Multi-packet regions in stabilized continuous

attractor networks

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Abstract

Continuous attractor neural networks are recurrent networks with center-surround

interaction profiles which are common ingredients in many neuroscientific models.

The basic CANN model is often augmented with mechanisms reflecting activity-

dependent cellular nonlinearities. In this paper, we study the balance between global

competition and the stabilizing effects of cellular nonlinearities, and derive the tran-

sitions between regions in the network parameter space with different numbers of

stable activity packets.

Key words: CANN, NMDA stabilization,

1 Introduction

Wilson and Cowan [1] derived a description of the population dynamics of

neurons with excitatory and inhibitory pools coupled with center-surround in-

teraction profiles. They identified various dynamical regimes in these networks

and speculated that these regimes might map to different brain areas, includ-

ing thalamic nuclei, visual neocortex, and prefrontal cortex. Grossberg [2] dis-

covered the important implications of center-surround organizations for brain

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processes and their behavioral effects. Continuous attractor neural networks (CANNs) are now common ingredients in models of information processing in the brain and are regarded as the principle model of cortical hypercolumns [3], place and head direction cells in the limbic system [4,5], and working memory [6,7].

Basic CANN models are now often augmented with biologically realistic, intrinsic cellular nonlinearites based on, for example, the properties of NMDA-mediated ion channels [8,9,6]. These mechanisms not only solve the problem of shifting activity packets in noisy systems, but also allow the stabilization of multiple activity packets [10,11]. In this paper, we study the transitions between regions in the network parameter space in which different numbers of activity packets can be stabilized. We also outline transitions in the strength of the weight matrix required to sustain activity packets.

2 CANN dynamics and training

We consider here a basic recurrent rate model with N nodes, although corresponding networks with spiking neurons have similar properties. The time evolution of the membrane state u_i of a node with index i is given by

$$\tau \frac{\mathrm{d}u_i(t)}{\mathrm{d}t} = -u_i(t) + \sum_i w_{ij} r_j(t) \Delta x + I_i^{\text{ext}}(t), \tag{1}$$

where τ is a time constant, I_i^{ext} is the external input applied to the network, $\Delta x = 2\pi/N$ is a scale factor, and r_i is a rate that is related to u_i by a nonlinear gain function r = g(u). We use a sigmoidal gain function $g(u) = 1/(1 + \exp(-\beta(u - \alpha)))$ with a slope parameter $\beta = 0.1$ and firing threshold α .

The weight matrix **w** is determined in a learning phase with Hebbian learning, $w_{ij} \propto \sum_{\mu} r_i^{\mu} r_j^{\mu}$ on patterns with index μ . Such recurrent models are often studied after training on random patterns, resulting in networks with discrete attractors. In contrast, we study this model trained with Gaussian patterns, where each pattern is centered around a different node in the network, $\mu = 1, ..., N$. This results in an excitatory Gaussian weight matrix with width $\sigma_w = \sqrt{2}\sigma_r$,

$$r_i^{\mu} = A_{\rm r} e^{-((i-\mu)\Delta x)^2/2\sigma_{\rm r}^2} \rightarrow w_{ij}^{\rm ex} = A_{\rm r} \sqrt{\pi} \sigma_{\rm r} e^{-((i-j)*\Delta x)^2/4\sigma_{\rm r}^2},$$
 (2)

which is then augmented by an appropriate constant global inhibition C and scaled by a global, learning rate-dependent, strength constant $A_{\mathbf{w}}$,

$$w_{ij} = A_{\mathbf{w}} \left(\frac{1}{A_{\mathbf{r}} \sqrt{\pi} \sigma_{\mathbf{r}}} w_{ij}^{\mathbf{ex}} - C \right). \tag{3}$$

Appropriate values for C and $A_{\rm w}$ must be chosen to sustain an activity packet following transient input. Such values are shown in Figure 1 from simulations with periodic boundary conditions trained on Gaussian patterns with $\sigma_r = 2\pi/40$. Figure 1A illustrates the size of the activity packet as a function of C (solid line). If inhibition is too weak then the entire network becomes active, but an activity packet exists for intermediate values of C. The size of the activity packet decreases with increasing global inhibition until a critical value is reached, at which point the packet collapses. This collapse is paralleled by the maximal value of the potential $u_{\rm max} = {\rm max}_i(u_i(t=100\tau))$ as shown by the dashed line in Figure 1A. The dependence of $u_{\rm max}$ on $A_{\rm w}$ for different values of C is shown in Figure 1B. This transition does not occur with a threshold gain function.

