Precise SO-ripple Coupling Facilitates the Signal Transmission During Slow-wave Sleep

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Abstract

The interaction between hippocampal ripples and cortical slow oscillations (SO) has been proposed to play a critical role in memory consolidation during sleep. However, the neuronal mechanisms underlying the transmission of ripples within cortical regions remain poorly understood. In this study, we used a computational model to investigate how ripple events propagate through cortical networks. We found that sparse and weak inter-areal connections impede ripple propagation, while dense and strong inter-areal connections facilitate it. Notably, our findings reveal that when cortical networks exhibit slow oscillations (SOs), characterized by alternating up and down states, ripples occurring before the SO peak can propagate to distant cortical areas even in the presence of sparse and weak inter-areal connections. These results indicate that the precise coordination between SO and the ripple promotes efficient communication in cortical regions during sleep. This study offers new mechanistic insights into the role of SOs during slow-wave sleep, deepening our understanding of the processes underlying memory consolidation.

Keywords: Ripples; slow oscillations; coupling; sleep; inter-areal communication

Introduction

The hippocampal ripple is a transient high-frequency oscillation critical for memory recall and consolidation during non-rapid eye movement (NREM) sleep (Marshall, Cross, Binder, & Dang-Vu, 2020). The ripple frequency is $\sim$ 80-150 Hz in humans (Staresina et al., 2015; Dickey et al., 2021, 2022; Staresina, Niediek, Borger, Surges, & Mormann, 2023), whereas in rodents is $\sim$ 110- 200 Hz (Buzsáki, 2015). These events are thought to replay activity patterns in the hippocampus, reinforcing synaptic connections (Staresina et al., 2023) and transferring stored information to cortical regions for long-term memory consolidation (Klinzing, Niethard, & Born, 2019; Marshall et al., 2020; Girardeau & dos Santos, 2021). In rodents, during slow-wave sleep (SWS, often referred to deep sleep or N3 stage), firing sequences associated with recent experiences are replayed synchronously in both the hippocampus and the neocortex in a temporally ordered manner (Ji & Wilson, 2007). Such replay was also observed in humans (Schreiner & Staudigl, 2020).

It has been suggested that declarative memory consolidation occurs through interactions between the hippocampus and neocortex during sleep, facilitating the long-term memory storage within the neocortex (Klinzing et al., 2019; Marshall et al., 2020). Previous studies have suggested that ripples are not confined in the hippocampus but may be transferred to cortical regions (Sirota, Csicsvari, Buhl, & Buzsáki, 2003; Buzsáki & Watson, 2012). For instance, pronounced coupled ripple oscillations between the medial temporal lobe and neocortex were observed for successful memory retrival (Vaz, Inati, Brunel, & Zaghloul, 2019). It was also reported that the co-occurred ripples were observed in widespread cortical regions during sleep (Dickey et al., 2021; Verzhbinsky et al., 2023). We hypothesized that the widespread ripples may result from the transmission of ripples and ripple-triggered activity in distant cortical regions, which organize the replay of related memory. However, previous studies suggested the breakdown of effective cortical connectivity during sleep (Massimini et al., 2005; Titone et al., 2023), especially the long-distance connectivity decreases during SWS (Spoormaker et al., 2010). Therefore, how communications can be maintained with decreasing connectivity during sleep is a key question.

Specifically, during SWS, slow oscillations (SO) dominate cortical activity (Marshall et al., 2020), orchestrating the temporal structure for other oscillatory phenomena, including ripples (Staresina, 2024). It was reported that there exists hierarchical nesting of slow oscillations, spindles and ripples in the human hippocampus during sleep (Staresina et al., 2015). The temporal coupling of these sleep rhythms was demonstrated as a key in memory consolidation, spindles nest in SO up states and in turn group ripples in individual cycles during their waxing phase (Helfrich et al., 2019; Skelin et al., 2021; Ngo, Fell, & Staresina, 2020). Both experimental and computational works supported that ripple events occur mainly in the cortical up states (Dickey et al., 2022), which promote up state initiation and induce long-term changes in synaptic conductances (Wei, Krishnan, & Bazhenov, 2016).

