Frequency Spectrum of Coupled Stochastic Neurons with Refractoriness

Mona Spiridon
MANTRA, Center for Neuro-Mimetic Systems, EPFL-DI
Swiss Federal Institute of Technology, 1015 Lausanne, Switzerland
Email: Mona.Spiridon@epfl.ch

Carson C. Chow
Center for Biodynamics and Department of Mathematics,
Boston University, Boston, MA 02215, USA.
Email: ccc@bu.edu@epfl.ch

Wulfram Gerstner
MANTRA, Center for Neuro-Mimetic Systems, EPFL-DI
Swiss Federal Institute of Technology, 1015 Lausanne, Switzerland
Email: Wulfram.Gerstner@epfl.ch

Abstract

Biological neurons are noisy and integrate their inputs over an extended period of time. On the other hand, the nervous system is able to transmit temporally-changing signals reliably. We address this problem with a homogeneous, fully connected model network. Neurons in our model have an absolute refractory period and fire with a stochastic rate that depends on the input they receive. An analytical solution of the noise spectrum of a population of such neurons is derived, which is in good agreement with simulations. We find that strong inhibitory couplings can considerably reduce the noise level in a certain frequency band. This allows the system to transmit signals with very low noise at frequencies close to the single-neuron mean firing rate.

1 Introduction

In many areas of the brain, neurons are organized in groups of cells with similar properties. Prominent examples are columns in the cortex and pools of motor neurons. What is the use of having large numbers of similar neurons in such a population? A natural answer is that averaging over many (noisy) elements improves the signal-to-noise ratio. Normally, if we average over N independent elements we will get an improvement in the signal-to-noise ratio

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which is proportional to the square root of N. With a suitable coupling, however, the neurons can be made to fire incoherently and moreover systematically *anticorrelated.* The anticorrelations improve the signal-to-noise ratio further – beyond the squareroot of N averaging improvement. We show in this paper how inhibitory coupling can significantly increase the signal to noise ratio in a given frequency band.

2 The Model

2.1 Population activity

We consider a network of stochastic neurons with absolute refractoriness. We idealize the biological situation and assume that the network is completely homogeneous, i.e., all neurons have the same parameters, and receive the same input. We define the activity of a population of N neurons as the proportion of active neurons in a short interval Δt:

$$A(t) = \lim_{\Delta t \to 0} \frac{1}{\Delta t} \frac{n_{\mathrm{act}}(t; t + \Delta t)}{N} = \frac{1}{N} \sum_{t^{(f)}} \delta(t - t^{(f)}) \tag{1}$$

where $n_{\mathrm{act}}(t; t + \Delta t)$ is the number of neurons that fire during the interval Δt and $t^{(f)}$ is the firing time of a pulse. The sum runs over all firings of all neurons in the population. If we assume that the population is large we can take the limit $N \to \infty$, which gives a continous variable A with units s^{-1}. The activity (1) corresponds to a spatially averaged rate.

2.2 The Model

Neurons in the population communicate with each other via spikes. Input from a presynaptic neuron will produce a change in the membrane potential which can be positive (EPSP) or negative (IPSP) and which is described by a kernel $\varepsilon(t - t^{(f)})$ where $t^{(f)}$ is the firing time of a pulse. To impose causality, $\varepsilon(s)$ must vanish for $s < \Delta_{ax}$, where Δ_{ax} is the axonal delay. The input is weighted with a synaptic efficacy J/N. Scaling with N^{-1} ensures consistent behavior in the limit of $N \to \infty$. The total potential is

$$h(t) = \frac{J}{N} \sum_{t^{(f)}} \varepsilon(t - t^{(f)}) + h^{\mathrm{ext}}(t) \tag{2}$$

where $h^{\mathrm{ext}}(t)$ is some external signal with zero temporal average and J/N is a coupling constant. The sum runs over all firing times of all neurons.
Using the activity (1) in the form $A(t) = N^{-1} \sum_{t^{(f)}} \delta(t - t^{(f)})$, we can rewrite Eq.(2)

$$h(t) = J \int_0^\infty \varepsilon(s) A(t - s)\, ds + h^{\mathrm{ext}}(t)\,. \tag{3}$$

Each neuron generates pulses stochastically with intensity $\rho[h(t)]$. If the potential is high, the neuron will have a high probability of firing and a low probability if it is low. This situation corresponds to a noisy threshold. After spike emission, there is an absolute refractory period d (dead time) during which the emission of a further spike is impossible. The activity may be described by

$$A(t) = \left\{ 1 - \int_0^d A(t-s)\, ds \right\} \rho[h(t)] + \xi(t)\,. \tag{4}$$

