

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

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(Received 14 May 2001; revised manuscript received 31 October 2001; published 8 February 2002)

We investigate the 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 postsynaptic oscillators.

DOI: 10.1103/PhysRevE.65.031902

PACS number(s): 87.18.Sn, 87.18.Hf, 87.19.La

Synchrony of neural activity is a widespread phenomenon occurring in different parts of the mammalian cerebral cortex [1] and it could play a substantial functional role. For example, it has been 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 mechanisms 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 postsynaptic neurons. If a presynaptic spike precedes a postsynaptic 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 the 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:

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

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

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

for  $i, j = 1, \dots, N$ , and where  $\theta_i(t)$  is the phase of an oscillator  $i$ ,  $\omega_i$  is its uncoupled frequency, and  $\kappa_{ij}$  is a connectivity matrix for a connection from oscillator  $j$  to  $i$ . It assumes the value 1 if the oscillator  $j$  is synaptically connected to  $i$ , and zero otherwise.  $Q(x)$  and  $H(x)$  are standard coupling functions representing, respectively, inhibitory and excitatory interactions. The parameters  $a$  and  $b$  are positive constants measuring the relative strength of inhibition and excitation. The symbol  $\epsilon$  denotes some numerical parameter that sets the scale for the magnitude of the coupling. We assume that the 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. The function  $u_i(t)$  characterizes the activity of the oscillator  $i$ , and its time evolution is given by Eq. (3), with  $\mu$  being a time constant related to a temporal window for pre- and postsynaptic spikes, in which spike-timing-dependent plasticity is possible. The function  $s_i(t)$  is the 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  $2\pi$ , and we choose  $s_i(t) = A_i \exp[-\beta(1 - \cos \theta_i)]$ , 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 1, and  $\beta$  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 a form equivalent to that reported experimentally recently [13–15].

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 the behavior of a larger network. Since our goal is to examine the 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  $\phi_1$  and  $\phi_2$  are some time-independent residual phases, and  $\Omega$  is the emerging 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  $\Omega$ . Using Eq. (3) and the expression for  $s_i$  one can determine that the time constant  $\tau_p$  associated with this change is determined completely by  $\omega_1, \omega_2$ , and  $\mu$  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: the slow  $\tau_p$  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

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

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 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 antiphase solution  $|\phi_1 - \phi_2| = \pi$ . Below, we show that only the synchronous solution is stable if we make a reasonable choice for the  $Q$  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  $\mu$  cannot be too small, i.e., the quantity  $\mu \Omega$  cannot approach zero, since then convergence to synchrony is very slow.