In this study, we addressed the question by examining how ripple events are transmitted to downstream cortical areas through a computational network model of cortex during sleep. The cortex was modeled as different modules interacted in a feed-forward manner. Ripples were modeled as high-intensity spike packets. The cortical excitatory neurons were tuned to alternate in up and down states to mimic the slow-wave sleep. We found that precise SO-ripple coupling facilitates the ripple transmission with sparse and weak inter-areal connections. We highlight the role of coupling between slow oscillations and ripples on the inter-areal communication. This study provides new mechanistic insights into the

role of SO-ripple coupling potentially enhancing our understanding of memory consolidation.

Materials and Methods

Computational Modeling

Cortical networks. The cortex was considered as a highly modular structure, hence we modeled it as a feed-forward neuronal network (FNN) (Kumar, Rotter, & Aertsen, 2010) including 6 layers (Area), with each consists of 500 excitatory and 125 inhibitory neurons. The sub-threshold dynamics of each neuron is described by following equation:

$$C\dot{V} = -g_{leak}[V(t) - V_{reset}] + I_{syn}(t) + \xi, \tag{1}$$

where V is the membrane potential, $C = 200$ is the capacitor of membrane, $g_{leak} = 10$ is the membrane leak conductance, and I_{syn} is the total synaptic input current. ξ is the background noise based on the Ornstein-Uhlenback process, and its amplitude is 5. An action potential (also referred to as a spike) is emitted as the potential reaches the threshold $V_{th} = -55\ mV$, and the membrane potential is reset to $V_{reset} = -70\ mV$ for a refractory period $\tau_{ref} = 2\ ms$.

The synaptic current I_{syn} is modelled as follows:

$$I_{syn} = -g_E(V - E_{AMPA}) + g_I(V - E_{GABA}) \tag{2}$$

$$\tau_{xy}\dot{g} = -g + \sum_j g_{xy}\delta(t - t_k), \tag{3}$$

where g is the dynamics of synaptic conductance, τ_{xy} is the decay time constant for different synapses, t_k is the pre-synaptic spike time, g_{xy} is the conductance amplitude in which x, y is excitatory (E) or inhibitory (I). All excitatory conductance amplitudes including recurrent excitatory ($g_{EE} = 0.1$) and excitatory to inhibitory ($g_{IE} = 0.1$) conductance were fixed. The recurrent inhibitory ($g_{II} = 0.4$) conductance was fixed while inhibitory to excitatory (g_{EI}) conductance was variable. $E_{AMPA} = 0$, $E_{GABA} = -80\ mV$, $\tau_{EE} = \tau_{IE} = 5\ ms$, $\tau_{II} = \tau_{EI} = 10\ ms$. All recurrent delay within cortical networks is 2 ms. For inter-area connections, synaptic delay is 5 ms.

To keep that the recurrent E-I network performs in a balance state, inhibitory plasticity was included for inhibitory to excitatory synpases (Vogels, Sprekeler, Zenke, Clopath, & Gerstner, 2011). In order to quantify the changes to each g_{EI}^{ij}, a synaptic trace x_i is assigned to each neuron. x_i increases with each spike when a spike arrives and decays otherwise:

$$\begin{cases} \tau_{STDP}\frac{dx_i}{dt} = -x_i, & \text{if neuron fires,} \\ x_i \to x_i + 1, & \text{otherwise} \end{cases} \tag{4}$$

The synaptic conductance g_{EI}^{ij} from neuron j to i is updated for every pre- or post-synaptic events:

$$\begin{cases} g_{EI}^{ij} = g_{EI}^{ij} + \eta(x_i - \alpha_{STDP}), & \text{for presynaptic spikes,} \\ g_{EI}^{ij} = g_{EI}^{ij} + \eta x_j, & \text{for postsynaptic spikes} \end{cases}, \tag{5}$$

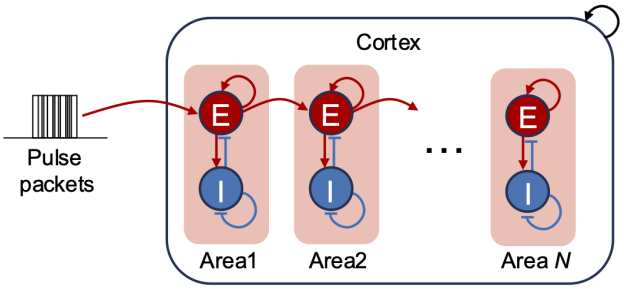

Figure 1: Network connectivity. Red denotes excitatory neurons (E), and blue denotes inhibitory neurons (I). Red arrow denotes excitatory synapses, and blue line with a bar denotes inhibitory synapses. The excitatory neurons in first cortical area receive pulse packets representing ripple activities from hippocampus. $N = 6$.

