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## Stochastic resonance in globally coupled nonlinear oscillators

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We consider a network of N globally coupled nonlinear overdamped oscillators subject to Langevin noise and a weak periodic modulation. Assuming that one of the oscillators relaxes to its steady state on a time scale far slower than the remaining oscillators in the network, we obtain its dynamics from the coupled stochastic differential equations describing the system, via adiabatic elimination. The bifurcation properties of this "reduced oscillator" model are discussed, together with cooperative stochastic effects (e.g., stochastic resonance) that result from the presence of the modulation.

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Recently, there has been an upsurge of interest in cooperative effects arising in networks of nonlinear oscillators interacting via mean-field-type couplings [1]. The interest has spread to applications in biophysics and neural networks where a recent interest in single or few neuron dynamics has led to a derivation of an effective single neuron model [2] starting from the connectionist neural network model of Hopfield [3], in the presence of Langevin and multiplicative fluctuations. The presence of a weak periodic signal in the noisy nonlinear dynamic system can lead to cooperative effects, e.g., "stochastic resonance" wherein small amounts of noise can actually enhance the flow of information through the system [4,5]. In a network of nonlinear oscillators with a linear meanfield interaction, the stochastic resonance effect is enhanced [6] over what would be expected for a single isolated element of the network.

In this work, we consider the influence of a large number of weakly nonlinear oscillators on the dynamics of a single (reference) nonlinear oscillator. The elements of the noisy network are assumed to include *a priori*, selfcoupling terms as well as a weak, low-frequency periodic modulation:

$$C_i \dot{u}_i = \sum_{j=1}^N J_{ij} \tanh u_j - \frac{u_i}{R_i} + F_i(t) + q \sin \omega t \quad . \tag{1}$$

An equation of this form describes a set of N nonlinear coupled bistable oscillators (the coupling is also nonlinear). The i = 1 index is taken to denote the reference oscillator and the indices i = 2, ..., N (where N is large) denote the "bath" oscillators. Systems of the form (1) have been used to describe connectionist-type electronic neural networks [7]. In such networks,  $u_i$  denotes the activation function (analogous to the membrane potential of a neurophysiological neuron) of the *i*th element,  $C_i$  and  $R_i$  denoting the input capacitance and transmembrane resistance. The coupling coefficients  $J_{ij}$  are usually determined via a learning rule. We assume the noise  $F_i(t)$  to be Gaussian,  $\delta$ -function correlated with zero mean and variance  $\sigma_i^2$  (the noise sources for different indices *i* are assumed to be uncorrelated).

We now assume that the time scale for relaxation of the reference oscillator is much longer than that for the bath:

$$C_i R_i \ll C_1 R_1 \quad (i > 1)$$
 (2)

This assumption allows us to adiabatically eliminate the bath variables from (1) and write down an equation for the variable  $u_1$  in terms of the bath variables. The procedure for doing this has been described in [2]. Specifically, an N-body Fokker-Planck equation is constructed from (1). Haken's slaving principle [8] then permits us to factorize the N-body probability density function describing the system (1) into a product of a singlebody density function for the slow variable  $u_1$  and a conditional density function for the remainder of the system. This, in turn, leads to a separation of the N-body Fokker-Planck equation (FPE) into a FPE for the probability density function of  $u_1$  (which contains the bath variables  $u_{i>1}$  and a FPE for the bath variables. The latter is solved in the long-time limit, after invoking a local equilibrium assumption for the bath variables (this is tantamount to a quasilinearization of the bath dynamics). We are ultimately led [9] to a closed FPE for the slow variable  $u_1$  whence a stochastic differential equation may

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be readily written down by inspection:

$$\dot{u}_1 = -\alpha u_1 + \beta \tanh u_1 + \delta \sin \omega t + (\sigma_e^2)^{1/2} F(t)$$
, (3)

where

$$\alpha = (R_1 C_1)^{-1} ,$$
  

$$\beta = C_1^{-1} \left[ J_{11} + \sum_{i>1} R_i G_i^{-1} J_{1i} J_{i1} \left[ 1 - \frac{\sigma_i^2 R_i}{2C_i} \right] \right] ,$$
  

$$\delta = \frac{q}{C_1} \left[ 1 + \sum_{i>1} R_i G_i^{-1} J_{1i} \left[ 1 - \frac{\sigma_i^2 R_i}{2C_i} \right] \right] ,$$
  

$$G_i \equiv 1 - J_{ij} R_i, \quad \sigma_e^2 \equiv \sigma_1^2 / C_1 ,$$
  
(4)

and F(t) is now Gaussian  $\delta$ -function-correlated noise having zero mean and unit variance. In carrying out the procedure leading to (3) we have assumed further that the modulation frequency is smaller than the Kramers frequency of the unmodulated system. This assumption is a cornerstone of the adiabatic theory of stochastic resonance [5] on which our subsequent results are based. Further, we assume that

$$\sigma_i^2 R_i < 2C_i \quad (i > 1) . \tag{5}$$

This assumption (quantified in [5]) guarantees the convergence of the steepest-descent techniques used to evaluate the coefficient  $\beta$  in (3) and places an upper limit on the noise strengths (with very large amounts of noise, the interesting cooperative behavior is lost). Note also the absence of terms involving coupling between pairs of bath oscillators; these terms are  $O(R_iR_j)$  (i, j > 1) or higher and are assumed to be negligible.

