrelated cells ($m=0$) and globally coupled cells ($m=42$), respectively. For both cases and low noise intensity values (a)–(c), the signal is most of the time subthreshold and occasionally some spiking occurs due to noise, Figs. 1(a) and 2(a), with period equal to the mean of the Poisson process, λ . The

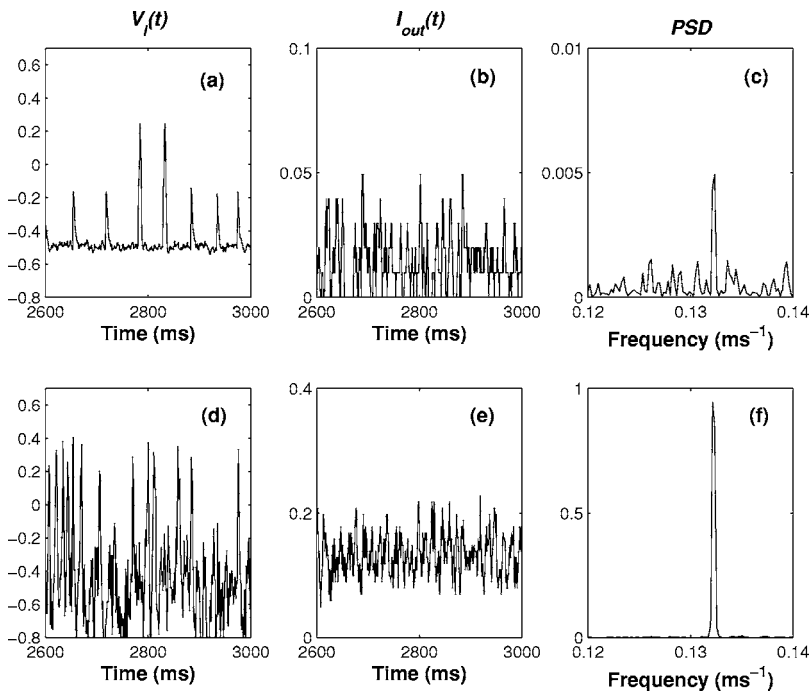


FIG. 1. Uncorrelated network ($m=0$). Time series of $V_i(t)$ and $I_{out}(t)$ and its power spectrum for two different values of the noise intensity: $D=2 \times 10^{-4} \mu\text{A}^2/\text{cm}^4$ (a)–(c) and $D=0.04 \mu\text{A}^2/\text{cm}^4$ (d)–(f).

output of the network, Eq. (6), shows poor correlation among neurons most of the time, only 0.2% of cells firing synchronously for $m=0\%$ and 8% for $m=42$; that is due to the Morris-Lecar network being a type I neural system. On the other hand, increasing the noise amplitude (d)–(f) leads to an increase in the spiking activity (d), although it is more regular for highly correlated cells than for uncorrelated ones. For $m=42$, the spiking behavior of a single cell is nearly periodic as well as the output of the network, but with a period smaller than λ . In both cases, $I^{out}(t)$ oscillates [Figs. 1(c), 1(f), 2(c), and 2(f)] with a frequency approximately equal to the mean frequency of oscillation of a neuron forced with a constant value of I^{ext} [24].

Figure 3 depicts the SNR output as a function of noise intensity D for three different values of m . Each curve presents a similar behavior, namely two maxima of the SNR develop for two different noise intensities. Besides, these maxima shift to higher values as the correlation length m increases. For the limit case $D \rightarrow 0$, the noise vanishes and the deterministic subthreshold case is recovered, so $I^{out}(t)$ remains zero always and $\text{SNR} \rightarrow 0$. For high noise intensities SNR goes to zero since the spectrum becomes broadband. Noise has the effect of randomizing the spike firing times, giving rise to useless spikes as well. In most neurons the saturation level is reached and the SNR diminishes as noise increases. Both maximum SNR values are more pronounced

