Synchrony arising from a balanced synaptic plasticity in a network of heterogeneous neural oscillators

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Abstract

We investigate dynamics of a recurrent network of coupled heterogeneous neural oscillators with, experimentally observed, spike-timing dependent synaptic plasticity. We show both theoretically and by computer simulations that, in a regime of a balance between synaptic potentiation and depression, the network of such oscillators converges to a stable synchronous state. The stability of this state is fostered by flexible synaptic weights which adjust themselves based on the relative timing of firing of pre- and post-synaptic oscillators.

Synchrony of neural activity is a widespread phenomenon occuring in different parts of the mammalian cerebral cortex [1] and it could play a substantial functional role. For example, it was suggested that synchrony may be important in cognitive tasks [2, 3], as well as in creating transient cell assemblies [4, 5], which can efficiently transmit information to downstream cortical networks. Various mechanism of neural synchrony have been investigated. It follows from those studies that in networks with inhibitory interactions synchrony is more robust than in networks with excitatory coupling alone [6–9].

Synchronous activity of neurons can also influence the strength of synaptic connections between cortical circuits [10, 11]; the phenomenon hypothesized to be linked to long-term memory [10, 12]. Recent experimental studies [13–15] indicate that excitatory synapses are very sensitive to the temporal order of firing of pre- and post-synaptic neurons. If pre-synaptic spike precedes a post-synaptic action potential then the synapse is potentiated, if the temporal order of firing is reversed then the synapse is depressed. The temporal window for these changes is of the order of 10-20 msec, suggesting that this type of spike-timing dependent synaptic plasticity is basically short-term, although it can have long-term consequences [16]. It has been demonstrated that the sensitivity of this synaptic rule to timing can be functionally useful, since it facilitates learning of a temporally varying input to a network [17].

In this paper, we study the influence of the spike-timing dependent synaptic plasticity on coherent states in heterogeneous networks of oscillators. In particular, we investigate if excitatory synapses with the above form of plasticity can generate a stable synchronous state and under what conditions. We find that, in a most competitive regime for synaptic plasticity, i.e. when there is a balance between synaptic potentiation and depression, the network of heterogeneous oscillators converges to a globally synchronous state under a wide range of the network parameters. This finding suggests that not only inhibitory coupling but also plastic excitatory synapses can foster global coherence.

The dynamics of the system composed of N oscillators is described by the following set of equations:

$$\frac{d\theta_i}{dt} = \omega_i + \frac{\epsilon}{(N-1)} \sum_{j=1}^{N} \kappa_{ij} \left[aQ(\theta_j - \theta_i) + bc_{ij}(t) H(\theta_j - \theta_i) \right], \tag{1}$$

$$\frac{dc_{ij}}{dt} = g_{ij}^{(+)} s_i(t) u_j(t) - g_{ij}^{(-)} s_j(t) u_i(t), \tag{2}$$

