Localized activity patterns in excitatory neuronal networks

Jonathan Rubin* Amitabha Bose†

January 15, 2004

Motivation: Sustained neuronal activity patterns that are localized in space, or bumps, have been correlated with working memory tasks (reviewed in [15, 1]), orientation selectivity in the visual system (e.g. [13, 7]), and activity in the mammalian head-direction system (reviewed in [14, 12]). Various mechanisms have been proposed as possible means by which biological neuronal networks could support such activity patterns (reviewed in [15]), and recent theoretical interest has focused on these mechanisms (reviewed in [5, 15, 1]). In particular, a standard "recipe" for generating localized activity bumps within model neuronal networks (as well as in a wide range of other physical systems) consists of local excitation, to promote activity, together with long-range inhibition, to control its spread. Synaptic connections that instantiate this so-called Mexican hat coupling pattern have been found to lead to bumps in a wide variety of model settings. Alternatively, theoretical work, motivated by thalamic networks lacking recurrent excitation, has shown that a Mexican hat connectivity is not necessary for bumps to arise. This leads to more general questions about what neuronal network features are sufficient and/or necessary for bump formation, which remain unanswered.

Summary: In this work, we present and explain a novel mechanism through which bumps can exist in *purely excitatory* neuronal networks. Examples of such bumps appear in the following figure.

Our results are of interest for a variety of reasons:

1. They add another synaptic architecture to the rather short list of those known to support bumps.

2. They show that *synaptic inhibition is not necessary* to localize activity.

3. They emphasize the role that intrinsic neuronal dynamics can play in shaping network activity patterns.

*Partially supported by NSF grant DMS-0108857; Department of Mathematics and Center for the Neural Basis of Cognition, University of Pittsburgh, Pittsburgh, PA 15260; rubin@math.pitt.edu

†Partially supported by NSF grant DMS-0315862; Department of Mathematical Sciences, New Jersey Institute of Technology, Newark, NJ 07102; bose@njit.edu