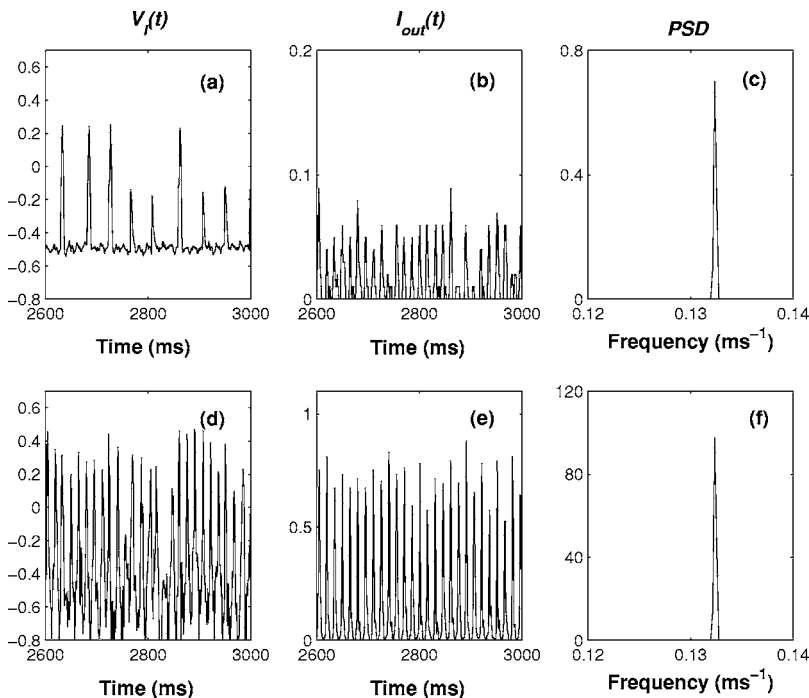


FIG. 2. Globally correlated network ($m=42$). Time series of $V_i(t)$ and $I_{out}(t)$ and its power spectrum for two different values of the noise intensity: $D=2 \times 10^{-4} \mu\text{A}^2/\text{cm}^4$ (a)–(c) and $D=0.04 \mu\text{A}^2/\text{cm}^4$ (d)–(f).

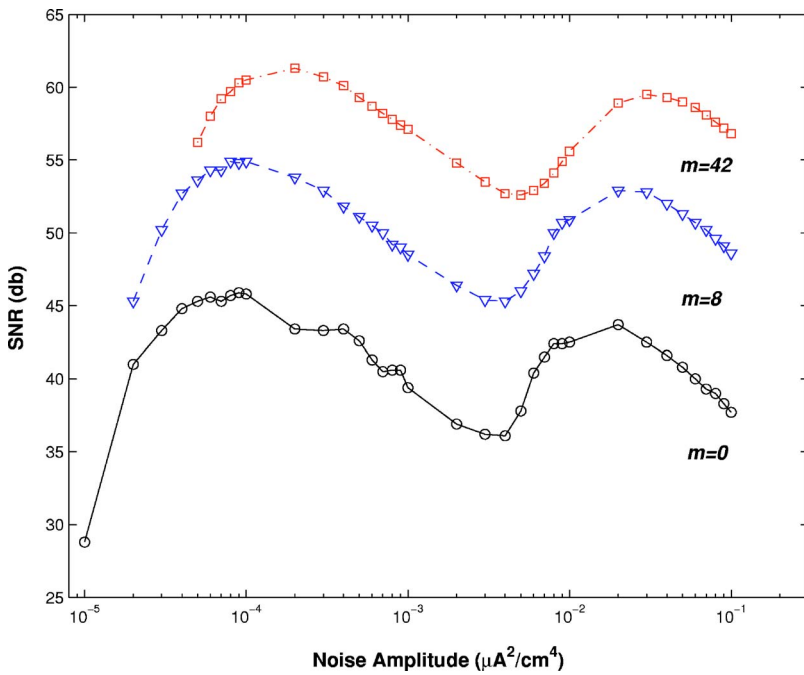


FIG. 3. (Color online) Signal-to-noise ratio (SNR) as a function of noise amplitude D for three different values of the correlation length m . Dots represent average values over 15 different realizations of noise seeds.

for relatively large values of m as shown in Fig. 3. The optimal noise intensity and the values of both SNR maxima shift towards larger values as the noise correlation length m increases as shown in Fig. 4. For the first maximum, noise is large enough to excite some neurons beyond the threshold only when a subthreshold peak (provided by the Poisson distribution) is available, raising the mean firing rate of the network. Increasing the noise intensity, the SNR diminishes as expected until above some critical value of the noise intensity each neuron oscillates periodically with a similar interspike interval to a coupled neuron forced with a constant value of I^{ext} . Then, a second maximum for the SNR develops.