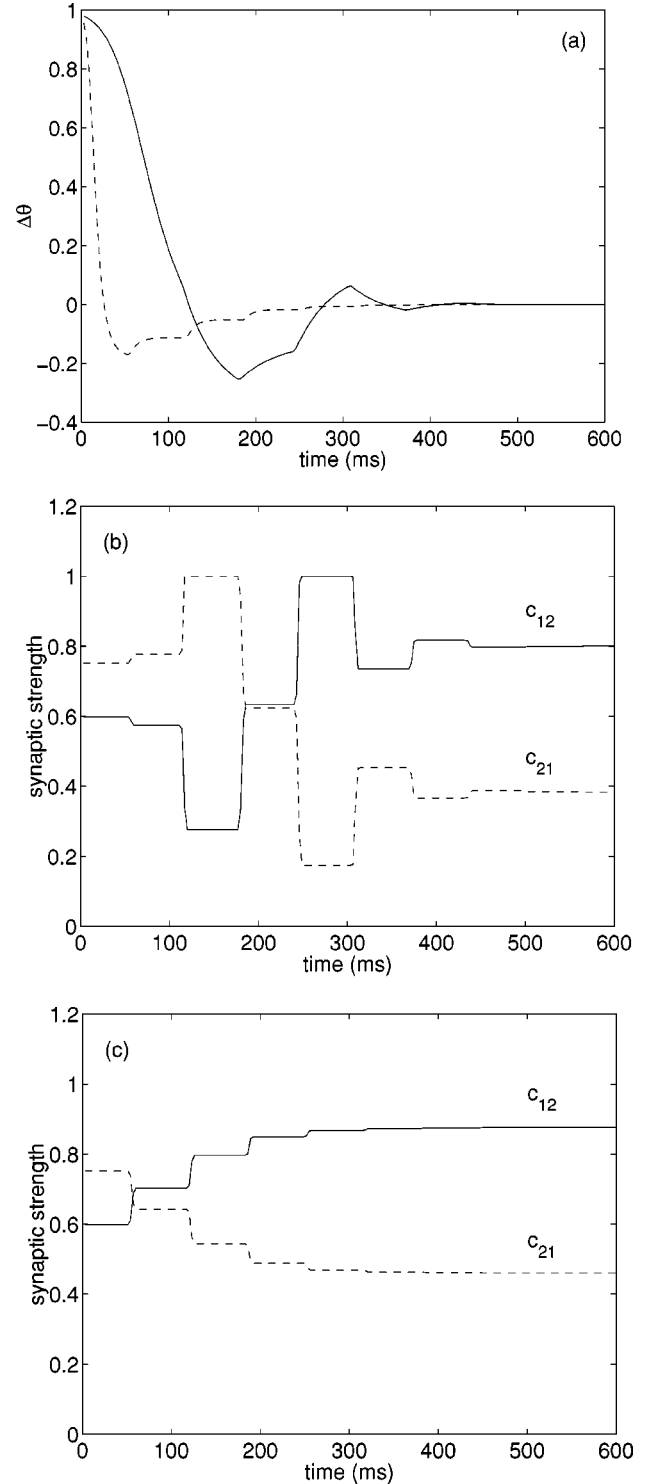


FIG. 1. Weak vs strong coupling and heterogeneity. (a) Dependence of the phase difference function  $\Delta\theta = \sin\{[\theta_1(t) - \theta_2(t)]/2\}$  between two oscillators on the time course. Solid line corresponds to weak coupling ( $\epsilon=0.1$ ); dashed line corresponds to strong coupling ( $\epsilon=0.5$ ). A corresponding dependence of the synaptic strength on the time course in the weak coupling (b) and strong coupling (c) limits. Parameters 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.

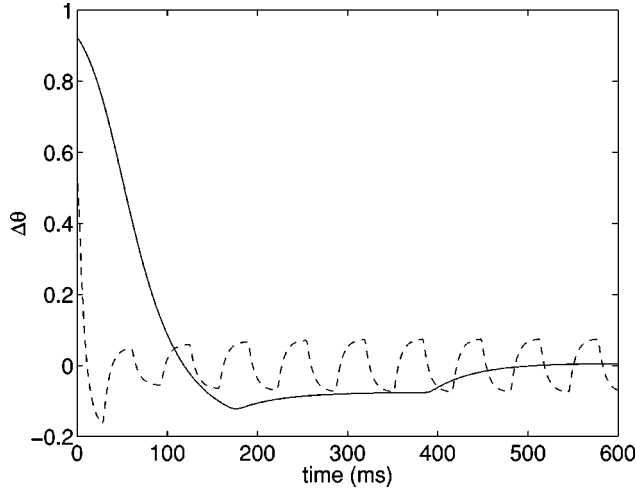


FIG. 2. Slow vs fast synaptic plasticity. Dependence of the phase difference function  $\Delta\theta$  between two oscillators on the time course. Solid line corresponds to slow synaptic plasticity ( $\mu\Omega \approx 0.2$ ); dashed line corresponds to fast synaptic plasticity ( $\mu\Omega \approx 1.3$ ). Parameters used in simulations: slow plasticity,  $\omega_1 = 0.017 \text{ ms}^{-1}$ ,  $\omega_2 = 0.023 \text{ ms}^{-1}$ ,  $a = 0.036$ ,  $b = 0.045$ ; fast plasticity,  $\omega_1 = 0.11 \text{ ms}^{-1}$ ,  $\omega_2 = 0.15 \text{ ms}^{-1}$ ,  $a = 0.24$ ,  $b = 0.30$ . Other parameters,  $\mu = 10 \text{ ms}$ ,  $\epsilon = 0.3$ ,  $\beta = 50$ , are the same in both cases.

In the synchronous state the global frequency of oscillations must satisfy two equations:  $\Omega = \omega_i + \epsilon[aQ(0) + b\bar{c}_{ij}(\infty)H(0)]$  for  $i, j = 1, 2$ . They suggest that the magnitude of the emerging frequency is of the order of  $\omega_1, \omega_2$ , and that the difference in the frequencies  $|\omega_1 - \omega_2| \sim O(\epsilon)$ . The latter condition implies that the oscillators' 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 (antiphase) solution. Expanding Eq. (1) for small  $\delta\phi_i$  and averaging over the period of oscillations, we find the time evolution of average  $\bar{\delta\phi}_i$  in the form

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

where  $i, j = 1, 2$  and  $i \neq j$ . Note that the 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 the time evolution of fluctuations in the synaptic strength as we did in the existence part. This procedure generates the following equations:

