



Research article

Magnetic flow drives hippocampal-cortical memory rhythm shifts through bifurcation dynamics

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Abstract: This study investigates a biologically constrained computational model of the hippocampal-cortical circuit incorporating magnetic-flow feedback to elucidate the nonlinear dynamical mechanisms through which magnetic stimulation modulates network states and drives transitions among memory-related neural rhythms. Bifurcation analysis characterizes the response of the hippocampal CA3 excitatory population to variations in the magnetic-flow feedback coefficients. One-parameter bifurcation analysis identifies critical bifurcations governing rhythm initiation, stability, and complexity, whereas two-parameter bifurcation diagrams show that cortical feedback expands the oscillatory regime more strongly than hippocampal feedback. Combined feedback produces the largest expansion and generates closed torus-bifurcation curves, indicating quasi-periodic dynamics. An automated sharp wave ripple (SWR) detection algorithm quantifies the incidence of model-derived SWR-related events across feedback conditions, thereby relating changes in the magnetic-flow feedback coefficients to changes in simulated SWR-related rhythmic activity. We further examine how SWR counts depend on two key CX1 microcircuit parameters: excitatory self-connection strength and local connectivity probability. Comparative analysis of SWR distributions illustrates how the network transitions from baseline quiescence or irregular firing to coherent, feedback-driven rhythmic discharge. SWR-count heatmaps further map the modulation of SWR-related activity across the parameter space. Collectively, these results establish a theoretical framework for understanding how magnetic-flow feedback modulates hippocampal-cortical dynamics and SWR-related activity, providing a basis for investigating targeted magnetic neuromodulation in memory-related neural processes.

Keywords: hippocampal-cortical memory networks; magnetic stimulation; sharp wave ripples; rhythmic transitions; bifurcation dynamics

1. Introduction

The hippocampal-cortical memory network constitutes a core theoretical framework for investigating the neural mechanisms underlying memory encoding and consolidation. Sharp wave ripples (SWRs) are distinct, high-frequency local field potential (LFP) oscillations originating in the hippocampus and are widely recognized as critical neural signatures of offline memory replay. SWR events emerge collectively from synaptic interactions and synchronized spiking activity within large ensembles of excitatory and inhibitory hippocampal neurons—not from membrane-potential changes in individual neurons. In the population-level formulation, each membrane-potential variable denotes the mean state of an excitatory or inhibitory neuronal population. Accordingly, the periodic oscillations of memory networks reflect SWR-associated population dynamics. These dynamics capture changes in population synchrony, rhythmic stability, and transitions among network states. To systematically elucidate the quantitative relationships between synaptic connectivity parameters and emergent network oscillatory dynamics, recent computational studies grounded in this framework have significantly advanced dynamical modeling [1–6]. Ghorbani et al. developed a cortical network model comprising coupled excitatory and inhibitory neurons and successfully recapitulated the bistable oscillatory transitions between up and down states, which have been observed experimentally in cortical circuits [7]. Foroutannia et al. subsequently employed bifurcation analysis and Lyapunov exponent calculations to demonstrate that minute modulations in synaptic connection strength can induce abrupt, qualitative shifts in the system's dynamical regime, thereby highlighting the pivotal role of synaptic weights in governing network state stability [8]. Bifurcation analysis—conducted through systematic variation of key parameters—is a well-established methodology for uncovering complex dynamical transitions and rigorously characterizing the behavior of nonlinear systems. It is extensively employed in the study of nonlinear dynamical systems [9, 10]. For example, in canonical models such as memristive neural networks and chaotic oscillators, this approach enables precise delineation of how system dynamics evolve across distinct regimes, including periodic oscillations, quasi-periodic motions, and chaotic attractors, as well as the bifurcations mediating transitions among them. To probe the cross-frequency coordination between slow wave oscillations and sharp wave ripples, Azimi et al. extended prior work by constructing a multi-regional coupled model [5]. Their simulations revealed that even subtle increases in recurrent excitatory connectivity within the CA3 subfield markedly enhance both the occurrence probability and duration of SWRs, indicating that CA3 recurrent excitation serves as a key gain-control parameter that regulates the fidelity and efficiency of memory replay and consolidation. Fan et al. introduced the septo-hippocampal glutamate–GABAergic (SHG) network model [11], which explicitly incorporates two functionally distinct hippocampal GABAergic interneuron subtypes—Teevra and Komal—to investigate their contributions to theta versus non-theta state transitions. This modeling study provided the first computational evidence for a dual-pathway mechanism, in which specialized GABAergic subtypes coordinate division of labor and functional synergy to enable dynamic switching between hippocampal memory encoding and consolidation states. Yang et al. established a biophysically constrained synaptic network model in which synaptic efficacy is governed by calcium-dependent CaMKII phosphorylation dynamics. Through systematic stimulation protocols, they characterized how CaMKII activation kinetics and AMPAR trafficking behavior vary across different stimulation frequencies and patterns and how these variations map onto distinct memory stages. Their findings

thus bridge molecular signaling cascades with network-level functional outcomes, identifying CaMKII–AMPA co-regulation as a decisive biochemical–electrophysiological switch that governs the transition from short-term memory retention to long-term synaptic consolidation [4, 12].

Based on this framework, a variety of external neural modulation interventions have been systematically applied to the study of neural network dynamics regulation, including optogenetic stimulation, electrical stimulation, magnetic stimulation, noise stimulation and multimodal stimulation coupling. Shi et al. investigated how noise influences the synchrony of neuronal network firing and revealed that noise plays a crucial role on shaping both the temporal structure and the population-level synchronization of neuronal discharge activity [13]. Since then, research in this domain has advanced along two complementary directions: clinical translation and mechanistic dissection.

At the clinical translation level, Fan et al. proposed a predictive control strategy that exploits known future information to anticipate seizure onset; they implemented this strategy in a closed-loop electrical stimulation system capable of delivering preictal intervention, thereby enabling early warning and prevention of epileptic seizures [14]. Pei et al. applied deep brain stimulation, a well-validated neuromodulation technique to patients with Parkinson's disease and observed significant improvements in motor function and quality of life. These findings confirm its therapeutic efficacy and translational viability [15]. Hou et al. introduced a three-phase delayed stimulation strategy designed to emulate the physiological conduction delay of synaptic transmission [16]. Through comprehensive computational modeling and simulation, this strategy was shown to suppress pathological synchronous oscillations while simultaneously promoting functional reorganization of the network—achieved by resetting neuronal phase relationships and adaptively reshaping synaptic weights. Compared with conventional monopolar or bipolar electrical stimulation, the three-phase approach employs alternating electrode activation, which substantially reduces risks of thermal injury and electrochemical tissue damage at equivalent therapeutic effect, thus lowering the incidence of postoperative complications [16]. Liu and Duan leveraged the millisecond-scale temporal precision and cell type specificity of optogenetic stimulation to achieve breakthrough control over respiratory rhythm generation [17]. Their work provides a transformative experimental platform for identifying the causal contributions of defined neuronal populations within the respiratory central pattern generator. Shen et al. further demonstrated in a computational model of the CA1 medial septum circuit that rhythmic light pulses delivered at a specific frequency can fully restore oscillatory amplitude deficits caused by potassium channel dysfunction, outperforming stochastic non-rhythmic optical stimulation in both consistency and magnitude of effect [18].

At the mechanistic level, Hua et al. systematically compared excitatory and inhibitory external stimuli and uncovered how each drives distinct membrane potential trajectories. They identified a four-phase mixed dynamical mode underlying clustered bursting, clarified the nonlinear mechanisms governing burst initiation and termination and explained counterintuitive phenomena—such as how inhibitory inputs can promote high-frequency oscillations. Critically, they developed the first unified theoretical model that quantitatively reproduces these effects and experimentally validated its predictions [19]. Wang et al. examined how sinusoidal sensory stimulation affects the seizure threshold in a cortical thalamic network model, parametrizing the input by its bias amplitude and conduction delay. Their analysis revealed that the spectral properties of the stimulus (e.g., dominant frequency, bandwidth) and its alignment with the network's intrinsic heterogeneity jointly determine

the efficiency of resonance with endogenous oscillations and the sensitivity of the global network response [20]. These findings emphasize that accurate characterization of thalamocortical conduction delays and phase coupling strengths is essential for designing effective neuromodulation protocols. Moreover, modern regulatory paradigms increasingly integrate multiple stimulation modalities in a coordinated fashion [21–23]. Recently, reinforcement learning frameworks have been deployed to dynamically adjust stimulation timing and intensity in real time [24], and multi-objective optimization algorithms have been used to concurrently tune stimulation parameters across distributed brain regions [25]. These data-driven approaches are now entering empirical validation stages and have markedly enhanced both the adaptability and energy efficiency of neural interventions. Therefore, optimizing stimulation strategies to the intrinsic dynamic signatures of specific neural circuits enables not only effective reversal of pathological states but also, from the perspective of computational neuroscience, furnishes a computable, reproducible, and experimentally testable framework for deciphering the organizational principles of brain function and the pathophysiological mechanisms underlying neurological disorders.

Among various physical stimulation modalities, magnetic and electromagnetic stimulation have emerged as prominent tools in translational neuroregulation due to their noninvasive or minimally invasive nature [26–28]. Electromagnetic induction stimulation has been shown to directionally modulate epileptic network dynamics: it demonstrates superior efficacy in controlling tonic seizures, yet yields comparatively weaker effects against atonic seizures [29]. Magnetic stimulation effectively suppresses pathological discharges in hippocampal neurons and has been experimentally confirmed to selectively inhibit abnormal oscillations within specific frequency bands—particularly in the motor cortex of Parkinson’s disease patients [30]. The functional specificity conferred by rhythmic magnetic stimulation on memory-related neural circuits is of particular importance. Hermiller et al. demonstrated that targeted synchronous stimulation delivered at the hippocampal-cortical theta frequency band (4–8 Hz) significantly improves the accuracy of episodic memory retrieval [31]. Liu et al. achieved cortical plasticity enhancement by precisely controlling both the spatial distribution and temporal onset of pulsed magnetic fields and focused ultrasound fields [32]. Their findings confirmed that this novel noninvasive magnetic-acoustic paired stimulation can induce long-term potentiation (LTP)-like plasticity in the human motor cortex. Phylactou et al. investigated the distributed neural mechanisms underlying working memory using a dual-coil transcranial magnetic stimulation (TMS) paradigm to deliver paired pulses between distinct cortical regions. Leveraging the principles of spike-timing-dependent plasticity, they provided causal evidence that the early visual cortex contributes to working memory maintenance through feedback projections from higher-order association areas. This work establishes a directly testable experimental paradigm for the distributed representation theory of working memory and underscores functional specialization across cortical nodes within the memory network [33]. Zhang et al. examined the effects of high-frequency repetitive TMS applied to individuals with subjective cognitive decline [34]. The study found that stimulation targeting the prefrontal–hippocampal network improved episodic memory performance by restoring long-term synaptic plasticity, thereby identifying the prefrontal cortex as a promising early intervention target for cognitive preservation. Whittaker et al. applied rhythmic TMS over fronto-parietal regions during auditory working memory training in healthy adults. Their results showed that combining rhythmic TMS with behavioral training enhances endogenous oscillatory activity, accelerates learning rates, and induces persistent neural plasticity changes [26].

