

The Gamma Cycle and its Role in the Formation of Assemblies

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Abstract. Rhythmic synchronization of activated neural groups in the gamma-frequency range (30-100 Hz) is observed in many brain regions. Interneuron networks are key to the generation of these rhythms. Motivated by the inhibitory effect of interneurons and summarizing experimental findings, it was recently proposed that the corresponding gamma cycle realizes a rapidly repeating winner-take-all algorithm. Here, this interpretation is considered from the modeling perspective, starting from an oscillatory network model with several stored patterns. A gradient formulation is used to include inhibitory pulses. The resulting dynamics is discussed, identifying temporal coding assemblies with coherent patterns. Thereby, the winner-take-all hypothesis is combined with binding-by-synchrony and confirmed.

1 Introduction

Rhythmic synchronization of activated neural groups in the gamma-frequency range (30-100 Hz) was observed in many brain regions. Several observations established that interneuron networks play an essential role for the generation of these rhythms; see the review and related references in [1]. It was proposed recently, in the context of the review in [1], that the gamma cycle may constitute a rapidly repeating winner-take-all algorithm. In the following, this proposal is discussed in the context of a recently proposed network model that combines synchronization with a desynchronizing mechanism, denoted as acceleration. The latter mechanism lets neural units oscillate with higher phase velocity in case of stronger and/or more coherent input from the other units [2, 3, 4].

In section 2, the model is described and extended with inhibitory couplings, corresponding to the inhibitory effect of interneurons. In section 3, an example is given that demonstrates the winner-take-all dynamics in combination with binding-by-synchrony. The conclusions and an outlook are given in section 4.

2 The Model with Pulse Activity and Inhibition

Consider a network with N units, where each unit k , $k = 1, \dots, N$, is described in terms of an amplitude $V_k = g(u_k) = (1 + \tanh(u_k))/2$, where g corresponds to

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the activation function of classical networks, and a phase θ_k that describes the temporal structure of the unit. In the following, we assume that the network dynamics is given by a phase-extension of the classical Cohen-Grossberg-Hopfield model:

$$\tilde{\tau}(u_k) \frac{du_k}{dt} = I_k - u_k + \frac{1}{N} \sum_{l=1}^N w_{kl}(\theta_k, \theta_l) V_l \quad (1a)$$

$$\tau \frac{d\theta_k}{dt} = \tau \omega_k(u, \theta) + \frac{1}{N} \sum_{l=1}^N s_{kl}(\theta_k, \theta_l) V_l, \quad (1b)$$

where τ is a time-scale, $\tilde{\tau}(u_k) = (1 - V_k)\tau > 0$, and I_k are input parameters. In the remainder of this section, we construct the w_{kl} , s_{kl} , and ω_k .

Following the approach that was described in [2], the dynamics of equation 1 is derived from a complex-valued gradient system:

$$\tau \frac{dz_k}{dt} = -\frac{\partial}{\partial \bar{z}_k} \mathcal{L}, \quad (2)$$

where $z_k^2 = V_k \exp(i\theta_k)$, $\bar{z}_k^2 = V_k \exp(-i\theta_k)$. The gradient function is given by

$$\mathcal{L} = \mathcal{P} + \mathcal{W}_{exc} + \mathcal{W}_{inh}, \quad (3)$$

where \mathcal{P} gives the dynamics of the single units, \mathcal{W}_{exc} describes the excitatory couplings, and \mathcal{W}_{inh} the inhibitory couplings.

In order to connect the model to the picture of the gamma cycle that was sketched in the introduction, we go beyond the approach discussed in [2, 3, 4] by describing pulses and specifying the inhibitory part \mathcal{W}_{inh} . Let us use

$$0 \leq \Upsilon^a(\theta) = \frac{1}{2^a} (1 + \cos(\theta))^a = \cos^{2a} \left(\frac{\theta}{2} \right) \leq 1. \quad (4)$$

to describe the pulsing character of the activity of the units (each unit should rather be understood as a population of neurons). We refer to $a \geq 0$ as peak parameter and abbreviate $\Upsilon^1 = \Upsilon$. With larger value of a , the inhibitory coupling is more narrowly peaked around $\theta = 0$. This may then be used to model the inhibitory couplings with

