Fast Convergence of Spike Sequences to Periodic Patterns in Recurrent Networks

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The dynamical attractors are thought to underlie many biological functions of recurrent neural networks. Here we show that stable periodic spike sequences with precise timings are the attractors of the spiking dynamics of recurrent neural networks with global inhibition. Almost all spike sequences converge within a finite number of transient spikes to these attractors. The convergence is fast, especially when the global inhibition is strong. These results support the possibility that precise spatiotemporal sequences of spikes are useful for information encoding and processing in biological neural networks.

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The dynamics of recurrent neural networks are thought to underlie many computations done in the brain's neural networks. Previously, rate based models of neural activity have shown that dynamics of recurrent networks can converge to attractors in the spiking rates [1]. This finding has led to many proposals on the way brain functions might be implemented with recurrent neural networks [1,2]. However, neurons interact through individual spikes, not through averaged spiking rates as assumed in the rate models. In this Letter we show that, with important modifications, the notion of attractors can be extended to more biologically realistic network models with neurons interacting through individual spikes. Here, instead of the spiking rate patterns, precise spike sequences are the attractors. Our results support the possibility that precise spatiotemporal sequences of spikes are useful for information encoding and processing in the brain [3]. Our work also provides a possible mechanism for generating precise spike sequences in groups of neurons, which have been observed in recent experiments on olfactory systems of insects [4] and song-related premotor areas of songbirds [5].

We analyze the dynamics of recurrent networks consisting of pulse-coupled leaky integrate-and-fire neurons driven by constant external inputs. In particular, we study a class of networks whose recurrent connections are dominated by global inhibition. The structures of the networks we study are general: The number of neurons, the distribution of the external inputs, and the network connectivity are all arbitrary; in addition, each neuron can have different spike threshold, resting membrane potential, and reset potential. We demonstrate that the spike sequences in these networks converge to stable periodic patterns with precise spike timings from almost all initial states of the neurons. Moreover, the convergence is rapid: The number of transient spikes preceding the periodic spike sequences is finite, and is small especially when the global inhibition is strong.

Spiking dynamics of recurrent neural networks have been studied previously [6–8], but most of these studies have focused on networks with simple structures characterized by simple connectivity, identical (or nearly identical) neurons, or uniform (or nearly uniform) external inputs. In some work, the number of neurons is limited to a few. The networks we study have more general structures and, therefore, are more biologically relevant.

The model.—The dynamics of our networks is described by the following equation:

$$\tau \frac{dV_j}{dt} = L_j + I_j - V_j - \sum_{i=1}^{N} \sum_{k=1}^{\infty} [G^E_{j,i} V_j + G^I_{j,i}(V_j - E_I)] \tau \delta(t - t_i^{(k)}), \tag{1}$$

where τ is the membrane time constant; N is the number of neurons; $V_j(t)$, L_j, I_j are the membrane potential, the resting membrane potential, and the external input of the jth neuron, respectively; $G^E_{j,i} \geq 0$ and $G^I_{j,i} > 0$ are the conductances of the excitatory and inhibitory synapses from neuron i to j (if there is no excitatory connection, $G^E_{j,i} = 0$; the conductances are scaled with the leak conductance of the neuron); E_I is the reversal potential of the inhibitory synapse (the reversal potential of the excitatory synapse is set to 0 mV); and finally, $t_i^{(k)}$ is the time of the kth spike of neuron i. When V_j reaches a threshold potential $\Theta_j < 0$, neuron j spikes, and V_j is reset to the reset potential R_j. The following relation holds: $\max(E_I, R_j, L_j) < \Theta_j$. All neurons get suprathreshold inputs; i.e., $I_j > \Theta_j - L_j \equiv C_j$, where C_j is the threshold current. In our networks, the global inhibition dominates. In other words, for any pair of neurons, a spike in one will not cause an immediate spike in the other. This requires that $G^I_{j,i}$ must be larger than a lower limit set by $G^E_{j,i}$ (the exact relation is given later in the Letter).