$$\frac{du_i}{dt} = -\frac{u_i(t)}{\mu} + s_i(t), \tag{3}$$

for $i, j = 1, ..., N$, and where $\theta_i(t)$ is a phase of an oscillator i, ω_i is its uncoupled frequency, and κ_{ij} is a connectivity matrix for a connection from oscillator j to i. It assumes value one if the oscillator j is synaptically connected to i, and it assumes value zero otherwise. $Q(x)$ and $H(x)$ are standard coupling functions representing, respectively, inhibitory and excitatory interactions. Parameters a and b are positive constants measuring relative strength of inhibition and excitation. Symbol ϵ denotes some numerical parameter, which sets the scale for the magnitude of the coupling. We assume that excitatory synaptic efficacy $c_{ij}(t)$ is time dependent, with dynamics given by eq. (2). The first term on the right-hand side in eq. (2) corresponds to a synaptic potentiation with amplitude $g_{ij}^{(+)}$, while the second term corresponds to a depression with amplitude $g_{ij}^{(-)}$. Both amplitudes are assumed to be c_{ij} independent constants. We also assume that the value of the synaptic efficacy is bounded between 0 and 1. Function $u_i(t)$ characterizes an activity of the oscillator i, and its time evolution is given by eq. (3), with μ being a time constant related to a temporal window for pre- and post-synaptic spikes, in which spike-timing dependent plasticity is possible. Function $s_i(t)$ is a probability of firing of the oscillator i per time unit. We assume that each oscillator fires when its phase is equal to a multiple of 2π, and we choose $s_i(t) = A_i \exp\left[-\beta\left(1 - \cos\theta_i\right)\right]$, where $A_i \approx \omega_i(\beta/2\pi)^{1/2}$ is a constant coming from normalization of $s_i(t)$ over one period of oscillation ($= 2\pi/\omega_i$) to one, and β is a unitless measure of noise amplitude in the system. Noise is absent when $\beta = \infty$, and it is maximal when $\beta = 0$. Finally, eqs. (2) and (3) together, describe the synaptic dynamics in the form equivalent to that reported experimentally recently [13–15].

Two coupled oscillators . First, we study a system of $N = 2$ coupled heterogenous oscillators ($\kappa_{12} = \kappa_{21} = 1$, $\kappa_{11} = \kappa_{22} = 0$). We perform both theoretical analysis and computer simulations. In the theoretical analysis we assume that the synaptic plasticity is slow and the coupling is weak, i.e. $\epsilon \ll 1$. In simulations we do not make these assumptions. The theoretical analysis of this small network enables us to gain some insight about a behavior of a larger network. Since our goal is to examine a possibility of coherent states in the network, we look for a phase-locked solution to the system described by eqs. (1-3), i.e. $\theta_1(t) = \Omega t + \phi_1$, and $\theta_2(t) = \Omega t + \phi_2$, where ϕ_1 and ϕ_2 are some time-independent residual phases, and Ω is an emergent global frequency of oscillations. In this phase-locked state the rate of change of the synaptic efficacy c_{ij} will oscillate in time with the frequency Ω. Using eq. (3) and the expression for s_i one can determine that the time constant τ_p associated with this change is determined completely by ω_1, ω_2, and μ and is given by $\tau_p \sim (\mu\omega_i\omega_j)^{-1} \sim (\mu\Omega^2)^{-1}$. The limit of slow synaptic plasticity corresponds to $\mu\Omega \ll 1$. In this limit, we have two separate time scales: slow τ_p and fast period of oscillations $2\pi/\Omega$, satisfying $\tau_p\Omega \gg 1$. This fact allows us to perform averaging of dc_{ij}/dt over the period of fast oscillations. After that operation we find

$$\frac{d\bar{c}_{ij}}{dt} = \frac{\beta\mu\omega_i\omega_j e^{-2\beta}}{2\pi}\int_0^\infty dx\, e^{-x}\left[g_{ij}^{(+)} I_0\left(2\beta|\cos\frac{\phi_i - \phi_j + \mu\Omega x}{2}|\right)\right.$$
$$\left. - g_{ij}^{(-)} I_0\left(2\beta|\cos\frac{\phi_j - \phi_i + \mu\Omega x}{2}|\right)\right], \qquad (4)$$

where $\bar{c}_{ij}$ is a time-averaged synaptic strength differing from the original c_{ij} by a quantity of the order of $O((\tau_p\Omega)^{-1})$ [18], and I_0 is a zero-order Bessel function. From this formula, it follows that the dynamics of the average synaptic strength depends mainly on the relative phase of the two coupled oscillators. This feature ensures that, when $g_{ij}^{(+)} \neq g_{ij}^{(-)}$, a fixed point solution for $\bar{c}_{ij}$ is achieved when there is a finite phase-lock, that is, $\phi_1 - \phi_2 \neq 0$. However, when $g_{ij}^{(+)} = g_{ij}^{(-)}$, the case of the perfect balance between the synaptic potentiation and depression, there are two fixed points: one corresponding to a synchronous state, i.e. $\phi_1 = \phi_2$, and the second corresponding to an anti-phase solution $|\phi_1 - \phi_2| = \pi$. Below, we

