Temporal decorrelation of collective oscillations in neural networks with local inhibition and long-range excitation

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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 quasi-periodic 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. Modeling and experimental studies 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. 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 cross-correlations arise because the network is in fact close to the onset of synchrony. 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.

We study a model of a small piece of cortex (2–6 mm²) which consists of two local networks representing two cortical columns. Each network comprises one inhibitory and one excitatory population of neurons. Oscillations are induced independently within each network by inhibitory to inhibitory interactions. The two networks interact via their excitatory populations, as consistent with anatomical evidences that excitation has a larger spread than inhibition. We show below, in both firing-rate and conductance-based models, that when this long-range excitation is sufficiently strong, the activity of the whole system displays synchronous but highly irregular oscillations with fast temporal decorrelation. This decorrelation of the rhythmic activity is associated with the emergence of stable chaotic attractors.

We start by a simplified model in which the activity of each excitatory and inhibitory population is described by a firing-rate variable:

\[ \tau_E \dot{m}_{1,2}^E(t) = -m_{1,2}^E(t) + \Phi \left[ h_{1,2}^E + S^{EE} m_{1,2}^E(t-D) \right] + S^{II} m_{1,2}^E(t-D) + L^{EE} m_{2,1}^E(t-D) \]

\[ \tau_I \dot{m}_{1,2}^I(t) = -m_{1,2}^I(t) + \Phi \left[ h_{1,2}^I + S^{II} m_{1,2}^I(t-D) \right] + S^{IE} m_{1,2}^I(t-D) + L^{IE} m_{2,1}^I(t-D) \]

where \( m_i^\alpha, \alpha = E, I \) and \( i = 1, 2 \), are the activities of population \( i \) in the local network \( \alpha \) 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 \( L^{\alpha E} > 0 \). The delays \( \bar{D} \) 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 \( \tau_E = \tau_I = 1 \), \( h_{1,2}^E = h_{1,2}^I = h_{\text{ext}} \), \( S^{II} = S^I, S^{EE} = S^I \) and \( L^{EE} = L^I \). Hence, in any attractor of the dynamics \( m_i^\alpha = m_i \equiv m_i \). The equations then reduce to:

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

where \( K_0 = S^I + S^E \) and \( K_1 = L^I \). The model describes two effective populations with a local (intra-population) interaction \( K_0 \) and a long-range (inter-population) interaction \( K_1 \).

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

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, \( m_i(t) = h_{\text{ext}}/(1-K_0) \), loses stability via a Hopf bifurcation. For \( D < \bar{D} \), \( K_0^{osc}(D) \sim -\pi/(2D) \). The activity, \( m_{\text{osc}}(t) \), of the population in the resulting oscillatory regime can be derived under certain conditions. It is also possible, under these same conditions, to compute the phase-response curve \( \mathcal{Z}(\phi_p) = \partial \phi_p/\partial h \), which quantifies the shift in the phase, \( \phi \), of the population oscillation, following a small current perturbation \( \delta h \) applied at phase \( \phi_p \).

When the populations are weakly coupled \( (K_1 \approx 0^+) \), the oscillations in their activity become phase-locked with a phase-shift, \( \Delta \phi \), which can be computed by combining the expressions for \( m_{\text{osc}}(t) \) and \( Z(\phi) \). This
Δφ depends on the local inhibition K₀ and on the delay D. We found that in general two regimes can be distinguished as a function of K₀ (see Fig. 1 inset). In the first regime, [K₀osc(D)] < |K₀| < |K₀(D)|, the activities of the two populations oscillate in anti-phase (Δφ = π). At K₀ = K₀ the supercritical pitchfork bifurcation occurs from anti-phase locking toward out-of-phase locking. For |K₀| > |K₀(D)|, two stable intermediate phase-shifts exist, Δφ = π ± ξ, 0 < ξ < π. Such dynamical configurations break the invariance of equations (2) under permutation of the two 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 K₁ is not too large (see Fig. 1). However, when K₁ increases sufficiently, phase-locked oscillations destabilize and a series of bifurcations leads eventually to chaos. The largest Lyapunov exponent λmax — evaluated by numerical integration of the linearized equations — in fact becomes strictly positive (colored background in Fig. 1). The scenario for the onset of chaos depends critically on the strength of local inhibition as we show below.