A detailed analysis of the full dynamics described by (3) is beyond the scope of this Brief Report and will be presented elsewhere [9]. In the following, we first discuss the cooperative effects that arise as a result of the interaction with the bath, as embodied in the renormalized coefficients  $\beta$  and  $\delta$  in (3). The bifurcation properties of the reduced system (3) may be studied (in the absence of the noise and modulation terms) via the potential function.

$$U(u_1) = \frac{\alpha}{2} u_1^2 - \beta \ln \cosh u_1 . \qquad (6)$$

For positive  $\alpha$  and  $\beta$ , the potential is bimodal (for  $\beta/\alpha > 1$ ) with minima located at  $c \approx (\beta/\alpha) \tanh(\beta/\alpha)$ . It is instructive to consider the effects of the bath dynamics on the transition to bimodality in the potential. To this end, we plot, in Fig. 1, the ratio  $\beta/\alpha$  as a function of the resistance  $R_2$ , for different values of the variance  $\sigma_2^2$  of the bath noise source. Throughout the remainder of this work, we assume that  $\sigma_i^2 \equiv \sigma_2^2$  and  $R_i \equiv R_2$  for the bath variables i > 1; also, we set  $C_i = 1$  for all *i*. Figure 1 is a plot of  $\beta/\alpha$  versus the resistance  $R_2$ . For this figure we have drawn the diagonal elements  $J_{ii}$  of the coupling matrix from a Gaussian set having mean zero and unit variance. The elements  $J_{1i}$  are drawn from a Gaussian set having unit mean and variance, and the back-coupling elements  $J_{i1}$  are drawn from a Gaussian set having unit mean and variance as well as from a Gaussian set having



FIG. 1. Effective nonlinearity parameter  $\beta/\alpha$  vs  $R_2$  for N=10,  $R_1=10$ ,  $J_{11}=1$ . Solid curves represent  $\overline{J}_{1i}=1=\overline{J}_{i1}$  and bath noise variance  $\sigma_2^2=0,1,2$ , reading from the top curve downward. Dotted curves correspond to  $\overline{J}_{i1}=-1$  with the same values of  $\sigma_2^2$ , reading from the bottom curve upward.  $\overline{J}_{ii}=0, \overline{J}_{ii}^2=1$  for i > 1.

a mean of -1 and unit variance. In a neurophysiological or neural network context, these selections would respectively correspond to the couplings being (almost) purely excitatory or a mixture of excitatory and inhibitory. Since in general N can be quite large, we must scale the coupling matrix by N to assure that the second term in (6) does not become inordinately large. Henceforth, all the elements  $J_{ij}$  will be taken to be the scaled quantities  $J_{ij}/N$ . From the expressions (4) we may obtain, approximately, the threshold value of  $R_2$  at which the potential becomes bimodal (all other parameters being fixed). In Fig. 1 we have arbitrarily set  $J_{11} = 1$ , i.e., we assume an excitatory self-feedback in the slow oscillator in the absence of any coupling to the bath. We also set  $R_1 = 10$  so that in the absence of any coupling to the bath, the single (i.e., isolated) oscillator potential is bimodal. In the presence of a preponderance of excitatory couplings  $(\overline{J}_{1i} = 1 = \overline{J}_{11} \text{ and } \overline{J}_{1i}^2 = 1 = \overline{J}_{11}^2)$  the ratio  $\beta/\alpha$  increases (for nonzero  $\sigma_2^2$ ) up to a maximum value, after which it decreases. The opposite effect is seen to occur for the case of a mix of excitatory and inhibitory couplings  $(\bar{J}_{1i}=1=-\bar{J}_{i1})$ . The curves corresponding to these two realizations of the off-diagonal elements for a given value of  $\sigma_2^2$  (for the cases in which an extremum exists) cross at the values  $R_2 = 0$  and  $2/\sigma_2^2$ , yielding the  $J_{1j} = 0$  result  $(\beta/\alpha = J_{11}R_1 = 10.0)$ . Beyond this intersection, the inequality (5) is violated and we do not expect the theory to yield accurate results.

