Transmission of Noise Coded versus Additive Signals through a Neuronal Ensemble

Benjamin Lindner and Lutz Schimansky-Geier
Institute of Physics, Humboldt-University at Berlin, Invalidenstrasse 110, D-10115 Berlin, Germany
(Received 25 August 2000)

Neuronal populations receive signals through temporally inhomogeneous spike trains which can be approximated by an input consisting of a time dependent mean value (additive signal) and noise with a time dependent intensity (noise coded signal). We compare the linear response of an ensemble of model neurons to these signals. Our analytical solution for the mean activity demonstrates the high efficiency of the transmission of a noise coded signal in a broad frequency band. For both kinds of signal we show that the transmission by the ensemble reveals stochastic resonance as well as a nonmonotonous dependence on the driving frequency.

DOI: 10.1103/PhysRevLett.86.2934 PACS numbers: 05.40.Ca, 87.10.+e

The processing of periodic stimuli by single stochastic neurons [1–5] or neuronal populations [6,7] has been intensely studied in the past. Most investigations have focused on models driven by an additive periodic signal and noise of a constant intensity. For higher ordered sensory or cortical neurons which receive trains of excitatory (positive amplitude) and inhibitory (negative amplitude) spikes such an additive forcing may arise due to temporal changes of the mean value (base current) of these components [8,9].

However, there is no reason to believe that the modulation of such an input spike train to a neuronal system is restricted to the mean value. Consider, for instance, the summed spike trains generated by $(an + bn^2)$ excitatory and bn^2 inhibitory neurons with equal time dependent spike rate $s(t)$ containing, e.g., a periodic signal. These neurons are part of a larger ensemble (input ensemble). The intensities of the spike trains are given by $\lambda_{\mathrm{exc}} = (an + bn^2)s(t)$ and $\lambda_{\mathrm{inh}} = bn^2 s(t)$. For postsynaptical potential amplitudes of the receiving neuron $a_e = -a_i = 1/n$ as in [8] and a large number of input neurons ($n \to \infty$) the diffusion approximation [10] allows one to replace the spike train by $as(t) + \sqrt{2b\ s(t)}\,\xi(t)$. Here, the signal $s(t)$ is in part effectively *noise coded* since it modulates the intensity of the fluctuations [white Gaussian noise $\xi(t)$] as well as the base current a. Similar inputs to a bistable system [11] and, recently, to neuronal models [12–14] were used by only a few authors. Pawelzik *et al.* [14] considered the response of an ensemble of model neurons to a steplike increase of the noise intensity and found an instantaneous response of the system's spike rate. This indicates a potential high frequency transmission of noise coded signals. Another component of the input may originate in the neuronal background. This part may be assumed to contribute temporally homogeneously to base current and noise intensity. Thus, the total input is given by

$$I(t) = as(t) + \mu_{bg} + \sqrt{2bs(t)}\,\xi(t) + \sqrt{2D_{bg}}\,\eta(t), \tag{1}$$

where $\eta(t)$ is white Gaussian noise. The functional role of the background noise could be understood in the context of *stochastic resonance* (SR): the detection of a weak signal can be enhanced by an appropriate amount of fluctuations [15]. There is much evidence for SR in the case of additive signals [1–6]. For noise coded signals in turn, its significance will be clarified here.

In this Letter, we study an ensemble of neurons which are driven by the input (1), i.e., by the input ensemble and by background noise. Arrangements of such unidirectionally coupled pools are the basis for synfire chains [16] and believed to represent processing pathways in the cortex. It will emerge that for *both* additive and noise coded parts of a periodic signal the ensemble exhibits SR. Moreover, only the noise coded part leads to an efficient high frequency transmission that might account for the high speed processing found, e.g., in the visual system [17]. The analytical results leading to these conclusions are valid for weak signal amplitudes and are not limited to low frequencies or small noise levels. They apply also to cases that have been treated numerically in the literature [2,3,6] and can readily be generalized to aperiodic signals.

