A Note on Cracking Neuronal Oscillations via Master Equations of Reduced Models

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This note aims to propose a potential research framework to study the neural oscillation dynamics emerging from spiking network models, such as theta and gamma oscillations. We here start from homogeneous networks, so the dynamics are unaffected by specific network structures. Therefore, this framework's spirit is the following: The dynamics can be described by the numbers of neurons in different states plus the drives (external drives, conductance, or current, etc.). If we can reduce the model to a small number of states, it would be possible to use classical methods like master equations and Fokker-Planck equations to study the probability transfer between different states of the network dynamics, from homogeneity to synchronization. Hence, this framework is potentially useful to quantify the detailed temporal dynamics of networks, such as neuronal oscillations.

Full Model

Inspired by Li et al. 2017[1], we first introduce a leaky-integrate-fire-like model to describe the dynamics of a population of neurons ($N = N_E + N_I$), including excitatory and inhibitory units. This model is referred as 'full model' throughout this note. The largest differences between the full model and a common leaky-integrate-fire (LIF) network are that

1. The membrane potential of each neuron is described by finite many discrete values $\Gamma = \{-M_r, \dots 0,1, \dots, M\} \cup \{R\}$, instead of continuous states. R for the refractory state.
2. The network structure is homogenized, i.e., symmetrical all-to-all network. When a neuron fires, a random set of postsynaptic neurons are chosen to 'receive' the spike (say p neurons from N neurons).
3. When consider the (excitatory/inhibitory) conductance of each neuron, it is described by the number of spikes received by the neuron yet have not taken effect. Each spike received by the neuron takes effect with an exponential random delay time (τ_E for excitatory spikes and τ_I for inhibitory spikes). 'Taking effect' means the membrane potential jumps up/down.

Generally, the full dynamics of each neuron is given by three values: membrane potential (V_i), as well as excitatory and inhibitory spikes received yet have not taken effect ($H_i^{E,I}$). The interested reader should refer to the original paper for the detail of the model. I would like to stress here is that Li et al. built a Markov-process model with discrete states $(\Gamma \times Z_+ \times Z_+)^N$ on discrete time steps, unlike most spiking neuronal network models currently used. They have also proved that such a Markov process is ergodic and exponentially converges to a unique invariant probability measure. Furthermore, they show that this network model produces gamma oscillations with different degrees of synchrony with appropriate conductance parameters ($\tau_{E,I}$).

[1] https://arxiv.org/pdf/1711.01487.pdf

Reduced Model

The first step is to simulate a reduced model and see if neuronal oscillation can be observed. Namely, the reduced model consists of $N_E + N_I$ neurons that

1. The membrane potential of each neuron is described by two or three states: $\Gamma_2 = \{B, G\}$ or $\{R, B, G\}$. R for the refractory state, before which a neuron fires and stay unexcitable for a while; B for the base state, where a neuron is excitable, yet away from the threshold; G for gate state, where a neuron is only one spike away from firing.
2. Unlike the second point for the full model, we here provide an equivalent setup of how spikes take effect, which benefits later analysis via master equations. For excitatory spikes, consider H^E as the size of the pool of spikes that have not taken effect. When a neuron fires, a random number with a mean of p is added to H^E. Each spike in the pool takes effect independently with an exponential random delay time τ_E, and when one does, a random neuron is selected as the target neuron.

   Now consider the three-state model. 1) If the target neuron is at R state, the spike takes no effect; 2) If a G state neuron receives a excitatory spike, the target neuron fires and reset to R state (or B state for 2-state models), and if receives a inhibitory spike, the target neuron has a probability to be kicked down to B state; 3) If the target neuron is at B state, it goes to G state with certain probability.

   Without any external input, all G (R) state neurons go back to B state with exponential delay time τ_{Leak} (τ_R).

The most important issue for this step is to tune the parameters. Though others can be reused from Li et al. 2017, the transfer probabilities between states are more subtle. While this reduced model is a direct reduction from the full model, we may need different designs of transfer probabilities between states to capture dynamics in different regimes.