The factor in curly brackets takes care of the fact that neurons which have fired between $t-d$ and t are refractory and can not immediately fire again [1]. ξ is noise term with zero mean and time-dependent variance $\sigma(t)$: $\langle \xi(t) \rangle = 0$ and $\langle \xi(t)\xi(t') \rangle = \sigma^2(t)\, \delta(t-t')$. To get an expression for σ^2 consider discrete time dynamics with time step Δt. In an interval $[t, t+\Delta t]$, the expected number of firings is $\overline{n} = \langle n_{act}(t; t+\Delta t) \rangle = N\, [1 - \int_0^d A(t-s) ds]\, \rho[h(t)] \Delta t$. For large N the actual number of firings $n_{\text{act}}(t; t+\Delta t)$ varies as $\sqrt{\overline{n}}$. To get the variance of the activity, we have to divide by $N^2 \Delta t^2$; cf. (1). Hence, we have $\sigma^2(t; t+\Delta t) = \overline{A}(t)/N\Delta t$ with $\overline{A}(t) = \overline{n}/N\Delta t$. Note that (in the limit of large N), the variance scales as N^{-1}. For $N \to \infty$ the noise vanishes and Eq.(4) reduces to the Wilson-Cowan population equation [1].

3 Noise Spectrum

3.1 Theory

We want to calculate the noise spectrum in the case of constant input ($h^{\text{ext}}(t) = h_0$). To do so, we take a piecewise linear stochastic intensity:

$$\rho[h] = h\, \mathcal{H}(h). \tag{5}$$

$\mathcal{H}(\mathrm{x})$ is the Heaviside function that assures a positive spiking probability.

For $N \to \infty$, the activity (4) tends to a fixed point $A(t) = a_0$ given by

$$a_0 = (1 - a_0 d)\, (J a_0 + h_0) \tag{6}$$

Stability of a_0 will be discussed below. If the fixed point is stable, then the main effect of a finite number N of neurons is to cause the activity to fluctuate around a_0. To calculate the spectrum of the fluctuations, we set $A(t) = a_0 + a_1(t)$ and linearize Eq.(4) around a_0. Taking the Fourier transform and squaring the result gives the spectrum

$$|\tilde{a}_1(\omega)|^2 = \frac{\sigma^2}{|1 - [1 - a_0\, d]\, J\, \tilde{\varepsilon}(\omega) + (J a_0 + h_0) \tilde{1}_{[0,d]}(\omega)|^2} \tag{7}$$

where the tilde denotes the Fourier transform and $1_{[0,d]}$ denotes the indicator function which is unity on the interval $[0, d]$ and vanishes otherwise. For finite Δt, the variance is $\sigma^2 = a_0/N\Delta t$. The fixed point is stable if the denominator of Eq.(7) has no poles.

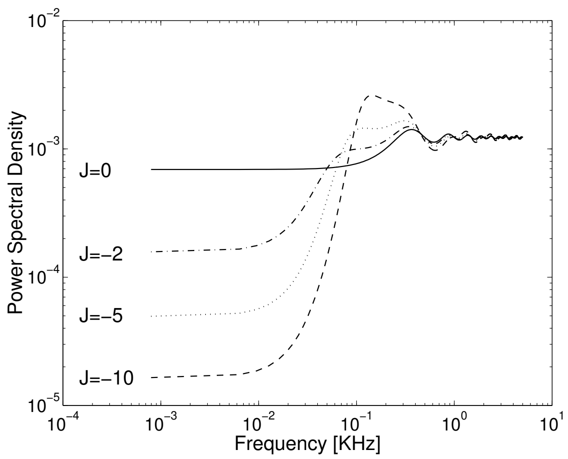

Figure 1: Theoretical noise spectrum for different coupling strenghs J on a log-log scale. The mean activity a_0 has been fixed to 125 Hz. The transmission delay $\Delta_{\rm ax}$ is equal to 1 ms and refractory period is 2 ms. The curve for J = 0 shows the effects of absolute refractoriness. For strong inhibitory coupling (J = -10), the noise level in the frequency band below 80 Hz is significantly reduced.

In the limit of $N \to \infty$ the variance $\sigma \to 0$ and the spectrum vanishes - as it should be since fluctations are levelled out by averaging. For finite N and in the absence of coupling ($J = 0$), the spectrum shows the effect of refractoriness [2]. In the limit of no refractoriness ($d = 0$) Eq.(7) reduces to the Hawkes formula for coupled point processes [3].

Figure 1 shows the theoretical spectrum for neurons with refractoriness $d = 2\,\mathrm{ms}$ firing at a mean activity $a_0 = 125$ Hz for different coupling strengths. The coupling kernel is $\varepsilon(s) = ((s - \Delta_{ax})/\tau^2)\,\exp(-(s - \Delta_{ax})/\tau)$ for $s > \Delta_{ax}$ with $\tau = 4\,\mathrm{ms}$ and a transmission delay of $\Delta_{\rm ax} = 1\,\mathrm{ms}$.