Basic idea.—To solve the spiking dynamics of the networks, we derive a nonlinear map. Nonlinear maps have been used previously for analyzing phase-locked states in spiking neural networks with a small numbers of neurons

(typically two) or uniform connections, with each neuron spiking with the same or slightly different frequencies [6]. Here we derive a novel map that is suitable for analyzing much more general situations with δ-pulse synaptic coupling. The derivation relies on two insights. First, the dynamics of our network is completely determined if the membrane potentials of all neurons are given at one point in time. Second, the dominance of the global inhibition over excitation guarantees that simultaneous spiking of multiple neurons is forbidden. With these two insights, we discretize the dynamics of the network into a mapping of the membrane potentials of all neurons right after one spike transmission to those right after the next spike transmission [9]. This mapping of the membrane potentials can be translated to that of *pseudo spike times (PST's),* which are linearly related to the membrane potentials and are related to the times it would have taken each neuron to spike if it were not interacting with the other neurons. The resulting *pseudo-spike-time map (PSTM)* is the basis for our analysis of the spiking dynamics of our networks.

In a network driven by a set of external inputs, different initial states of the neurons can lead to different spike sequences. A key property of the PSTM is that it is a contracting map: The PST's of the PSTM's that generate a common segment of a spike sequence converge to a common set of values. The convergence is exponentially fast with the length of the common segment. With this convergence property, we find a criterion for *stable spike sequences,* which are defined as those that do not change under small but finite perturbations of the membrane potentials at any time. We further prove with the convergence property that all stable sequences end up in periodic patterns. Intuitively, the PSTM is a contracting map for three reasons. First, in between spikes, the membrane potentials of the neurons converge from any initial states to the values determined by the external inputs. Second, right after a neuron spiked, its membrane potential is reset to a fixed value. Finally, at each spike, the membrane potentials of all neurons jump towards the *effective reversal potentials* set by the inhibitory and excitatory conductances of the synapses from the neuron that spiked (see the next section).

The PSTM.—Between two consecutive spikes in the network, neurons do not interact. Let $t = 0^+$ be the time right after the transmission of the nth spike in the network, let p label the neuron that spiked, and let the membrane potential of neuron j at this time be $V_{j,p}^{(n)+}$. Integrating Eq. (1), we find that it will take neuron j a time $T_{j,p}^{(n)} = \tau \log \Gamma_{j,p}^{(n)}$ to reach the spike threshold. Here, $\Gamma_{j,p}^{(n)}$ is the pseudo spike time of neuron j relative to the nth spike of the network and is defined as

$$\Gamma_{j,p}^{(n)} \equiv 1 + (\Theta_j - V_{j,p}^{(n)+})/(I_j - C_j). \tag{2}$$

The neuron that will spike next has the smallest PST; therefore, its label q is found according to

$$q = \arg(\min_{j=1,\ldots,N} \Gamma_{j,p}^{(n)}). \tag{3}$$

With the neuron that spikes next identified, we can calculate the membrane potentials $V_{j,q}^{(n+1)}$ of all neurons *right before* the $(n+1)$th spike transmission. This is done by integrating Eq. (1) until time $T \equiv T_{q,p}^{(n)}$. Because of the reset, we have $V_{q,q}^{(n+1)} = R_q$.

We now calculate the effects of the $(n+1)$th spike to find out the membrane potentials *right after* the transmission of this spike. Integrating Eq. (1) over the spike at $t = T$, we find

$$V_{j,q}^{(n+1)+} - V_{j,q}^{(n+1)} = -\alpha_{j,q}(V_{j,q}^{(n+1)} - \beta_{j,q}E_I), \tag{4}$$

where $\alpha_{j,q} \equiv 1 - \exp(-G_{j,q}^E - G_{j,q}^I)$ and $\beta_{j,q} \equiv G_{j,q}^I/(G_{j,q}^I + G_{j,q}^E)$ are constants. We require that neuron j will not spike immediately because of the spike transmission; this leads to the condition $\beta_{j,q}E_I < \Theta_j$. With this condition, the net effect of inhibitory and excitatory inputs is an inhibitory input with an effective reversal potential $\beta_{j,q}E_I$.