We digress briefly to consider the case in which the potential is monomodal in the absence of any coupling to the dendritic bath (this can be achieved by setting  $J_{11}R_1 < 1$ ). Then, one readily calculates [9] the value of  $R_2$  (for given noise variance  $\sigma_2^2$  and configuration of the matrix **J**) above which the effective potential is bimodal. Increasing  $R_2$  leads to a transition to bimodality only for the case in which the sum  $\sum_{i>l} J_{1i} J_{i1}$  is positive [keeping in mind the constraint imposed by the inequality (5)]. This may be realized by imposing the same sign on the vast majority of the off-diagonal elements  $J_{1i}$  and  $J_{i1}$ . Increasing the noise variance  $\sigma_2^2$  degrades the effect. It is apparent that the coupling to the bath may actually introduce a phase-transition-like behavior into the neuron dynamics. Effects such as this *coupling-induced bimodality* are a hallmark of multiplicative noise [10] and have earlier been examined [11] in simple models of the form (3). The opposite effect can also occur: depending on the magnitude and sign of each element  $J_{ij}$ , a potential that is bistable in the absence of the bath coupling can be rendered monostable by the bath.

We now consider the effects of the deterministic modulation, specifically stochastic resonance, wherein a small amount of noise can introduce correlated switching events in the dynamics (3), corresponding to the system described by the effective potential (6). An adiabatic theory, valid for very low frequency  $\omega$  and weak amplitude q (such that there is no switching in the absence of noise) has been developed by McNamara and Wiesenfeld [5]. The central result of this theory is that if one computes the signal-to-noise ratio (SNR) of a bistable system of the form (3) as a function of the noise variance, then the SNR passes through a maximum at a noise variance approximately equal to the potential barrier height. Stochastic resonance has been investigated [12] for a single (isolated) oscillator of the form (3), with arbitrary  $\alpha$  and  $\beta$ . In the current context, we define the deterministic switching threshold as the critical value  $\delta_c$  of the scaled modulation amplitude  $\delta$ , above which one would obtain deterministic switching in the  $\sigma_1^2 = 0 = \sigma_2^2$  case. This critical value can be easily found to be given by  $\delta_c =$  $-\alpha u_c + \overline{\beta} \tanh u_c$ , where  $u_c \equiv \ln[\sqrt{\beta/\alpha} + \sqrt{(\beta/\alpha)} - 1]$ and  $\overline{\beta} \equiv (\beta)_{\sigma_2^2 = 0}$ . Then, in order to satisfactorily explain stochastic resonance using adiabatic theory we must ensure that  $\delta < \delta_c$  and  $\omega < \omega_K$ , the Kramers rate for the unmodulated system. The adiabatic conditions can be satisfied in the reduced dynamics (3) if we ensure that there is no deterministic switching in the isolated  $(J_{1i}=0)$  case and we operate within the realm of validity [defined by (5)] of the theory.

In Fig. 2 we show the SNR, obtained via the adiabatic theory [5], for the reduced Eq. (3). The bottom curve shows the SNR that would be obtained for the isolated case  $(J_{1i}=0, i>1)$  with the remaining curves showing the effects of including the bath coupling with different values of the bath noise strength  $\sigma_2^2$ . The maximum enhancement is seen to occur for  $\sigma_2^2=0$ ; increasing  $\sigma_2^2$  degrades this enhancement. The important result is that the coupling to the bath enhances the SNR even in the presence of noise [recall that the inequality (5) imposes an upper limit on the noise]. Similar effects have been observed recently in a mean-field model of linearly coupled bistable oscillators [6]. The enhancement of the SNR may be explained by observing (see Fig. 1) that increasing  $R_2$  from zero causes the ratio  $\beta/\alpha$  (and therefore, the potential barrier height  $U_0$ ), for this configuration of **J**, to initially decrease and then increase. The renormalized modulation amplitude  $\delta$ , however, can only increase since



FIG. 2. SNR vs variance  $\sigma_1^2$  for  $(J_{11}, R_1, R_2, q, \omega, N) \equiv (1, 10, 0.6, 0.1, 0.1, 100)$ .  $\bar{J}_{1i} = 1 = -\bar{J}_{i1}$ ,  $\bar{J}_{i1}^2 = 1 = J_{1i}^2$ .  $\bar{J}_{ii} = 0$ ,  $\bar{J}_{ii}^2 = 1$ , (i > 1). Bottom curve:  $J_{1i} = 0$  (isolated case). Remaining curves:  $\sigma_2^2 = 0$  (top), 1 (middle), and 2 (lower).

we have taken the set  $J_{1i}$  to be mainly excitatory in nature. Hence, one obtains a marked increase in the SNR as the potential barrier height decreases. Past the extremum of  $\beta/\alpha$  in Fig. 1, the opposite effect occurs. A similar enhancement of the SNR occurs for the case of all the off-diagonal elements of J being excitatory (i.e.,  $\bar{J}_{1i}=1=\bar{J}_{i1}$ ). However, the enhancement will not be as great as that obtained for the case when the elements of J describe a mix of excitatory and inhibitory couplings. As the barrier height increases the SNR will decrease since there are fewer switching events. Figure 3 shows the peak SNR (normalized to its value for the isolated, i.e.,  $J_{1j}=0$  case) as a function of  $R_2$  for different bath noise strengths. This figure clearly shows that increasing the bath noise leads to a lower enhancement in the SNR.