A substantial body of empirical evidence demonstrates that, during non-rapid eye movement (NREM) sleep, hippocampal sharp wave ripples (SWRs), cortical slow oscillations (SOs), and thalamic sleep spindles exhibit robust, phase-organized temporal coordination. This cross-regional rhythmic coupling is widely regarded as a core physiological mechanism underlying hippocampal-cortical information transfer and sleep-dependent memory consolidation [35]. Staresina et al. identified a hierarchical temporal coupling among hippocampal SWRs, cortical SOs, and thalamic spindle waves using intracranial electroencephalographic recordings in human participants [36]. Geva-Sagiv et al. demonstrated, via real-time closed-loop deep brain stimulation during natural sleep, that phase-locked prefrontal stimulation—timed to the up-state of endogenous medial temporal lobe slow oscillations—enhances thalamic spindle power and strengthens cross-regional coupling between medial temporal ripple activity and cortical oscillations; this intervention also improved subsequent memory recognition accuracy [37]. Helfrich et al. revealed bidirectional, millisecond-precise temporal coordination among prefrontal SOs, thalamic spindles, and hippocampal ripples through high-density intracranial electrophysiological recordings in humans [38]. Finally, Fan et al. developed a computational model of the hippocampal-thalamic-cortical network grounded in neural population dynamics. Their framework not only elucidates how phase-specific stimulation modulates large-scale network synchronization but also reproduces empirically observed coupling phenomena and proposes optimized inhibitory and non-invasive magnetic-acoustic stimulation protocols [6]. However, current research has not yet clarified how magnetic stimulation alters hippocampal neuronal discharge patterns, modulates synaptic transmission, or affects the dynamical mechanisms underlying SWR generation in the hippocampal-cortical network. In particular, how variations in regulatory parameters influence neuronal synchronization, network oscillations, and SWR-related activity remains insufficiently understood. To address this gap, we develop a hippocampal-cortical coupled network model incorporating magnetic flow feedback to investigate the nonlinear regulatory mechanisms underlying network oscillations and SWR-related activity.

To systematically investigate the regulatory mechanisms of magnetic stimulation on the hippocampal-cortical memory network, this study integrates dynamical systems theory with numerical simulation techniques across three complementary analytical levels. At the theoretical level, we employ one-parameter bifurcation analysis to characterize the dynamic transition pathways of the membrane potential in hippocampal CA3 excitatory neurons as the magnetic flow feedback coefficient varies, identifying critical bifurcation points—including Hopf, period-doubling, and torus bifurcations—that govern rhythm generation, stability and complexity. Extending this framework, we construct two-parameter bifurcation diagrams in the P_{ee}^{CX1} and J_{ee}^{CX1} parameter plane under four distinct magnetic flow feedback configurations: (a) no stimulation; (b) hippocampal-only; (c) cortical-only; and (d) combined hippocampal-cortical stimulation. P_{ee}^{CX1} and J_{ee}^{CX1} were selected as bifurcation parameters due to their distinct yet complementary functional roles in shaping network dynamics. Specifically, P_{ee}^{CX1} governs the probability of synaptic connectivity between excitatory cortical neurons, thereby influencing both signal propagation pathways and the underlying network topology. In contrast, J_{ee}^{CX1} quantifies the synaptic strength of these connections and directly modulates the gain of inter-population interactions. Consequently, systematic variation of either parameter enables controlled modulation of network stability, synchronization propensity, and transitions among qualitatively distinct oscillatory regimes. This analysis reveals how the oscillatory

regime expands differentially across feedback configurations: cortical feedback induces significantly greater expansion than hippocampal feedback alone, while combined stimulation yields maximal expansion—and uniquely generates closed torus bifurcation curves, indicating the emergence of quasi-periodic dynamics. At the visualization level, we generate heatmaps of the root-mean-square (RMS) amplitude and dominant frequency of oscillatory waveforms, complementing the theoretical analysis. Systematic comparison between these heatmap features and the bifurcation structures establishes a quantitative correspondence: the spatial boundaries of high-amplitude and high-frequency regions align closely with bifurcation-bounded oscillatory regimes, confirming that theoretically predicted dynamical boundaries quantitatively constrain observed waveform characteristics. At the electrophysiological level, we apply an automated sharp wave ripple (SWR) detection algorithm to quantify SWR incidence across different magnetic flow feedback coefficients and parameter spaces, thereby linking bifurcation-predicted dynamical transitions to SWR-related rhythmic activity. Through this integrated tri-level framework—combining bifurcation theory, heatmap-based visualization, and SWR quantification—we elucidate the nonlinear dynamical mechanisms by which magnetic flow feedback modulates hippocampal-cortical rhythmic activity and establish a mechanistic, parameter-grounded framework for optimizing neuromodulation strategies in memory-related applications.

The structure of this article is as follows. Section 2 introduces the hippocampal-cortical memory neural network model incorporating magnetic stimulation. Section 3 presents a systematic dynamical analysis and quantitative characterization of rhythm-related neural features. One and two-parameter analyses characterize the dynamic transition pathways of hippocampal CA3 excitatory neurons and delineate oscillatory regimes in the P_{ee}^{CX1} and J_{ee}^{CX1} parameter plane under varying magnetic flow feedback coefficients. Section 4 summarizes the key findings and discusses their theoretical implications for SWR-related rhythmic regulation and their potential relevance to memory-related neural processes.

2. Model description

Based on [5], we incorporate magnetic flow and the associated induced currents into the hippocampal-cortical memory model. These currents arise from time-varying magnetic fields. Subsequent analyses are performed on this stimulated model. The full memory neural network model under magnetic stimulation comprises 16 ordinary differential equations. The specific formulation is as follows:

$$\begin{aligned}
 \frac{dV_e^k}{dt} &= -\frac{V_e^k}{\tau_e} - x_1 N_i^{k_n} P_{iq_n}^{k_{n_1}} J_{iq_n}^{k_{n_1}} r(V_{q_n}^{k_n}) + \sum_{m=1}^3 x_m N_e^{k_m} P_{eq_m}^{k_{m_1}} J_{eq_m}^{k_{m_1}} r(V_e^{k_m}) - \gamma_1^k V_e^k (\alpha + 3\beta\varphi_k^2), \\
 \frac{dV_i^k}{dt} &= -\frac{V_i^k}{\tau_i} - x_1 N_i^{k_n} P_{iq_n}^{k_{n_1}} J_{iq_n}^{k_{n_1}} r(V_{q_n}^{k_n}) + \sum_{m=1}^3 x_m N_e^{k_m} P_{eq_m}^{k_{m_1}} J_{eq_m}^{k_{m_1}} r(V_e^{k_m}), \\
 \frac{dc^k}{dt} &= -\frac{c^k}{\tau_c} + \sum_{n=1}^3 y_n N_e^{k_n} P_{ee}^{k_{n_1}} \Delta c^k r(V_e^{k_n}), \\
 \frac{d\varphi_k}{dt} &= V_e^k - \gamma_2^k \varphi_k.
 \end{aligned} \tag{2.1}$$

Here, V_e^k and V_i^k denote the population-averaged membrane potentials of the excitatory and inhibitory neuronal populations, respectively, in brain region k , where $k \in \{CA3, CA1, CX1, CX2\}$. These regions correspond to the hippocampal subregions CA3 and CA1 and the cortical subregions CX1 and CX2. The variable c^k represents the population-level synaptic adaptation variable in region k , capturing the adaptive modulation of synaptic coupling strength and population membrane-potential dynamics in response to external inputs and internal feedback. The parameters τ_e , τ_i , and τ_c denote the time constants governing the dynamics of the excitatory and inhibitory membrane potentials and the synaptic adaptation variable, respectively. The binary variables x_1 , x_m , and y_n , with $m, n \in \{1, 2, 3\}$, indicate the presence or absence of specific population-level coupling pathways: a value of 1 signifies an active functional connection and signal-transmission pathway between the corresponding neuronal populations, whereas a value of 0 indicates its absence. N denotes the size of the respective neuronal population, P the average probability of synaptic connectivity between populations, and J the population-averaged synaptic coupling strength. The function $r(V)$ denotes the mean firing rate of a neuronal population as a monotonic, sigmoidal function of its population-averaged membrane potential V . Finally, Δc^k represents the population-averaged increment in the adaptation variable induced by incoming neuronal activity.

Since only excitatory-to-excitatory synapses are affected by dendritic spike frequency adaptation (DSFA), the synaptic connection strength between excitatory neurons in the cortex, denoted as J_{ee} , is modeled as an S-shaped function of the adaptation variable c^k , which characterizes the adaptation level of excitatory neuronal dendrites to long-term input signals. The specific form is given by:

$$J_{ee}^k(c) = \frac{J_{ee}^k(0)}{1 + e^{\frac{c-c^*}{\delta c}}}. \quad (2.2)$$

In equation (2.1), the subscripts e and i denote excitatory and inhibitory neurons, respectively. The index iq_n represents signal transmission from inhibitory neurons to neuron q_n , while eq_m represents signal transmission from excitatory neurons to neuron q_m , where $q_n, q_m \in \{e, i\}$. The indices $k_n, k_{n_1}, k_m, k_{m_1}$ denote connections between different brain regions in the system, where $k_n, k_{n_1} \in \{CA3, CA1, CX1, CX2\}$, $k_m, k_{m_1} \in \{CA3, CA1, CX1, CX2, CA3-CA1, CA1-CX1, CA1-CX2, CX1-CA1, CX2-CA3, CX2-CA1\}$. The parameter values used in the system are provided in Appendix A.1 and are drawn from four complementary sources. First, membrane and adaptation time constants, population sizes, local connection probabilities, and synaptic strengths are adopted from experimentally constrained hippocampal-cortical population models. These models, in turn, are grounded in anatomical and electrophysiological data. Second, parameters of the sigmoidal firing-rate function and synaptic adaptation formulation follow electrophysiologically informed values used in these established models [5, 7, 39]. Third, interregional connection strengths and probabilities reflect empirically reported hippocampal-cortical projection specificity [5, 35]. Fourth, magnetic-flux parameters and the feedback formulation are grounded in established memristive neuronal models and their network-level extensions [6, 40, 41]. The feedback coefficients γ_1^k are subsequently varied as dimensionless control parameters across ranges selected to characterize transitions among the model's dynamical regimes; they do not correspond to direct measurements of physical stimulation intensity.

This study employs a modeling simplification wherein magnetic stimulation is assumed to act directly on the excitatory neuronal population, while inhibitory populations are modulated indirectly via excitatory-inhibitory synaptic interactions. For each brain region k , φ_k denotes the effective

magnetic-flux state variable associated with the neuronal membrane. The differential equation $\frac{d\varphi_k}{dt} = V_e^k - \gamma_2^k \varphi_k$ governs the temporal evolution of φ_k , wherein V_e^k drives flux accumulation and $\gamma_2^k \varphi_k$ mediates flux dissipation. The induced feedback current is given by $-\gamma_1^k V_e^k \rho(\varphi_k)$, where $\rho(\varphi_k) = \alpha + 3\beta\varphi_k^2$ represents the memductance of a flux-controlled memristive element. Here, γ_1^k scales the gain of the feedback loop, α sets the baseline (linear) coupling strength, and β governs the quadratic nonlinearity in the flux dependence. Variations in φ_k modulate the memductance and thereby shape the magnitude and sign of the induced current impinging on the excitatory population. This feedback mechanism formalizes the bidirectional coupling between magnetic-flux dynamics and population-level membrane-potential dynamics, enabling the model to reproduce nonlinear, history-dependent neuronal responses to magnetic stimulation. Crucially, γ_1^k is an internal, dimensionless model parameter—not a physical proxy for magnetic-field intensity. All parameter values are tabulated in Appendix A.1.

3. The regulatory mechanism of magnetic stimulation on the hippocampal-cortical memory Dsystem

To explore how magnetic flow feedback regulates hippocampal-cortical rhythmic activity in system (2.1), we analyze the dynamical effects of this model parameter through bifurcation analysis and SWR-related activity quantification. It directly controls both the steady state level of network excitability and the stability of oscillatory patterns. Therefore, this section focuses on how this coefficient modulates the system. Using dynamical systems analysis, we examined how variations in the magnetic stimulation feedback coefficient within the hippocampal-cortical region affect neural network activity and rhythms associated with memory. These analyses reveal the dynamical mechanisms that maintain network stability and regulate SWR-related rhythmic activity.