With Eq. (4), we can relate $V_{j,q}^{(n+1)+}$ to $V_{j,q}^{(n+1)}$. We can further relate $V_{j,q}^{(n+1)}$ to $V_{j,p}^{(n)+}$ by integrating Eq. (1) from $t = 0^+$ to T. Therefore, a mapping from $\{V_{j,p}^{(n)+}\}$ to $\{V_{j,q}^{(n+1)+}\}$ can be found. Since the PST's are linearly related to the membrane potentials, we can also derive the mapping from $\{\Gamma_{j,p}^{(n)}\}$ to $\{\Gamma_{j,q}^{(n+1)}\}$, and the results are

$$\Gamma_{j,q}^{(n+1)} = \psi_{j,q} + \varepsilon_{j,q}\Gamma_{j,p}^{(n)}/\Gamma_{q,p}^{(n)}. \tag{5}$$

Here $\varepsilon_{j,q}$ and $\psi_{j,q}$ are constants and are given by $\varepsilon_{j,q} \equiv 1 - \alpha_{j,q}$, and $\psi_{j,q} \equiv \alpha_{j,q}[1 + (\Theta_j - \beta_{j,q}E_I)/(I_j - C_j)]$ for $j \neq q$ and $\psi_{q,q} \equiv \alpha_{q,q} + (\Theta_q - \varepsilon_{q,q}R_q - \alpha_{q,q}\beta_{q,q}E_I)/(I_q - C_q)$.

Equations (3) and (5) complete the pseudo-spike-time map: Once the PST's of all neurons after one spike are known, the neuron that will spike next can be determined with Eq. (3); then, with Eq. (5), the PST's of all neurons after the next spike can be calculated. Iterating the PSTM gives the spiking dynamics of the network.

Convergence theorems.—Driven by a set of external inputs, a network can generate different spike sequences because of different initial membrane potentials of the neurons. We consider the convergence properties of these sequences. Central to our analysis is the following lemma:

Lemma: Consider two PSTM's that generate the same spike sequence $(s_1, \ldots, s_P)$ of length P. Here s_n is the neuron label of the nth spike of the network. Denoting the PST's of one PSTM as $\Gamma_{j,s_n}^{(n)}$ and those of the other as $\Omega_{j,s_n}^{(n)}$, we find

$$\begin{aligned}\max_j(|\Gamma_{j,s_n}^{(n)} - \Omega_{j,s_n}^{(n)}|) &< \lambda^{n-1}2D \max_j(|\Gamma_{j,s_1}^{(1)} - \Omega_{j,s_1}^{(1)}|) \\ &< \lambda^{n-1}2D^2\end{aligned} \tag{6}$$

for $n = 2, \ldots, P$. Here, $D = 1 + \max_j\{[\Theta_j - \min(L_j, R_j, E_I)]/(I_j - C_j)\}$, and

$$\lambda = De^{-G_{\min}}/(\psi_{\min} + De^{-G_{\min}}) < 1, \tag{7}$$

where $G_{\min} = \min_{j,q}(G^I_{j,q} + G^E_{j,q})$, $\psi_{\min} = \min_{j,q}(\psi_{j,q})$. Quantity D is an upper limit of the PST and is obtained from Eq. (2) and the fact that $\min(L_j, R_j, E_I) \le V^{(n)+}_{j,q}$. The proof of the Lemma is in the Appendix.

With the Lemma, we consider a class of spike sequences generated by the PSTM's with finite *margins*. The margin Δ of a PSTM that generates a spike sequence $(s_1, s_2, \ldots)$ is defined as

$$\Delta = \min_{n=1,\ldots,\infty} \min_{j \neq s_{n+1}} (\Gamma^{(n)}_{j,s_n} - \Gamma^{(n)}_{s_{n+1},s_n}). \tag{8}$$

Here $\Gamma^{(n)}_{j,s_n}$ are the PST's. At a spike, the neuron that will spike next has the smallest PST; the margin measures how much smaller this PST is compared to those of the other neurons. We prove three theorems.

Theorem I: A spike sequence generated by a PSTM with nonzero margin Δ is stable: If perturbations of the membrane potentials of the neurons leave the PST's within a range $\Delta/4D$ from the unperturbed values, the spike sequence does not change; moreover, the timings of the spikes return to unperturbed values exponentially fast with the number of spikes after the perturbations.