In order to clarify the existence of both maxima in Fig. 3, we have drawn both components of the SNR, $S(D)$ and $B(D)$, in Fig. 5. $B(D)$ clearly grows logarithmically, while $S(D)$ shows two plateaus just after a sudden increase at some critical noise intensity. As a result, the ratio S/B shows two maxima. In terms of the mean firing rate of the network, these two plateaus correspond to a different activity of the ensemble of neurons which is shown in terms of the asymptotic value of $I^{out}(t)$ in Figs. 5(b) and 5(c). Note that the activity of the network linearly increases with noise amplitude, but with a larger slope above some critical noise intensity. Then, all pulses are excited above the threshold and

the network shows a collective behavior close to synchronization. In this sense, Fig. 6 shows the cross-correlation coefficient C_0 as a function of noise amplitude, quantifying the input-output correlation for two different values of the correlation length m . Curves with $m \neq 0$ exhibit a minimum that corresponds to the optimal noise level for maximum information transfer. For $m \rightarrow 0$ the minimum diminishes to nearly zero, since $I^{out} \approx \langle I^{out} \rangle_t$ [see Fig. 1(e)]. For large noise amplitudes, an overall increase of C_0 is observed as $I^{out} - \langle I^{out} \rangle_t > 0$ most of the time. C_0 is negative near the minimum value, because the variables $I^{out}(t)$ and $P(t)$ exhibit a time delay between the sequence of spikes in the input Poisson signal and the response of the system. The optimal noise intensities coincide with those obtained for the second maximum of the SNR above, describing once more that in this case most of the cells spike together at the rate indicated by the Poisson subthreshold process. On the other hand, $C_0(D)$ does not show a singular behavior for noise intensity values corresponding to the first maximum of the SNR shown in Fig. 3. For this interval of noise amplitudes, input-output signal coincidence rarely occurs and $C_0(D) \approx 0$.

The influence of the mean period of the Poisson process λ and the number of neurons N are depicted in Fig. 7. The network is more sensitive to the input signal, Eq. (3), when the mean of the Poisson process λ is equal to the mean

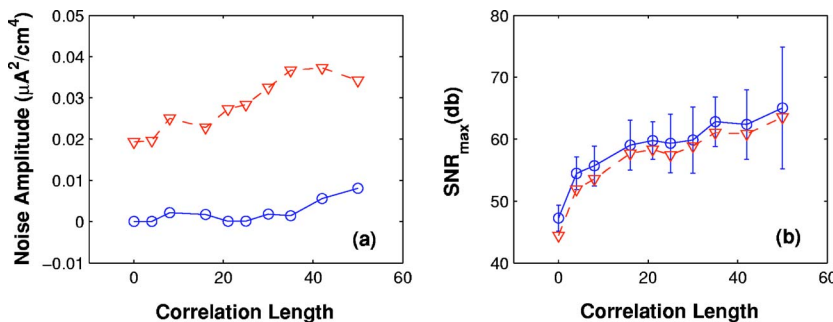


FIG. 4. (Color online) Noise intensity (a) and maximum SNR (b) as a function of the correlation length m . Solid line (circles) corresponds to the first peak in Fig. 3, while the dashed line (triangles) corresponds to the second peak. Error bars corresponding to the different noise realizations are only shown for the first peak in order not to complicate the figure.