Model.—We consider an ensemble of N leaky integrate-and-fire (LIF) neurons which are driven by (1). We restrict ourselves to the simple case that the input rate is harmonically modulated $s(t) = 1 + \varepsilon \cos\Omega t$ ($\varepsilon < 1$) and no correlations occur between inputs to different neurons, i.e., the number of common input connections for two neurons within the ensemble is negligible. The input parameters a, b, and μ_{bg} are assumed to be constant implying a constant base current $\mu = \mu_{bg} + a$ and fixed effective amplitudes $\varepsilon_\alpha = a\varepsilon$, $\varepsilon_\beta = b\varepsilon$ of the additive and noise coded signal, respectively. The time independent part of the effective noise intensity reads $D = D_{bg} + b$ and may be varied changing D_{bg}.

Theory.—The depolarization voltage v_i of the cell membrane of a single neuron is described by a periodically driven Ornstein-Uhlenbeck process with absorbing boundary at the firing threshold $v_{\mathrm{thr}} = 1$. The membrane time constant is set to one, thus, the resting level is given by $v_{\mathrm{rest}} = \mu$. If the voltage reaches the threshold the neuron fires a δ spike and will be absolutely refractory for the

 0031-9007/01/86(14)/2934(4)$15.00

time τ. Afterwards the voltage will be reset to $v_{\text{reset}} = 0$. The N neurons with voltage $v_i(t)$ share the same periodic stimuli, however, the fluctuative parts of their input are entirely independent. Therefore the probability density of the population factorizes and it will be sufficient to look at $P(v,t) = \langle \sum_1^N \delta(v - v_i(t)) \rangle$. This density obeys the Fokker-Planck equation (FPE) of a single neuron

$$\partial_t P = \partial_v [v - \mu - \varepsilon_\alpha \cos\Omega t + (D + \varepsilon_\beta \cos\Omega t)\partial_v] P . \quad (2)$$

A part of the population denoted by $P_\tau(t)$ will be in the absolute refractory state. The total density is normalized as $\int_{-\infty}^{1} dv\, P(v,t) + P_\tau(t) = N$. Furthermore, the reset mechanism leads to the following conditions: (i) the density must be zero at the absorbing boundary; (ii) the density outflux at the threshold equals the influx at the reset point delayed by the absolute refractory period; (iii) the density $P(v,t)$ needs to be continuous for the entire range $v \in (-\infty, v_{\text{thr}}]$.

For large N, the output spike train of the population forms an inhomogeneous Poissonian spike train [6] the rate of which is given by

$$R(t) = Nr(t),$$
$$r(t) = -\frac{1}{N}(D + \varepsilon_\beta \cos\Omega t)\left.\frac{\partial P}{\partial v}\right|_{v=1}, \quad (3)$$

with $r(t)$ being the mean activity of the population, an ensemble averaged rate that is independent of N. Note, that $r(t)$ coincides with the time dependent mean output (instantaneous firing rate) of a single neuron.

For weak signals ($\varepsilon_\beta \ll D$, $\varepsilon_\alpha/\sqrt{1+\Omega^2} \ll 1 - \mu$), the asymptotic solution of the FPE (2) obeying the above conditions can be calculated by linear response theory [15]. Inserting into (3) yields the asymptotic activity

$$r(t) = r_0 + \varepsilon_\alpha |\alpha| \cos(\Omega t - \phi_\alpha) + \varepsilon_\beta |\beta| \cos(\Omega t - \phi_\beta). \quad (4)$$

Here $r_0 = 1/[\tau + \sqrt{\pi}\int_{(\mu-1)/\sqrt{2D}}^{\mu/\sqrt{2D}} dz\, e^{z^2}\text{erfc}(z)]$ denotes the stationary spike rate of a single neuron in the absence of a signal [18]. The complex functions α and β in (4) are given by

$$\alpha = \frac{r_0 i\Omega/\sqrt{D}}{i\Omega - 1} \frac{\mathcal{D}_{i\Omega-1}(\frac{\mu-1}{\sqrt{D}}) - e^{(2\mu-1)/(4D)}\mathcal{D}_{i\Omega-1}(\frac{\mu}{\sqrt{D}})}{\mathcal{D}_{i\Omega}(\frac{\mu-1}{\sqrt{D}}) - e^{(2\mu-1)/(4D)}e^{i\Omega\tau}\mathcal{D}_{i\Omega}(\frac{\mu}{\sqrt{D}})}, \quad (5)$$