Proof: Let $S = (s_1, s_2, \ldots)$ be the unperturbed spike sequence, and let Γ_{j,s_n} be the underlying PST's. Let $S' = (s'_1, s'_2, \ldots)$ be the spike sequence generated by the perturbed dynamics, and let $\Omega^{(n)}_{j,s'_n}$ be the underlying PST's. Without losing generality, we can assume that the perturbations are applied after the first spike of the network, so that $s'_1 = s_1$. With the Lemma, we prove the theorem recursively. Suppose that the perturbations are such that $|\Gamma^{(1)}_{j,s_1} - \Omega^{(1)}_{j,s_1}| < \Delta/4D < \Delta/2$ Together with Eq. (8), this relation implies $\Omega^{(1)}_{s_2,s_1} < \Omega^{(1)}_{j,s_1}$ for all $j \neq s_2$. Therefore, the next neuron to spike in the perturbed dynamics is neuron s_2, i.e., $s'_2 = s_2$. Now suppose $s'_k = s_k$ for all $k = 1, \ldots, P$. Applying the Lemma, we find $|\Gamma^{(P)}_{j,s_P} - \Omega^{(P)}_{j,s_P}| < \lambda^{P-1} 2D\Delta/4D < \Delta/2$. This relation and Eq. (8) again imply that $\Omega^{(P)}_{s_{P+1},s_P} < \Omega^{(P)}_{j,s_P}$ for all $j \neq s_{P+1}$. Therefore, $s'_{P+1} = s_{P+1}$. This proves that $S' = S$. The returning of the spike timings to unperturbed values follows directly from the Lemma.

Theorem II: Consider two stable spike sequences $S_1 = (\ldots, i_1, \ldots, i_P, i_{P+1}, \ldots)$ and $S_2 = (\ldots, j_1, \ldots, j_P, j_{P+1}, \ldots)$. Suppose that two subsequences of length P from S_1 and S_2 are identical, i.e., $i_n = j_n$ for $n = 1, \ldots, P$. Then S_1 and S_2 will be identical for all $n > P$ if $P \ge P^*$, where

$$P^* = \log(\Delta/8D^3)/\log(\lambda) + 1. \tag{9}$$

Proof: Denote the PST of neuron j at the nth spike in S_1 as $\Gamma^{(n)}_{j,i_n}$, and that in S_2 as $\Omega^{(n)}_{j,j_n}$. Using the Lemma, we find $|\Gamma^{(P)}_{j,P} - \Omega^{(P)}_{j,P}| < \lambda^{P^*-1} 2D^2 = \Delta/4D$. Then, applying Theorem I, we find that $i_k = j_k$ for all $k > P$.

Theorem III: All stable spike sequences will end up in periodic spiking patterns. Moreover, the numbers of transient spikes before settling down to the periodic patterns are finite and are *at most* $N^{P^*} + P^* - 1$.

Proof: Theorem II implies that a stable spike sequence will be trapped in a periodic spike pattern as soon as a subsequence of length P^* appears twice in the sequence. Any sequence of length $N^{P^*} + P^*$ contains at least two copies of a subsequence of length P^*.

Discussion—Almost all spike sequences in our networks are stable. Unstable sequences can be generated with PSTM's with the margins equal to 0. Such a situation can arise only from a set of restricted initial states of the neurons.

The upper limit of the number of transient spikes given in Theorem III is not a least upper bound. This is mainly because many neurons with small external inputs will never spike after the first spike of the network—they are suppressed by the spiking neurons. A similar process has been analyzed for the winner-take-all computation in a spiking network [10]. Therefore, the number of neurons that can participate in the spiking dynamics is typically much smaller than N. To illustrate this point, we performed simulations of random recurrent networks with global inhibition by numerically iterating the PSTM. A typical example is shown in Fig. 1. As can be seen in the figure, only a fraction of neurons spike when the network is driven with random external inputs. Moreover, the number of transient spikes is small. In general, the number of transient spikes is small if the global inhibition is strong. This follows from Eqs. (7) and (9).