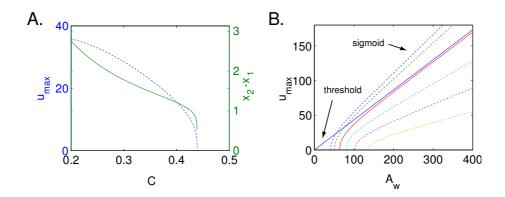


Fig. 1. (A) The width of the activity packet (solid line) and the maximal value of the potential u (dashed line) as a function of the inhibition constant C for $A_w = 70$ (B) The maximal value of the potential u as a function of the weight scaling factor A_w . The curved solid line shows the results with an inhibition constant of C = 0.4 for simulations with the sigmoidal gain function $g(u) = (1 + \exp(-0.1u))^{-1}$. The straight solid line represents the results from simulations with a threshold gain function $g(u) = \Theta(u)$ with otherwise unchanged parameters. Results from simulations with the sigmoidal gain function and different equidistant values for the inhibition constant C are shown as dashed lines, from C = 0.2 (top) to C = 0.7 (bottom).

3 Stabilization of the activity packet

A well known problem in CANN models is that noise in the weight matrix leads to a drift of the activity packet [12], though it has been argued that drift slows down with increasing network sizes [5,6]. Activity packets are also stabilized when activity dependent nonlinearities in the excitability of the neurons are taken into account [8,9]. It has also been shown that such stabilization mechanisms enable the simultaneous stabilization of multiple activity packets [10]. Here, we study the transitions between regions where different numbers of activity packets are stable.

Activity dependent nolinearities can result from intrinsic cellular bistabili-

ties such as those based on $\operatorname{Ca^{2+}}$ -gated ion channels [13] or the properties of NMDA receptors [14,15]. Regardless of the specific biological implementation, the effect of such mechanisms on node activity in rate models is captured by an activity-dependent variation of the threshold parameter α . Several specific models relating the change of *alpha* to the firing rate of the model are possible. Here, we use a simple model where the threshold is reduced instantaneously when the firing rate reaches half the maximal firing rate. Thus, the change of the threshold $\Delta \alpha$ is given by

$$\Delta \alpha = \alpha_0 \Theta(u), \tag{4}$$

where $\Theta(u)$ is the heaviside function. This mechanism works well in stabilizing the location of an activity packet [9]. Indeed, this mechanism can be used to stabilize multiple activity packets [10], though only a small number of packets can be stabilized simultaneously [11].

4 Stabilization versus competition

The stabilization mechanism outlined in the previous section is sufficient to sustain neural activity after transient external stimuli without further support by other neurons in the neural layer [14]. However, a network without the lateral connections typical of CANN models cannot implement the competition between stimuli that is essential to much of the brain processing for which CANN networks were proposed. Here we ask how strong the stabilization can be without losing the competitive nature of the network.

To test if a given number of activity packets can be simultaneously stabilized,

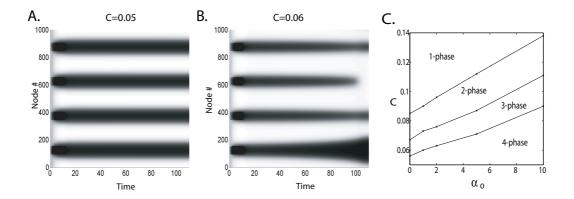


Fig. 2. (A) Network activity over time for a simulation with external input at nodes 125, 375, 625, and 875 in a 1000 node network until $t = 10\tau$ with inhibition constant C = 0.05. (B) Corresponding simulations for C = 0.06. (C) Transitions between n-phases dependent on inhibition C and threshold adjustment α_0 . The transitions between the n and n + 1 phases were thereby studied with n + 1 of the original 4 input bands.

we started the network on external inputs with different numbers of activity bands, where one input was slightly larger than the other. An example of 4 stable activity packets is shown in Figure 2A for a simulation with parameters $\sigma_r = 2\pi/80$, $A_w = 200$, $a_0 = 0.1$, and C = 0.05. The sustained activity demonstrates the stability of the packets. The results of a simulation with slightly increased inhibition constant, C = 0.06, is shown in Figure 2B. Inhibition is now strong enough to introduce sufficient competition in the network such that one activity packet disappears within the time of $t = 100\tau$ following removal of the external stimulus. We call the area where n equidistant activity packets will not considerably decay within the time of $t = 100\tau$ an n-phase. The results of several simulations with varying values of a_0 and C are summarized in Figure 2C for different numbers of initial activity bands. Stabilizing more than 4 activity packets with the current parameters is impractical.

5 Discussion and conclusion

In the literature, much attention has been paid to stabilizing activity packets in CANN models of working memory, but surprisingly little research has focused on balancing stabilization such that the model retains its winner-take-all functionality. Stabilization mechanisms are important to the biological plausibility of CANN models, but stabilization that sustains multiple activity packets does so at the expense of the global competition that makes the network useful. Strong stabilization effectively partitions the network into a series of local networks with winner-take-all characteristics similar to networks with short range inhibition [16].

The location of transitions between n-phases depends on the specific choice of parameters such as the width of training patterns and the closeness of activity bands in external inputs. Regardless of the specific transition locations, stabilization must be restricted to achieve winner-take-all functionality in a CANN model. This relationship between stabilization and inhibition with respect to the winner-take-all functionality is demonstrated by the transition between the 1-phase and 2-phase in this paper. What is unclear, however, is whether stabilization weak enough to preserve winner-take-all functionality is strong enough to prevent drift. This issue should be investigated further.

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