$$\beta = \frac{r_0 i\Omega(i\Omega - 1)}{D(2 - i\Omega)} \times \frac{\mathcal{D}_{i\Omega-2}(\frac{\mu-1}{\sqrt{D}}) - e^{(2\mu-1)/(4D)}\mathcal{D}_{i\Omega-2}(\frac{\mu}{\sqrt{D}})}{\mathcal{D}_{i\Omega}(\frac{\mu-1}{\sqrt{D}}) - e^{(2\mu-1)/(4D)}e^{i\Omega\tau}\mathcal{D}_{i\Omega}(\frac{\mu}{\sqrt{D}})}, \quad (6)$$

where $\mathcal{D}_a(z)$ denotes the parabolic cylinder function [19]. The phase shifts ϕ_α and ϕ_β are the complex phases of α and β, respectively.

From (4) it becomes apparent that in linear response theory the two different signals yield additive contributions to the mean activity. In the following, we discuss and compare the relative amplitudes and phases of both components. In all data shown, the absolute refractory period is set to $\tau = 0.1$ and for the base current we choose $\mu = 0.8$.

Amplitude and phase for the additive signal.—The response amplitude $|\alpha|$ exhibits a maximum with respect to the noise intensity for all frequencies (Fig. 1) [6]. Moreover, $|\alpha|$ displays also an overall maximum with respect to both driving frequency and noise intensity. The nonmonotonous dependence on the frequency occurring at large but subthreshold base current ($0.5 < \mu < 1$), was studied in the case of a single LIF neuron in terms of the spectral signal-to-noise ratio [2] and also found in the FitzHugh-Nagumo neuronal model [4,5]. At large base current and moderate noise intensity, the relative refractory period, i.e., the time for relaxation from reset level ($v_{\text{reset}} = 0$) to resting level ($v_{\text{rest}} = \mu$) will be comparable with the escape time from resting level to threshold ($v_{\text{thr}} = 1$). The long refractory period gives rise to a more regular firing even in the absence of a signal, i.e., the neuron possesses in this case a noise induced eigenfrequency [5]. Hence, the nonmonotonous dependence on the driving frequency is nothing other than a classical resonance with respect to this eigenfrequency.

In addition, for a finite τ and large noise intensity, the spike rate becomes large and the absolute refractory period causes a more regular timing of the neuron's firing. The associated eigenfrequency and its higher harmonics lead to resonances revealed by the small maxima in Fig. 1. The influence of the signal on the neuronal dynamics, however, is diminished for further increasing noise intensity, therefore, the maxima vanish in the limit $D \to \infty$. We note that high frequent signals are suppressed by the ensemble, $|\alpha|$ decreases in proportion to $1/\sqrt{\Omega}$ when $\Omega \to \infty$.

The phase shift ϕ_α depicted in Fig. 2 vanishes in the adiabatic case ($\Omega \to 0$) and shows otherwise a delayed response to the additive signal. It attains a maximum and saturates at $\pi/4$ in the high frequency limit. At large noise strength several minima and maxima are obtained (thick line in Fig. 2) and even a negative phase shift may

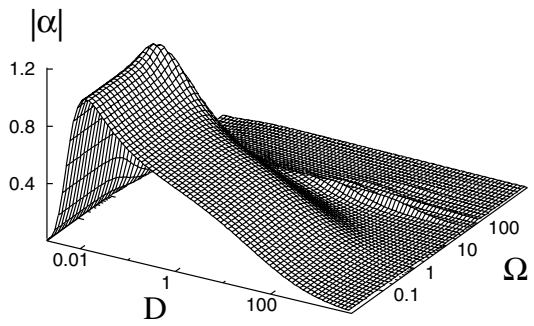

FIG. 1. Amplitude of response to the additive signal.