3.1. One-parameter dynamic regulatory mechanism of magnetic stimulation on the hippocampal-cortical network

We first fix the self-connection strength of cortical excitatory neurons at $J_{ee}^{CX1} = 0.55$ and analyze the system's dynamical behavior as a function of the magnetic stimulation feedback coefficients ($\gamma_1^{CA3}, \gamma_1^{CA1}, \gamma_1^{CX1}, \gamma_1^{CX2}$) and the self-connection probability P_{ee}^{CX1} of cortical excitatory neurons. The corresponding one-parameter bifurcation diagrams are shown in Figure 1. In Figure 1, the horizontal panels (left to right) depict how system dynamics evolve as the cortical magnetic stimulation feedback coefficients ($\gamma_1^{CX1}, \gamma_1^{CX2}$) increase, while the hippocampal coefficients ($\gamma_1^{CA3}, \gamma_1^{CA1}$) remain fixed. The vertical panels (top to bottom) depict how system dynamics evolve as the hippocampal coefficients ($\gamma_1^{CA3}, \gamma_1^{CA1}$) increase, while the cortical coefficients ($\gamma_1^{CX1}, \gamma_1^{CX2}$) remain fixed. In each bifurcation diagram: Unstable equilibrium points are indicated by black dashed lines; stable equilibrium points by red solid lines; unstable limit cycles (USLC) by thick blue dashed lines; and stable limit cycles (SLC) by thick green dashed lines. All remaining model parameters are listed in Appendix A.1.

3.1.1. One-parameter bifurcation analysis with respect to P_{ee}^{CX1}

As shown in Figure 1, the dynamical behavior of system (2.1) exhibits marked diversity and stage-dependent evolution. Figures 1(1) to 1(10) share similar topological structures, whereas Figures 1(11) to 1(16) represent a distinct topological class. We first analyze the dynamical behavior of these two configuration types in detail.

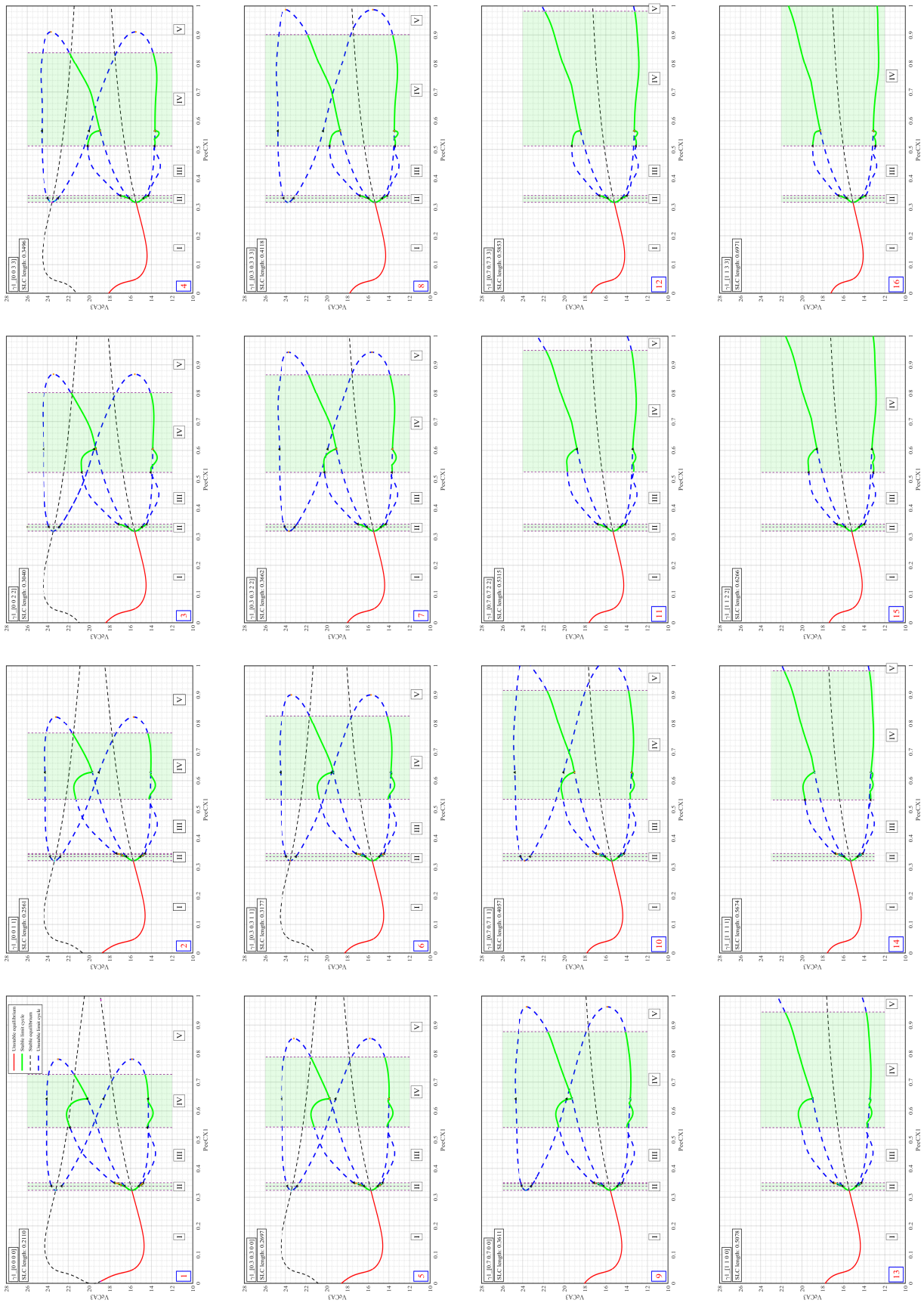


Figure 1. One-parameter bifurcation diagram of system (2.1) with respect to the parameter P_{cc}^{CX1} under the varying feedback coefficients of magnetic stimulation in the hippocampus and cortex.

In Figure 1(1) to Figure 1(16), the system undergoes a sequence of qualitative transitions across five parameter regions: In region I, the system exhibits coexisting stable and unstable equilibria. In region II, it exhibits coexisting stable and unstable limit cycles. In region III, there exist multiple unstable limit cycles. In region IV, stable and unstable limit cycles reappear, and the stable cycle exhibits markedly increased amplitude relative to region II. In region V, the system loses stability entirely.

Corresponding bifurcations occur at critical boundaries and within regions: A Hopf bifurcation arises at the I and II boundary; period-doubling, torus, and saddle-node bifurcations of limit cycles occur within region II; a period-doubling bifurcation occurs at the III and IV boundary; a saddle-node bifurcation of limit cycles occurs within region IV; and a torus bifurcation occurs at the IV and V boundary. Notably, in Figure 1(7) to Figure 1(10), the high-amplitude unstable equilibrium points vanish.

Figure 1 illustrates that increasing the cortical magnetic stimulation feedback coefficients γ_1^{CX1} and γ_1^{CX2} induces significant topological reorganization of the system. Comparative analysis across Figure 1(1) to Figure 1(10) and Figure 1(11) to Figure 1(16) reveals that higher-amplitude unstable limit cycles, which were previously present in regions II–V, vanish; stability in region IV is markedly enhanced; and the parameter range occupied by the green stable region expands substantially. Consequently, overall system stability is strengthened.

3.1.2. Analysis of the dynamical evolution of the system under magnetic stimulation

We further analyzed the regulatory effect of magnetic stimulation on the dynamics of the hippocampal-cortical coupling system (2.1). By examining one-parameter bifurcation diagrams, we characterized how variations in the magnetic flow feedback coefficients of the cortical or hippocampal regions modulate system-level oscillation patterns and stability.

First, we fix the magnetic stimulation feedback coefficients γ_1^{CA3} and γ_1^{CA1} of the two neuronal populations in the hippocampal region and examine how system dynamics evolve as the magnetic stimulation feedback coefficients γ_1^{CX1} and γ_1^{CX2} of the two cortical neuronal populations increase. The results show that, as the cortical magnetic flow feedback coefficients (γ_1^{CX1} and γ_1^{CX2}) increase, the parameter range of the self-connection probability P_{ee}^{CX1} supporting stable limit cycles progressively expands. At small cortical magnetic flow feedback coefficients, stable periodic oscillations occur only within a narrow interval of P_{ee}^{CX1} —specifically, $P_{ee}^{CX1} \in (0.3, 0.4)$ —and are accompanied by large amplitude fluctuations and heightened susceptibility to instability. As the cortical magnetic flow feedback coefficients increase, the P_{ee}^{CX1} range supporting stable limit cycles broadens symmetrically at both ends; moreover, even for relatively small P_{ee}^{CX1} values, oscillation amplitude and frequency become increasingly regular, with substantially reduced fluctuations and markedly improved stability relative to baseline conditions. These findings demonstrate that cortical magnetic stimulation enhances the robustness of network-level rhythmic output and attenuates the system's sensitivity to variations in intrinsic synaptic connectivity parameters. This dynamical behavior indicates that cortical magnetic flow feedback expands the basin of attraction for stable periodic oscillations in parameter space, facilitating a transition from locally unstable dynamics to more stable periodic activity and thereby improving the stability and regularity of network rhythmic output. These results provide a mechanistic basis for understanding how external electromagnetic stimulation modulates local cortical network oscillations.

Next, we fix the cortical magnetic stimulation feedback coefficients γ_1^{CX1} and γ_1^{CX2} and gradually

increase the hippocampal magnetic stimulation feedback coefficients γ_1^{CA3} and γ_1^{CA1} to analyze the dynamical response of the membrane potential of hippocampal CA3 excitatory neurons as a function of P_{ee}^{CX1} . The results show that hippocampal stimulation exerts a significant modulatory influence on the system's dynamical structure. In the absence of cortical stimulation, the parameter range supporting stable limit cycles is relatively narrow. As the hippocampal magnetic flow feedback coefficients increase, longitudinal comparison across panels reveals that region IV transitions from a complex dynamical regime—characterized by coexistence of stable and unstable limit cycles—to a simplified state dominated by a single stable limit cycle.

The topological properties of this stable limit cycle undergo marked changes: its existence interval along the P_{ee}^{CX1} axis broadens substantially; the corresponding membrane potential amplitude shifts upward overall; and the stability region extends toward lower values of P_{ee}^{CX1} .

These findings indicate that increasing the hippocampal magnetic flow feedback coefficients within a certain range promotes a transition to a more stable rhythmic state. However, when the hippocampal magnetic flow feedback coefficients exceed a critical model threshold, the system begins to exhibit rhythmic instability. Thus, the model exhibits a finite effective regulatory range for hippocampal magnetic flow feedback: feedback coefficients beyond this range reduce oscillatory stability and may even disrupt synchronous network activity.

Collectively, the magnetic stimulation feedback coefficients in both the hippocampal and cortical regions critically regulate system stability and oscillatory characteristics. Cortical stimulation expands the parameter domain of stable oscillations and enhances its robustness, whereas hippocampal stimulation reshapes the bifurcation thresholds and stability boundaries of the oscillatory regime. Their synergistic interaction jointly regulates the dynamic balance and rhythmic stability of the hippocampal-cortical system. This analysis provides a rigorous theoretical foundation for understanding how externally applied electromagnetic stimulation modulates local network oscillations—and highlights the therapeutic potential of spatially targeted, multi-region stimulation strategies in clinical neuromodulation.