show that only the synchronous solution is stable if we make a reasonable choice for Q and H functions. The case of the perfect balance between potentiation and depression leads to the most competitive situation for synaptic weights terminating on the same postsynaptic neuron [16, 19]. Also note that the time constant μ cannot be too small, i.e. the quantity $\mu\Omega$ cannot approach zero, since then convergence to synchrony is very slow.

In the synchronous state the global frequency of oscillations must satisfy two equations: $\Omega = \omega_i + \epsilon\left[aQ(0) + b\overline{c}_{ij}(\infty)H(0)\right]$ for $i, j = 1, 2$. They suggest that the magnitude of the emergent frequency is of the order of ω_1, ω_2, and that the difference in the frequencies $|\omega_1 - \omega_2| \sim O(\epsilon)$. The latter condition implies that oscillators's heterogeneity must be of the same order of magnitude as the synaptic coupling strength, that is weak, in order to obtain a synchronous solution.

Next, we perform a linear stability analysis for the two fixed points. We look for a solution in the form $\theta_1(t) = \Omega t + \delta\phi_1(t)$, $\theta_2(t) = \Omega t + k\pi + \delta\phi_2(t)$, and $c_{ij}(t) = c_{ij}(\infty) + \delta c_{ij}(t)$, $(i, j = 1, 2)$, where $k = 0$ $(k = 1)$ for the synchronous (anti-phase) solution. Expanding eq. (1) for small $\delta\phi_i$ and averaging over the period of oscillations, we find the time evolution of average $\overline{\delta\phi_i}$ in the form

$$\frac{d\overline{\delta\phi_i}}{dt} = \epsilon\left(\left[aQ'((-1)^j k\pi) + bc_{ij}(\infty)H'((-1)^j k\pi)\right](\overline{\delta\phi}_j - \overline{\delta\phi}_i) + bH((-1)^j k\pi)\overline{\delta c}_{ij}\right), \quad (5)$$

where $i, j = 1, 2$ and $i \neq j$. Note that fluctuations in residual phases are slowly varying in time, in particular on a much slower time scale than the period of oscillations. This information enables us to use the same averaging method in determining time evolution of fluctuations in the synaptic strength as we did in the existence part. This procedure generates the following equations:

$$\frac{d\overline{\delta c}_{ij}}{dt} = \frac{\beta\mu\omega_i\omega_j e^{-2\beta}}{\pi} F(\Omega, \mu, k)\left(\overline{\delta\phi}_j - \overline{\delta\phi}_i\right), \quad (6)$$

where $i, j = 1, 2$ $(i \neq j)$ and function $F(\Omega, \mu, k)$ is given by

$$F(\Omega,\mu,k)=\int_0^\infty dx\; e^{-x} I_1\left(2\beta|\cos\frac{\mu\Omega x+k\pi}{2}|\right) \times|\cos\frac{\mu\Omega x+k\pi}{2}|\tan\frac{\mu\Omega x+k\pi}{2}, \tag{7}$$

where $I_1(x)$ is a first-order Bessel function.