Conclusion.—To conclude, we find that the spiking dynamics of the recurrent networks with global inhibition are attracted to stable periodic patterns with exact spike timings. The spike sequences of the network end up in periodic patterns in a finite number of transient spikes. When the inhibition is strong, the number of transient spikes is small. In future work, it will be important to elucidate the effects of finite time synaptic dynamics,

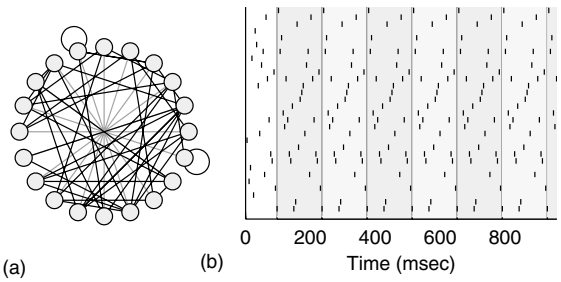

FIG. 1. (a) The network. In the simulation, $N = 1000$ [only 20 are shown (grey circles)]. The connections are randomly chosen: $G^I_{i,j}$ from the range (0.4, 0.6) (grey lines), and $G^E_{i,j}$ from (0, 0.05) [only those with $G^E_{i,j} > 0.005$ are shown (black lines)]. (b) Spike raster plot for a typical run. Parameters: $L_j = -70$ mV, $\Theta_j = -54$ mV, $R_j = -64$ mV, $\tau = 40$ msec, and $E_I = -75$ mV. $I_j - L_j$ are randomly sampled from 0 to 100 mV. Initial membrane potentials are randomly selected from $(-70, -54)$ mV. Only neurons that spiked at least once are shown (31 neurons). The periodic spike patterns are shaded with alternating grey areas.

spike transmission delays, interneurons, different neuron models, nonconstant external inputs, and noise.

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Appendix.—Here we outline the derivation of Eq. (6). For $n = 1, \ldots, P$, define $N \times 1$ vectors $X^{(n)}$ and $W^{(n)}$, whose jth elements are defined as $X_j^{(n)} \equiv (\Gamma_{j,s_n}^{(n)} - \Omega_{j,s_n}^{(n)})/\Omega_{j,s_n}^{(n)}$ and $W_j^{(n)} \equiv (\Omega_{j,s_n}^{(n)} - \Gamma_{j,s_n}^{(n)})/\Gamma_{j,s_n}^{(n)}$, respectively. Using Eq. (5), we obtain the following iteration relations for the X's and W's:

$$X^{(n)} = r_{n-1}\Lambda^{(n-1)}X^{(n-1)}, \qquad W^{(n)} = r_{n-1}^{-1}\Phi^{(n-1)}W^{(n-1)}. \tag{10}$$

Here $r_{n-1} \equiv \Omega_{s_n,s_{n-1}}^{(n-1)}/\Gamma_{s_n,s_{n-1}}^{(n-1)}$; $\Lambda^{(n-1)}$ and $\Omega^{(n-1)}$ are $N \times N$ matrices with their entries defined as $\Lambda_{i,j}^{(n-1)} = \Lambda_i^{(n-1)}(1 - \delta_{i,s_n})(\delta_{i,j} - \delta_{j,s_n})$ and $\Phi_{i,j}^{(n-1)} = \Phi_i^{(n-1)}(1 - \delta_{i,s_n})(\delta_{i,j} - \delta_{j,s_n})$, where $\Lambda_i^{(n-1)} \equiv 1 - \psi_{i,s_n}/\Omega_{i,s_n}^{(n)}$ and $\Phi_i^{(n-1)} \equiv 1 - \psi_{i,s_n}/\Gamma_{i,s_n}^{(n)}$. Here $\delta_{i,j} = 1$ if $i = j$ and 0 otherwise. Equation (5) gives $\Omega_{j,s_n}^{(n)} < \psi_{j,s_n} + \varepsilon_{j,s_n}\Omega_{j,s_{n-1}}^{(n-1)}$ since $\Omega_{s_n,s_{n-1}}^{(n-1)} > 1$. This leads to $\Lambda_i^{(n-1)} < \varepsilon_{j,s_n}\Omega_{j,s_{n-1}}^{(n-1)}/(\psi_{j,s_n} + \varepsilon_{j,s_n}\Omega_{j,s_{n-1}}^{(n-1)}) \le \lambda$, with λ defined in Eq. (7). Similarly, $\Omega_i^{(n-1)} < \lambda$. Iterating Eq. (10) for $m = n, \ldots, 2$, we obtain $X^{(n)} = \rho_{n-1}\mathcal{P}^{(n-1)}X^{(1)}$, where $\rho_{n-1} \equiv r_{n-1}\cdots r_2 r_1$ and $\mathcal{P}^{(n-1)} \equiv \Lambda^{(n-1)}\cdots\Lambda^{(2)}\Lambda^{(1)}$. Therefore, $|X^{(n)}|_\infty \le \rho_{n-1}||\mathcal{P}^{(n-1)}||_\infty|X^{(1)}|_\infty$. Here $|\cdot|_\infty$ is the vector norm and is defined as the maximum of the absolute values of the vector components, and $||\cdot||_\infty$ is the matrix norm and is obtained by summing the absolute values of the matrix entries in each row and picking the maximum of these sums. Similarly, we obtain $|W^{(n)}|_\infty \le \rho_{n-1}^{-1}||\mathcal{Q}^{(n-1)}||_\infty|W^{(1)}|_\infty$, where $\mathcal{Q}^{(n-1)} \equiv \Phi^{(n-1)}\cdots\Phi^{(2)}\Phi^{(1)}$. Multiplying the preceding two inequalities leads to

