Stochastic resonance tuned by correlations in neural models

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The idea that neurons might use stochastic resonance (SR) to take advantage of random signals has been extensively discussed in the literature. However, there are a few key issues that have not been clarified and thus it is difficult to assess that whether SR in neuronal models occurs inside plausible physiology parameter regions or not. We propose and show that neurons can adjust correlations between synaptic inputs, which can be measured in experiments and are dynamical variables, to exhibit SR. The benefit of such a mechanism over the conventional SR is also discussed.

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I. INTRODUCTION

The stochastic resonance is referred to the phenomenon that the input-output signal relationship is optimized by the assistance of noise. Due to the ubiquitous noise in neural systems, this idea is very illuminating and might be helpful for understanding how the brain codes information, a question that has been studied for a century and remains elusive [1–3]. It is argued in the literature that the tunable noise that causes the SR can stem from either the input or the fluctuation of membrane potential (see for example, [4] on p. 259). A few authors ([4] and references therein) have shown that the stochastic resonance does happen in neuronal models ranging from the integrate-and-fire model to biophysical models, by exclusively adjusting the noise level.

In order to apply the SR to neurobiology, however, there are three key issues, as partly discussed in [5] that have to be clarified. (a) There is a consensus that a neuron [6–9] receives signals in the form of Poisson processes, or more generally, renewal processes, i.e., the signal strength is proportional to its noise level, although in recent years many other approaches have been proposed [10]. In the Poisson process case, it is impossible to adjust the noise level alone, while leaving the signal unchanged. (b) At the single neuron level, membrane potential fluctuations are governed by a large number of ionic channels and it is hard to tune such a large number of ionic channels so that the cell operates in the regions where the SR occurs (however, see [11]). (c) There is no evidence to show that the parameter regions where the SR happens are inside physiologically plausible parameter regions of neuronal models.

In this paper we overcome all the three shortcomings mentioned above: we consider neuronal models with Poisson inputs, assume that the stochastic resonance is tuned by the correlation between synapses that is a dynamical variable as verified from experimental data, and confine ourselves inside the physiological parameter regions of neuronal models. For simplicity of expression here only results from the integrate-and-fire model are presented.

Correlations between synapses have been observed and exactly calculated from experimental data [12]. It is not difficult to imagine that due to the large number of afferent synapses, a tiny change of the correlation between synapses can result in a dramatic change in the model behavior. Both theoretically and experimentally, the impact of positive correlation between synapses has been studied and its role on information processing has been explored [13–15]. Here we reveal the functional role of the negative correlation: the efferent signals might be optimized by adjusting the negative correlation.

II. THE MODEL

Suppose that a cell receives excitatory postsynapstic potentials (EPSPs) at p excitatory synapses and inhibitory postsynaptic potentials (IPSPs) at q inhibitory synapses. The activities among excitatory synapses and inhibitory synapses are correlated but, for simplicity of notation here, we assume that the activities of the two classes are independent of each other. When the membrane potential V_t is between the resting potential V_{rest} and the threshold V_{thre},

$$dV_t = -\frac{1}{\gamma} V_t dt + a\sum_{i=1}^{p} dE_i(t) - b\sum_{j=1}^{q} dI_j(t), \qquad (2.1)$$

where $1/\gamma$ is the decay rate, $E_i(t), I_i(t)$ are Poisson processes with rate $\lambda_E(t)$ and $\lambda_I(t)$, respectively, and a,b are magnitude of each EPSP and IPSP. Once V_t crosses V_{thre} from below, a spike is generated and V_t is reset to V_{rest}. This model is termed the integrate-and-fire model. The ith interspike interval T_i of the efferent spike process is

$$T_i = \inf\left\{ t - \sum_{j<i} T_j : V_t \geqslant V_{thre} \right\}$$

with $T_0 = 0$.

