Simple Framework for Constructing Functional Spiking Recurrent Neural Networks

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Abstract

Cortical microcircuits exhibit complex recurrent architectures that possess dynamically rich properties. The neurons that make up these microcircuits communicate mainly via discrete spikes, and it is not clear how spikes give rise to dynamics that can be used to perform computationally challenging tasks. In contrast, continuous models of rate-coding neurons can be trained to perform complex tasks. Here, we present a simple framework to construct biologically realistic spiking recurrent neural networks (RNNs) capable of learning a wide range of tasks. Our framework involves training a continuous-variable rate RNN with important biophysical constraints and transferring the learned dynamics and constraints to a spiking RNN in a one-to-one manner. We validate our framework on several cognitive task paradigms to replicate previously observed experimental results. We also demonstrate different ways to exploit the biological features of our models to elucidate neural mechanisms underlying cognitive functions.

Introduction

Understanding how seemingly irregular and chaotic neural activity facilitates information processing and supports complex behavior is a major challenge in neuroscience. Previous studies have employed models based on recurrent neural networks (RNNs) of continuous-variable rate units to characterize network dynamics underlying neural computations [1–6].

Methods commonly used to train rate networks to perform cognitive tasks can be largely classified into three categories: recursive least square (RLS)-based, gradient-based, and reward-based algorithms. The First-Order Reduced and Controlled Error (FORCE) algorithm, which utilizes RLS, has been widely used to train RNNs to produce complex output signals [2] and to reproduce experimental results [3, 7, 8]. Gradient descent-based methods, including Hessian-free methods, have been also successfully applied to train rate networks in a supervised manner and to replicate the computational dynamics observed in networks from behaving animals [4, 9, 10]. Unlike the previous two categories (i.e. RLS-based and gradient-based algorithms), reward-based learning methods are more biologically plausible and have been shown to be as effective in training rate RNNs as the supervised learning methods [11, 12]. Even though these models have been vital in uncovering previously unknown computational mechanisms, continuous rate networks do not incorporate basic biophysical constraints such as the spiking nature of biological neurons.

Training spiking network models where units communicate with one another via discrete spikes is more difficult than training continuous rate networks. The non-differentiable nature of spike signals prevents the use of gradient descent-based methods to train spiking networks directly, although several differentiable models have been proposed [13, 14]. Due to this challenge, FORCE-based learning algorithms have been most commonly used to train spiking recurrent networks. While recent advances have successfully modified and applied FORCE training to construct functional spike RNNs [5, 15–18], FORCE training is computationally inefficient and unstable when connectivity constraints, including separate populations for excitatory and inhibitory populations (Dale's principle) and sparse connectivity patterns, are imposed [16]. Consistent with these limitations, there are only few examples of biologically realistic spiking RNN models trained via the FORCE algorithm. In these examples, moderately sparse spiking networks that obey Dale's principle were trained to produce simple oscillatory output signals [16, 18].

Here we present a computational framework for constructing functional spiking neural networks that can easily incorporate biophysical constraints. Our method involves training a continuous-variable rate RNN using a gradient descent-based method, and transferring the learned dynamics

of the rate network along with the constraints to a spiking network model in a one-to-one manner. The gradient descent learning algorithm allowed us to easily optimize many parameters including the connectivity weights of the network and the synaptic decay time constant for each unit. In addition, Dale's principle and additional connectivity patterns can be enforced without significantly affecting computational efficiency and network stability using the recurrent weight matrix parametrization method proposed by Song et al. [10]. We demonstrate the flexibility and the versatility of our framework by constructing spiking networks to perform several tasks ranging from a simple Go-NoGo task to a more complex task that requires input integration and working memory. Furthermore, we demonstrate how biologically realistic spiking RNNs constructed from our framework allow us to utilize both rate- and spike-based measures to better understand the network dynamics underlying cognitive behavior.

Results

Here we provide a brief overview of the two types of recurrent neural networks (RNNs) that we employed throughout this study (more details in Methods): continuous-variable firing rate RNNs and spiking RNNs. The continuous-variable rate network model consisted of N rate units whose firing rates were estimated via a nonlinear input-output transfer function [1, 2]. The model was governed by the following set of equations:

$$\tau_i \frac{dx_i}{dt} = -x_i + \sum_{j=1}^{N} w_{ij}^{rate} r_j^{rate} + I_{ext} \tag{1}$$

$$r_i^{rate} = \phi(x_i) \tag{2}$$

where τ_i is the synaptic decay time constant for unit i, x_i is the synaptic current variable for unit i, w_{ij}^{rate} is the synaptic strength from unit j to unit i, and I_{ext} is the external current input to unit i. The firing rate of unit i (r_i^{rate}) is given by applying a nonlinear transfer function ($\phi(\cdot)$) to the synaptic current variable. In order to make the network biologically realistic, we chose the transfer function to be a non-negative saturating function (standard sigmoid function) and parametrized the connectivity matrix ($w_{ij}^{rate} \in W^{rate}$) to enforce Dale's principle and additional connectivity constraints (see Methods).