where η is the learning rate and $\alpha_{STDP} = 2\rho_0\tau_{STDP}$ is the depression factor based on a pre-defined excitatory firing rate ρ_0. $\eta = 0.1$, $\tau_{STDP} = 20$, $\rho_0 = 5$, $\alpha_{STDP} = 0.2$.

Network connectivity. Considering the modular structure of cortex, an $N-$layer FNN was built to investigate the neural activity transmission (Fig. 1). In a biologically plausible scenario, a FNN was embedded in a large recurrent network (Kumar, Rotter, & Aertsen, 2008; Chenkov, Sprekeler, & Kempter, 2017). The recurrent network incorporated 4000 excitatory and 1000 inhibitory neurons interacted with a sparse random background connectivity ($p = 0.02$). Each layer of FNN was formed by selecting 400 excitatory and 125 inhibitory neurons and connecting with tighter random connectivity ($p = 0.04$). The FNN was then formed by connecting these isolated E-I networks in a feed-forward manner, in which the feed-forward connection probability is denoted as p_{ff} and the feed-forward synaptic strength is denoted as g_{ff}.

External input. As spiking activity of neurons during ripples is highly organized (Wilson & McNaughton, 1994), we used pulse packets consisting of high-intensity short pulses to mimic the hippocampal ripple. The pulse packets were injected into the excitatory neurons in the first cortical area. In addition, external periodic stimulation ($\sim$1 Hz) were applied to the cortical excitatory neurons to mimic the slow oscillations during SWS.

Data Analysis

Population activity. We quantified the representation of responses to external input in each layer based on its population activity, which is defined as:

$$A(t) = \frac{1}{\Delta t}\frac{n(t; t + \Delta t)}{N}, \tag{6}$$

where Δt is a given short time interval centered at time t, whose value here is taken as 5 ms, $n(\Delta t)$ is the total amount of spikes in the neuronal population during the given time interval. The given time interval moves with time step of 1 ms.