Eqs. (5) and (6) represent the stability equations, which can be rewritten in a shorter notation as $d(\delta X)/dt = \mathbf{M}\,\delta X$, with a 4-dimensional vector $\delta X = (\overline{\delta\phi}_1, \overline{\delta\phi}_2, \overline{\delta c}_{12}, \overline{\delta c}_{21})$, and a 4×4 stability matrix $\mathbf{M}$. This matrix has two degenerate zero eigenvalues. The first being a consequence of the translational phase invariance (the functions Q and H depend only on the relative phase of the oscillators), and the second corresponding to a conservation law associated with the total synaptic strength between two reciprocally connected oscillators (i.e., $c_{12}(t)+c_{21}(t)$ is time independent). The remaining two eigenvalues are negative, i.e. the corresponding fixed point is stable, only if the following two conditions are satisfied: (i) $a[Q'(k\pi)+Q'(-k\pi)]+b[\overline{c}_{12}(\infty)H'(k\pi)+\overline{c}_{21}(\infty)H'(-k\pi)]>0$, and (ii) $b[H(k\pi)+H(-k\pi)]F(\Omega,\mu,k)>0$, where prime denotes a derivative. For many biophysical models [20, 21] the coupling functions H and Q can be computed [22] and we have that: $H(0)>0$, $H'(0)>0$, $Q'(0)>0$, and $H'(\pm\pi)<0$, $Q'(\pm\pi)<0$. Using these inequalities we find that only the conditions corresponding to the synchronous fixed point ($k=0$) are satisfied. The anti-phase solution ($k=1$) breaks condition (i). These results suggest that only the synchronous state is stable.

It is interesting to ask how sensitive the synchronization is to the assumption of weak coupling between oscillators, their weak heterogeneity and slow synaptic plasticity. In Fig. 1 we display the results of simulations of two coupled oscillators with different levels of synaptic coupling and oscillator's heterogeneity. We used random initial conditions for values of phases and synaptic strengths, and we took $Q(x)=\sin x$ and $H(x)=\cos x+\gamma\sin x$ (γ is small positive constant). There is a close relationship between the strength of coupling and the level of heterogeneity. In the case of a weak heterogeneity ($2|\omega_1-\omega_2|/(\omega_1+\omega_2)\approx 0.08$), the coupling must be weak too, in order to obtain a synchronous state. For

strong heterogeneity ($2|\omega_1 - \omega_2|/(\omega_1 + \omega_2) \approx 0.70$), synchrony emerges only in the strong coupling limit. These results suggest that the key factor in achieving synchrony is that the synaptic coupling must be of the same order of magnitude as the oscillators heterogeneity, and additionally that this heterogeneity does not have to be weak. Interestingly, for the strong coupling/heterogeneity regime, the synchrony is achieved even faster than for the weak coupling/heterogeneity. The synapses achieve equilibrium at about the same time that the synchronous state emerges (Figs. 1b and 1c). Note also that the sum of synaptic strengths does not depend on time, which is a consequence of eq. (2). We also performed simulations of two coupled oscillators with slow and fast synaptic plasticity (Fig. 2). This small network converges to a synchronous state only when the synaptic plasticity time constant τ_p is much larger than the period of oscillations $2\pi/\Omega$ (slow synapses). When the period is increased, i.e. $\tau_p\Omega \sim 1$ (fast synapses), then the synchrony is unstable and the phase difference $\theta_2 - \theta_1$ oscillates in time around zero (Fig. 2). These oscillations also manifest themselves in corresponding changes in the synaptic strengths (not shown).

Network of coupled oscillators . In Figs. 3 and 4, we present the results of simulations on the dynamics of a large network containing $N = 50$ heterogeneous oscillators. The uncoupled frequencies of oscillations ω_i $(i = 1, ..., N)$ are distributed uniformly between some ω_{min} and ω_{max}, the coupling is moderate ($\epsilon = 0.3$), and elements of the connectivity matrix κ_{ij} are random and non-symmetric with diagonal elements equal to zero. We are primarily interested in how (i) sparseness in synaptic connections, (ii) an imbalance in the synaptic potentiation and depression, and (iii) topology of the connectivity matrix influence the synchronization. In the regime of the perfect balance between potentiation and depression the synchrony turns out to be insensitive to the degree of the network connectivity up to a very low connectedness. As an example, in Fig. 3 we display the activity of oscillators as a function of time in a sparsely connected random network (25% average connectivity). Note that synchrony is achieved after only few spikes. A regime of imbalance between potentiation and depression is modeled by putting: $g_{ij}^{(+)} = (1 + q\eta_{ij})a_{ij}$ and $g_{ij}^{(-)} = a_{ij}$, where a_{ij} is some non-symmetric random matrix and η_{ij} are uniformly distributed between -1 and 1. The latter choice implies that in a population of oscillators there are approximately as many