3.1.3. The regulatory effect of magnetic stimulation on sharp wave ripple patterns

Based on the preceding dynamical analysis, we apply a sharp wave ripple (SWR) detection algorithm (Details can be found in Appendix A.2) to quantify the number of sharp wave ripples generated within a fixed time window. This quantification is performed under varying magnetic flow feedback coefficients and across the full range of cortical excitatory neuronal self-connection probability $P_{ee}^{CX1} \in [0, 1]$, while holding J_{ee}^{CX1} constant at 0.55. Figure 2 shows the resulting comparison, which illustrates how SWR incidence varies with both P_{ee}^{CX1} and the magnetic flow feedback coefficients.

Figure 2 presents the temporal evolution characteristics of SWR-related events in the hippocampal and cortical regions under different magnetic flow feedback coefficients. The results illustrate how variations in magnetic flow feedback modify the emergence, temporal distribution, and synchronization patterns of SWR-related events, reflecting the dynamic response of the hippocampal-cortical network to feedback regulation.

However, combined stimulation targeting both the hippocampus and cortex does not consistently yield additive effects. In certain stimulation configurations, the number of SWRs fails to exceed that observed under single-region stimulation. In some cases, it declines markedly. This implies that

cross-regional stimulation can elicit nonlinear interactions that suppress synergistic neural activation. Furthermore, the system's response exhibits dependence on functional connectivity strength: maximal modulation efficacy occurs within a narrow optimal range of connection strength, whereas both weaker and stronger connections attenuate regulatory effects. This attenuation may reflect intrinsic neuronal properties such as adaptive homeostasis or refractory period constraints. Collectively, these findings confirm that magnetic stimulation modulates neural rhythms, yet they also reveal that this modulation is inherently nonlinear and highly sensitive to stimulation and network parameters. Future studies should systematically investigate how self-connection strength interacts with stimulation parameters to identify the optimal configuration for targeted neuromodulation.

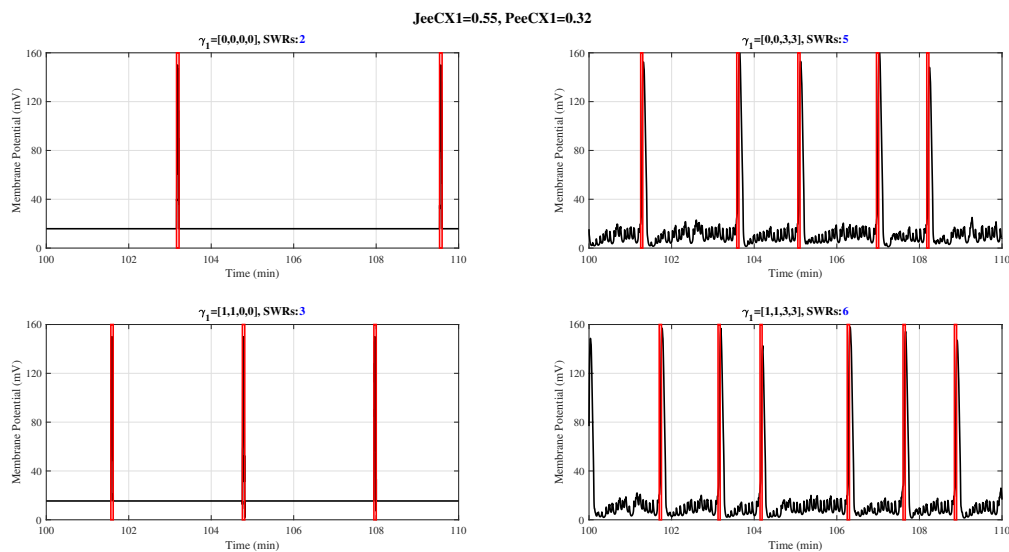


Figure 2. This figure presents the stimulation effects of system (2.1) under varying magnetic stimulation feedback coefficients. These coefficients— γ_1^{CA3} , γ_1^{CA1} , γ_1^{CX1} and γ_1^{CX2} —are applied to excitatory neurons in the hippocampal subregions CA3 and CA1 and in the cortical region, respectively. The simulations are conducted with fixed synaptic coupling strength $J_{ee}^{CX1} = 0.55$ and external input gain $P_{ee}^{CX1} = 0.32$. In each subplot, the black annotation at the top specifies the value of the feedback coefficient assigned to the corresponding brain region; the blue annotation indicates the total number of SWRs observed during the simulation period.

3.1.4. Analysis of magnetic stimulation–induced modulation of hippocampal sharp wave ripple incidence

To further quantify the dependence of SWR-related activity on magnetic flow feedback coefficients, we performed a systematic parameter-space analysis using SWR count heatmaps. We investigate how the incidence of sharp wave ripples in system (2.1) varies with the self-connection probability $P_{ee}^{CX1} \in [0, 1]$ and self-connection strength $J_{ee}^{CX1} \in [0, 2]$ of cortical excitatory neurons under magnetic stimulation. Figure 3 presents heatmaps quantifying SWR incidence across combinations of magnetic stimulation feedback coefficients γ_1^{CA3} , γ_1^{CA1} , γ_1^{CX1} and γ_1^{CX2} .

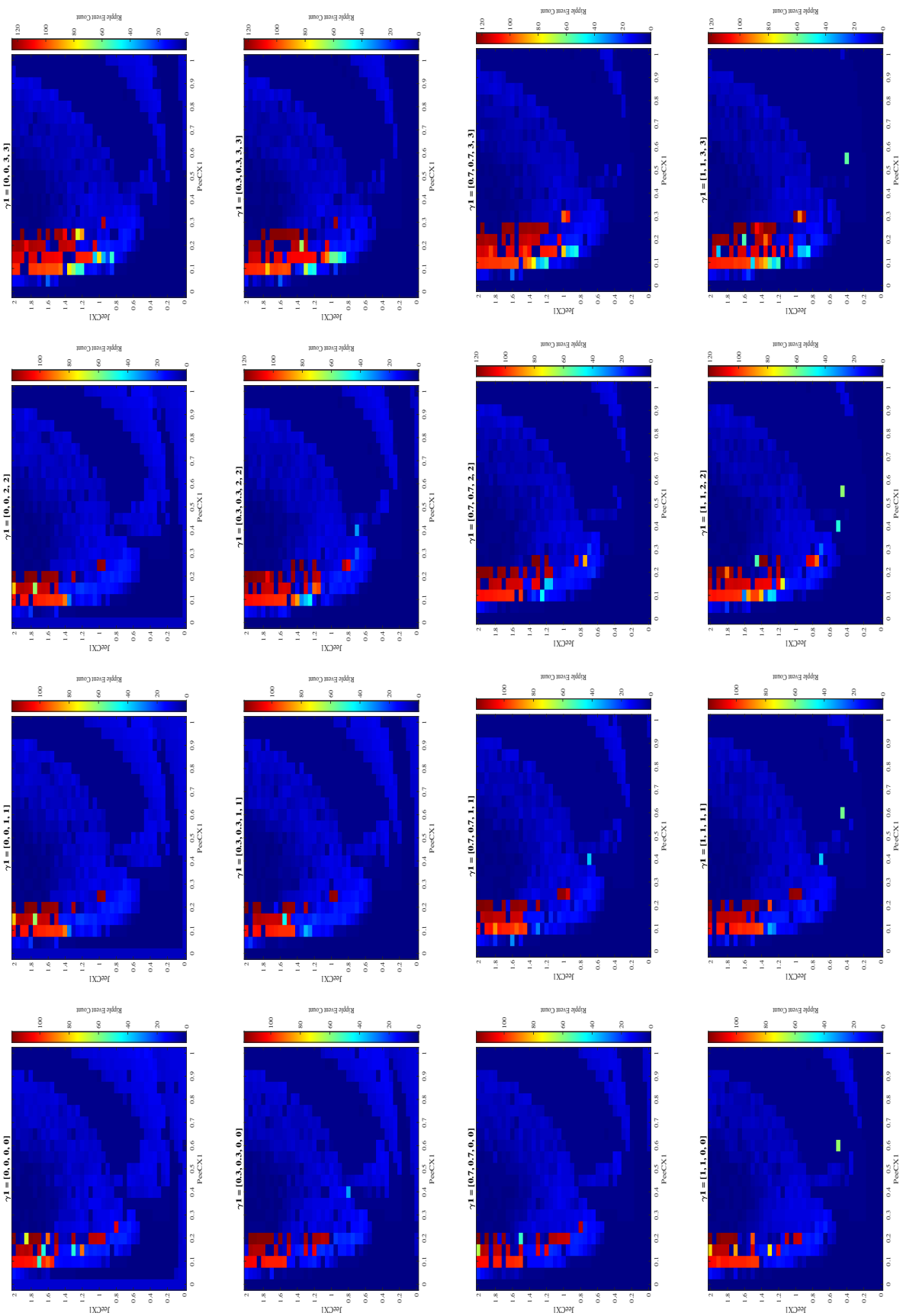


Figure 3. Heatmaps illustrating how the number of SWRs in hippocampal-cortical regions varies with magnetic flow-mediated feedback coefficients γ_1^{CA3} , γ_1^{CA1} , γ_1^{CX1} , γ_1^{CX2} —as defined in system (2.1)—under parametric variation of $P^{CX1} \in [0, 1]$ and $J^{CX1} \in [0, 2]$.

First, we fix the magnetic stimulation feedback coefficients γ_1^{CA3} and γ_1^{CA1} for the two hippocampal neuronal populations and systematically examine how SWR incidence varies across the full parameter space of cortical excitatory neuron self-connection probability $P_{ee}^{CX1} \in [0, 1]$ and self-connection strength $J_{ee}^{CX1} \in [0, 2]$, while incrementally increasing the cortical feedback coefficients γ_1^{CX1} and γ_1^{CX2} from 0 to 3. Horizontal comparison across subplots in Figure 3 reveals that this parametric increase induces substantial reorganization of both the spatial distribution and statistical properties of SWR events. Specifically, when γ_1^{CX1} and γ_1^{CX2} rise from 0 to 1, the maximum, mean and standard deviation of SWR incidence all increase markedly. This indicates that small cortical magnetic flow feedback coefficients are associated with enhanced neuronal excitability and increased SWR-related activity in the model. When γ_1^{CX1} and γ_1^{CX2} further increase from 1 to 3, SWR incidence rises across the cortical domain, and the amplitude of temporal fluctuations gradually increases. This suggests that intermediate-to-large cortical magnetic flow feedback coefficients are associated with broader SWR-related activity and increased spatiotemporal heterogeneity in the model. Collectively, these findings demonstrate a positive relationship between the cortical magnetic flow feedback coefficients and SWR-related activity within the investigated parameter range, with larger feedback coefficients associated with increased event frequency and variability. These results further indicate that cortical magnetic flow feedback significantly modulates SWR-related network dynamics.

Next, we fix the magnetic stimulation feedback coefficients γ_1^{CA3} and γ_1^{CA1} for the two hippocampal neuronal populations and systematically examine how SWR incidence varies across the full parameter space defined by cortical excitatory neuron self-connection probability $P_{ee}^{CX1} \in [0, 1]$ and self-connection strength $J_{ee}^{CX1} \in [0, 2]$, while independently increasing γ_1^{CX1} and γ_1^{CX2} . Figure 3 shows that applying hippocampal magnetic stimulation—by gradually increasing γ_1^{CA3} and γ_1^{CA1} from 0 to 1—induces pronounced reorganization of both the spatial distribution and statistical properties of SWR events. As γ_1^{CA3} and γ_1^{CA1} increase, SWR incidence first rises steeply and then plateaus. Specifically, when γ_1^{CA3} and γ_1^{CA1} increase from 0 to 0.3, the maximum, mean and standard deviation of ripple incidence all increase markedly. This indicates that small-to-intermediate hippocampal magnetic flow feedback coefficients enhance neuronal excitability and promote SWR-related activity in the model. When γ_1^{CA3} and γ_1^{CA1} are further increased to 0.7 and 1.0, ripple incidence remains elevated but the rate of increase diminishes substantially. This suggests the existence of a finite effective range for hippocampal magnetic stimulation, beyond which facilitatory effects saturate. Collectively, these results demonstrate that hippocampal magnetic flow feedback enhances SWR-related activity within a defined model parameter range by modulating neuronal excitability. This modulation reflects changes in hippocampal network dynamics associated with SWR generation.

