Signal Compression in the Sensory Periphery

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Abstract

In this contribution we address the issue of signal compression in the peripheral nervous system. We propose a simple mechanism by which the narrow dynamical range observed experimentally in isolated sensory neurons, translates into a wide dynamical range as a result of the collective phenomenon of self-limited amplification. The mechanism is illustrated by means of different models in which excitable elements are coupled by lateral excitatory connections. The models are based on recent experimental findings that gap junctions are present in the sensory periphery.

Key words: Gap junctions, Olfactory Bulb, Retina
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1 Introduction

Live organisms have to deal with sensory stimuli whose intensities usually span several orders of magnitude. The Psychophysics literature illustrates how humans cope with that task [8]: the relation of psychological sensation to stimulus intensity is usually fit by *signal compressing* functions, such as the

logarithm (Weber-Fechner law), a power law with exponent $\alpha < 1$ (Stevens law), or variants of the Hill function. An important theoretical question then arises: how does the nervous system implement signal compression?

To prevent early saturation, it is natural to assume that this signal compression should occur already at the peripheral nervous system. However, the response function (spike frequency vs. stimulus intensity) of isolated sensory neurons usually presents a linear saturating behavior. This implies that their dynamical range, defined as the number of decades spanned by stimulus intensity between 10% and 90% of the response saturation, is usually very narrow. For instance, in isolated olfactory receptor cells of the frog [7], the stimulus intensity can be properly discriminated in a dynamical range of only about 10 dB (one decade).

While the signal compression of *individual* sensory neurons is typically poor (narrow dynamical range), we propose that the dynamical range can be extended by means of a *collective* phenomenon. The model we present is based on recent experimental evidence that electrical synapses (gap junctions) are present in the peripheral nervous system (see e.g. Ref. [2] for results concerning the retina, and Refs. [5,9] for the olfactory bulb). We make use of excitable elements to represent the sensory neurons, and connect them with passive conductances to account for the experimentally observed gap junctions. In order to illustrate the robustness of the mechanism, we have employed three different modeling levels for the excitable elements, namely: cellular automata (CA), coupled map lattices (CML) and the Hodgin-Huxley (HH) equations. All three models yield similar results: the dynamical range is greatly enhanced due to the coupling.

2 The model

The three models have complementary aspects: despite their lack of biological realism, CA are extremely useful tools for investigating the issue of signal compression. Large networks can be simulated due to their low computational cost (discrete time, discrete space) and, more importantly, the collective response of the network can be calculated under appropriate approximations. We employed the Greenberg-Hastings CA model for a neuron [3,1], which spikes (state 1) only if stimulated while at rest (state 0), undergoing an absolute refractory period (states 2 through $n-1$) before recovery.

The framework of the HH equations, on the other hand, is much more biologically plausible, allows the continuous variation of relevant parameters, the inclusion of extra currents and more detailed compartmental modeling. However, its higher computational cost limited the maximum size of the simulated networks. The values of the parameters used in our single compartment cells,

set the system in its excitable regime [4], with gap junction resistances of 100 MΩ.

CMLs stand in between the CA and HH formalisms. With continuous state variables and discrete time, maps have proven computationally inexpensive for large scale simulations, yet retaining much of the desirable dynamical features of a model neuron. We have employed the three-variable map of Ref. [6]:

$$\begin{aligned} x_i(t+1) &= \tanh\left(\frac{x_i(t) - K y_i(t) + z_i(t) + H + I_i(t) + \sum j G_{ij}(x_j - x_i)}{T}\right) , \\ y_i(t+1) &= x_i(t) , \\ z_i(t+1) &= (1-\delta)\, z_i(t) - \lambda\,(x_i(t) - x_R) \ , \end{aligned} \tag{1}$$

with $K = 0.6$, $H = -0.05$, $T = 0.34$, $\lambda = \delta = 0.1$ and $x_R = -0.85$. The membrane potential of the i-th sensory neuron at time t is represented by $x_i(t)$, while $y_i(t)$ is a recovery variable and $z_i(t)$ is a slow current. The sum runs over its neighbors j, and for simplicity we have used a constant conductance $G_{ij} = 0.3$. $I_i(t)$ represents the external stimulus on neuron i at time t.

