Temporal Decorrelation of Collective Oscillations in Neural Networks with Local Inhibition and Long-Range Excitation

Demian Battaglia, Nicolas Brunel, and David Hansel
Université Paris Descartes, Laboratoire de Neurophysique et Physiologie; CNRS UMR 8119; 45, Rue des Saints-Pères, 75270 Paris Cedex 06, France
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We consider two neuronal networks coupled by long-range excitatory interactions. Oscillations in the gamma frequency band are generated within each network by local inhibition. When long-range excitation is weak, these oscillations phase lock with a phase shift dependent on the strength of local inhibition. Increasing the strength of long-range excitation induces a transition to chaos via period doubling or quasiperiodic scenarios. In the chaotic regime, oscillatory activity undergoes fast temporal decorrelation. The generality of these dynamical properties is assessed in firing-rate models as well as in large networks of conductance-based neurons.

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Fast synchronous gamma rhythms (30–100 Hz) are observed in the neuronal activity of cortical areas [1–4]. Modeling [5–8] and experimental studies [6,9,10] suggest that these oscillations can be generated within local networks of GABAergic inhibitory interneurons.

Gamma oscillatory episodes lose temporal coherence in several tens of milliseconds [11,12]. This decorrelation could be due to noisy extrinsic feed-forward inputs, but this would require substantial spatial correlations in their fluctuations on the scale of the circuits generating the rhythm. Another possibility is that damped crosscorrelograms arise because the network is in fact close to the onset of synchrony [8]. In this case, temporal decorrelation is a finite size effect. Here, we explore an alternative mechanism in which the decoherence of gamma oscillations emerges as a collective phenomenon.

\[
\tau_E m_{1,2}^E(t) = -m_{1,2}^E(t) + \Phi[h_{1,2}^E + S_{EE} m_{1,2}^E(t - D) + S_{EI} m_{1,2}^I(t - D) + L_{EE} m_{2,1}^E(t - D)]
\]

\[
\tau_I m_{1,2}^I(t) = -m_{1,2}^I(t) + \Phi[h_{1,2}^I + S_{II} m_{1,2}^I(t - D) + S_{IE} m_{1,2}^E(t - D) + L_{IE} m_{2,1}^I(t - D)]
\]

(1)

where \(m_i^\alpha, \alpha = E, I, \text{ and } i = 1, 2, \) are the activities of population \(\alpha\) in the local network \(i\) and \(h_i^\alpha\) are external driving currents, constant in time. The interaction between populations \(\alpha\) and \(\beta\) within a local network is denoted by \(S_{\alpha\beta}\), and the interaction between the two networks by \(L^{\alpha\beta} > 0\) \((\alpha, \beta = E, I)\). The delays \(D\) and \(\bar{D}\) (in the intra- and inter-network interactions, respectively) represent synaptic and conduction delays. We choose a threshold-linear transfer function \(\Phi[x] = [x]_+ = x\) if \(x > 0\), 0 otherwise.

For simplicity, we take \(\tau_E = \tau_I = 1\), \(h_{1,2}^E = h_{1,2}^I = h_{ext}\), \(S_{EI} = S_{II}\), \(S_{EE} = S_{IE}\), and \(L_{EE} = L_{IE}\). Hence, in any attractor of the dynamics, \(m_i^E = m_i^I = m_i\). The Eqs. (1) then reduce to

\[
m_{1,2}(t) = -m_{1,2}(t) + [h_{ext} + K_0 m_{1,2}(t - D) + K_1 m_{2,1}(t - \bar{D})]_+
\]

(2)

where \(K_0 = S_{II} + S_{IE}\) and \(K_1 = L_{IE} > 0\). Now the model describes two effective populations with a local (intrapopulation) interaction \(K_0\) and a long-range (interpopulation) interaction \(K_1\).

The analysis of Eq. (2) simplifies if we assume \(D = \bar{D}\) as we do in most of the Letter. However, the results described below remain qualitatively valid for a broad range of values of \(D\) and \(\bar{D}\) even if \(D \neq \bar{D}\) [17].

Let us first consider the dynamics of one isolated population \((K_1 = 0)\). For sufficiently strong local inhibition, at \(K_0 = K_0^{osc}(D)\), the fixed point, \(m_i(t) = h_{ext}/(1 - K_0)\), loses stability via a Hopf bifurcation. For \(D \ll 1\), \(K_0^{osc}(D) \sim -\pi/(2D)\). The activity, \(m_{osc}(t)\), of the population in the resulting oscillatory regime can be derived under certain conditions [14,17]. It is also possible, under these same conditions, to compute the phase-response...
When the populations are weakly coupled ($K_1 \to 0^+$), the oscillations in their activity become phase-locked with a phase-shift, $\Delta \phi$, which can be computed by combining the expressions for $n^{\text{osc}}_i(t)$ and $Z(\phi)$ [17]. This $\Delta \phi$ depends on the local inhibition $K_0$ and on the delay $D$. We found that in general, two regimes can be distinguished as a function of $K_0$ (see Fig. 1, inset). In the first regime, $|K_{0c}^{\text{osc}}(D)| < |K_0| < |K_{0c}(D)|$, the activities of the two populations oscillate in antiphase ($\Delta \phi = \pi$). At $K_0 = K_{0c}$, a supercritical pitchfork bifurcation occurs from antiphase locking toward out-of-phase locking. For $|K_0| > |K_{0c}(D)|$, two stable intermediate phase-shifts exist, $\Delta \phi = \pi \pm \xi$, $0 < \xi < \pi$. Such dynamical configurations break the invariance of Eqs. (2) under permutation of the populations and a leader population acquires a phase advance with respect to a laggard population (spontaneous symmetry breaking). These two regimes of phase-locking persist if $K_1$ is not too large (see Fig. 1). However, when $K_1$ increases sufficiently, phase-locked oscillations destabilize and a series of bifurcations leads eventually to chaos. The largest Lyapunov exponent $\lambda_{\text{max}}$—evaluated by numerical integration of the linearized equations—increases further, an abrupt transition occurs to a symmetric chaotic state in which the fluctuations have comparable magnitudes in the two populations (see Fig. 2). Eventually, the activity of both populations goes to infinity (rate instability) when the positive feedback loop between them becomes exceedingly strong.