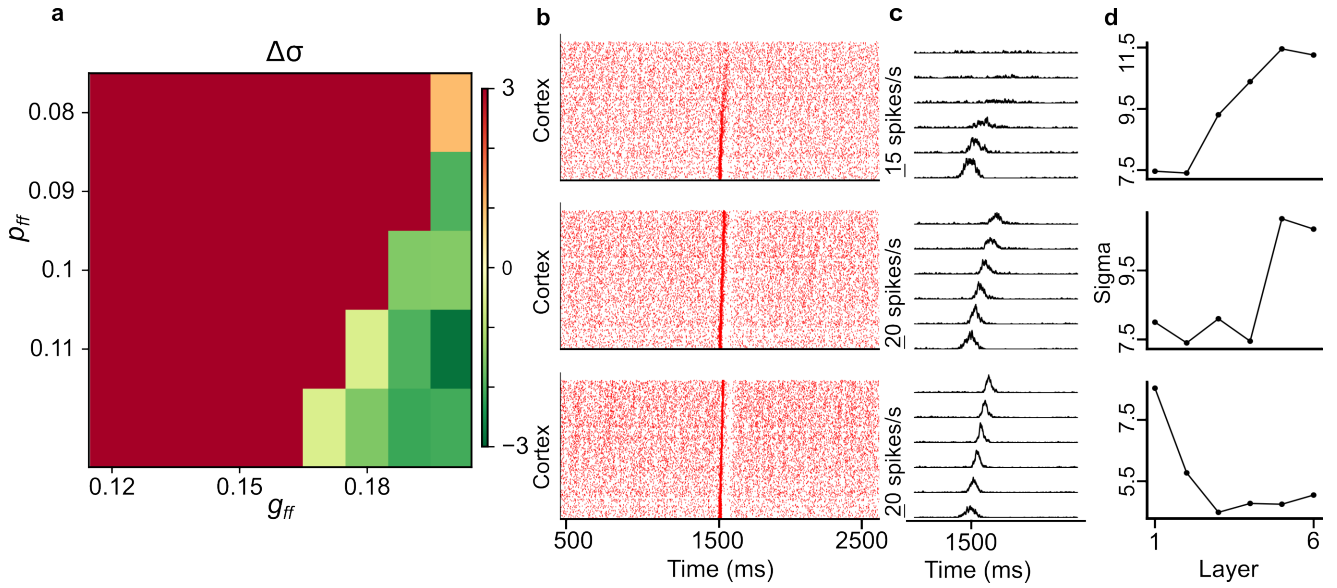

Figure 2: Ripple transmission in cortical networks with the absence of SOs. (a) The co-effect of p_{ff} and g_{ff} on the measurement of inter-areal communication – $\Delta\sigma$. Color encodes the $\Delta\sigma$, green denotes $\Delta\sigma < 0$, red denotes $\Delta\sigma > 0$. (b) Spiking raster plot of cortical excitatory neurons, top: $p_{ff} = 0.08, g_{ff} = 0.18$; middle: $p_{ff} = 0.1, g_{ff} = 0.18$; bottom: $p_{ff} = 0.1, g_{ff} = 0.2$. (c) The population firing rate for each area centered around the ripple spikes corresponding to (b) from bottom to top. (d) The temporal dispersion of ripple spikes in each area corresponding to (b) from bottom to top.

Inter-areal correlation lag. We calculated the cross-correlation between the population activities of first area and the downstream areas to determine the inter-areal lag as the time delay corresponding to the maximum correlation, computed by

$$\rho_{1,j}(\tau) = \frac{\left\langle [A_1(t) - \bar{A_1}][A_j(t+\tau) - \bar{A_j}] \right\rangle_t}{\sqrt{\left\langle [A_1(t) - \bar{A_1}]^2 \right\rangle_t \left\langle [A_j(t+\tau) - \bar{A_j}]^2 \right\rangle_t}}, \quad (7)$$

where $\bar{A}$ denotes the average of population activity, $\langle \cdot \rangle_t$ denotes the expectation over time. τ is the delay between signals. The inter-areal lag was used to determine the time window that the ripple activity arrives.

Measurement of transmission. In cortical networks, a population of pyramidal cells transmit a message encoded as a volley of spikes, which is also known as pulse packets (Diesmann, Gewaltig, & Aertsen, 1999). This spike volley propagates through diverging and converging axons and synapses to reach a target network, which generates its own spike volley in response. Communication is considered successful if the message's statistical properties (α: the spike number; σ: the temporal dispersion) are maintained or improved during transmission. However, a decrease in α or an increase in σ diminishes the signal quality, indicating communication failure (Hahn, Ponce-Alvarez, Deco, Aertsen, & Kumar, 2019). We mainly consider the difference of σ between input and output area ($\Delta\sigma$), in which $\Delta\sigma > 0$ denotes the failed communication and $\Delta\sigma < 0$ denotes the successful communication.

$$\Delta\sigma = \sigma_i - \sigma_1, \quad (8)$$

$$\sigma_i = std(\{t_i^j\}), (i = 1, .., N) \quad (9)$$

, where σ_i is the temporal dispersion of ripple spikes in i-th layer. $\{t_i^j\}$ denotes the set of spike times during ripple window in the i-th layer, where t_i^j represents the timing of the j-th spike. *std* denotes the standard deviation.

Statistical analysis. The obtained data distributions were statistically evaluated against a null hypothesis of zero using paired-samples t-tests. This method was employed to determine whether there was a significant difference in the means of the paired observations, reflecting deviations from the hypothesized baseline.

Computational Methods

Network simulations were performed in Euler method using the simulator tool BrainPy with a time step of 0.02 ms (Wang et al., 2023). To ensure the balance of excitation and inhibition in the cortical network, the large recurrent network was priorly balanced with plastic inhibitory-to-excitatory (noted as I2E) connections. After 5-second simulation, the I2E weights were saved locally. In the simulation of whole networks, the I2E weights were loaded to keep the cortical networks into a state of asynchronous irregular (AI) firing. The simulation time is 2500 ms, whereas the first 100 ms is ignored due to the initial instability.