For the three dynamical systems considered, the stimulus is modeled as a Poisson process of supra-threshold events of stereotyped amplitude: the probability of a stimulus event occurring during a time step is given by $\lambda = 1 - e^{-r\tau}$, where r is the stimulus rate and τ is the duration of a time step (= 1ms in the discrete time models). We present results for one- and two-dimensional lattices, where N is the total number of neurons.

3 Results

After stimulating the network during a sufficiently long time T, the mean firing rate per neuron F was calculated by dividing the total number of spikes in the network by NT. Note that this is the proper normalization for a fair comparison with the firing rate f of an isolated neuron.

The curves $F(r)$ and $f(r)$ can be compared in Fig. 1 for both the CML and the HH models in a one dimensional lattice, with each neuron connected to its two first neighbors. The qualitative behavior is the same for both models (as well as for the CA model, whose data is not shown for clarity). In comparison with the results for isolated neurons (open symbols), one observes a clear amplification in the response of the network with electrical synapses (full symbols). This amplification is associated to the excitable waves which propagate due to the lateral connections. For sufficiently low values of r, the wave generated at a site will propagate undisturbed through the entire lattice, and

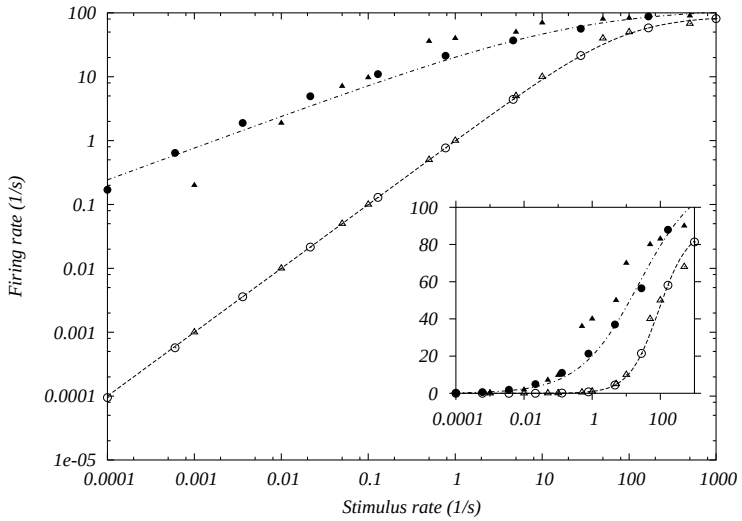

Fig. 1. Log-log plot of the mean firing rate per neuron as a function of the stimulus rate for a one-dimensional system. Circles: CML model with 2000 neurons. Triangles: HH model with 200 neurons. Full symbols: connected system. Open symbols: isolated neurons. Inset: same data, but for a linear-log plot.

the amplification F/f due to the coupling will be of the order of the system size N. For larger values of r, wave fronts will meet and annihilate each other, leading to less amplification. For even larger values of r, the coupled system is nearly undistinguishable from an isolated receptor neuron, due to the near saturation. The gap junctions therefore manage to implement a self-limited amplification, which leads to signal compression.

The effect of a change in the dimensionality of the lattice can be seen in Fig. 2, which shows the result for the CA in a two-dimensional triangular lattice, where each neuron is connected to its six nearest neighbors. The amplification is larger than in the one-dimensional case, specially for low r. In that regime, the time for a wave starting at the center of the lattice to reach the border scales with $\sqrt{N}$, instead of N for the one-dimensional case. Note that the linear regime $F \simeq Nr$ can be clearly seen in Fig. 2, stressing the relevance of the system size. The effect of the coupling for signal compression has been to enhance the dynamical range from ~ 1.8 decade, for an isolated neuron, to ~ 4 decades in the two-dimensional system.