Master Equations for The Reduced Model

Since both the full model and reduced model concern homogeneous networks, the network dynamics are entirely determined by the number of neurons in each state and the size of pools of spikes. For the two-state model, specifically, since $N_B^E + N_G^E = N^E$, the current state of excitatory neurons can be described by (N_G^E, H^E), and (N_G^I, H^I) for inhibitory neurons. Therefore, we can consider the master equation for the probability of the network state on state $(N_G^E, H^E; N_G^I, H^I)$.

Consider a 100-neuron network where 75 are excitatory, and 25 are inhibitory, where the maximum sizes of spike pools are 300 and 100, solving the invariant probability measure equivalents to solving a 75*25*300*100 = 56250000-dimensional linear equation. While this may be computationally expensive for personal computers, I believe that the sizes of spike pools can be much smaller, since the high-number states of pools may make up very little probabilities.

The sparseness of the linear equation of invariant measure can be guaranteed by the following setup: we assume that the discrete time interval we use during the simulation is small enough that no more than 1 spike in the pool can take effect simultaneously within one step, hence no more than one neuron can fire in each step. Due to the larger time scales of τ_{Leak} and τ_R, we conclude that no more than one neuron may change its state within one step.

Further Reducing the Master Equations

The master equation of the Markov process we described above is still $O(n^4)$, and even $O(n^6)$ for three-state models, which is horrible for large-scale networks. For such purpose, we consider potential methods to further relieve our burden.

One potential method is to use an analogy of finite element methods in PDE to combine neighboring states when taking $N \to \infty$. However, this method may fail to capture the size effect.

Another method is to consider the dynamics of excitatory/inhibitory neurons separately since, in most cases, the inhibitory synapses have longer time scales than the excitatory ones. Namely, we can consider (N_G^E, H^E) as the averaged values given the current state of inhibitory neurons. With such assumptions, the Markov process is restricted to the state space of (N_G^I, H^I) with a complexity of $O(n^2)$ (or $O(n^3)$ for three-state models).

Specifically, for the mean-field regime, the core assumption is that: Comparing with the size of the network, the firing rate is very low such that firing events are temporally uncorrelated. In this regime, the numbers of states for $H^{E,I}$ are small, comparing with $N^{E,I}$.

Finally, if none of the reduction methods works, there is an ultimate one. We can simulate the network dynamics of the reduced model and collect the "trajectories" of states, then use traditional machine learning method (LLE, tSNE, etc.) to see if there is a low-dimensional structure for specific dynamics like neuronal oscillations.

What I Want to Observe

With the master equations of network states and the invariant measure, we may observe the following:

1. For neuronal oscillations, we may observe a circle around which accumulates the invariant measure. One may test how different parameters affect the concentration and size of the circle, i.e., the synchrony in the oscillation.
2. The frequency of oscillations can be observed by obtaining the most possible trajectories around the circle. With this framework, we may investigate how different parameters quantitatively alter the oscillation frequency.
3. The combination of different neuronal oscillations, like the coupling of theta and gamma, are discovered in many cortical areas. People suspect that these phenomena come from the coupling of different types of inhibitory neurons in the network. In our framework, we may add another slower inhibitory neuron and repeat everything above, if a reduction method is applicable. Hence, we may observe the coupling of theta and gamma in the invariant measure as well as the most possible trajectories.
4. Propagation of gamma across populations. Several studies like Li et al. 2018[2] and Chariker et al. 2016[3] have reported modeling the propagation and coupling of gamma oscillations across different populations of neurons representing functional hypercolumns in V1 cortex.

[2] https://arxiv.org/pdf/1805.07246.pdf

[3] https://www.jneurosci.org/content/jneuro/38/40/8621.full.pdf

More interesting ideas can be added to this list with your input. But anyway, the key point to this framework is to use a reduced model to describe network dynamics, hence the reproduction of specific dynamics like gamma oscillation may take most of the time.