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

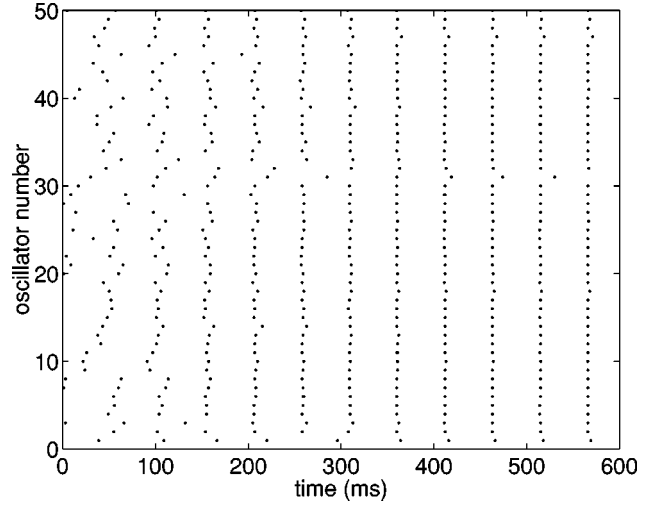


FIG. 3. Effect of sparseness of connections on synchrony. Activity of  $N = 50$  oscillators as a function of time. Dots denote firing times of a given oscillator. For every excitatory synapse we took  $g_{ij}^{(+)} = g_{ij}^{(-)}$ . Parameters used:  $\mu = 5 \text{ ms}$ ,  $\epsilon = 0.3$ , average connectivity 25%,  $a = 0.48$ ,  $b = 0.60$ ,  $\beta = 50$ . Frequencies  $\omega_i$  are uniformly distributed between  $0.08 \text{ ms}^{-1}$  and  $0.12 \text{ ms}^{-1}$ .

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

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

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

Equations (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 four-dimensional vector  $\delta X = (\delta\phi_1, \delta\phi_2, \delta c_{12}, \delta c_{21})$  and a  $4 \times 4$  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)  $a[Q'(k\pi) + Q'(-k\pi)] + b[\bar{c}_{12}(\infty)H'(k\pi) + \bar{c}_{21}(\infty)H'(-k\pi)] > 0$ , and (ii)  $b[H(k\pi) + H(-k\pi)]F(\Omega, \mu, k) > 0$ , where the 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 antiphase 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 heterogeneity. We used random initial conditions for the values of phases and synaptic strengths, and we took  $Q(x) = \sin x$  and  $H(x) = \cos x + \gamma \sin x$  ( $\gamma$  is a 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 oscillator heterogeneity, and additionally that this heterogeneity does not have to be weak. Interestingly, for the strong coupling/heterogeneity regime, synchrony is achieved even faster than for weak coupling/heterogeneity. The synapses achieve equilibrium at about the same time that the synchronous state emerges [Figs. 1(b) and 1(c)]. 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  $\tau_p$  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).

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  $\omega_i$  ( $i = 1, \dots, N$ ) are distributed uniformly between some  $\omega_{\min}$  and  $\omega_{\max}$ , the coupling is moderate ( $\epsilon = 0.3$ ), and the elements of the connectivity matrix  $\kappa_{ij}$  are random and nonsymmetric 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) the topology of the connectivity matrix influence the synchronization. In the regime of perfect balance between potentiation and depression the synchrony turns out to be insensitive to the degree of 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 a few spikes. A regime of imbalance between potentiation and depression is modeled by putting  $g_{ij}^{(+)} = (1 + q\eta_{ij})a_{ij}$  and

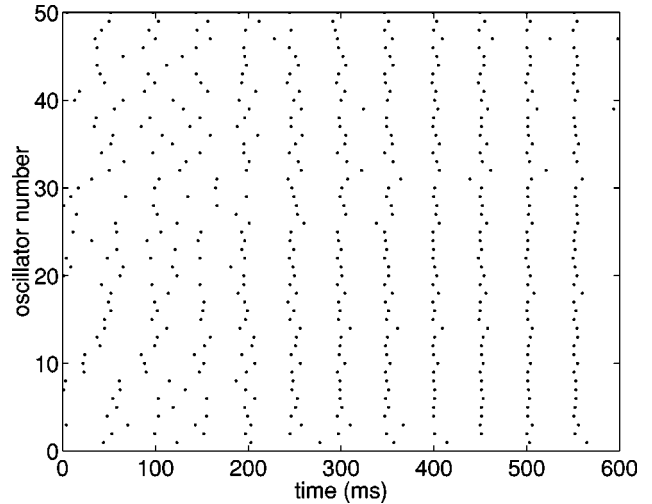


FIG. 4. Impact of synaptic plasticity imbalance on synchrony. The amplitude of imbalance between potentiation and depression is  $q = 0.2$ . Frequencies  $\omega_i$  are uniformly distributed between  $0.07 \text{ ms}^{-1}$  and  $0.13 \text{ ms}^{-1}$ . Other parameters and notation are the same as in Fig. 3.

$g_{ij}^{(-)} = a_{ij}$ , where  $a_{ij}$  is some nonsymmetric random matrix and  $\eta_{ij}$  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 the imbalance parameter  $q$  from zero to some small finite value and examined 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 for both symmetric (reciprocal) and nonsymmetric matrices  $\kappa_{ij}$ .

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, as 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.

The work was supported by NSF Grant No. DMS 9972913.

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