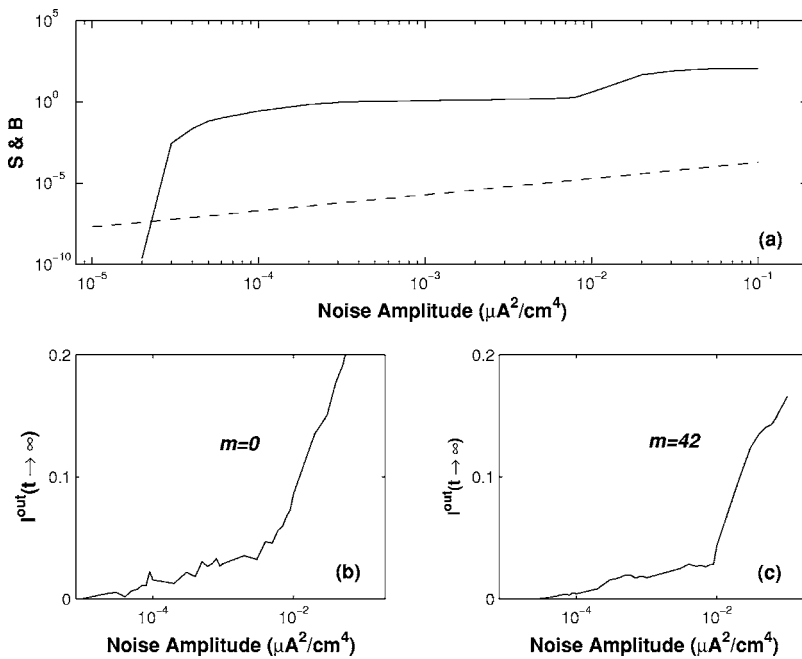


FIG. 5. (a) Maximum signal strength in the power spectrum $S(D)$ (solid line) for $I^{\text{out}}(t)$ and mean amplitude of the background noise $B(D)$ (dashed line) as a function of noise ($m=42$). Asymptotic value $I^{\text{out}}(t \rightarrow \infty)$ as a function of noise amplitude for two different values of m (b,c).

period of oscillation of a single neuron. This resonant behavior is shown in Figs. 7(a) and 7(b), where both maxima of the SNR take a relatively large value for $\lambda^* \approx 8 \pm 2$ ms, independently of the correlation length m . For $m \rightarrow 0$, the noise intensity corresponding to $\text{SNR}_{\text{max}}^1$ diminishes, so $V_i(t)$ is subthreshold most of the time, and the expected resonant behavior at λ^* is masked by the decreasing behavior of $\text{SNR}_{\text{max}}^1(\lambda)$. That is, the network is more sensitive to a correlated noise input forced with a subthreshold signal within a range of mean periods. This interval of λ 's enlarges or reduces with m , but does not change for large enough values of N . In fact, the maximum SNR diminishes, attaining a nearly constant value with increasing N [Fig. 7(c)].

IV. CONCLUSIONS

We have investigated the effect of noise correlation length on a Morris-Lecar neural network. Stochastic perturbations, inherent to the neural system, play an important role in its dynamical behavior as they can act as a coupling parameter. Increasing the noise intensity leads to two maximum values of the SNR output, which are clearly shown as the noise correlation length increases. The first maximum corresponds to the case where cells occasionally are excited above the threshold, but the cooperative behavior between cells I^{out} is low enough to contribute to the SNR. Here, the combined effect of the subthreshold random Poisson process plus the correlated Gaussian noise contributes to the generation of

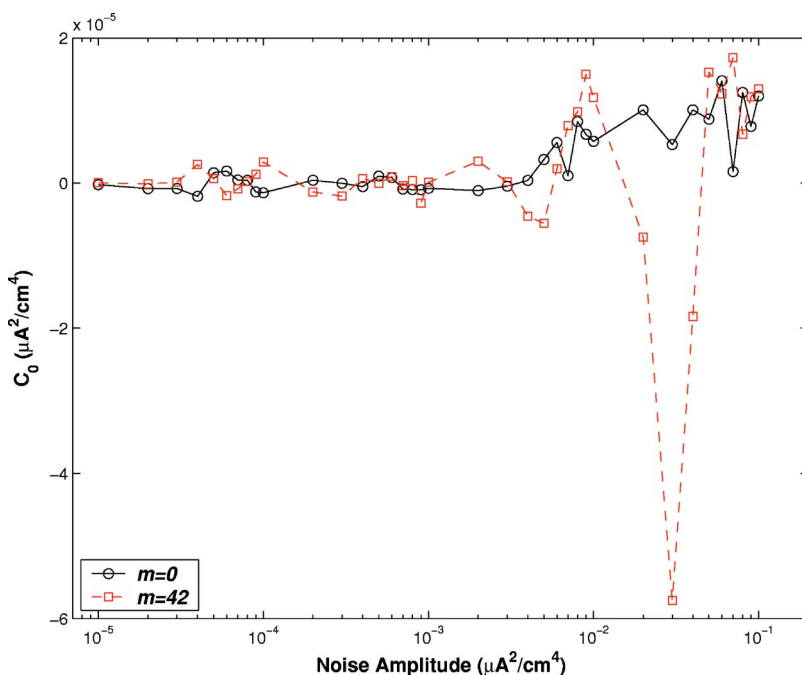


FIG. 6. (Color online) Cross-correlation coefficient C_0 as a function of noise amplitude D for two different values of the correlation length m . Dots represent average values over 15 different realizations of noise seeds.