potentiated as depressed synapses. We varied an imbalance parameter q from zero to some small finite value and looked how this affects the synchrony. Again, it turns out that the degree of synchrony is quite high even for substantial values of q, strong heterogeneity, and low random connectedness (Fig. 4). It is interesting to note that the synchrony is insensitive to the topology of oscillator connectivity, i.e. the synchrony is stable both for symmetric (reciprocal) and non-symmetric matrices κ_{ij}.

We investigated a network of heterogeneous neural oscillators with plastic excitatory synapses. In the regime in which there is a balance, even imperfect, between potentiation and depression in the synaptic efficacies, we find that the network converges to a stable synchronous state. Such a balanced state could be produced by some, yet unknown, slow process, which regulates the interplay between potentiation and depression. Note that synchronization, which is a product of this balanced state, is fostered by the plastic excitation and inhibition does not play a part in it. This fact could have functional implications for synchronization between cortical areas, which are connected primarily by excitatory synapses.

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Figure Captions

Fig. 1

Weak vs. strong coupling and heterogeneity. (a) Dependence of the phase difference function $\Delta\theta = \sin([\theta_1(t) - \theta_2(t)]/2)$ between two oscillators on the time course. Solid line corresponds to weak coupling ($\epsilon = 0.1$), dashed line corresponds to strong coupling ($\epsilon = 0.5$). A corresponding dependence of the synaptic strength on the time course in the weak coupling (b) and strong coupling (c) limits. Paramters used in simulations: weak coupling: $\epsilon = 0.1$, $\mu = 5$ ms, $\omega_1 = 0.085$ ms^{-1}, $\omega_2 = 0.092$ ms^{-1}; strong coupling: $\epsilon = 0.5$, $\mu = 5$ ms, $\omega_1 = 0.029$ ms^{-1}, $\omega_2 = 0.061$ ms^{-1}. Other parameters: $a = 0.12$, $b = 0.15$, $\beta = 50$, are the same in both cases.

Fig. 2

Slow vs. fast synaptic plasticity. Dependence of the phase difference function $\Delta\theta$ between two oscillators on the time course. Solid line correspondes to slow synapstic plasticity ($\mu\Omega \approx 0.2$), dashed line corresponds to fast synaptic plasticity ($\mu\Omega \approx 1.3$). Parameters used in simulations: slow plasticity - $\omega_1 = 0.017$ ms^{-1}, $\omega_2 = 0.023$ ms^{-1}, $a = 0.036$, $b = 0.045$; fast plasticity - $\omega_1 = 0.11$ ms^{-1}, $\omega_2 = 0.15$ ms^{-1}, $a = 0.24$, $b = 0.30$. Other parameters: $\mu = 10$ ms, $\epsilon = 0.3$, $\beta = 50$ are the same in both cases.

Fig. 3

Effect of sparseness of connections on synchrony. Activity of $N = 50$ oscillators as a function of time. Dots denote firing times of a given oscillator. For every excitatory synapse we took $g_{ij}^{(+)} = g_{ij}^{(-)}$. Parameters used: $\mu = 5$ ms, $\epsilon = 0.3$, average connectivity= 25%, $a = 0.48$, $b = 0.60$, $\beta = 50$. Frequencies ω_i are uniformly distributed between 0.08 ms^{-1} and 0.12 ms^{-1}.

Fig. 4

Impact of synaptic plasticity imbalance on synchrony. The amplitude of imbalance between potentiation and depression is $q = 0.2$. Frequencies ω_i are uniformly distributed between 0.07 ms^{-1} and 0.13 ms^{-1}. Other parameters and notation are the same as in Fig. 3.