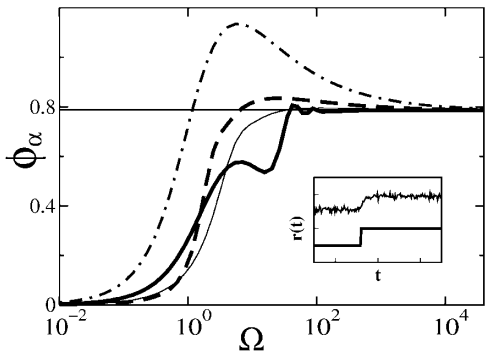

FIG. 2. Phase shift to the additive signal. $D = 2 \times 10^{-3}$ (dash-dotted line), 2×10^{-2} (dashed line), 0.1 (thin solid) and 2 (thick solid). Inset: the response of the activity to a steplike signal (thick) in the base current $\mu \to \mu + \varepsilon$ at $D = 0.1$ and $\varepsilon = 0.05$.

occur—an effect which is again due to the absolute refractory period.

Amplitude and phase for the noise coded signal.—The amplitude $|\beta|$ shown in Fig. 3 reveals stochastic resonance for the noise coded signal with respect to D. At an appropriate noise intensity the ensemble will transmit *all* frequencies. There are two simple limits belonging to different mechanisms for SR. On the one hand, the quasistatically driven system ($\Omega = 0$) responds as $|\beta| \longrightarrow dr_0/dD$. This can be regarded as an effective modulation of the potential barrier at the threshold similar to that caused by varying the base current. In this limit, the SR phenomenon is based on the fact that the sensitivity of the stationary spike rate to a change in the noise strength is maximal at a moderate noise level. On the other hand, for infinite driving frequency, the ensemble cannot follow the signal and persists in its stationary state. The time dependent response is thus determined by the direct modulation of the diffusion current in Eq. (3). It becomes proportional to the stationary rate with an effective amplitude ε_β/D, i.e., $\beta \to r_0/D$ as $\Omega \to \infty$. Since r_0 decreases exponentially for decreasing noise intensity D and saturates for large D at $1/\tau$, the response amplitude must vanish in either limits $D \to 0$ and $D \to \infty$. For moderate D this high frequency limit (thick line in Fig. 3) is finite and possesses a maximum. Hence, the transmission of noise coded signals with high frequencies is possible and also for these signals stochastic resonance can be found.

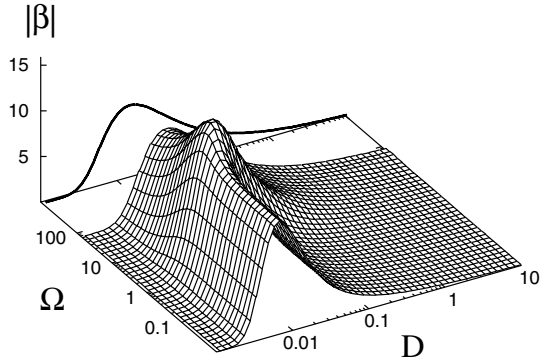

FIG. 3. Amplitude of response to the noise coded signal.

Furthermore, the overall maximum of $|\beta|$ is attained at finite frequency. Primarily, the refractory period and thus the noise induced eigenfrequency of the neuron is again responsible for the nonmonotonous dependence on the driving frequency, since the overall maximum is considerably diminished for the case of vanishing total refractory period ($v_{\text{reset}} = v_{\text{rest}}, \tau = 0$) (not shown).

The response amplitude for the noise coded signal exceeds the amplitude in the additive case by 1 order of magnitude for low up to moderate noise intensities (note the different scales in Figs. 1 and 3)—a striking difference to the findings for an asymmetric bistable system [11] in which always the response to the additive signal is stronger than the response to the noise coded signal.