$$|X^{(n)}|_\infty|W^{(n)}|_\infty \le ||\mathcal{P}^{(n-1)}||_\infty||\mathcal{Q}^{(n-1)}||_\infty|X^{(1)}|_\infty|W^{(1)}|_\infty. \tag{11}$$

To evaluate the upper limit of $f \equiv ||\mathcal{P}^{(n-1)}||_\infty$, we observe that f can be regarded as a collinear function of $(n-1)N$ independent variables $\Lambda_i^{(m)}$, where $i = 1, \ldots, N$ and $m = 1, \ldots, n-1$ (in other words, f depends linearly on each $\Lambda_i^{(m)}$). These variables are bounded: $0 < \Lambda_i^{(m)} < \lambda$. Since the maximum of a collinear function is achieved at one of the vertices of the hypercube that confines the independent variables, the maximum of f is achieved at one of the $2^{(n-1)N}$ possible combinations of setting each $\Lambda_i^{(m)}$ at either 0 or λ. Therefore,

$$||\mathcal{P}^{(n-1)}||_\infty < \lambda^{n-1} \max_{k=1,\ldots,2^{(n-1)N}}(||\mathcal{L}^{(n-1,k)}||_\infty). \tag{12}$$

Here, $\mathcal{L}^{(n-1,k)} = \Xi^{(n-1,k)}\cdots\Xi^{(2,k)}\Xi^{(1,k)}$, and $\Xi^{(m,k)}$ is a $N \times N$ matrix with the entries defined as $\Xi_{i,j}^{(m,k)} = \sigma_i^{(m,k)}(1 - \delta_{i,s_{m+1}})(\delta_{i,j} - \delta_{j,s_{m+1}})$, where $\sigma_i^{(m,k)}$ is either 0 or 1. Note that matrix $\Xi^{(m,k)}$ has the same shape as the matrix $\Lambda^{(m)}$, except that its entries take values of 0, 1, -1. Also, all $\Xi^{(m,k)}$ are elements of a set M of matrices. An element in this set is a $N \times N$ matrix with the following characteristics: All entries are zero except that (a) entries in one column, say, the mth column, can be -1; (b) one entry is 1 in the row with its mth column equal to -1. Obviously, the norm of all matrices in set M is 2 except that of the matrix with all its elements equal to zero. It is easy to show that products of matrices in set M still belong to M. Therefore, $\mathcal{L}^{(n-1,k)}$ belongs to M, and $||\mathcal{L}^{(n-1,k)}||_\infty = 2$, except for the special case $\mathcal{L}^{(n-1,k)} = 0$, whose norm is 0. From this and Eq. (12), we obtain $||\mathcal{P}^{(n-1)}||_\infty < 2\lambda^{n-1}$. With similar reasoning, we get $||\mathcal{Q}^{(n-1)}||_\infty < 2\lambda^{n-1}$. From these two relations, Eq. (11), the relation $\max_j(|\Gamma_{j,s_n}^{(n)} - \Omega_{j,s_n}^{(n)}|^2/D^2) \le |X^{(n)}|_\infty|W^{(n)}|_\infty$, and the relation $|X^{(1)}|_\infty|W^{(1)}|_\infty < \max_j(|\Gamma_{j,s_1}^{(1)} - \Omega_{j,s_1}^{(1)}|^2)$, we obtain Eq. (6).

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