$$\mathcal{W}_{inh} = \frac{\gamma}{2N} \sum_{k,l=1}^N c_{kl} \Upsilon^a(\theta_k) \Upsilon^a(\theta_l) V_k V_l, \quad (5)$$

where $\gamma > 0$ and the $c_{kl} = c_{lk} \geq 0$ describe the strengths of the inhibitory couplings between units k and l . The inhibitory character of these couplings results from the signature of \mathcal{W}_{inh} , while the usage of equation 4 implies that the inhibitory coupling between units k and l is present only when the oscillators are active, where ‘‘active’’ refers to values around $V_k \simeq 1$ and $\theta_k \simeq 0$.

Let us now study the role that \mathcal{W}_{inh} may have in conjunction with pattern recognition, corresponding to the formation of temporal coding assemblies. It was stated that the mentioned proposal regarding the winner-take-all character of the gamma cycle “is clearly distinct from the binding by binding-by-synchrony hypothesis” [1] that was described, for example, in [5, 6, 7]. (See also the comments on this remark at the beginning of section 3.) Correspondingly, we implement the binding-by-synchrony hypothesis with an oscillator model \mathcal{P} and couplings \mathcal{W}_{exc} that complement the couplings \mathcal{W}_{inh} in equation 3.

As mentioned in the introduction, we use the recently discussed approach that is given by the gradient functions \mathcal{P} and \mathcal{W}_{exc} that may be found in [2, 3, 4]. Combining these with equation 5 leads to

$$w_{kl}(u, \theta) = h_{kl} \left(\alpha + \frac{\sigma}{2} \cos(\theta_l - \theta_k) - \frac{\tau\omega_3}{2} \sin(\theta_l - \theta_k) \right) \quad (6)$$

$$- \gamma c_{kl} \Upsilon^a(\theta_k) \Upsilon^a(\theta_l) \quad (7)$$

$$s_{kl}(\theta_k, \theta_l) = \sigma h_{kl} \sin(\theta_l - \theta_k) + a \gamma c_{kl} \sin \theta_k \Upsilon^{a-1}(\theta_k) \Upsilon^a(\theta_l) \quad (8)$$

and

$$\omega_k(u, \theta) = \omega_{1,k} + \omega_{2,k} V_k + \frac{\omega_3}{N} \sum_{l=1}^N h_{kl} \cos(\theta_l - \theta_k) V_l. \quad (9)$$

(For the derivation, use equations 2, 3, 5, and [2, sections 2.3 to 2.5].) The h_{kl} describe Hebbian couplings, realizing the storage of P patterns $\xi_k^p \in \{0, 1\}$, $p = 1, \dots, P$. As usual, $h_{kl} = \sum_p \lambda_p \xi_k^p \xi_l^p$, where the $\lambda_p > 0$ weight the contribution of the patterns to the memory. The σ gives the strength of synchronization, while α describes a classical, that is, phase-independent coupling strength. (In section 3, the examples use $\alpha = 0$.)

The parameters $\omega_{1,k}$ and $\omega_{2,k}$ describe eigenfrequencies and shear parameters, respectively. References to and comparisons with other phase models may be found in [2, 3, 4, 8]. The approach of equation 1 is special in using the third term on the r.h.s. of equation 9, parameterized with $\omega_3 > 0$, thereby establishing the acceleration mechanism that was described in the introduction.

3 Gamma Cycle and Binding-by-Synchrony

We will now illustrate the dynamics of equation 1 with an example.

In section 2, we mentioned the complementary character of the proposed gamma cycle mechanism and the binding-by-synchrony hypothesis. The latter mechanism is “probably based, in part, on experience-dependent refinement of intra- and inter-areal connections. In contrast, the hypothesis” related to the gamma cycle mechanism “relies on basic biophysiological dynamics that unfolds primarily within a local neuronal group” [1].

