Signal Compression in the Sensory Periphery

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Abstract

We consider the signal compression problem 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 excitable waves and synchronization phenomena which provides self-limited amplification. The mechanism is illustrated by means of different models in which excitable elements are coupled by lateral electrical connections. The models are based on recent experimental findings that gap junctions are present in the olfactory sensory periphery.

Key words: Gap junctions, Olfaction, Retina, Ephaptic interaction, Excitable media, Synchronization, Neural code, Dynamical range

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 [1]: 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 (or Michaelis-Menten) function. An important 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 [1]. However, the response function (spike frequency f vs. stimulus intensity I) of isolated sensory neurons usually presents a Hill saturating behavior $f(I) = f_{max} I^{\alpha}/(C + I^{\alpha})$

with a Hill exponent $\alpha \geq 1$ (we refer to the case $\alpha = 1$ as the "linear-saturating" case). 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 [2], the stimulus intensity can be properly discriminated in a dynamical range of only about 10 dB (one decade).

The standard mechanism proposed to bypass the dynamic range problem is lateral inhibition mediated by inhibitory (chemical) synapses [3]. This enlarges the dynamic range by preventing saturation of individual neurons. In general terms this is achieved by subtracting the mean local activity from the individual neuron input. However, it must be noted that the primary motivation for lateral inhibition is not intensity coding but specificity coding, that is, to fine-tune responses from broadly-tuned sensory cells [3].

Here we propose a complementary mechanism where the dynamic range can be extended by means of a collective phenomenon: traveling waves in excitable networks. Our primary concern is not preventing saturation due to high level of input, but to enhance the sensitivity to very low input rates (say, single photon or picomolar odor detection). The model we present is motivated by recent experimental evidence that electrical coupling by gap junctions plays a key role in the detection of very low input rates [4], that ganglion cells are electrically coupled and fire together with small latency [5], by similar evidence in the olfactory periphery [6] and also by theoretical and experimental suggestions that ephaptic interactions could produce amplification of signals in the tightly packed bundles of unmyelinated olfactory nerve axons [7]. Our model works at the same descriptive level of the work of Cleland and Linster [8], that is, we compare the dynamic range of the individual olfactory sensory neuron with the global dynamic range produced by the fiber bundle of axons prior to glomerular computation. Our level of analysis is on computational principles implemented in biological networks. The next level is to search for specific biological implementations of these principles and the proposal of experiments that can test our mechanism (like to measure odor intensity coding under the presence of gap junction blockers).

We make use of excitable elements to represent the sensory neurons (or, perhaps, the sensory axons), and connect them with passive conductances to account for the experimentally observed electrical coupling (not distinguishing if they are mediated by gap junctions or by ephaptic interactions). 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 Hodgkin-Huxley (HH) equations. All three models yield similar results: sensitivity to small input levels and the dynamical range is greatly enhanced due to the coupling.

2 The models

The three models have complementary aspects: despite their lack of biological realism, CA are extremely useful tools. Large networks can be simulated due to their low computational cost 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 [9], 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 (voltage gated conduction models written in terms of nonlinear coupled ordinary differential equations), on the other hand, is much more traditional. It allows the continuous variation of relevant parameters, the inclusion of extra currents and eventually compartmental modeling. However, its computational cost limits the maximum size of the simulated networks and the volume in parameter space to be explored. The values of the parameters used in our single compartment cells are the same used in Ref. [10], 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: realistic spikes, bursts, adaptation, correct bifurcation scenarios [11]. We have employed the three-variable map of Ref. [11], which can be thought of as a discrete-time version of Hindmarsh-Rose systems.

We present results for one- and two-dimensional lattices, where N is the total number of neurons. The input to the network is modeled as a Poisson process: the probability of an element being excited 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). As a concrete biological scenario, the excitable elements could be thought of as unmielynated axons inside an olfactory nerve fascicle (which are known to pertain to a single class of receptors and innervate a single glomerulus), λ is the probability of a single fiber being activated by olfactory receptor activation, r would be a stimulus rate which we assume proportional to the concentration of odorant molecules and G is the ephaptic coupling strength between neighboring axons. Notice that the lattices represent transversal sections of the fascicles: G_{ij} are couplings between axons, here modeled by single compartment elements. The one-dimensional case apparently is not realistic but indeed is a good approximation for the case where each axon is coupled only to two neighbors, being a standard approximation in Statistical Mechanics. Also note that longitudinal propagation of the action potential along the fiber is not modeled (see the electrical coupling sites (b) and (c) in Fig. 1 of Lowe [12], which lie prior to the glomerulus).