We further assume that the correlation coefficient between ith excitatory (inhibitory) synapse and jth excitatory (inhibitory) synapse is

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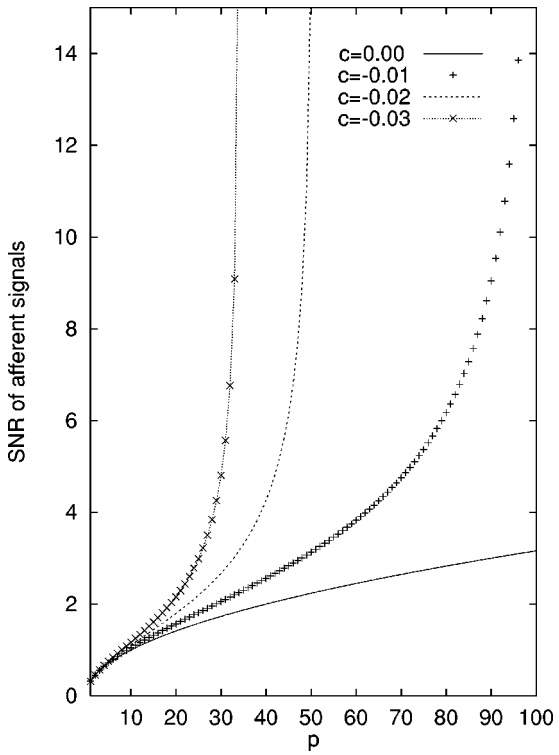

FIG. 1. Signal-to-noise ratio (SNR) of inputs vs p. When the mutual correlation between neurons is around $c=-0.01$, around 100 active neurons will ensure the SNR goes to infinity and so a perfect signal can be extracted. When the correlation is about $c=-0.02$, only 50 neurons are needed for extracting a perfect signal; a correlation of $c=-0.03$ requires 33 neurons for a perfect signal. However, for independent neuronal activities $c=0.00$, we have to resort to infinity the number of neurons to obtain a perfect signal.

$$c=\frac{\langle[E_i(t)-\langle E_i(t)\rangle][E_j(t)-\langle E_j(t)\rangle]\rangle}{\sqrt{\langle[E_i(t)-\langle E_i(t)\rangle]^2\rangle\langle[E_j(t)-\langle E_j(t)\rangle]^2\rangle}},$$

where c is constant.

III. RESULTS

The integrate-and-fire model without reversal potentials, i.e., Eq. (2.1), can be approximated by

$$dv_t=-\frac{1}{\gamma}v_t dt+a\sum_{i=1}^{p}\lambda_E dt-b\sum_{i=1}^{q}\lambda_I dt + a\sqrt{\lambda_E}\sum_{i=1}^{p}dB_i^E(t)-b\sqrt{\lambda_I}\sum_{i=1}^{q}dB_i^I(t), \quad (3.1)$$

where B_i^E $[B_i^I]$ are Brownian motions corresponding to $E_i(t)$ $[I_i(t)]$. Since the summation of Brownian motions is again a Brownian motion, we can rewrite the equation above as follows:

$$dv_t=-\frac{1}{\gamma}v_t dt+(ap\lambda_E-bq\lambda_I)dt + \sqrt{a^2p\lambda_E+b^2q\lambda_I+a^2\lambda_E p(p-1)c+b^2\lambda_I q(q-1)c}\times dB(t), \quad (3.2)$$