3.2 Simulation

We simulate the population activity of 1000 homogenous neurons in a fully connected network using the neuronal model described in section 2.2. We apply a short initial pulse and then let the system evolve freely. For every neuron the firing of any other neuron will modify the membrane potential after an axonal delay Δ_{ax}. Noise is introduced as follows: at each time step, each neuron has a probability $\rho(t)$ of firing that depends on the state of its membrane potential via Eq. 5. The mean activity depends on the coupling strength and the refractory period (Eq 6). In order to compare simulation results performed with different couplings J, we adjust ρ_0 so as to fix the mean activity at a value of 125 Hz. This value corresponds to a mean interspike interval of 8 ms. Figure 2 shows that the noise spectrum found from the simulation is in excellent agreement with the analytical noise spectrum, which validates the above analytical calculation. Note that this is a zero-parameter fit.

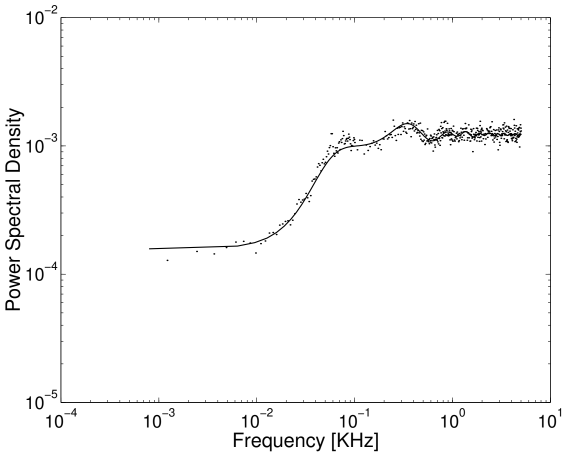

Figure 2: Comparison between the theoretical noise spectrum (solid line) and the one obtained by simulations (points) on a log-log scale. The mean activity a_0 has been fixed to 125 Hz. We use inhibitory coupling of strengh J = -2 and a transmission delay $\Delta_{\rm ax}$ of 1 ms.The refractory period is 2 ms. For the simulations, a network with 1000 neurons has been used.

3.3 Parameter dependence of the noise spectrum

Figure 1 shows that even in the absence of connections the spectrum is not completely flat, as would be expected for a pure Poisson process. Due to the refractory period, low frequencies are significantly attenuated compared to high frequency noise. Strong inhibitory coupling between the neurons will reduce the noise level at low frequencies even further (<100 Hz) (Figure 1). The signal-to-noise ratio can be up to 100 times greater at low frequencies than at high frequencies. The axonal delay, the refractory period and the time constants of the EPSP modulate the shape of the noise spectrum. Decreasing the axonal delay or increasing the refractory period will decrease the amount of noise present at low frequencies. However, an increase in the refractory period may destroy the incoherent state of the system, and the network will tend to oscillate with a period corresponding to the mean spike interval of a single neuron ($= 1/a_0$). If we decrease the time constant of the EPSP (decribed by the kernel $\varepsilon(s)$), the shape of the noise spectrum is modified (Fig. 3). For a small value of τ ($\tau = 0.5$ ms), the frequency band where noise is low is increased but the overall noise level in the band is slightly higher. The frequency band in which the signal-to-noise ratio is improved by coupling can be calculated from Eq.(7).

4 Conclusion

In this paper we have shown that a suitable choice of inhibitory connections can significantly improve the signal-to-noise ratio in the low-frequency band. A

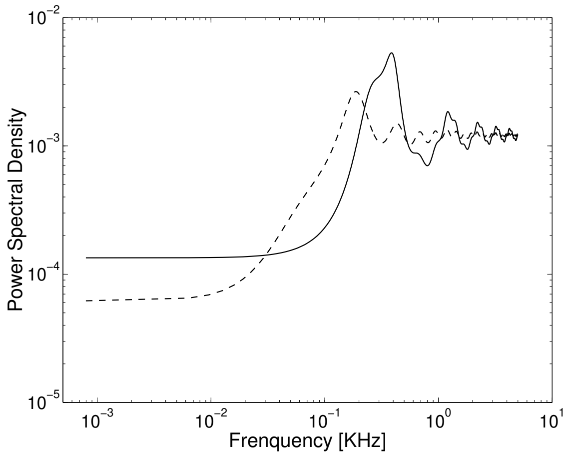

Figure 3: Theoretical noise spectrum for two EPSP time constants, $\tau = 4.0$ ms (dashed line) and $\tau = 0.5$ ms (solid line). The mean activity a_0 has been fixed to 125 Hz. The coupling strengh J = -5, the transmission delay $\Delta_{\rm ax}$ is 1 ms and the refractory period is 4 ms.

theory has been developped which is valid for stochastic neurons with absolute refractoriness. We have checked in additional simulations that similar results also holds for integrate-and-fire neurons. Strong inhibition with $J = -10$ seems to be somewhat academic, but the effect remains qualitatively the same (albeit weaker) for less efficient inhibition.

Finally we remark that the improvement of signal-to-noise ratio by coupling is related to the principle of noise-shaping via suitable feedback in commercial A/D converters. This gives rise to the hope that a better understanding of noise spectra in populations of spiking neurons could also help to improve applications of noise shaping [4].

References

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