3.2. Two-parameter dynamic regulatory mechanism of magnetic stimulation on the hippocampal-cortical network

In this section, we investigate the dynamical behavior of the hippocampal-cortical network with respect to the magnetic stimulation feedback coefficients (γ_1^{CA3} , γ_1^{CA1} , γ_1^{CX1} , γ_1^{CX2}), together with the self-connection probability P_{ee}^{CX1} and self-connection strength J_{ee}^{CX1} of cortical excitatory neurons. Four two-parameter bifurcation diagrams are constructed under distinct magnetic stimulation feedback coefficients, as presented in Figure 4.

It should be noted that, from a neurophysiological perspective, the cortical excitatory self-connection probability P_{ee}^{CX1} is biologically constrained to the interval $[0, 1]$, as it represents a

normalized synaptic connectivity likelihood between neuronal populations. However, in the present bifurcation analysis, we extend the parameter range to $P_{ee}^{CX1} \in [0, 2]$ to systematically uncover a broader spectrum of dynamical phenomena—including the emergence, evolution, and annihilation of complex oscillatory regimes—that remain only partially resolvable within the physiologically bounded interval $[0, 1]$. This extension enables full characterization of key bifurcation structures: specifically, the onset and termination loci of stable limit cycles, as well as the precise parameter coordinates of period-doubling and torus bifurcations. Consequently, it reveals the complete topological organization of the dynamical landscape—particularly the global connectivity of coexisting attractors and the boundaries separating distinct oscillatory, quiescent, and chaotic regimes. All analyses in this section therefore adopt $P_{ee}^{CX1} \in [0, 2]$, unless otherwise stated.

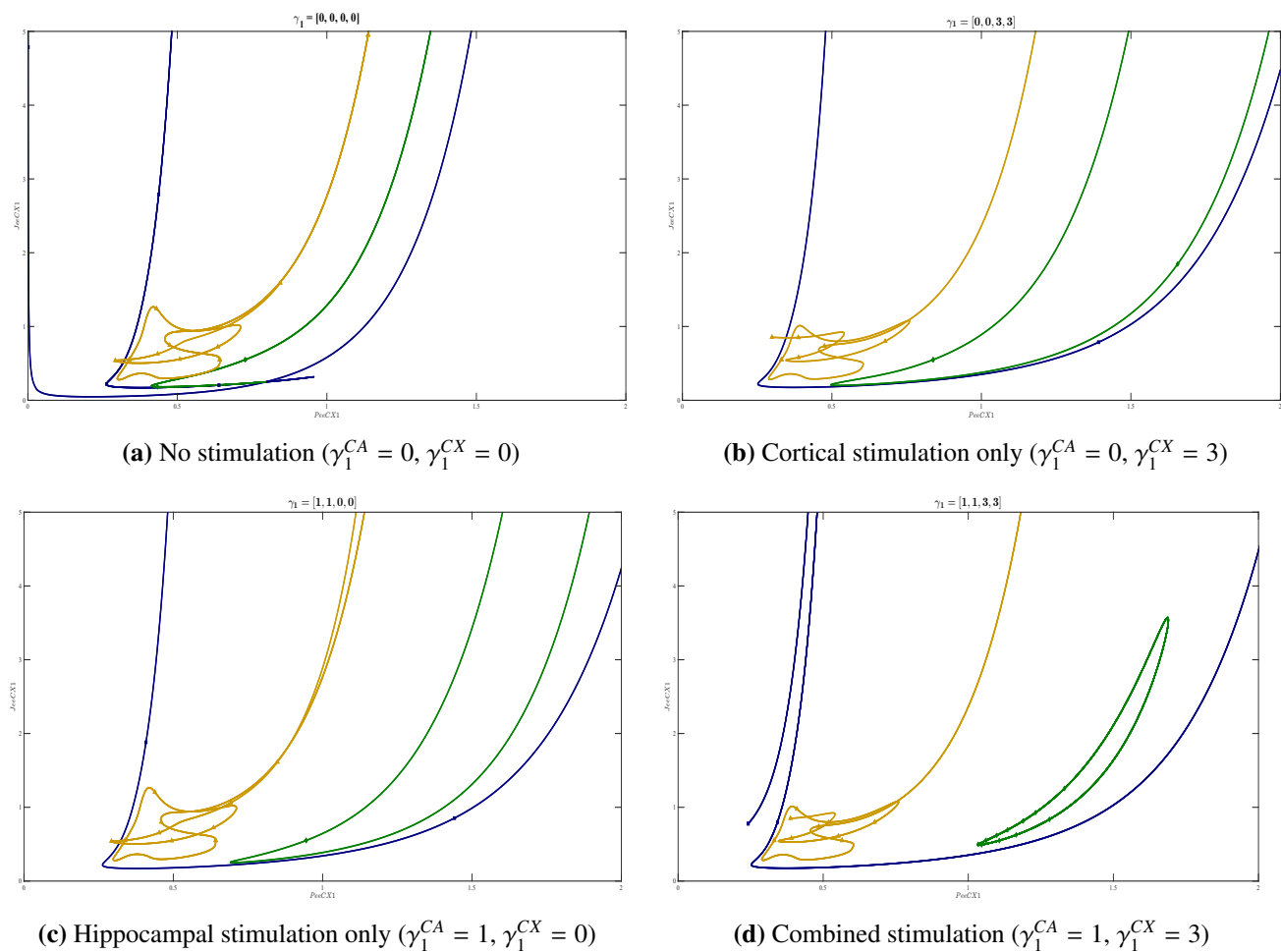


Figure 4. Two-parameter bifurcation diagrams of the hippocampal-cortical network with respect to the self-connection probability P_{ee}^{CX1} and self-connection strength J_{ee}^{CX1} of cortical excitatory neurons under the varying magnetic stimulation feedback coefficients. The blue, orange, and green curves denote Hopf, period-doubling (PD), and torus (TR) bifurcations, respectively.

3.2.1. Two-parameter bifurcation analysis of the hippocampal-cortical network under magnetic stimulation

As shown in Figure 4, we investigate the evolution of system dynamical states through two-parameter bifurcation analysis, under the combined action of magnetic stimulation feedback coefficients applied to excitatory neurons in both the hippocampal and cortical regions. In Figure 4, the blue, orange, and green curves denote Hopf bifurcation, period doubling bifurcation (PD), and torus bifurcation (TR) bifurcation, respectively. The four subfigures represent four distinct combinations of magnetic stimulation feedback coefficients: Figure 4(a) corresponds to the no-stimulation condition (hippocampal feedback coefficient $\gamma_1^{CA} = 0$, cortical feedback coefficient $\gamma_1^{CX} = 0$); Figure 4(b) corresponds to cortical stimulation alone ($\gamma_1^{CA} = 0$, $\gamma_1^{CX} = 3$); Figure 4(c) corresponds to hippocampal stimulation alone ($\gamma_1^{CA} = 1$, $\gamma_1^{CX} = 0$); and Figure 4(d) corresponds to combined stimulation ($\gamma_1^{CA} = 1$, $\gamma_1^{CX} = 3$).

Horizontal comparison of Figure 4(a),(b) shows that, with the hippocampal feedback coefficient fixed at 0, increasing the cortical feedback coefficient from 0 to 3 induces a substantial expansion of the oscillatory regime, indicating that cortical magnetic flow-mediated feedback alone is sufficient to drive large-scale oscillations. By contrast, vertical comparison of Figure 4(a),(c) reveals that increasing the hippocampal feedback coefficient from 0 to 1 yields only a modest expansion of the oscillatory regime, confined primarily to marginal shifts near the baseline. Collectively, these findings demonstrate that cortical feedback exerts a quantitatively dominant and functionally privileged influence on oscillatory dynamics relative to hippocampal feedback, underscoring a pronounced inter-regional asymmetry in neuromodulatory control of network rhythms.

From the comparison between Figure 4(a),(d), increasing the hippocampal feedback coefficient from 0 to 1 while concurrently increasing the cortical feedback coefficient from 0 to 3 induces the most pronounced expansion of the oscillatory regime: it grows from an initially narrow region to occupy the majority of the parameter plane. Relative to either lateral-only or longitudinal-only stimulation, the expansion under combined stimulation exceeds the sum of the individual effects, indicating synergistic interaction between hippocampal and cortical magnetic flow-mediated feedback. This synergy enhances oscillatory recruitment more effectively than either pathway acting in isolation.

However, dynamical richness does not imply stronger neuromodulatory efficacy: although subfigure Figure 4(c) exhibits nontrivial complexity (e.g., isolated periodic windows), its oscillatory regime remains markedly narrower than that in Figure 4(b), where cortical feedback operates in isolation. Thus, cortical feedback exerts a quantitatively superior facilitatory effect on oscillation generation—whether deployed alone or in combination with hippocampal feedback.

Across subfigures (a)–(d), the oscillatory regime monotonically expands in size according to the stimulation condition: (a) < (c) < (b) < (d). This ordering demonstrates that cortical feedback alone exerts a stronger facilitatory effect on oscillations than hippocampal feedback alone, and that their combination yields the maximal expansion—indicating synergistic neuromodulatory interaction. Furthermore, in all four cases, oscillatory activity is confined to an intermediate band of self-connection strength: both excessively low and excessively high values drive the system to quiescent fixed points, while only moderate values permit sustained rhythmic dynamics. This non-monotonic dependence on self-connection is preserved invariantly across all stimulation configurations.

3.2.2. Heatmap characterization of the RMS amplitude and dominant frequency of oscillatory waveforms

Figure 5 presents heatmaps of the root-mean-square (RMS) amplitude of oscillatory waveforms in the hippocampus and cortex for system (2.1) under distinct magnetofluid-mediated feedback configurations. A vertical comparison across the four subfigures shows that in the baseline (unstimulated) state—Figure 5(a), with $\gamma_1^{CA} = 0$ and $\gamma_1^{CX} = 0$ —the RMS amplitude remains negligible across most of the $(P_{ee}^{CX1}, J_{ee}^{CX1})$ parameter plane, with large regions exhibiting exactly zero amplitude; only a confined domain ($P_{ee}^{CX1} \in [0, 2]$, $J_{ee}^{CX1} \in [0, 5]$) supports measurable oscillations. Increasing the cortical feedback coefficient from 0 to 3 (Figure 5(b)) drives a substantial expansion of the high-amplitude regime toward higher J_{ee}^{CX1} values, accompanied by a marked increase in peak amplitude magnitude. In contrast, increasing the hippocampal feedback coefficient from 0 to 1 (Figure 5(c)) induces only a modest expansion—significantly less than that observed under cortical stimulation—without notable amplitude intensification. Under combined cortical–hippocampal feedback (Figure 5(d)), the high-amplitude regime achieves its maximal spatial extent and amplitude intensity across all conditions.