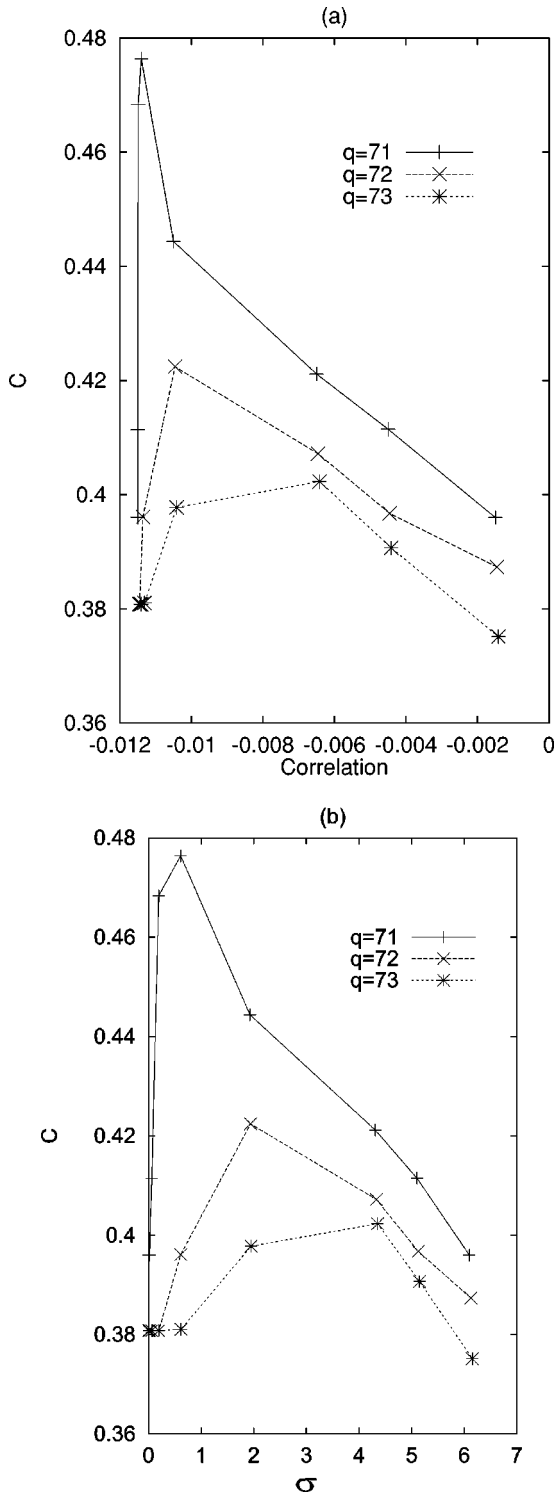

FIG. 2. C vs (a) the correlation and (b) σ defined by Eq. (3.5). Only $q=71,72,73$ are shown here. Equation (3.2) is solved by the Euler scheme using step size 0.001. One thousand spikes are generated to estimate the instantaneous firing rate.

where B is a standard Brownian motion. When $c=0$, Eq. (3.2) gives rise to the same results as in the literature [16] for independent inputs. When $q=0$, the signal-to-noise ratio $\mathcal{R}$ (SNR) of inputs given by Eq. (3.2) is

$$\mathcal{R}=\frac{p\lambda_E}{\sqrt{p\lambda_E+p(p-1)\lambda_E c}}, \quad (3.3)$$

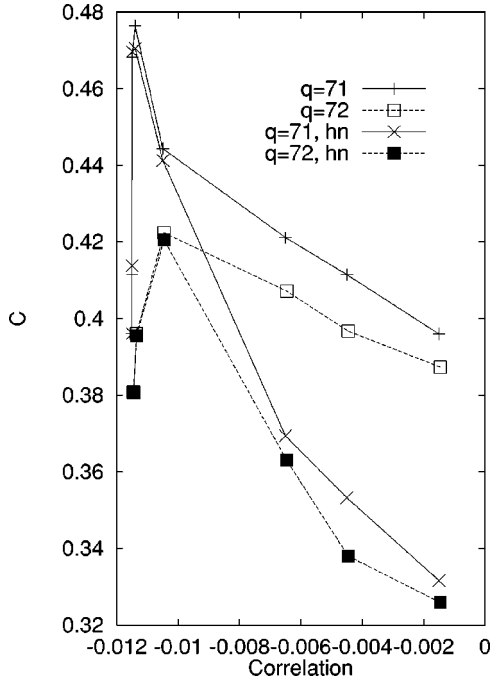

FIG. 3. A comparison between the dynamics Eq. (3.2) with $dx_t = -x_t/\gamma dt + \sigma dB_t$, where σ is given by Eq. (3.5). Results of x_t are indicated by homogeneous noises (HN).

which coincides with that in [12], Fig. 3(a). It is reported that c is between -0.5 and 0.5 [12]. In this paper we only consider the case $c \leqslant 0$, in comparison to the authors in [12] who only take into account positive correlations.