FIG. 3. Peak SNR (normalized to its value for  $J_{1i} = 0$  case) vs  $R_2$  for  $(J_{11}, R_1, q, \omega, N) \equiv (1, 10, 0, 1, 0, 1, 100)$  and  $\sigma_2^2 = 0$  (top curve), 1 (middle curve), 2 (bottom curve).  $\overline{J}_{ii} = 0$ ,  $\overline{J}_{ii}^2 = 1 = (i > 1), J_{i1}^2 = 1 = \overline{J}_{1i}^2$ . Solid curves:  $\overline{J}_{1i} = 1 = \overline{J}_{i1}$ . Dotted curves:  $\overline{J}_{1i} = 1 = -\overline{J}_{i1}$ .

Note that the curves in Fig. 3 are peaked (for nonzero  $\sigma_2^2$ ) at the same values of  $R_2$  for which the critical curves of Fig. 1 display their extrema. The effects outlined here are described in greater detail in [9]. Before concluding, we must point out that the enhancement of the SNR through the bath coupling will not be as striking as depicted in Fig. 2 if the set  $J_{1i}$  is purely inhibitory. This is evident from (4). If most of the terms in the summation are negative, the net effect is to decrease  $\delta$  while  $\beta$  may increase or decrease depending on the relative magnitudes and signs of the product terms in the summation. This situation is qualitatively similar to that encountered for the case of mainly excitatory  $J_{ij}$ . It is also important to point out that the bath may actually induce stochastic resonance through the coupling-induced bimodality (for the case when the slow oscillator is monostable in the absence of the bath) described in the preceding paragraph.

The above analysis underscores the importance of the magnitudes as well as the signs of the interactions  $J_{ij}$ . This is also obvious from the definitions (3) wherein we observe that  $\beta$  (and hence the potential barrier height) depends on the product  $J_{1j}J_{j1}$ , whereas the effective modulation term  $\delta$  depends only on  $J_{1i}$ . In neurophysiological terms, we could argue that having a statistical mix of excitatory and inhibitory couplings provides superior performance to having all the couplings of the same sign. The latter situation is unlikely to occur in neurophysiology, although it could certainly be realized in electronic neural networks and in more general oscillators governed by dynamics of the form (3) (note that for a general network of nonlinear oscillators, such as that considered in this work, the coefficients  $J_{ij}$  could take on any values, as long as the system remains stable, i.e.,  $\alpha \ge 0$ ).

In conclusion, it is important to reiterate the beneficial effects of coupling the reference oscillator to the bath with a faster time constant. The coupling clearly enhances the information flow (measured by the SNR) to the system output. Clearly, in order to achieve the best possible SNR (in the context of the adiabatic theory employed here) the barrier height should be decreased and the effective modulation increased via the coupling to the bath. If the noise strength  $\sigma_2^2$  is approximately known, then the optimum SNR can be achieved by setting  $R_{2c} \approx \sigma_2^{-2}(1+h)$ , where h satisfies the quadratic (taking the negative square root),

$$(1 - \sigma_2^{-2}H_1 + \frac{5}{2}\sigma_2^{-4}H_2)h^2 + (2\sigma_2^{-4}H_2 - 1)h + \frac{1}{4}\sigma_2^{-4}H_2 + \frac{1}{2}\sigma_2^{-2}H_0 = 0$$

with the definitions  $H_1 \equiv \sum_{i>1} J_{1i} J_{i1} J_{ii},$  $H_2$  $\equiv \sum_{i>1} J_{1i} J_{i1} J_{ii}^2$ , and  $H_0 \equiv \sum_{i>1} J_{1i} J_{i1}$ . The critical value  $R_{2c}$  corresponds to the location of the maxima of the solid curves in Fig. 1 (in the large N limit, the scaling of the coupling matrix J yields  $R_{2c} \approx \sigma_2^{-2}$ ). If the external noise  $\sigma_1^2$  and/or the signal amplitude q are approximately known, then further optimization can be achieved by adjusting the coupling parameters  $J_{ij}$  such that the potential barrier height is roughly equal to  $\sigma_1^2$  and, the effective signal amplitude  $\delta$ , in the reduced (3), is very close to the deterministic switching threshold  $\delta_c$ . When suitably optimized, a network of oscillators such as (3) may well provide signal processing or detection capabilities that are far beyond the purview of the single (i.e., isolated) oscillator defined by  $J_{1i} = 0$ .

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