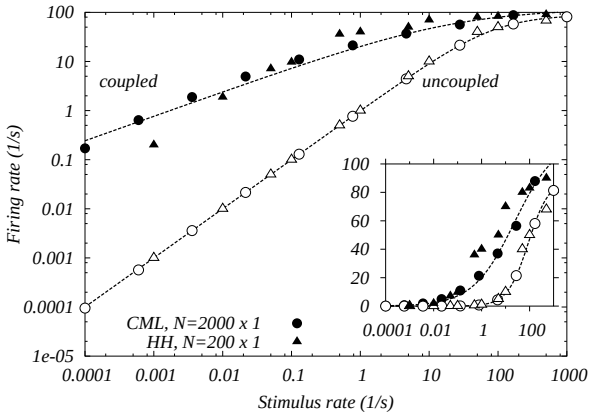

Fig. 1. Log-log (inset: linear-log) plots of $F(r)$ and $f(r)$ for a 1D system. Circles: CML model with $N = 2000 \times 1$. Triangles: HH model with $N = 200 \times 1$. Full (open) symbols: connected (isolated) neurons. Lines are fits of the Hill function: $\alpha = 1$ (uncoupled) and $\alpha = 0.5$ (coupled).

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. The qualitative behavior is the same for the CML and the HH models, as well as for the CA model (Fig. 2). We conclude that all models present the same qualitative behavior although the CA and CML computational costs are much lower than for the HH system. One observes a strong amplification of weak signals, which is due to the propagation of excitable waves. For low values of r, the wave initiated at a site propagates through the entire lattice, and one observes that $F \simeq Nf$. For higher input r, wave fronts annihilate each other, leading to less amplification. For saturating input levels, the coupled system is nearly undistinguishable from an isolated receptor neuron. A good fit of the data is obtained with a Hill function with $\alpha = 1$ for isolated neurons and $\alpha = 0.5$ for the network.

We show in Fig. 2 results 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. Note that the linear regime $F \simeq Nr$ can be clearly seen for the two-dimensional system in Fig. 2, stressing the relevance of the system size. The dynamical range is enhanced from ~ 18 dB, for an isolated neuron, to ~ 38 dB in 1D and ~ 40 dB in the 2D system. Results for the CML and HH systems are similar, but need detailed discussion about new phenomena (reflecting waves, spiral residual waves etc.) that we postpone to a more extensive report.

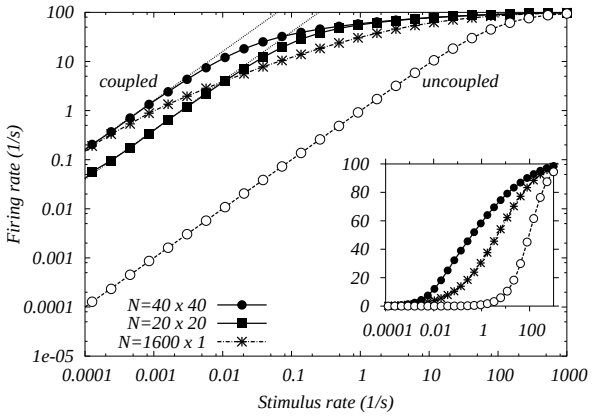

Fig. 2. Log-log (inset: linear-log) plots of $F(r)$ and $f(r)$ for a 2D and 1D CA model with 10 states. Circles and squares (2D): $N = 40 \times 40$ and 20×20, respectively. Stars (1D): $N = 1600 \times 1$. Full (open) symbols: connected (isolated) neurons.

4 Concluding Remarks

We have discussed the collective effects of electrical coupling in a network of excitable elements. Excitable waves induced by the stimuli lead to a self-limited amplification, yielding an enhancement of the dynamical range. The model is based on experimental findings that electrical coupling is present in the sensory periphery. We are currently investigating experimental proposals for testing our mechanism, such as the measurement of olfactory nerve response under the action of gap junction blockers.

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