Figure 6 displays heatmaps of the dominant frequency of oscillatory waveforms in system (2.1) under different magnetic flow–mediated feedback configurations, computed over the steady-state interval $t \in [100, 110]$ min. In the unstimulated state (Figure 6(a)), clear frequency components appear only within the confined parameter region corresponding to the high-amplitude area in Figure 5, exhibiting a spatially uniform frequency distribution. Following cortical stimulation (Figure 6(b)), the frequency-distributed region markedly expands and becomes stratified within the high-amplitude area into two distinct bands: a lower-frequency band and a higher-frequency band. Hippocampal stimulation alone (Figure 6(c)) yields only marginal expansion of the frequency-distributed region, with a distribution pattern closely resembling that of the unstimulated state. Under combined stimulation (Figure 6(d)), the frequency coverage reaches its broadest observed extent, and multiple frequency “patches” emerge across the parameter plane, indicating that the system is capable of generating a richer repertoire of frequency components.

3.2.3. Correspondence between heatmap-derived features and bifurcation structures

Comparison of the heatmaps in Figures 5 and 6 with the bifurcation diagram in Figure 4 reveals a high degree of spatial correspondence across all three representations. Specifically, the oscillatory region bounded by the Hopf bifurcation curve (blue) in Figure 4 aligns closely—both in location and contour—with the high-amplitude region in Figure 5 and the frequency-supporting region in Figure 6 on the $(P_{ee}^{CX1}, J_{ee}^{CX1})$ parameter plane. The spatial partitioning of dynamical regimes revealed by the heatmaps is broadly consistent with the theoretical boundaries derived from bifurcation analysis; this correspondence demonstrates that the dominant-frequency metric reliably captures the system’s parameter-dependent dynamical transitions.

Further comparison across the four stimulation conditions shows that cortical feedback exerts a dominant role on oscillation induction. In Figure 4, the expansion of the oscillatory region from subfigure (a) to (b) (cortical feedback only) substantially exceeds that from (a) to (c) (hippocampal feedback only). This asymmetry is consistently reproduced in Figures 5 and 6: the increase in RMS amplitude magnitude and the extent of frequency-supporting parameter space under isolated cortical

feedback are markedly greater than those under isolated hippocampal feedback. The ordering of oscillatory region size—(a) < (c) < (b) < (d)—holds identically across the enclosed area of the Hopf curve in Figure 4, the high-amplitude region in Figure 5, and the frequency-supporting region in Figure 6. This invariant ranking reveals a consistent hierarchical and asymmetric dependence of the system's oscillatory behavior on feedback location, with cortical magnetic flow-mediated feedback exerting quantitatively stronger regulatory efficacy than hippocampal feedback.

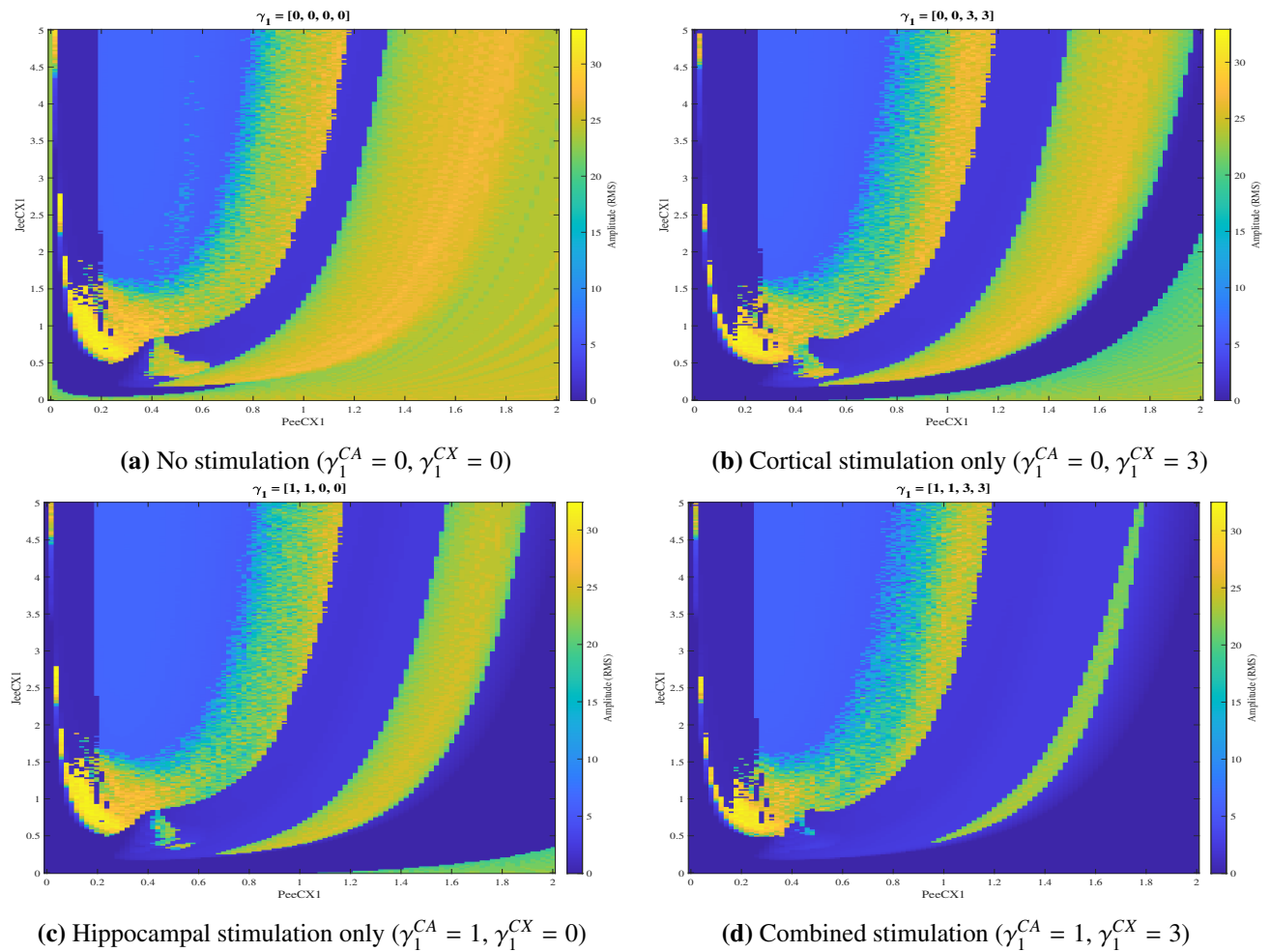


Figure 5. Heatmaps of the root-mean-square (RMS) amplitude of oscillatory waveforms in system (2.1), computed over the interval $t \in [100, 120]$ min, parametrized by $P_{ee}^{CX1} \in [0, 2]$ and $J_{ee}^{CX1} \in [0, 5]$ under the varying magnetic stimulation feedback coefficients.

Furthermore, the internal dynamical structures revealed in the bifurcation diagrams are faithfully reproduced in the heatmap results. In Figure 4(d) (combined feedback), a closed torus bifurcation curve (green) appears within the oscillatory region, signaling the emergence of quasi-periodic dynamics. Correspondingly, Figure 5(d) exhibits multiple local amplitude maxima and steep spatial gradients within the high-amplitude regime; likewise, Figure 6(d) displays multiple frequency “patches” confined to the parameter subregion $P_{ee}^{CX1} \in [0.6, 1.2]$ and $J_{ee}^{CX1} \in [2, 4]$. Critically, the heatmap regions showing the strongest color gradients align precisely with the loci of the

period-doubling bifurcation curves (orange) and torus bifurcation curves (green) in Figure 4—indicating that qualitative transitions in oscillatory behavior occur strictly at these bifurcation boundaries.

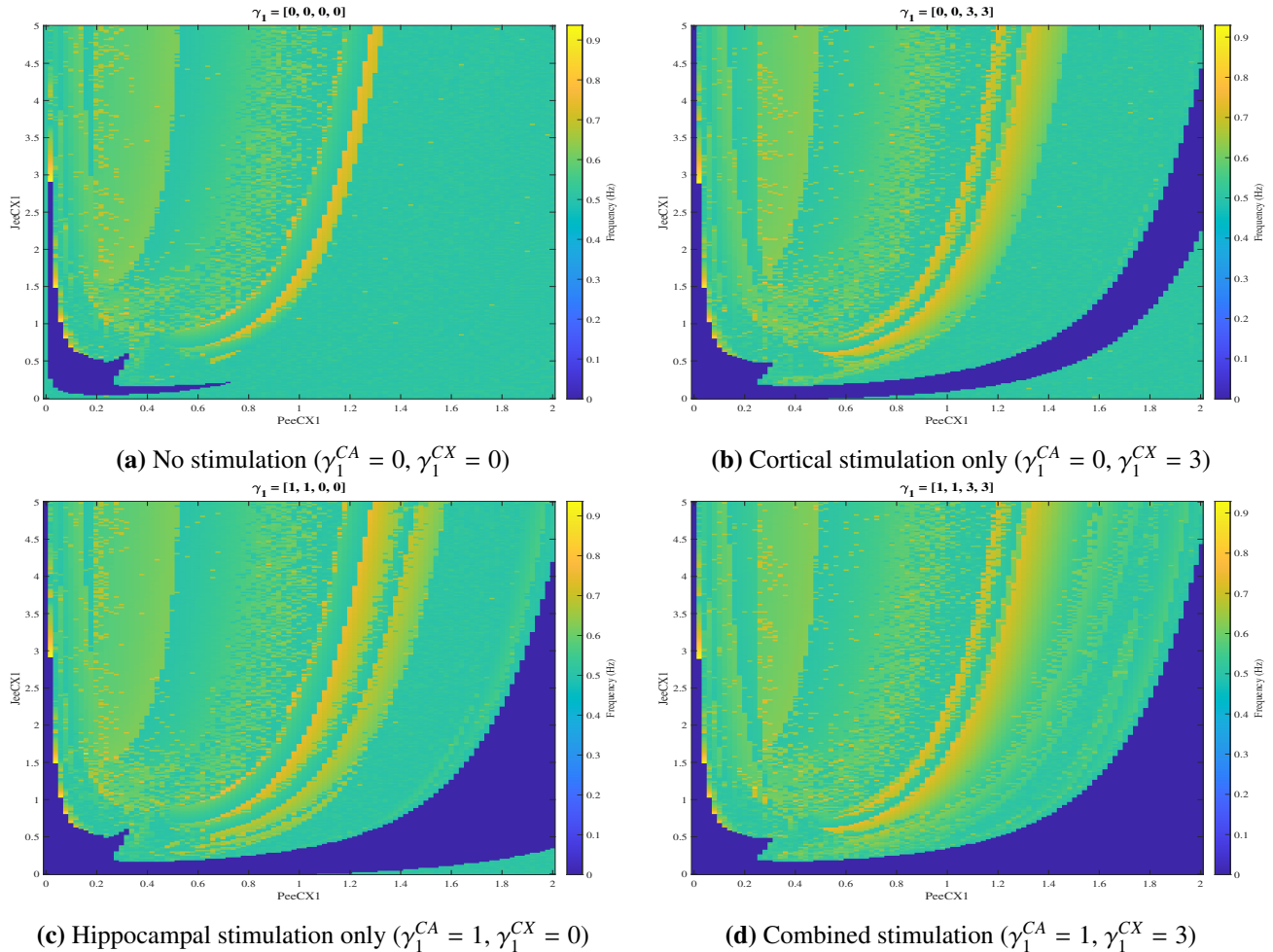


Figure 6. Heatmaps of the dominant frequency of oscillatory waveforms in system (2.1), computed over the interval $t \in [100, 120]$ min, parametrized by $P_{ee}^{CX1} \in [0, 2]$ and $J_{ee}^{CX1} \in [0, 5]$ under the varying magnetic stimulation feedback coefficients.

In summary, bifurcation analysis provides a theoretical framework for delineating the system's dynamical boundaries and parameter-space partitions, whereas the heatmaps offer a numerical visualization of the resulting oscillatory behaviors across those same parameter regimes. The two methodologies mutually corroborate one another: the structural features predicted by bifurcation theory are quantitatively confirmed by simulation-based heatmaps, and conversely, the heatmap patterns provide empirical grounding for the theoretical bifurcation structure. Together, they robustly validate the nonlinear dynamical mechanisms through which magnetofluid-mediated feedback coefficients regulate hippocampal-cortical network oscillations.