For sufficiently strong inhibition (region Feig. in Fig. 1), chaos originates from out-of-phase locking via period doubling. This Feigenbaum scenario is confirmed by the numerical estimations of the constants δ ≈ 4.66(9) ± 0.002 and α = 2.502(8) ± 0.0001, close to their universal values [18, 29]. The bifurcation diagram shown in Fig. 2 shows the Feigenbaum cascade and the chaotic regime for K₀ = −500 and D = D = 0.1. Dots in the figure and in the inset correspond to values of the activity at a local maxima for each population. Remarkably, out-of-phase locking which occurs at weak-coupling gives rise to asymmetric chaos, for not too large K₁ (K₁ ≲ 27). This can be seen in Fig. 2 where fluctuations are considerably larger in the laggard population than in the leader. The leader population oscillations are almost periodic and can be thought of as driving the laggard. A very similar bifurcation sequence is found, indeed, if one takes the excitation between the two populations to be unidirectional [29]. As a matter of fact, it is well known that such a system can exhibit chaotic behaviors [19]. As K₁ 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 anti-phase locked periodic state (see Fig. 1). The corresponding bifurcation diagram is shown in Fig. 3. In this scenario no symmetry breaking occurs. Quasi-periodic oscillations and eventually chaotic oscillations emerge as K₁ 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 λmax > 0 is associated with the sudden broadening of the Fourier peaks (inset of Fig. 3). This behavior corresponds to the Newhouse-Ruelle-Takens quasi-periodic scenario for the onset of chaos [20]. As shown in Figs. 1 and 3 chaos is intertwined with quasi-periodic and resonant windows of period-doubled regular oscillations (doublets).

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 inter-population connections [29].

The connectivity patterns are random with a probability of connection $p'$ (resp. $p^E$) for two neurons in the same (resp. different) populations. Synaptic couplings are modeled as time-varying conductances (peak conductances $g^{I,E}$, rise and decay time $\tau_1$ and $\tau_2$, delay $d$ [29]). 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 $p^E = 0$ [29]. The strength of the inter-population 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 autocorrelationograms (ACs) and the crosscorrelogram (CC) of the average neuronal voltages $\langle V^{(\alpha)} \rangle = \frac{1}{N} \sum_j V^{(\alpha)}_j$, where $V^{(\alpha)}_j$ represents the voltage of the $j$-th neuron ($j = 1, \ldots, N$) in population $\alpha = 1, 2$. Results for $N = 32000$ are shown in Fig. 4 and in Fig. 5 for a strong and relatively weak local inhibition, respectively.

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 laggard population and then in both populations. As a result, the ACs of $\langle V \rangle$ become rapidly damped with decorrelation times on the order of tens of milliseconds (right column of Fig. 4, laggard in blue, leader in red). Note that a high degree of synchrony within each population is maintained even when the oscillations are irregular (ACs are normalized $\chi = (\sigma^2_{V_j} / \langle \sigma^2_{V_j} \rangle)^{1/2}$ [21]).

If the local inhibition is not too strong, the oscillations of the two populations lock in anti-phase for small $p^E$. They gradually become more irregular when $p^E$ is increased, but the fluctuations are now similar in the two populations. Characteristic modulations in the envelope of the ACs hint at a quasi-periodic scenario for the emergence of irregular activity (see Fig. 5).

The sequences of raster plots displayed for increasing $p^E$ in Figs. 4 and 5 reproduce the main qualitative features of the corresponding Figs. 2 and 3. Finally, a more realistic network in which each population contains separate excitatory and inhibitory cells, and in which excitatory cells fire at lower rates than inhibitory cells, exhibit qualitatively similar dynamics (see [29]).

Our combined analytical and numerical study therefore suggests that large scale networks in which local interactions are predominantly inhibitory can exhibit two robust routes to synchronized chaotic states as the strength of long-range excitation is increased. In our model, oscillations are generated locally through delayed inhibition, leading to a stochastic synchronized state in which neurons fire typically less than one spike per cycle of the oscillations [3]. Our scenario is consistent with

![FIG. 3: Quasi-periodic scenario ($K_0 = -50, D = D = 0.1$). Bifurcation diagram. Inset: power spectra. Side panels: activity traces ($\tau$ units). Bottom to top: anti-phase ($K_1 = 1$), quasi-periodic ($K_1 = 8$), chaotic ($K_1 = 11.8$), doublets ($K_1 = 12$) and chaotic doublets ($K_1 = 16$).](image_url)
experimental observations in vivo [22]. However, other scenarios have been proposed in which gamma oscillations emerge due to spike-to-spike synchrony, driven by mutual inhibition, or to the excitation-inhibition feedback loop (see e.g. [6,23]). 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 [21,25,26]. 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 [24]. Besides, effective long range interactions between populations of neurons in primary visual cortex may be modulated by the spatial patterns of a visual stimulus [28]. Our work predicts then stimulus-dependent decoherence on synchronous activity evoked in the visual cortex.

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FIG. 5: Weak local inhibition (spiking neurons): $I_E = 0.1$, $p_I = 18 \mu S/cm^2$ (other parameters in [29]). Left: raster plots (red, first; blue, second population). Right: autocorrelograms (both shown for asymmetric states) and crosscorrelograms. Bottom to top: anti-phase ($p_E = 0.08$); quasi-periodic ($p_E = 0.017$); irregular ($p_E = 0.032$); doublets ($p_E = 0.035$); irregular doublets ($p_E = 0.062$).