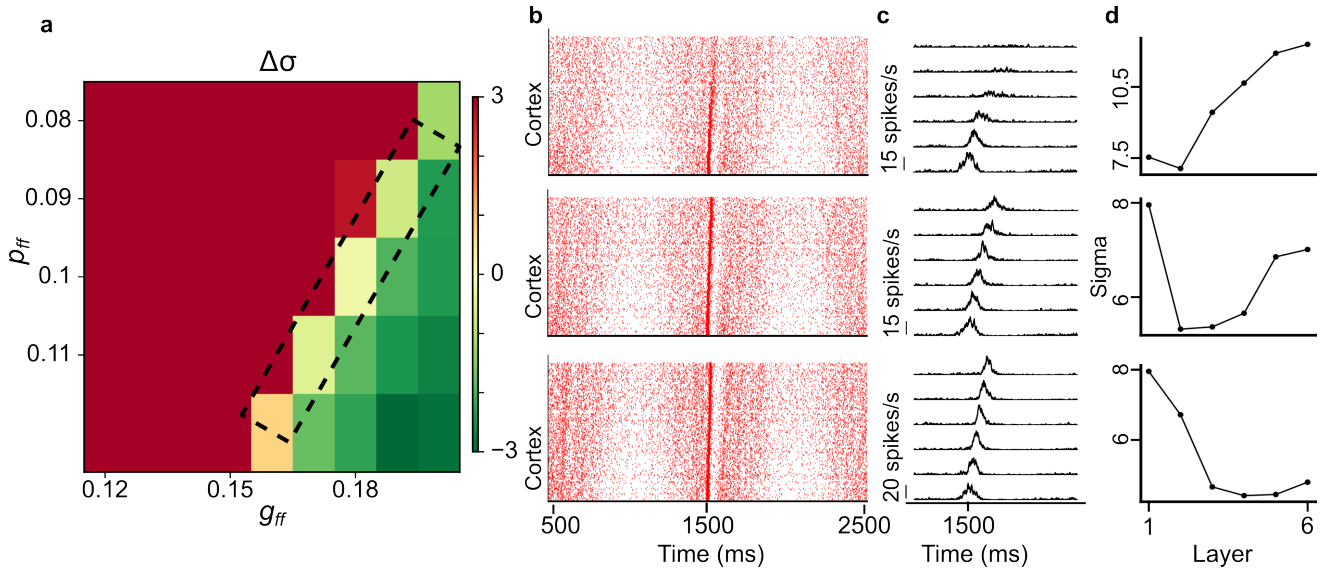

Figure 3: Ripple transmission in cortical networks with the presence of SOs. (a) The co-effect of p_{ff} and g_{ff} on the measurement of inter-areal communication – $\Delta\sigma$. Color encodes the $\Delta\sigma$, green denotes $\Delta\sigma < 0$, red denotes $\Delta\sigma > 0$. The area in the dashed rectangle denotes the new connectivity configurations supporting successful communications, with the presence of SOs. (b) Spiking raster plot of cortical excitatory neurons, top: $p_{ff} = 0.08, g_{ff} = 0.18$; middle: $p_{ff} = 0.1, g_{ff} = 0.18$; bottom: $p_{ff} = 0.1, g_{ff} = 0.2$. The ripple occurs 50 *ms* earlier than the SO peak. (c) The population firing rate for each area centered around the ripple spikes corresponding to (b) from bottom to top. (d) The temporal dispersion of ripple spikes in each area corresponding to (b) from bottom to top.