They conclude that for fixed $c>0$ there is p_0 such that when $p>p_0$, SNR is optimized and finite. Although their data clearly show negative correlations, they have not addressed its functional consequence. In fact, it is easily seen from Eq. (3.3) and Fig. 1 that for fixed c, when p approaches $1-1/c$, SNR$\to\infty$, there is no bound on SNR at all. Therefore negative correlations in input signals play a totally different role, compared to positive correlations.

In the following, as in the literature [17–19,14], we assume that $\lambda_E(t)=\lambda_I(t)$, fix $p=100$, and take $\gamma=20.2$ msec. In the literature, two quantities are introduced to measure the output signals: SNR of efferent signals and the similarity between input and output signals [11,4]. We simply use the latter one as the measurement of efferent signals because here even without the drift term in Eq. (3.2) v_t is still a nonhomogeneous process (see below). Hence it is difficult to estimate the interspike intervals T_i (see [20] and reference therein on estimating T_i), which are needed to calculate the SNR of efferent spike trains.

In Fig. 2 numerical simulations with $q=71,72,73$ are shown with

$$\lambda_E(t)=[2+\cos(2\pi t/100)]/30. \tag{3.4}$$

The input signal $\lambda_E(t)$ is chosen so that each synapse fires at a maximum rate of 100 Hz, which is inside the physiological region of cell recordings. For the efferent spike trains T_i we calculated the instantaneous firing rate with a bin of 1 msec, denoted as $r(t)$. Define

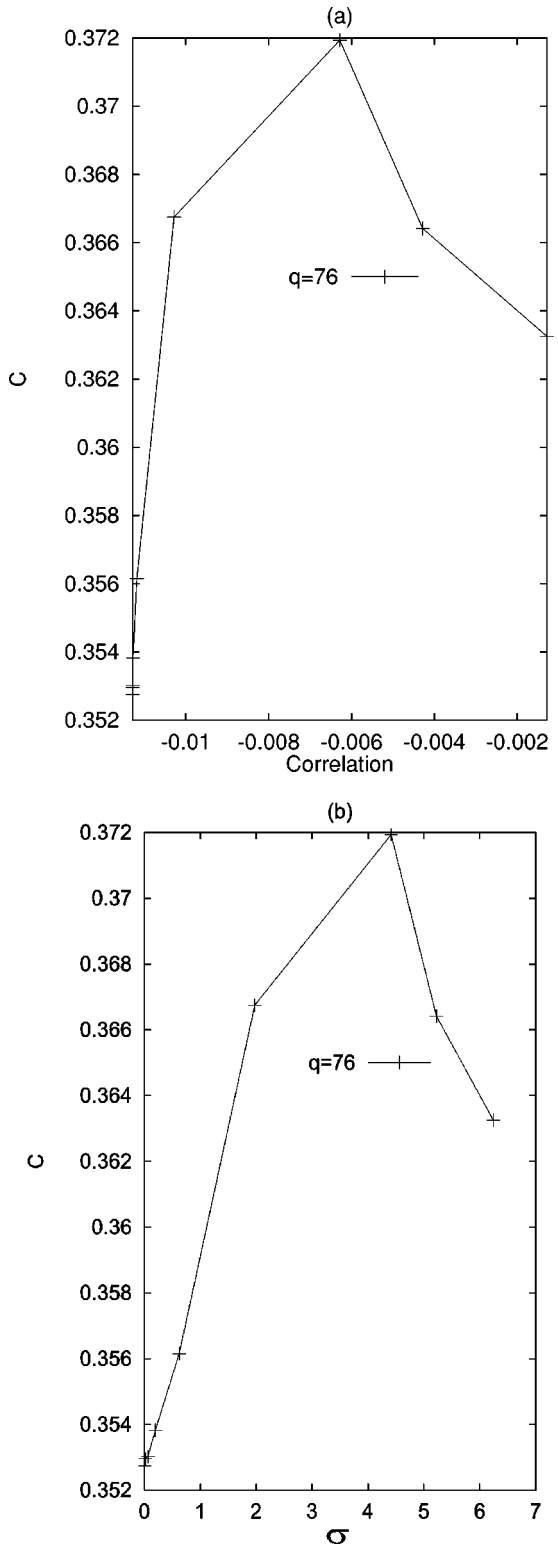

FIG. 4. C vs (a) the correlations and (b) σ with $\lambda_E(t)$ given by Eq. (3.6).