4. Conclusions and discussion

In this paper, we investigate a hippocampal-cortical coupled memory neural network model, focusing on the dynamical response of the membrane potential of excitatory neurons in the hippocampal CA3 region of system (2.1) to external magnetic stimulation. Our primary objective is to elucidate how magnetic stimulation modulates rhythmic neuronal activity within the hippocampal-cortical memory network.

Through one-parameter bifurcation analysis, we fix the self-connection strength J_{ee}^{CX1} of hippocampal CX1 excitatory neurons and systematically examine the synergistic effects of two key parameters—the magnetic stimulation feedback coefficient (γ_1^{CA} or γ_1^{CX}) and the cortical excitatory self-connection probability P_{ee}^{CX1} —on network dynamics. We find that increasing the magnetic flow feedback coefficient induces a canonical sequence of dynamical transitions: fixed-point instability, Hopf bifurcation, and period-doubling cascade. Concurrently, both the amplitude of stable limit cycles and the parameter range over which they persist expand monotonically. Extending this to two-parameter bifurcation diagrams in the $(P_{ee}^{CX1}, J_{ee}^{CX1})$ parameter plane, we observe progressive expansion of the oscillatory regime across the four stimulation conditions, with area ordering (a) < (c) < (b) < (d). This ordering demonstrates that cortical stimulation alone exerts a stronger facilitatory effect on oscillation generation than hippocampal stimulation alone, while combined stimulation yields maximal expansion—indicating synergistic neuromodulatory interaction. Crucially, internal bifurcation structures—specifically the period-doubling (orange) and torus (green) curves—partition the oscillatory regime into distinct dynamical subregions, revealing the emergence of quasi-periodic and higher-complexity dynamics exclusively under combined stimulation.

To complement theoretical bifurcation analysis, we construct heatmaps of the root-mean-square (RMS) amplitude and dominant frequency of oscillatory waveforms, visualizing actual oscillatory behaviors across parameter regimes. These heatmaps show that high-amplitude and high-frequency-supporting regions spatially align with the bifurcation-bounded oscillatory regimes—quantitatively confirming that theoretically predicted dynamical boundaries broadly govern observed waveform characteristics. Moreover, steep gradient variations and multiple frequency “patches” in the heatmaps faithfully reproduce the loci of period-doubling and torus bifurcation curves, indicating that qualitative transitions in oscillatory behavior occur precisely at these bifurcation boundaries. This integrated bifurcation–heatmap framework establishes a consistent bidirectional correspondence between dynamical systems theory and numerical simulation, providing a mechanistic foundation for understanding how magnetic flow feedback coefficients regulate hippocampal-cortical rhythmic activity.

To assess how magnetic flow feedback modulates SWR-related neural activity, we apply an automated sharp wave ripple (SWR) detection algorithm to generate time-series plots of SWR counts under varying magnetic flow feedback coefficients. These plots reveal both monotonic and nonmonotonic variation patterns in SWR occurrence as the magnetic flow feedback coefficients change. We further construct SWR-count heatmaps as functions of the cortical excitatory self-connection strength J_{ee}^{CX1} and self-connection probability P_{ee}^{CX1} , sampled across physiologically plausible ranges and under distinct magnetic flow feedback configurations. Results show that concurrent hippocampal-cortical stimulation yields a gradual, bounded increase in SWR counts with rising feedback coefficients—within a well-defined operational range—and exhibits complex

spatiotemporal distribution patterns. Specifically, under cortical-only feedback, SWR counts increase approximately linearly with the cortical magnetic flow feedback coefficients within the investigated parameter range, accompanied by an expansion of the spatiotemporal distribution of SWR-related events. In contrast, hippocampal-only feedback produces a strongly nonlinear response: SWR counts rise rapidly at small feedback coefficients but gradually plateau beyond a finite parameter range, while further increases in the feedback coefficients reduce the facilitatory effect and may destabilize network activity. Consequently, precise, amplitude-tuned control is essential for effective and safe hippocampal neuromodulation.

From the perspective of dynamical systems theory, SWR count variations directly reflect evolving stability properties of the hippocampal-cortical network under magnetic stimulation. Comparative analysis of bifurcation diagrams reveals that, as the magnetic flow feedback coefficient increases, the total length of the stable limit cycle (SLC) exhibits a marked upward trend—signifying expansion of the parameter regime supporting sustained oscillations, thereby establishing a dynamical basis for increased SWR frequency and duration. Larger SLC regions indicate that the CA3 excitatory membrane potential (V_e^{CA3}) maintains stable oscillations across broader ranges of the magnetic flow feedback coefficients. At relatively large hippocampal magnetic flow feedback coefficients, membrane potential dynamics remain stable across a broader range of cortical parameters, indicating an expanded stable oscillatory regime in the model. This is mechanistically consistent with SWR heatmap saturation: the SWR count growth rate decelerates markedly at large hippocampal magnetic flow feedback coefficients. Furthermore, topological reconfigurations of SLCs—such as fragmentation, merging, or abrupt boundary shifts—correspond directly to nonlinear modulations in SWR quantity, temporal periodicity, and spatiotemporal distribution. Correspondingly, SWR statistics reflect this dynamical change: heatmap analysis shows that SWR counts initially rise and then plateau, while statistical fluctuations increase markedly at large magnetic flow feedback coefficients. This empirical regularity maps directly onto bifurcation diagrams: beyond critical thresholds, the system undergoes period-doubling or torus bifurcations, or transitions involving annihilation/emergence of coexisting attractors (e.g., stable equilibria and limit cycles), inducing abrupt changes in oscillation amplitude, frequency, or existence. Thus, a consistent bidirectional correspondence exists between bifurcation diagrams and SWR heatmaps: bifurcation theory explains the nonlinear growth and saturation of SWR counts, while macroscopic SWR statistics serve as empirically accessible, quantitatively calibrated outputs that validate theoretically predicted dynamical transitions. It should be noted that the above correspondence reflects dynamical consistency at the model level, rather than direct reconstruction of experimental SWR waveforms. The morphology and amplitude of experimentally observed SWRs are influenced by factors including transmembrane current sources and sinks, CA3-CA1 synaptic drive, cell-type-specific spiking of pyramidal neurons and interneuron subtypes, and recording location within hippocampal laminae. SWRs also exhibit functional heterogeneity. For instance, larger-amplitude SWRs show preferential coupling to hippocampal-prefrontal reactivation and predict subsequent memory performance [42]. As this model represents the population-averaged membrane potential, the waveforms in Figure 2 serve solely as a basis for identifying SWR-related model events to enable comparative assessment of relative SWR activity modulation across feedback conditions. Since the taxonomy and functional distinctions among SWR subtypes remain incompletely resolved, our future work will further improve the model and integrate experimental LFP data to refine this mapping.

By comparing one-parameter bifurcation diagrams with SWR-count heatmaps generated by the hippocampal-cortical neuronal model under varying magnetic flow feedback coefficients, we identify three key patterns. First, the regulatory effect of hippocampal magnetic flow feedback is not uniformly facilitatory: bifurcation analysis reveals that when the hippocampal feedback coefficient exceeds a critical model threshold, originally stable limit cycles undergo marked topological restructuring—evidenced by shifts in oscillation amplitude bands, loss of local stability, and disappearance of Hopf and period-doubling bifurcation points. These changes collectively demonstrate a strongly nonlinear dependence of network dynamics on the magnetic flow feedback coefficient. Second, SWR-count heatmaps corroborate this nonlinearity: SWR incidence rises rapidly at small feedback coefficients but subsequently decelerates and saturates as the feedback coefficient increases. The inflection point of this curve defines a well-characterized effective facilitation window for hippocampal magnetic stimulation. Within this model parameter range, magnetic flow feedback enhances SWR-related rhythmic activity, whereas further increases in the feedback coefficient may lead to network destabilization or suppression of rhythmic activity. Third, the differential robustness of hippocampal versus cortical stimulation arises from fundamental neuroanatomical and biophysical constraints. The cortex lies on the brain's surface, with relatively fixed spatial coordinates and straightforward structural localization—enabling magnetic stimulation energy to be delivered with higher spatial precision and greater field uniformity. This improves modulation consistency and reproducibility. In contrast, the hippocampus is deeply embedded within the medial temporal lobe and surrounded by heterogeneous tissue layers—including white matter tracts, cerebrospinal fluid-filled ventricles, and overlying cortical gyri—that distort, scatter, and attenuate the induced electric fields. Consequently, achieving precise, focal, and reliable stimulation of hippocampal circuits remains technically challenging. Therefore, when designing practical neuromodulation protocols, one must weigh multiple factors—including operational complexity, targeting accuracy, safety margins, implementation cost, and response stability. Under equivalent experimental or clinical conditions, cortical-targeted magnetic stimulation consistently demonstrates superior robustness, reproducibility, and cost-effectiveness compared with hippocampal-targeted approaches.

The integrated bifurcation–heatmap framework developed in this study also opens several avenues for future investigation. First, the asymmetric expansion of oscillatory regimes in the two-parameter bifurcation diagrams—greater under cortical than hippocampal stimulation—quantitatively supports prioritizing cortical targets for reliable rhythm enhancement, while reserving hippocampal stimulation for fine-tuned, window-constrained interventions. Second, the closed torus bifurcation curves observed exclusively under combined feedback indicate the emergence of quasi-periodic and higher-complexity dynamics, reflecting richer patterns of network rhythmic activity. Systematic characterization of these regimes across broader parameter ranges could identify stimulation configurations that selectively enhance specific frequency bands (e.g., theta–gamma coupling). Third, the strong spatial correspondence between bifurcation boundaries and heatmap gradient discontinuities demonstrates that the theoretical framework serves as a predictive tool for identifying parameter regions of maximal stimulation efficacy—thereby guiding mechanism-driven, target-optimized intervention design. Ultimately, integrating dynamical systems theory with data-driven visualization establishes a principled foundation for precision neuromodulation: stimulation parameters are optimized not empirically, but according to the intrinsic bifurcation architecture of the targeted neural circuit—ensuring reliable, reproducible, and functionally specific

modulation of memory-related rhythms. However, the present conclusions are based on theoretical analysis and numerical simulations. A quantitative mapping between the dimensionless feedback coefficient γ_1^k employed in the model and experimentally accessible stimulation parameters has not yet been established. Specifically, these parameters include magnetic-field strength and induced electric-field amplitude. Accordingly, establishing this mapping and validating the model-predicted network dynamics against empirical electrophysiological or neuroimaging data will constitute a main direction of our future work.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Data availability

The datasets and code used in this study are available from the corresponding author upon reasonable request.

Conflict of interest

Professor Lixia Duan is an editorial board member for Electronic Research Archive and was not involved in the editorial review and the decision to publish this article. The authors declare there are no conflicts of interest.

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Appendix

A.1. Model parameters

Table A.1. The biological parameters of the hippocampal-cortical neural memory model [43].