Results

The Effect of Inter-areal Connections on Ripple Transmission

Distant cortical areas were suggested to be coordinated by the long-range excitatory projections (Leong et al., 2016). Considering the decrease of connectivity during sleep especially long-range connectivity during N3, we then systematically investigated how the key properties of inter-areal connections including feed-forward connection probability (p_{ff}) and strength (g_{ff}) effect on the transmission of the ripple activity with the absence of SOs (Fig. 2). Through the 2D map of p_{ff} and g_{ff}, we found that the change of temporal dispersion ($\Delta\sigma$) are co-modulated by p_{ff} and g_{ff} (Fig. 2a). As the number and strength of feedforward synaptic connections increase, $\Delta\sigma$ is decreased to negative values and attracted into the green zone ($\Delta\sigma < 0$), corresponding to the successful communications. Three samples from the $p_{ff} - g_{ff}$ map were selected for illustration. To better compare these samples, we re-named them with three orders: *A* ($p_{ff} = 0.08, g_{ff} = 0.18$, Fig. 2b-d top), *B* ($p_{ff} = 0.1, g_{ff} = 0.18$, Fig. 2b-d middle), and *C* ($p_{ff} = 0.1, g_{ff} = 0.2$, Fig. 2b-d bottom). When comparing *A* and *B*, although both of them exhibit failed communication ($\Delta\sigma > 0$, first two rows in Fig. 2c, d), the denser inter-areal connections in *B* enabled ripples to propagate to the distant cortical areas. In contrast, the sparse inter-areal connections in *A* cause ripples to dissipate into the background noise within shallow layers. When comparing *B* and *C*, strong inter-areal connections in *C* enabled the successful communication ($\Delta\sigma < 0$, bottom in Fig. 2c, d).

In a word, the increasing number and strength of feedforward synaptic connections support the reliable transmission of ripple activity in the distant cortical areas.

SOs Facilitate Ripple Transmission Within a Precise Time Window

To investigate the role of SOs on the ripple transmission during SWS, we modulated the cortical networks to alternate between up and down states.

The change of temporal dispersion ($\Delta\sigma$) with the presence of SO in cortical networks demonstrated that SO facilitated the ripple transmission across cortical areas (Fig. 3a). We also selected the same three samples shown in Fig. 2b-d for comparison under the conditions with (Fig. 3b-d) and without SOs (Fig. 3b-d). For very weak (case *A*) and strong (case *C*), there is no significant difference between conditions with (Fig. 3b-d, top and bottom) and without SOs (Fig. 2b-d, top and bottom) in the ripple propagation. Specifically, the transmission still failed for case *A* ($\Delta\sigma > 0$) and succeeded for case *C* ($\Delta\sigma < 0$). However, for intermediate synaptic strengths (case *B*), the presence of SOs reversed the failed communication (Fig. 2b-d, middle) into successful communication (Fig. 3b-d, middle). Comparing the results in Fig. 2a and Fig. 3a, the presence of SOs expands the margin of successful region in the p_{ff}-g_{ff} phase plane towards the left and upward, corresponding to a decrease in the number

and strength of feed-forward synaptic connections (Fig. 3a, dashed rectangle). These results indicate that SOs enabled cortical networks with sparser and weaker inter-areal connections to propagate the ripple activity more effectively, thereby enhancing inter-areal communication.

The temporal structure between sleep events was believed to be critical for memory consolidation during sleep (Marshall et al., 2020). We further investigated the effect of different relative timings between ripples and SO peaks (Fig. 4a), with the connectivity parameters from the extended margin of successful communications (Fig. 3a, dashed rectangle). We found that ripples are more likely to occur before SO peaks during the successful communication (Fig. 4b, t-test against zero, two-sided: $p < 0.001$), whereas no significant preferred timing was observed during the failed communication (Fig. 4c). Moreover, the temporal precedence of ripples relative to SO peaks aligns with experimental observations (Dickey et al., 2022).

Our results highlight the role of SOs in ripple transmission, with the precise temporal coupling between SOs and ripples playing a crucial role in facilitating the efficiency of inter-areal communication.

Discussion

In this study, we used computational modeling to investigate the role of SO-ripple coupling in ripple transmission within cortical networks during slow-wave sleep. Our findings demonstrated that SOs facilitate ripple transmission with sparse and weak inter-areal connections. The precise timing of SO-ripple coupling plays a crucial role in modulating the inter-areal communication, with ripples occurring before the SO peak propagating faithfully to distant cortical areas.

Memory consolidation occurs during sleep, particularly during slow-wave sleep, and requires the coordination and information exchange of multiple brain regions. However, the reduction in connectivity during sleep (Massimini et al., 2005), especially the decreased connectivity during N2 and N3 stages (Spoormaker et al., 2010; Titone et al., 2023), presents a likely paradoxical situation. To investigate the underlying mechanisms, our work systematically investigated how inter-areal communication can be sustained despite limited inter-areal coupling strength and connectivity probability. Our results suggested that the presence of SOs in local cortical regions can facilitate the signal transmission between the weak-coupling regions. Niknazar et al suggested that SOs promote long-range effective communication in a breakdown network during sleep through a EEG study (Niknazar, Malerba, & Mednick, 2022), which is aligned with our findings. Our model offers a theoretical framework to elucidate the role of SOs in facilitating inter-areal communication.