The example architecture has to take this complementarity into account by choosing inhibitory and excitatory couplings that are local and non-local, respectively. Such an architecture is easily realized by splitting the N units into

M modules of U units, $N = M \times U$, where each pattern ξ_k^p has at most one on-state unit, $\xi_k^p = 1$, for each module (thus, the pattern may have at most M on-state units), while the inhibitory couplings are non-vanishing, $c_{kl} = 1$, only among non-identical units ($c_{kk} = 0$) that belong to the same module, and $c_{kl} = 0$ whenever k and l belong to different modules. As an example, we use an architecture with $M = 32$, $U = 4$.

We choose $P = 8$ patterns that overlap as follows:

$$(\mathcal{O}^{pq}) = \begin{pmatrix} 26 & 4 & 6 & 2 & 3 & 6 & 1 & 2 \\ \cdot & 18 & 1 & 2 & 1 & 1 & 1 & 3 \\ \cdot & \cdot & 17 & 0 & 0 & 3 & 1 & 1 \\ \cdot & \cdot & \cdot & 15 & 2 & 1 & 2 & 0 \\ \cdot & \cdot & \cdot & \cdot & 15 & 1 & 3 & 4 \\ \cdot & \cdot & \cdot & \cdot & \cdot & 13 & 1 & 1 \\ \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & 12 & 1 \\ \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & 11 \end{pmatrix}, \quad (10)$$

where the symmetric elements are not repeated. For example, pattern $p = 3$ has $N_3 = 17$ units and is overlapping with pattern $p = 1$ at six units. The patterns are ordered so that $N_p \geq N_{p+1}$. With our examples, these patterns enter Hebbian weights with equal weight, $\lambda_p = 1/P$ for every p .

The parameters are chosen as $\tau = 1$, $\alpha = 0$, $\sigma = \tau\omega_3 = \pi N$, $a = 1$, and $\omega_{1,k} = \omega_{2,k} = 2\pi/\tau$, for every k . The inputs are chosen as $I_k = 0$. The discretization is obtain as in [2]. As initial values we use random u_k with small magnitudes and θ_k that are randomly distributed between 0 and 2π .

In order to describe the collective dynamics of the system, we introduce pattern activity A_p , coherence C_p , and phase Ψ_p through [3]:

$$A_p = \frac{1}{N_p} \sum_{k=1}^N \xi_k^p V_k, \quad C_p \exp(i\Psi_p) = \frac{1}{A_p N_p} \sum_{k=1}^N \xi_k^p V_k \exp(i\theta_k), \quad (11)$$

where N_p is the number of units k with $\xi_k^p = 1$. Moreover, we may describe the corresponding background quantities A_p^c , C_p^c , and Ψ_p^c by replacing $\xi_k^p \rightarrow 1 - \xi_k^p$ in equations 11.

Let us begin to demonstrate the dynamics with vanishing inhibition, $\gamma = 0$ (example 1); see figure 1A-C. Then, due to vanishing input, $I_k = 0$ for every k , we find that pattern $p = 1$ dominates due to the highest number of on-state units and gets segmented trough taking a state of enduring coherence; see figure 1B and C. This is due to the pattern segmenting effect of acceleration (a non-vanishing input could have made another pattern dominating and caused this to get segmented with respect to coherence; see [3]). We may define pattern peaks in analogy to equation 4 describing the collective dynamics; see figure 1A.

Next, we compare the situation with inhibition present, $\gamma = 3N$ (example 2); see figure 1D-F. Comparing the ratio of maximal pulse amplitudes, $A_1 C_1 / (A_1^c C_1^c)$, we find that the dominating pattern is now clearly enhanced over the background activity; see figure 1D. This confirms the winner-take-all