4 Concluding Remarks

We have discussed the collective effects of lateral excitatory connections in a network of excitable cells. The excitable waves which arise with the stimuli are responsible for a self-limited amplification that leads to an enhancement of the dynamical range. The model is based on experimental findings that gap

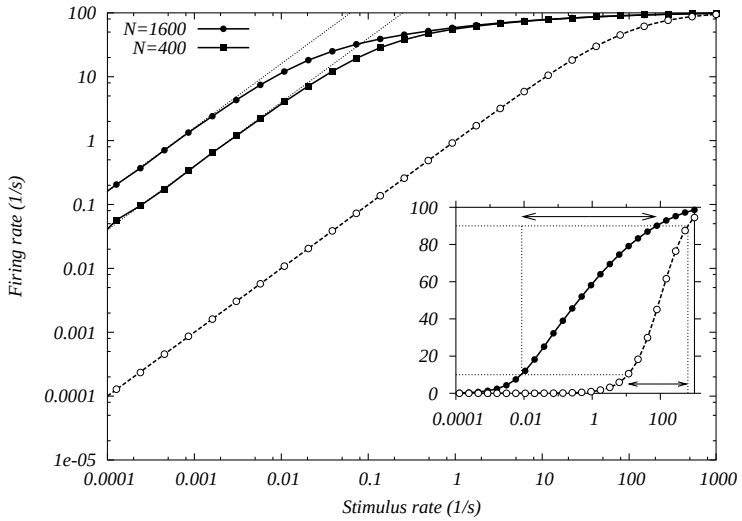

Fig. 2. Log-log plot of the mean firing rate per neuron as a function of the stimulus rate for a two-dimensional triangular lattice. CA model with 10 states. Circles: $N = 40 \times 40$ neurons. Squares: $N = 20 \times 20$ neurons. Full symbols: connected system. Open symbols: isolated neurons. Inset: linear-log plot, the same legend applies. The arrows indicate the dynamical ranges for the coupled and uncoupled systems.

junctions are present in the sensory periphery. In fact, experiments carried out for the mammalian retina show that the dynamic range of ganglion cells can drop dramatically when the gene responsible for connexin-36 is knocked out [2], yielding response functions remarkably similar to the those of Fig. 2. This phenomenon might also occur in the olfactory bulb at the glomerular layer, where gap junctions have been identified as well [9]. We are currently investigating the robustness of the mechanism under the change of topology and connectivity patterns, results will be presented elsewhere.

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References

[1] M. Copelli, A. C. Roque, R. F. Oliveira, O. Kinouchi, Physics of Psychophysics: Stevens and Weber-Fechner laws are transfer functions of excitable media, Phys. Rev. E 65 (2002) 060901(R).

[2] M. R. Deans, B. Volgyi, D. A. Goodenough, S. A. Bloomfield, D. L. Paul, Connexin36 is essential for transmission of rod-mediated visual signals in the mammalian retina, Neuron 36 (2002) 703–712.

[3] J. M. Greenberg, S. P. Hastings, Spatial patterns for discrete models of diffusion

in excitable media, SIAM J. Appl. Math. 34 (1978) 515–523.

[4] C. Koch, Biophysics of Computation, Oxford University Press, New York, 1999.

[5] T. Kosaka, K. Kosaka, Neuronal gap junctions in the rat main olfactory bulb, with special reference to intraglomerular gap junctions, Neuroscience Research 45 (2003) 189–209.

[6] S. M. Kuva, G. F. Lima, O. Kinouchi, M. H. R. Tragtenberg, A. C. Roque, A minimal model for excitable and bursting elements, Neurocomputing 38-40 (2001) 255–261.

[7] J.-P. Rospars, P. Lánský, P. Duchamp-Viret, A. Duchamp, Spiking frequency versus odorant concentration in olfactory receptor neurons, BioSystems 58 (2000) 133–141.

[8] S. S. Stevens, Psychophysics: Introduction to its Perceptual, Neural and Social Prospects, Wiley, New York, 1975.

[9] C. Zhang, D. Restrepo, Heterogeneous expression of connexin 36 in the olfactory epithelium and glomerular layer of the olfactory bulb, J. Comp. Neurol. 459 (2003) 426–439.

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