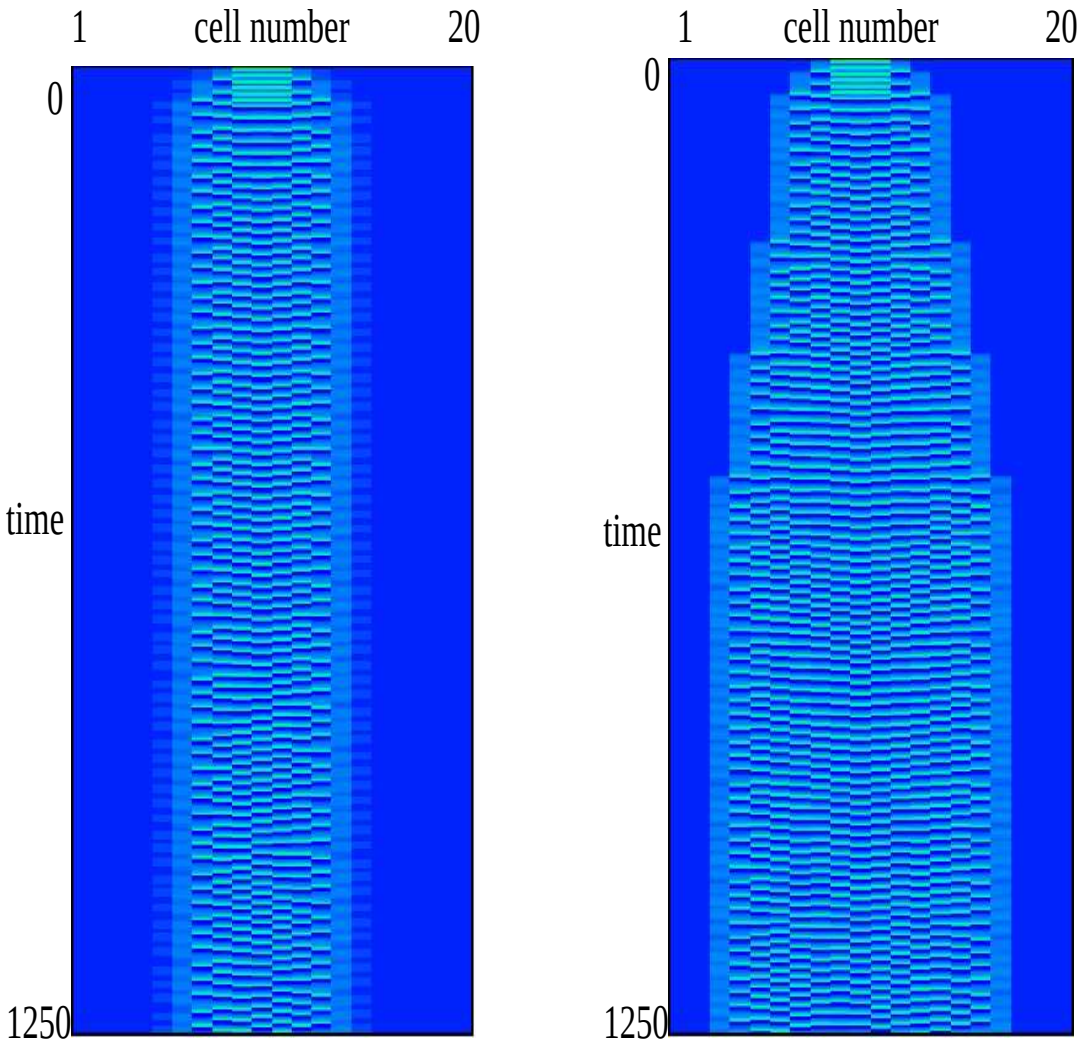

Figure 1: Bumps in a purely excitatory network. A 20 cell ring was simulated with XPPAUT [3], with each cell governed by equation (2) and with coupling to each cell from its 3 nearest neighbors on each side and itself. Activity was initiated by a transient input of positive current to three cells (shown in the center of the plot). Time evolves from top to bottom, with different cells' voltage traces appearing in different vertical columns in the plot. Pale blue regions (or light grey in greyscale) correspond to spiking while dark corresponds to silent periods. Time steps of .0025 time units were used, and values of v were only plotted once every 25 time steps. LEFT: Although we only show the first 1250 time units, this bump remained stable for 10000 simulation time units. RIGHT: A slightly different bump was obtained with a slight increase in coupling strengths. This bump appeared to remain at the size attained by time 1250, but our numerical accuracy was unable to resolve this definitively due to the sensitivity discussed below.

4. They provide a geometric elucidation of the mathematical mechanism underlying bump existence.

Details: We consider a discrete chain or ring of Type I neurons, which can exhibit oscillations at arbitrarily low frequencies, depending on the input they receive, and are taken to be quiescent without input. Within the chain, synaptic connections are purely excitatory, local (each cell is connected to 6 others and to itself), and homogeneous (the connection pattern is the same througout the chain). Synaptic strengths between connected cells depend on the distance between them in a decreasing manner. It is important to note that the self-coupling is not sufficiently strong to make isolated cells bistable.

We study two general kinds of Type I neuronal models. The first is the "theta model" [6, 2, 8], in which the dynamics of a neuron are governed by the one-dimensional equation

$$\theta' = 1 - \cos\theta + (1 + \cos\theta)(b + I_{syn}) \tag{1}$$

for synaptic current I_{syn}. The second has govening equations that are two-dimensional, typified by the Morris-Lecar model [9, 11]. A general model of this class takes the form

$$\begin{aligned} v' &= f(v, w) + I_{syn} \\ w' &= [w_\infty(v) - w]/\tau_w(v), \end{aligned} \tag{2}$$

where the v-nullcline, given by the set $\{(v, w) : f(v, w) + I_{ext} = 0\}$, is a cubic curve.

The networks that we consider can display quiescent states, where no cells are firing, and active states, with all cells firing. It is also well-known that starting with the network inititally at rest, spatially localized, transient inputs can lead to wave propagation in such networks [4, 10]. We show that appropriate brief inputs to small numbers of cells can also generate regions of sustained, localized activity, with only some subset of cells in the network firing and with active cells remaining active indefinitely (Figure 1). Moreover, these networks exhibit multistability of bump solutions of different sizes.