Parameter	Value	Unit	Definition
J_{ee}^{CX1}	[0,5]	mV	Strength of connection from excitatory to excitatory neurons in CX1
P_{ee}^{CX1}	[0,2]	–	Probability of connection from excitatory to excitatory neurons in CX1
τ_e	0.02	s	Membrane relaxation time constant for excitatory neurons
τ_i	0.01	s	Membrane relaxation time constant for inhibitory neurons
τ_c	0.5	s	Adaptation recovery time
r_0	0.1	Hz	Minimum firing rate in completely depolarized state
r_1	70	Hz	Maximum firing rate in completely depolarized state
$c_{\max}$	10	mV	Adaptation threshold level
g_c	3	mV	Sharpness of excitatory-excitatory connection dependence on adaptation variable
g_e^{CA3}	6	mV	Sharpness of firing rate dependence on membrane potential from CA3 excitatory neurons
g_i^{CA3}	3	mV	Sharpness of firing rate dependence on membrane potential from CA3 inhibitory neurons
g_e^{CA1}	0.5	mV	Sharpness of firing rate dependence on membrane potential from CA1 excitatory neurons
g_i^{CA1}	0.25	mV	Sharpness of firing rate dependence on membrane potential from CA1 inhibitory neurons
g_e^{CX1}	5	mV	Sharpness of firing rate dependence on membrane potential from CX1 excitatory neurons
g_i^{CX1}	2	mV	Sharpness of firing rate dependence on membrane potential from CX1 inhibitory neurons
g_e^{CX2}	5	mV	Sharpness of firing rate dependence on membrane potential from CX2 excitatory neurons
g_i^{CX2}	2	mV	Sharpness of firing rate dependence on membrane potential from CX2 inhibitory neurons
V_e^{*CA3}	40	mV	Firing threshold potential for excitatory neurons in CA3 region
V_i^{*CA3}	25	mV	Firing threshold potential for inhibitory neurons in CA3 region
V_e^{*CA1}	19	mV	Firing threshold potential for excitatory neurons in CA1 region
V_i^{*CA1}	20	mV	Firing threshold potential for inhibitory neurons in CA1 region
V_e^{*CX1}	30	mV	Firing threshold potential for excitatory neurons in CX1 network
V_i^{*CX1}	30	mV	Firing threshold potential for inhibitory neurons in CX1 network
V_e^{*CX2}	30	mV	Firing threshold potential for excitatory neurons in CX2 network
V_i^{*CX2}	30	mV	Firing threshold potential for inhibitory neurons in CX2 network
J_{ee}^{CA3}	0.7	mV	Base connection strength between excitatory neurons in CA3
J_{ei}^{CA3}	0.3	mV	Connection strength from excitatory to inhibitory neurons in CA3
J_{ie}^{CA3}	0.51	mV	Connection strength from inhibitory to excitatory neurons in CA3
J_{ei}^{CA1}	0.22	mV	Connection strength from excitatory to inhibitory neurons in CA1

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Parameter	Value	Unit	Definition
J_{ie}^{CA1}	0.87	mV	Connection strength from inhibitory to excitatory neurons in CA1
J_{ii}^{CA1}	0.25	mV	Connection strength between inhibitory neurons in CA1
J_{ei}^{CX1}	0.42	mV	Connection strength from excitatory to inhibitory neurons in CX1
J_{ie}^{CX1}	1.4	mV	Connection strength from inhibitory to excitatory neurons in CX1
J_{ii}^{CX1}	0.3	mV	Connection strength between inhibitory neurons in CX1
J_{ee}^{CX2}	0.38	mV	Base connection strength between excitatory neurons in CX2
J_{ei}^{CX2}	0.48	mV	Connection strength from excitatory to inhibitory neurons in CX2
J_{ie}^{CX2}	1.25	mV	Connection strength from inhibitory to excitatory neurons in CX2
J_{ii}^{CX2}	0.57	mV	Connection strength between inhibitory neurons in CX2
J_{ee}^{CA3CA1}	0.15	mV	Connection strength from CA3 excitatory to CA1 excitatory neurons
J_{ei}^{CA3CA1}	0.04	mV	Connection strength from CA3 excitatory to CA1 inhibitory neurons
J_{ee}^{CX2CA3}	1.88	mV	Connection strength from CX2 excitatory to CA3 excitatory neurons
J_{ei}^{CX2CA3}	0.31	mV	Connection strength from CX2 excitatory to CA3 inhibitory neurons
J_{ee}^{CA1CX1}	2	mV	Connection strength from CA1 excitatory to CX1 excitatory neurons
J_{ei}^{CA1CX1}	1	mV	Connection strength from CA1 excitatory to CX1 inhibitory neurons
J_{ee}^{CX1CA1}	1.04	mV	Connection strength from CX1 excitatory to CA1 excitatory neurons
J_{ei}^{CX1CA1}	0.21	mV	Connection strength from CX1 excitatory to CA1 inhibitory neurons
J_{ee}^{CA1CX2}	2	mV	Connection strength from CA1 excitatory to CX2 excitatory neurons
J_{ei}^{CA1CX2}	1	mV	Connection strength from CA1 excitatory to CX2 inhibitory neurons
J_{ee}^{CX2CX1}	0.67	mV	Connection strength from CX2 excitatory to CX1 excitatory neurons
J_{ei}^{CX2CX1}	1.8	mV	Connection strength from CX2 excitatory to CX1 inhibitory neurons
J_{ee}^{CX1CX2}	0.039	mV	Connection strength from CX1 excitatory to CX2 excitatory neurons
J_{ei}^{CX1CX2}	1.62	mV	Connection strength from CX1 excitatory to CX2 inhibitory neurons
Δ_c^{CA3}	0.0064	–	Average adaptive parameter per incoming action potential in CA3
Δ_c^{CA1}	0.002	–	Average adaptive parameter per incoming action potential in CA1
Δ_c^{CX1}	0.005	–	Average adaptive parameter per incoming action potential in CX1
Δ_c^{CX2}	0.0054	–	Average adaptive parameter per incoming action potential in CX2
p_{ee}^{CA3}	0.25	–	Connection probability between excitatory neurons in CA3
p_{ei}^{CA3}	0.3	–	Connection probability from excitatory to inhibitory neurons in CA3
p_{ie}^{CA3}	0.5	–	Connection probability from inhibitory to excitatory neurons in CA3
p_{ei}^{CA1}	0.3	–	Connection probability from excitatory to inhibitory neurons in CA1
p_{ie}^{CA1}	0.8	–	Connection probability from inhibitory to excitatory neurons in CA1
p_{ii}^{CA1}	0.25	–	Connection probability between inhibitory neurons in CA1
p_{ei}^{CX1}	0.2	–	Connection probability from excitatory to inhibitory neurons in CX1
p_{ie}^{CX1}	0.2	–	Connection probability from inhibitory to excitatory neurons in CX1
p_{ii}^{CX1}	0.2	–	Connection probability between inhibitory neurons in CX1
p_{ee}^{CX2}	0.2	–	Connection probability between excitatory neurons in CX2
p_{ei}^{CX2}	0.2	–	Connection probability from excitatory to inhibitory neurons in CX2
p_{ie}^{CX2}	0.2	–	Connection probability from inhibitory to excitatory neurons in CX2
p_{ii}^{CX2}	0.2	–	Connection probability between inhibitory neurons in CX2
p_{ee}^{CA3CA1}	0.6	–	Connection probability from CA3 excitatory to CA1 excitatory neurons
p_{ei}^{CA3CA1}	0.2	–	Connection probability from CA3 excitatory to CA1 inhibitory neurons
p_{ee}^{CX2CA3}	0.02	–	Connection probability from CX2 excitatory to CA3 excitatory neurons
p_{ei}^{CX2CA3}	0.02	–	Connection probability from CX2 excitatory to CA3 inhibitory neurons

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Parameter	Value	Unit	Definition
P_{ee}^{CA1CX1}	0.002	–	Connection probability from CA1 excitatory to CX1 excitatory neurons
P_{ei}^{CA1CX1}	0.002	–	Connection probability from CA1 excitatory to CX1 inhibitory neurons
P_{ee}^{CX1CA1}	0.03	–	Connection probability from CX1 excitatory to CA1 excitatory neurons
P_{ei}^{CX1CA1}	0.03	–	Connection probability from CX1 excitatory to CA1 inhibitory neurons
P_{ee}^{CA1CX2}	0.002	–	Connection probability from CA1 excitatory to CX2 excitatory neurons
P_{ei}^{CA1CX2}	0.002	–	Connection probability from CA1 excitatory to CX2 inhibitory neurons
P_{ee}^{CX1CX2}	0.01	–	Connection probability from CX1 excitatory to CX2 excitatory neurons
P_{ei}^{CX1CX2}	0.01	–	Connection probability from CX1 excitatory to CX2 inhibitory neurons
P_{ee}^{CX2CX1}	0.01	–	Connection probability from CX2 excitatory to CX1 excitatory neurons
P_{ei}^{CX2CX1}	0.01	–	Connection probability from CX2 excitatory to CX1 inhibitory neurons
N_e^{CA3}	1000	–	Number of excitatory neurons in CA3 hippocampal network
N_i^{CA3}	100	–	Number of inhibitory neurons in CA3 hippocampal network
N_e^{CA1}	1000	–	Number of excitatory neurons in CA1 hippocampal network
N_i^{CA1}	100	–	Number of inhibitory neurons in CA1 hippocampal network
N_e^{CX1}	8000	–	Number of excitatory neurons in CX1
N_i^{CX1}	2000	–	Number of inhibitory neurons in CX1
N_e^{CX2}	8000	–	Number of excitatory neurons in CX2
N_i^{CX2}	2000	–	Number of inhibitory neurons in CX2
γ_2^{CA3}	3	–	Decay coefficient of magnetic flux in CA3
γ_2^{CA1}	3	–	Decay coefficient of magnetic flux in CA1
γ_2^{CX1}	3	–	Decay coefficient of magnetic flux in CX1
γ_2^{CX2}	3	–	Decay coefficient of magnetic flux in CX2
α	0.6	–	Linear coupling parameter (constant component of inductance)
β	0.00002	–	Nonlinear modulation parameter (quadratic coefficient of inductance)

A.2. Sharp wave ripple detection criteria and parameter settings

To provide a clear and reproducible definition of the sharp wave ripple (SWR) events used in the quantitative analysis, we summarize here the detection criteria and corresponding parameter settings. The detection procedure is based on the electrophysiological definition of an SWR as the temporal coincidence of a hippocampal sharp-wave event and a ripple event.

Candidate sharp-wave events are first identified from deep hippocampal local field potential (LFP) signals. The signal is band-pass filtered between 2 and 50 Hz. A candidate sharp-wave event is identified when the filtered signal exceeds a threshold of 2.5 standard deviations (SD) relative to baseline activity. Events with durations shorter than 20 ms or longer than 500 ms are excluded.

Candidate ripple events are identified from superficial hippocampal LFP signals. The signal is band-pass filtered within the ripple frequency range of 80–250 Hz, and the oscillatory power within this frequency band is calculated. A candidate ripple event is identified when the ripple-band power exceeds a threshold of 2.5 SD relative to the baseline level. Events with durations shorter than 25 ms are discarded.

An SWR event is identified only when a candidate sharp-wave event and a candidate ripple event overlap in time. Therefore, temporal coincidence between the sharp-wave and ripple components is required for an event to be classified as an SWR. Each temporally coupled event satisfying the above criteria is counted as one SWR event.

The main detection criteria and parameter settings are summarized in Table A.2.

Table A.2. Criteria and parameter settings for sharp wave ripple detection.

Component	Detection criterion	Threshold	Duration criterion
Sharp wave	2–50 Hz band-pass filtered signal	2.5 SD	20–500 ms
Ripple	80–250 Hz ripple-band power	2.5 SD	≥ 25 ms
SWR	Temporal overlap between a sharp-wave event and a ripple event	–	Temporal coincidence required

For the computational analysis in this study, the above criteria provide the reference definition for identifying SWR-related events in the simulated hippocampal activity. The same detection criteria and parameter settings are used consistently across all magnetic flow feedback configurations and network parameter combinations. Consequently, differences in the reported SWR counts reflect changes in the simulated network dynamics under identical event-detection criteria rather than changes in the detection thresholds.



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