Previous studies (Kumar et al., 2010) on inter-areal neuronal communication have suggested that neural information is transmitted through synchronization-based mechanisms. One leading hypothesis is the communication-through-coherence (CTC) framework (Fries, 2005), which posits that inter-areal communication is facilitated when neuronal groups exhibit synchronous oscillatory activity. Supporting this idea, Kumar et al. demonstrated that synchrony increases the probability of successful signal transmission across neuronal layers (Kumar et al., 2010). These findings highlight the critical role of synchronization and temporal dynamics in regulating inter-areal communication in the brain, which is consistent with our observations.

We further study the requirements of the inter-areal communication within limited connectivity. Our results demonstrated the precise temporal coupling of SOs and ripples facilitates inter-areal communication during SWS. In fact, previous studies have suggested the critical role of temporal coupling between rhythmic event in memory consolidation during NREM sleep (Marshall et al., 2020; Staresina, 2024). It was suggested that memory consolidation during SWS relies on the precise coupling mechanism: ripples nest into the excitable troughs of the spindle oscillation and spindles themselves are nested in the excitable up state of the neocortical SOs (Staresina et al., 2015). From the perspective of inter-areal communication, we propose that precise SO-ripple temporal coupling enhances inter-areal communication during SWS, thereby improving memory consolidation efficiency. Our findings offer new insights into the mechanisms underlying this process. In this study, we use ripple activity as the signal for transmission. We hypothesized that the widespread ripples and ripple-triggered activity in distant cortical regions may organize the replay of related memory stored in distributed regions. Future research will integrate sleep-memory experiments with computational neuronal models to validate these findings and assumptions, and further explore the role of SO-ripple coupling in memory consolidation.

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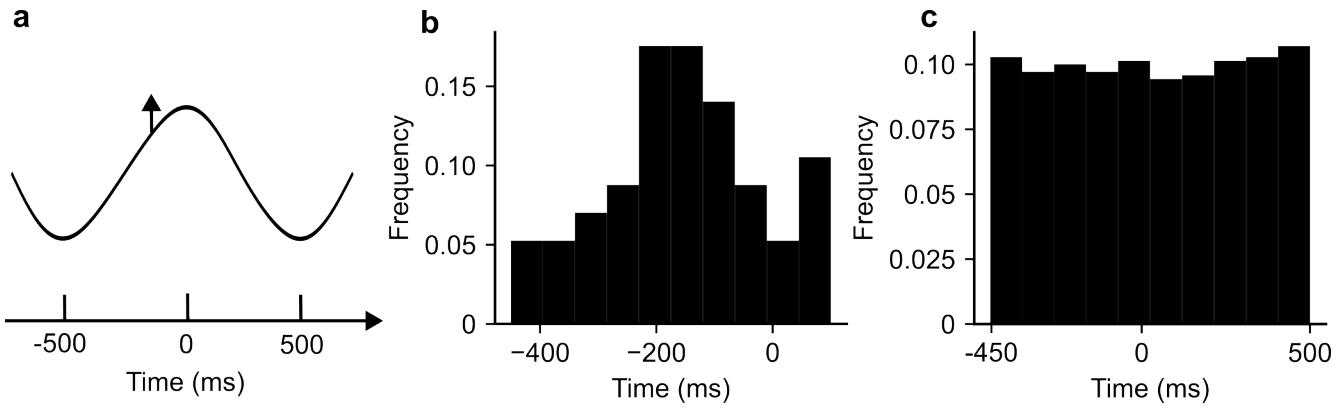

Figure 4: (a) An illustration of relative timing between ripple (arrow) and the SO peak. 0 ms indicates the peak of SO. (b-c) The probability of successful (b) and failed communication events (c) as a function of the timing of ripple occurrence relative to the SO peak. For successful communications (b), ripples preferentially occur before the SO peak (t-test against zero, two-sided: $p < 0.001$, mean ≈ -145 ms). In contrast, for failed communications (c), the ripple timing showed no significant preference relative to the SO peak.

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