We use a dynamical systems approach to understand how localized bumps of activity form. We show how geometric phase plane techniques allow us to determine which cells in a network become part of the bump and which stay out. In particular, we find that transient synchrony among the population is important in recruiting cells to the bump, while the eventual desynchrony of these same cells is important both for curtailing the spread of excitation and for sustaining activity of those cells already within the bump. In fact, too much synchrony in the network can cause it to stop oscillating.

Through simulations and analysis, we find that details of bump formation, such as the size of the active region in a bump, are quite sensitive to initial conditions and changes in parameters. Nonetheless, we are able to explain several characteristics that we observed in bump formation. In particular, we explain how to control bump size, why variable delays occur in the recruitment of additional cells into the activity pattern, and how synaptic depression affects network activity.

Ermentrout [2] has shown that for networks of weakly coupled Type I spiking neurons, excitation is desynchronizing. While we don't restrict ourselves to weak coupling, a similar effect is seen in our networks. In fact, it leads directly to one of the main points of this paper: the delay in firing in response to excitation that can occur in Type I neurons can lead to desynchronization, which can in turn decrease the flow of excitatory synaptic current and stop the spread of activity. This yields an an inhibition-free way to achieve spatially localized firing. On the other hand, the sensitivity of the details of the bumps we observe suggests that inhibition, when present, may play a functional role in adding robustness to neuronal network activity patterns.

References

[1] N. Brunel. Dynamics and plasticity of stimulus-selective persistent activity in cortical network models. *Cereb. Cortex*, **13**:1151–1161, 2003.

[2] B. Ermentrout. Type I membranes, phase resetting curves, and synchrony. *Neural Comput.*, **8**:979–1001, 1996.

[3] B. Ermentrout. *Simulating, Analyzing, and Animating Dynamical Systems*. SIAM, Philadelphia, 2002.

[4] B. Ermentrout and J. Rinzel. Reflected waves in an inhomogeneous excitable medium. *SIAM J. Appl. Math.*, **56**:1107–1128, 1996.

[5] G. Ermentrout. Neural nets as spatio-temporal pattern forming systems. *Reports on Progress in Physics*, **61**:353–430, 1998.

[6] G. Ermentrout and N. Kopell. Parabolic bursting in an excitable system coupled with a slow oscillation. *SIAM J. Appl. Math.*, **46**:233–253, 1986.

[7] D. Hansel and H. Sompolinsky. Modeling feature selectivity in local cortical circuits. In C.Koch and I. Segev, editors, *Methods in Neuronal Modeling: From Ions to Networks*, pages 499–567. The MIT Press, Cambridge, MA, second edition, 1998.

[8] F. Hoppensteadt and E. Izhikevich. *Weakly Connected Neural Networks*. Springer-Verlag, New York, Berlin, and Heidelberg, 1997.

[9] C. Morris and H. Lecar. Voltage oscillations in the barnicle giant muscle fiber. *Biophys. J.*, **35**:193–213, 1981.

[10] R. Osan, J. Rubin, and B. Ermentrout. Regular traveling waves in a one-dimensional network of theta neurons. *SIAM J. Appl. Math.*, **62**:1197–1221, 2002.

[11] J. Rinzel and G. Ermentrout. Analysis of neural excitability and oscillations. In C.Koch and I. Segev, editors, *Methods in Neuronal Modeling: From Ions to Networks*, pages 251–291. The MIT Press, Cambridge, MA, second edition, 1998.

[12] P. Sharp, H. Blair, and J. Cho. The anatomical and computational basis of the rat head-direction cell signal. *Trends Neurosci.*, **24**:289–294, 2001.

[13] D. Somers, S. Nelson, and M. Sur. An emergent model of orientation selectivity in cat visual cortical simple cells. *J. Neurosci.*, **15**:5448–5465, 1995.

[14] J. Taube. Head direction cels and the neurophysiological basis for a sense of direction. *Prog. Neurobiol.*, **55**:225–256, 1998.

[15] X.-J. Wang. Synaptic reverberation underlying mnemonic persistent activity. *Trends Neurosci.*, **24**:455–463, 2001.