Stochastic dimensional reduction on large biological neural networks

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The structure-function relationship is a classical – yet difficult – problem of network neuroscience [1]. In its simplest form, one tries to predict the effect of network topology on emergent properties of the neural activity, such as synchronization [2]. Dimension-reduction is a common approach used for relating structure to function. For instance, in their seminal work [3], Wilson and Cowan combined this approach with mean-field approximations to get a simplified dynamical model and identify two key factors for neural oscillations: the presence of inhibitory neurons (i.e., nodes with negative out-weight) and the subdivision of the network into neuron populations (i.e., community structures). However, their model fails to predict some observed oscillatory behaviors, especially when the size of the network remains finite [4].

We here refine the analysis of Wilson and Cowan. We consider a network of \( N \) nodes whose states evolve stochastically according to a Markovian dynamics related to the Greenberg–Hastings cellular automata [5]. Specifically, the state of node \( j \) at time \( t \) is the random variable \( X_j(t) \) with three possible values, 0, 1, and \( i \), respectively representing the sensible, active, and refractory states. The possible states and allowed stochastic transitions are illustrated in Fig. 1, with corresponding rates \( \alpha \), \( \beta \), and \( \gamma \). The activation rate \( \alpha \) of a node is a nonlinear function of the states of its neighbors while the other transition rates are constants. We have chosen the values 0, 1, and \( i \) to facilitate the analytical calculations. Yet, without additional hypotheses, the evolution of the states remains intractable since it is governed by a system of \( 3^N \) differential equations.

To reduce the dimension of the problem, we first split the network into \( n \) populations sharing similar properties, as in Fig. 2 (a). We then introduce population analogs to the state of a node: the random variables \( A_j \) and \( R_j \), which are the arithmetic means of the \( \text{Re } X_j \)’s and of the \( \text{Im } X_j \)’s in the population \( j \). Moreover, to capture even more stochastic aspects of neural interactions, we consider the covariances of the \( A_j \)’s and the \( R_j \)’s as dynamical variables. Finally, we take the expected values \( A_j := \mathbb{E}[A_j] \) and \( R_j := \mathbb{E}[R_j] \) as well as those related to the covariances and end up with a reduced dynamical system containing only \( n(2n + 3) \) equations. When all covariances are neglected and some aspects of refractory states are simplified, the reduced system tends to the \( n \) equations of the Wilson–Cowan model. Our theoretical work and numerical simulations prove that the \( n(2n + 3) \)-dimensional reduced system, even in the simplest case of \( n = 2 \) populations illustrated in Fig. 2 (c), possesses new oscillatory behaviors that would otherwise be missed.

\[ \begin{align*}
\beta & \\
\gamma & \\
\end{align*} \]

\( \text{Fig. 1: States and transitions.} \)

\[ \begin{align*}
\beta & \\
\gamma & \\
\end{align*} \]

\( \text{Fig. 2: (a) Network with } n = 2 \text{ populations of neurons: excitatory (blue) and inhibitory (red). (b) Schematic of the reduced network corresponding to (a). (c) Two solutions of the reduced dynamical system obtained numerically, both in the case of two populations as in (a) and (b). The same parameters were used in both cases. The solution on top is obtained when taking into account the covariances, while the one at the bottom is obtained when neglecting them. The labels } E \text{ and } I \text{ mean “excitatory” and “inhibitory”, respectively.} \)

\[ \text{[1] C.W. Lynn and D.S. Bassett, Nature Reviews Physics, 1, 318 (2019).} \]
\[ \text{[3] H.R. Wilson and J.D. Cowan, Biophysical journal, 12, 1 (1972).} \]