A different scenario occurs when considering the destabilization of the antiphase locked periodic state (see Fig. 1). The corresponding bifurcation diagram is shown in Fig. 3. In this scenario, no symmetry breaking occurs. Quasiperiodic oscillations and eventually chaotic oscillations emerge as $K_1$ increases. This is revealed by spectral analysis. As the excitation becomes stronger, two and then three incommensurate frequencies appear in the power spectrum. The first occurrence of $\lambda_{\text{max}} > 0$ is associated with the sudden broadening of the Fourier peaks (inset of Fig. 3). This behavior corresponds to the Newhouse-Ruelle-Takens quasiperiodic scenario for the onset of chaos [21]. As shown in Figs. 1 and 3, chaos is intertwined with quasiperiodic and resonant windows of period-doubled regular oscillations (doublets).

![FIG. 1 (color online). Phase diagram of the two-population rate model ($D = \bar{D} = 0.1$). Shading of the background indicates positive $\lambda_{\text{max}}$. Only the discussed bifurcation lines are plotted. (*) designs the stable homogeneous fixed-point region, (▼) a region of multistability not analyzed in the present Letter. Inset: phase shift in the weak-coupling limit ($K_0^* \approx -54.8$).](image1)

![FIG. 2 (color online). Period-doubling scenario ($K_0 = -500, D = \bar{D} = 0.1$). Bifurcation diagram for the laggard and for the leader populations. Side panels: activity traces ($\tau$ units). Bottom to top: out-of-phase ($K_1 = 1$), asymmetric ($K_1 = 13$), and symmetric chaos ($K_1 = 30$).](image2)
We conjecture that the dynamical properties described above and the destruction of coherence by long-range excitatory interactions are in fact a general feature of neuronal networks in which population synchronous oscillations are induced by local inhibition. We verified this claim in a large network model of conductance-based spiking neurons consisting of two populations of Hodgkin-Huxley type neurons [5]. Interactions among cells within a local population are purely inhibitory. For simplicity, these same cells are allowed to establish excitatory interpopulation connections [17].

The connectivity patterns are random with a probability of connection \( p^l \) (resp. \( p^E \)) for two neurons in the same (resp. different) populations. Synaptic couplings are modeled as time-varying conductances (peak conductances \( g^{ik}(t) \), rise and decay time \( \tau_1 \) and \( \tau_2 \), delay \( d \) [17]). The parameters of the network and of the external tonic input are fixed in order to obtain fast synchronous oscillations in the gamma frequency band when \( p^E = 0 \) [17]. The strength of the interpopulation excitation is then modulated by varying \( p^E \). The temporal decorrelation of the oscillations and the phase relation between the population activities are characterized by the autocorrelagrams (ACs) and the crosscorrelagram (CC) of the average neuronal voltages \( \langle V^{(a)} \rangle = \frac{1}{N} \sum_j V_j^{(a)} \), where \( V_j^{(a)} \) represents the voltage of the \( j \)-th neuron \( (j = 1, \ldots, N) \) in population \( a = 1, 2 \).

Results for \( N = 32000 \) are shown in Fig. 4 and in Fig. 5 for a strongly and relatively weak local inhibition, respectively. As a result, the oscillations are induced by local inhibition. We verified this claim in a large network model of conductance-based spiking neurons consisting of two populations of Hodgkin-Huxley type neurons [5]. Interactions among cells within a local population are purely inhibitory. For simplicity, these same cells are allowed to establish excitatory interpopulation connections [17].

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In the case of strong local inhibition, out-of-phase oscillations are observed when \( p^E \) is small (see CCs in the right column in Fig. 4). For increasing \( p^E \), the oscillations gradually become more irregular, at first only in the lag-gard population and then in both populations. As a result,
to the excitation-inhibition feedback loop (see, e.g., [6,24]). Further experimental studies are necessary to elucidate the mechanisms underlying the generation of gamma oscillations in vivo.

Previous modeling studies have considered the role of long-range excitation in synchronizing the activity of distant neuronal assemblies [25–27]. Here, we have found that chaotic activity naturally emerges when the long-range excitation is sufficiently strong. Thus, the locally generated rhythmic activity undergoes temporal decorrelation, even though, at zero time lag, the degree of synchronization between the populations increases for larger excitatory coupling. Such tightly synchronized firing might be an effective way to drive the connectivity between these populations through synaptic plasticity [28]. Besides, effective long-range interactions between populations of neurons in primary visual cortex may be modulated by the spatial patterns of a visual stimulus [29]. Our work predicts then stimulus-dependent decoherence on synchronous activity evoked in the visual cortex.

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