The periodic modulation of the noise intensity leads to surprising features of the phase shift ϕ_β (Fig. 4). First, it tends to zero for both vanishing and infinite driving frequency since in these limits the activity $r(t)$ becomes proportional to the signal. The latter limit implies a transmission of high frequent signals *without delay*. Second, the phase shift attains negative values for moderate to large noise intensity and not too large frequencies. This can be qualitatively understood by considering a steplike excitation $D \to D + \varepsilon$, a problem which was studied in [14]. This signal and the change of the activity in response to it is depicted in the inset of Fig. 4; for comparison we show the equivalent excitation $\mu \to \mu + \varepsilon$ in the inset of Fig. 2. While the latter additive signal results in a slow growth towards the new stationary activity, the switch of the noise intensity causes an instantaneous jump of the activity to $r_0 + r_0\varepsilon/D$. At a moderate noise intensity this value exceeds the asymptotic stationary activity, hence, the subsequent relaxation is mainly decreasing. In the case of

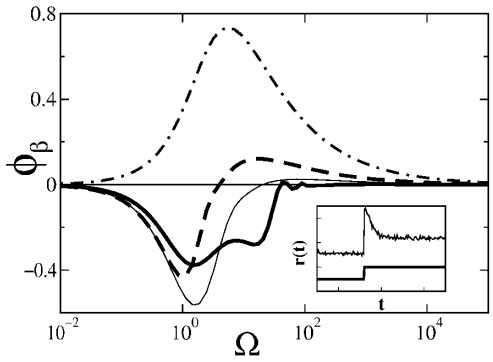

FIG. 4. Phase shift to the noise coded signal. $D = 2 \times 10^{-3}$ (dash-dotted line), 2×10^{-2} (dashed line), 0.1 (thin solid line), and 2 (thick solid line). Inset: The response of the activity to a steplike signal (thick line) in the noise intensity $D \to D + \varepsilon$ at $D = 0.1$ and $\varepsilon = 0.05$.

a continuous signal this kind of response implies a delayed component in *antiphase* to the signal. Combined with the instantaneous response (no phase shift) a negative total phase shift results. For large noise intensity we find again a multipeaked function due to the absolute refractory period.

In order to give an impression of the distinct responses we show in Fig. 5 simulations of the time dependent mean activity of an ensemble of LIF neurons which are either subject to a noise coded or to an additive signal. Clearly, the noise coded signal results in an activity with pronounced negative phase shift and larger amplitude than the additive signal. Similar simulations were carried out for high frequencies (not shown), confirming the finite response to a noise coded signal.

There are, however, two restrictions for the high frequency transmission by an ensemble of real neurons. First, increasingly high frequencies necessitate an increasingly large ensemble size. Otherwise, the relative error of the spatial average of the activity ($\approx 1/\sqrt{r_0 NT}$) in a time window T ($\ll 1/\Omega$) will be too large and a neuron receiving the input from the ensemble will not be able to extract the signal from the spike train within this time window. Second, the threshold condition of the leaky integrate-and-fire model which is responsible for the finite response at high frequencies is only an approximation. It replaces the dynamics of certain fast variables in more realistic neuronal systems like the FitzHugh-Nagumo or Hodgkin-Huxley model. Hence, the transmission should be observable at least up to frequencies which are comparable to those corresponding to the neglected small time scales. These time scales, however, can be several orders of magnitude smaller than the membrane time constant [10]. Noise coded signals of a large frequency range are therefore expected to be efficiently transmitted also by large ensembles of real neurons.

The stochastic resonance considered in this Letter relies on the presence of an effective potential barrier at the threshold as well as on a possible variation of the noise strength by D_{bg}. Consequently, SR can be observed with respect to the background noise level D_{bg} *only* for sufficiently small a and b. On the one hand, the parameter a determines the signal amplitude ε_α as well as the base current μ. If these effective parameters are too large ($\mu > 1$ or $\varepsilon_\alpha \gg 1 - \mu$) the ensemble neurons operate either in an oscillatory mode or receive additive suprathreshold signals and no SR effect is expected. On the other hand, b gives a lower bound for a possible variation of the overall noise intensity D. Therefore, if the response attains its maximum at values larger than b a better transmission is obtained adding background noise. In the opposite case of large b an increasing background noise deteriorates the performance and $D_{bg} = 0$ is optimal. Generally speaking, SR may assist the signal processing, unless the periodic input generated by the ensemble is strong. This is consistent with the conventional notion that in the majority of cases SR is relevant for the detection of weak signals, only.