$$C=\overline{[\lambda_E(t)-\overline{\lambda_E(t)}][r(t)-\overline{r(t)}]}/\{\overline{[\lambda_E(t)-\overline{\lambda_E(t)}]^2[r(t)-\overline{r(t)}]^2}\}^{1/2}$$

where overline represents the temporal average [11] and C reflects the similarity between input and output signals. Figure 2(a) shows clearly the stochastic resonance phenomenon with respect to the correlation: for example, when $q=71$ and the correlation is -0.0113, C reaches its maximum value. Too small or too large a correlation will not optimize the signal transmission. In Fig. 2(b), C vs

$$\sigma=\max_t\sqrt{a^2p\lambda_E+b^2q\lambda_I+a^2\lambda_Ep(p-1)c+b^2\lambda_Iq(q-1)c} \tag{3.5}$$

is depicted, which corresponds to the familiar pictures of SR. However, we emphasize here that the noise term in Eq. (3.2) is a dynamical variable, proportional to input signals.

Another interesting phenomenon is the dependence of C on q, the number of active inhibitory synapses. From Fig. 2 we could expect that when q becomes smaller ($q<71$), the maximum point of C moves toward small correlation, i.e., with smaller noise. This could be understood from the dynamics Eq. (3.2). The smaller the q is, the stronger the input signals. Hence the input signal gradually becomes a superthreshold stimulation and so the smaller the noise, the better the quality of signal transmission. On the other hand, when q becomes larger, the neuron will become almost silent. Only a large noise will push the membrane potential across the threshold and so a large noise, which carries signals as well, is required to have a better signal transmission. Figure 2 clearly shows this phenomenon.

It is a natural and interesting question to compare the stochastical resonance phenomena observed above with that of the homogeneous noise case, i.e., the conventional SR. Figure 3 shows such a comparison. An interesting finding here is that the model with signal-dependent noise is more widely tuned for optimizing output. Commencing from small correlations (Fig. 3), we see that the system with or without signal-dependent noise has similar C value. After passing through the optimal point of C for the correlations, the system with signal-independent noise drops much more rapidly than that with signal-dependent noise. In fact, this phenomenon can be easily understood and might reflect one of the functional roles of noises in the neuronal systems. Essentially, the stimulation we consider here is subthreshold. The neuron is most likely to fire when the input signal reaches its maximum value, particularly if a strong, positive noise signal is presented concurrently. When the signal is weak, and if the noise is weak as well, the neuron is less likely to fire. In other words, for the dynamics of Eq. (3.2) there is a signal inside the noise term [see Eq. (3.2)] and we would naturally expect a better signal transmission to be attained, compared to the case of constant noise.

We have considered the case of a fast varying input signal in the discussion above, i.e., Eq. (3.4), which oscillates at a rate of 10 Hz. How does this compare to slowly varying signals? In Fig. 4 we show numerical results with a slowly varying signal, oscillating at a rate of 1 Hz,

$$\lambda_E(t)=[2+\cos(2\pi t/1000)]/30. \tag{3.6}$$

Similar phenomena are observed as for the fast varying signals.

In summary, we show the existence of the SR phenomenon in the integrate-and-fire model with all its parameters in physiologically plausible regions and with Poisson inputs. Our novel approach where the SR is tuned by the negative correlation in synaptic inputs opens up a few issues to be discussed further. For example, how does the SR depend on q, what is the optimal input signal frequency at which the largest C could be attained, etc.? Furthermore an application of the present approach to biophysical models would be interesting, in particular when reversal potentials [16] are included in the model. These are the subject of another study and will be reported elsewhere. Finally we want to emphasize that although we show in this paper the parameter regions in which the SR occurs are physiologically plausible, it does not imply neurons do use it to transmit signals: it might be just epiphenomena and a final answer to this question can only be provided by experiments.

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