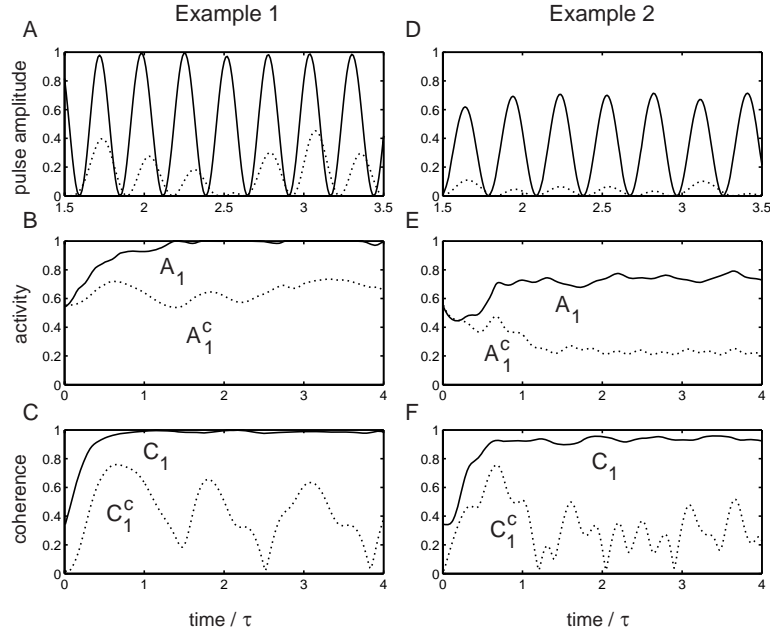


Fig. 1: (A-C) Example 1: without inhibition ($\gamma = 0$). (D-F) Example 2: with inhibition ($\gamma > 0$). (A) and (D) The solid lines show the pulse amplitudes of the dominating pattern $p = 1$, that is, $R_1 \Upsilon(\Psi_1) = R_1(1 + \cos(\Psi_1))/2$, $R_1 = A_1 C_1$, for $t = 1.5\tau$ to $t = 3.5\tau$. The dotted lines show the corresponding background values, given by A_1^c , C_1^c , and Ψ_1^c . (B) and (E) The activities A_1 (solid line) and A_1^c (dotted), for $t = 0\tau$ to $t = 4\tau$. (C) and (F) The coherences C_1 (solid line) and C_1^c (dotted), for $t = 0\tau$ to $t = 4\tau$. See the discussion in section 3.

effect of including the inhibition that constitutes the local interaction that was attributed to the gamma cycle.

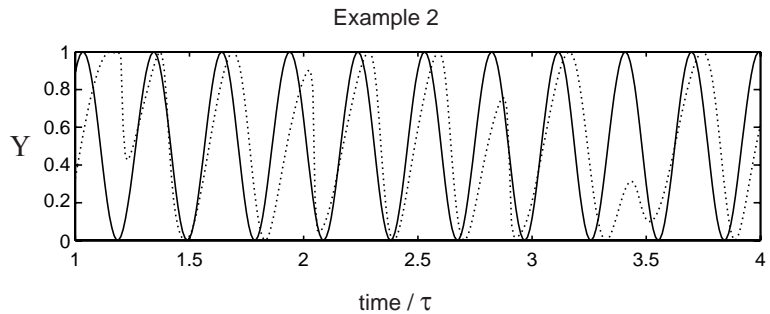


Fig. 2: The solid line gives $\Upsilon(\Psi_1) = (1 + \cos(\Psi_1))/2$, the dotted line $\Upsilon(\Psi_1^c)$. Notice the phase differences and see the remarks in section 4

4 Conclusions and Outlook

Considering a proposal regarding the functional (local) role of the gamma cycle in combination with the (non-local) binding-by-synchrony hypothesis (see [1] and references therein), we confirmed the proposed winner-take-all effect in the context of a recently discussed oscillatory neural network model. This was done by straightforwardly extending the model described in [2, 3, 4] with locally inhibitory couplings that were obtained from a gradient system. We demonstrated with an example that “although the two hypothesis are distinct, they are fully compatible to each other” [1].

Notice, in this context, another part of the proposal described in [1], related to the early firing of the winning neurons with respect to the gamma cycle that is observed in biological experiments. It is interesting to see that also this early firing is reproduced by the model; see the related phase shifts between the maxima of the winning pattern and the background in figure 2. Within the framework of the present discussion, the effect of this early firing could not be judged as being essential for the winner-take-all behavior. However, such phase shifts may be understood as being a direct consequence of the accelerating mechanism that constitutes the discussed model (see, for example, related comments in [4, section 6]). Thus, it would be of interest to explore whether the phase shifts that are observed in the brain are an experimental signal of acceleration.

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