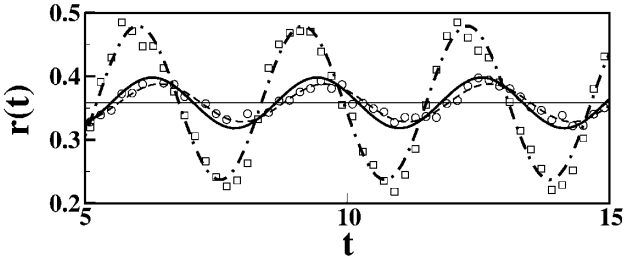

FIG. 5. Mean activity of 40 000 LIF neurons ($D = 0.1$, $\Omega = 2$) either driven by an additive ($\varepsilon_\alpha = 0.04, \varepsilon_\beta = 0$, circles) or by a noise coded signal ($\varepsilon_\alpha = 0, \varepsilon_\beta = 0.04$, squares). Theoretical curves (dashed and dot-dashed lines) and signal plus constant rate (solid line) are plotted for comparison.

We are grateful to Jarlath McHugh and Ulrike Laitko for their help. This work was supported by DFG: GK 268 and Sfb 555.

[1] A. Longtin, J. Stat. Phys. **70**, 309 (1993); K. Wiesenfeld *et al.,* Phys. Rev. Lett. **72**, 2125 (1994); X. Pei, K. Bachmann, and F. Moss, Phys. Lett. A **206**, 61 (1995); A. R. Bulsara *et al.,* Phys. Rev. E **53**, 3958 (1996); S. M. Bezrukov and I. Vodyanoy, Nature (London) **385**, 319 (1997); A. Longtin and D. R. Chialvo, Phys. Rev. Lett. **81**, 4012 (1998); D. Nozaki *et al.,* Phys. Rev. Lett. **82**, 2402 (1999).
[2] H. E. Plesser and T. Geisel, Phys. Rev. E **59**, 7008 (1999).
[3] T. Shimokawa, K. Pakdaman, and S. Sato, Phys. Rev. E **59**, 3427 (1999).
[4] S. R. Massanés and C. J. P. Vicente, Phys. Rev. E **59**, 4490 (1999).
[5] B. Lindner and L. Schimansky-Geier, Phys. Rev. E **61**, 6103 (2000).
[6] T. Shimokawa *et al.,* Phys. Rev. E **59**, 3461 (1999).
[7] M. Spiridon and W. Gerstner, Netw. Comput. Neural. Syst. **10**, 257 (1999).
[8] P. Lansky, Phys. Rev. E **55**, 2040 (1997).
[9] H. E. Plesser, Ph.D. thesis, Universität Göttingen, 1999, Ch. 2.1.
[10] H. C. Tuckwell, *Introduction to Theoretical Neurobiology* (Cambridge University Press, Cambridge, United Kingdom, 1988).
[11] M. I. Dykman *et al.,* Phys. Rev. A **46**, R1713 (1992).
[12] N. Brunel, J. Comput. Neurosci. **8**, 183 (2000).
[13] J. Feng and B. Titozzi, Phys. Rev. E **61**, 4207 (2000).
[14] K. Pawelzik, oral presentation.
[15] L. Gammaitoni, P. Hänggi, P. Jung, and F. Marchesoni, Rev. Mod. Phys. **70**, 223 (1998).
[16] M. Abeles, *Corticonics* (Cambridge University Press, Cambridge, 1991).
[17] S. Thorpe, D. Fize, and C. Marlot, Nature (London) **381**, 520 (1996).
[18] L. M. Ricciardi, *Diffusion Processes and Related Topics on Biology* (Springer-Verlag, Berlin, 1977).
[19] *Handbook of Mathematical Functions,* edited by M. Abramowitz and I. A. Stegun (Dover, New York, 1970).