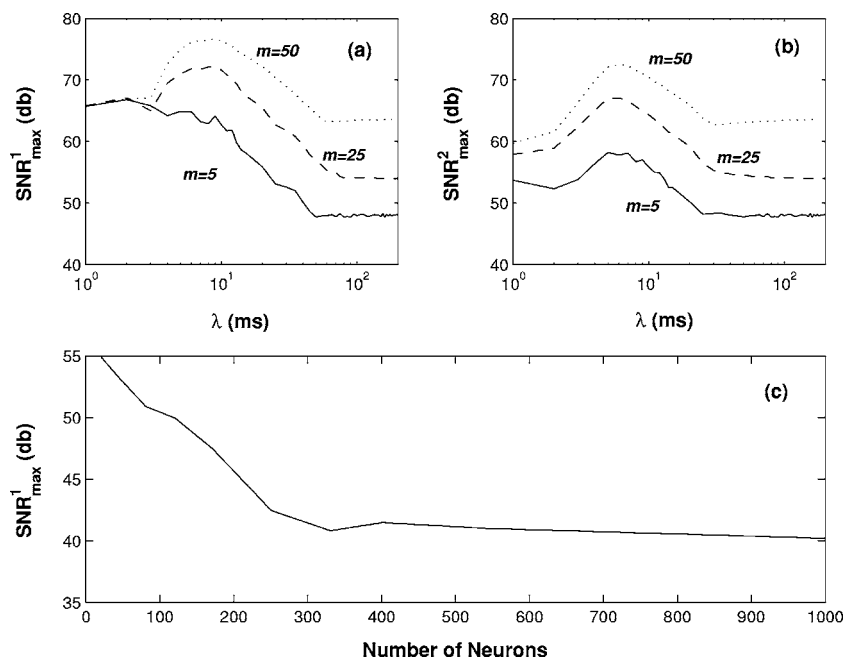


FIG. 7. First (a) and second (b) maximum of the SNR as a function of the mean period λ for three different values of the noise correlation length m . (c) First maximum value of the SNR as a function of the number of neurons N ($m=5$).

superthreshold spikes and then to an increase of the SNR with noise. On the other hand, for the second maximum most of the neurons are periodically excited at nearly the mean frequency of a single neuron, and the synchronization among cells reaches the maximum possible value, thus giving rise to a larger value of S in the SNR than for the first maximum, but, due to the larger noise intensity, the second peak amplitude is smaller. In this case, the correlated Gaussian noise is the most important contribution to the existence of this second peak. In addition, for the second peak the information transfer is maximized by the presence of noise, as it is shown by the unimodal structure of the curve $C_0(D)$ in Fig. 6, and it is optimized for an intermediate noise intensity level.

Our results suggest that increasing the noise correlation length leads to an increase of the SNR output. This behavior is different to that recently observed by Wang *et al.* [14], as their model correspond to type II neurons. Moreover, as the number of neurons that receive a correlated input increases, a range of mean periods for the subthreshold signal is selected as the most indicated in order to maximize the SNR. For type I deterministic neurons, as the value of $I_i^{ext}(t)$ in Eq. (3) is smoothly increased, the activity of the network increases as well. Neurons reach a saturation value, but synchronization is never attained ($I^{out} < 1$). For the stochastic network, as the correlation length is increased, higher values of the noise intensity are also needed in order to attain a maximum value for the SNR (Fig. 4). Then, a saturation level is attained and the mean value of I^{out} is always smaller than 1 as for the deterministic case. This result corroborates the fact that neu-

ral networks must be weakly connected to work correctly, i.e., increasing the correlation among neurons, beyond some critical value, does not contribute significantly to the dynamical behavior of the network.

In conclusion, in this paper we have shown that spatially correlated noise can play a constructive role in optimizing neuronal response to aperiodic subthreshold stimuli. Because the input signal is aperiodic, the behavior described in this paper can be interpreted as a form of ASR. The signal-processing capabilities of the Morris-Lecar network are optimized in terms of enhancement of the information transfer at intermediate noise levels and on time scales of the aperiodic subthreshold input signal that are comparable to the characteristic time scale of the responding system. On the other hand, increasing the correlation length, similarly to increasing the coupling strength, leads to an enhancement of the SNR and the cross-correlation coefficient as it happens for AESR, that in our case could be named *array enhanced aperiodic stochastic resonance* (AEASR). Nevertheless, the study should continue with a more complex configuration of the stochastic input provided for the use of more realistic spatial correlations, and the use of temporal correlations, for example Ornstein-Uhlenbeck processes [26]. The use of other aperiodic input signals with different underlying dynamics should also be considered.

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