The second RNN model that we considered was a network composed of N spiking units. Throughout this study, we focused on networks of leaky integrate-and-fire (LIF) units whose mem-

brane voltage dynamics were given by:

$$\tau_m \frac{dv_i}{dt} = -v_i + \sum_{j=1}^{N} w_{ij}^{spk} r_j^{spk} + I_{ext} \tag{3}$$

where τ_m is the membrane time constant (set to 10 ms throughout this study), v_i is the membrane voltage of unit i, w_{ij}^{spk} is the synaptic strength from unit j to unit i, r_j^{spk} represents the synaptic filtering of the spike train of unit j, and I_{ext} is the external current source. The discrete nature of r_j^{spk} (see Methods) has posed a major challenge for directly training spiking networks using gradient-based supervised learning. Even though the main results presented here are based on LIF networks, our method can be generalized to quadratic integrate-and-fire (QIF) networks with only few minor changes to the model parameters (see Supplementary Table 1, Supplementary Notes, Supplementary Fig. 4).

For each example shown in the study, we present the results obtained from a representative trained model, but all the results were robust and reproducible from multiple trained networks with random initialization conditions. Continuous rate network training was implemented using the open-source software library TensorFlow in Python, while LIF/QIF network simulations along with the rest of the analyses were performed in MATLAB.

Transfer Learning from Continuous Rate Networks to Spiking Networks. In order to construct functional spiking networks that perform cognitive tasks, we developed a simple procedure that directly maps dynamics of a trained continuous rate RNN to a spiking RNN in a one-to-one manner. The first step of our method involves training a continuous rate RNN to perform a task. Throughout this study, we used a gradient-descent supervised method, known as Backpropagation Through Time (BPTT), to train rate RNNs to produce target signals associated with a specific task [19].

The units in a rate RNN are sparsely connected via W^{rate} and receive a task-specific input signal through weights (W_{in}) drawn from a normal distribution with zero mean and unit variance. The network output (o^{rate}) is then computed using a set of linear readout weights:

$$o^{rate}(t) = W_{out}^{rate} \cdot \boldsymbol{r}^{rate}(t) \tag{4}$$

where W_{out}^{rate} is the readout weights and $\boldsymbol{r}^{rate}(t)$ is the firing rate estimates from all the units in the network at time t. The recurrent weight matrix (W^{rate}), the readout weights (W_{out}^{rate}), and the synaptic decay time constants ($\boldsymbol{\tau}$) are optimized during training, while the input weight matrix (W_{in}) stays fixed (see Methods).

Once the rate network model is trained, the three sets of the weight matrices (W_{in}, W^{rate}, and W^{rate}_{out}) along with the tuned synaptic time constants ($\boldsymbol{\tau}$) are transferred to a network of LIF spiking units. The spiking RNN is initialized to have the same topology as the rate RNN. The input weight matrix and the synaptic time constants are simply transferred without any modification, but the recurrent connectivity and the readout weights need to be scaled by a constant factor (λ) in order to account for the difference in the firing rate scales between the rate model and the spiking model (see Methods).

In Fig. 1, we trained a small continuous rate network of $N = 200$ units (162 excitatory and 38 inhibitory units) on a simple task modeled after a Go-NoGo task to demonstrate our framework. Using BPTT, the network was trained to produce a positive mean population activity approaching +1 after a brief input pulse (Fig. 1A). For a trial without an input pulse, the network was trained to maintain its output close to zero. The trained rate RNN performed the task correctly on all test trials with a mean synaptic decay time constant of 28.2 ± 9.4 ms (Fig. 1B and 1C).

Next, we directly transferred the input weight matrix (W_{in}) and the optimized synaptic time constants to a network of LIF units. The connectivity matrix (W^{rate}) and the readout weights (W^{rate}_{out}) were scaled by a factor of $\lambda = 0.02$ (see Methods on how it was computed) and transferred to the spiking network. When the weights were not scaled (i.e. $\lambda = 1$), the spiking network could not perform the task (output signals for both Go and NoGo trials converged) and produced largely fluctuating signals (Fig. 1D top). With an appropriate value for λ, the LIF network performed the task with the same accuracy as the rate network (Fig. 1D bottom), and the LIF units fired at rates similar to the "rates" of the continuous network units (Fig. 1E).

Our framework also allows seamless integration of additional functional connectivity constraints. For example, a common cortical microcircuitry motif where somatostatin-expressing interneurons inhibit both pyramidal and parvalbumin-positive neurons can be easily implemented in our framework (see Methods and Supplementary Fig. 1). In addition, Dale's principle is not required for our framework (Supplementary Fig. 2).

Excitatory and inhibitory dynamics during autonomous oscillatory network activities. Next, we tested our framework on an autonomous oscillation task where a rate RNN was first trained to produce a periodic output signal in the absence of external input signals (Fig. 2A). The target signal used to train the rate network was a simple 1 Hz sine wave. The rate network composed of 98 excitatory units and 102 inhibitory units was successfully trained to produce the target sinusoidal signal autonomously (Fig. 2B top). An LIF model was endowed with the same

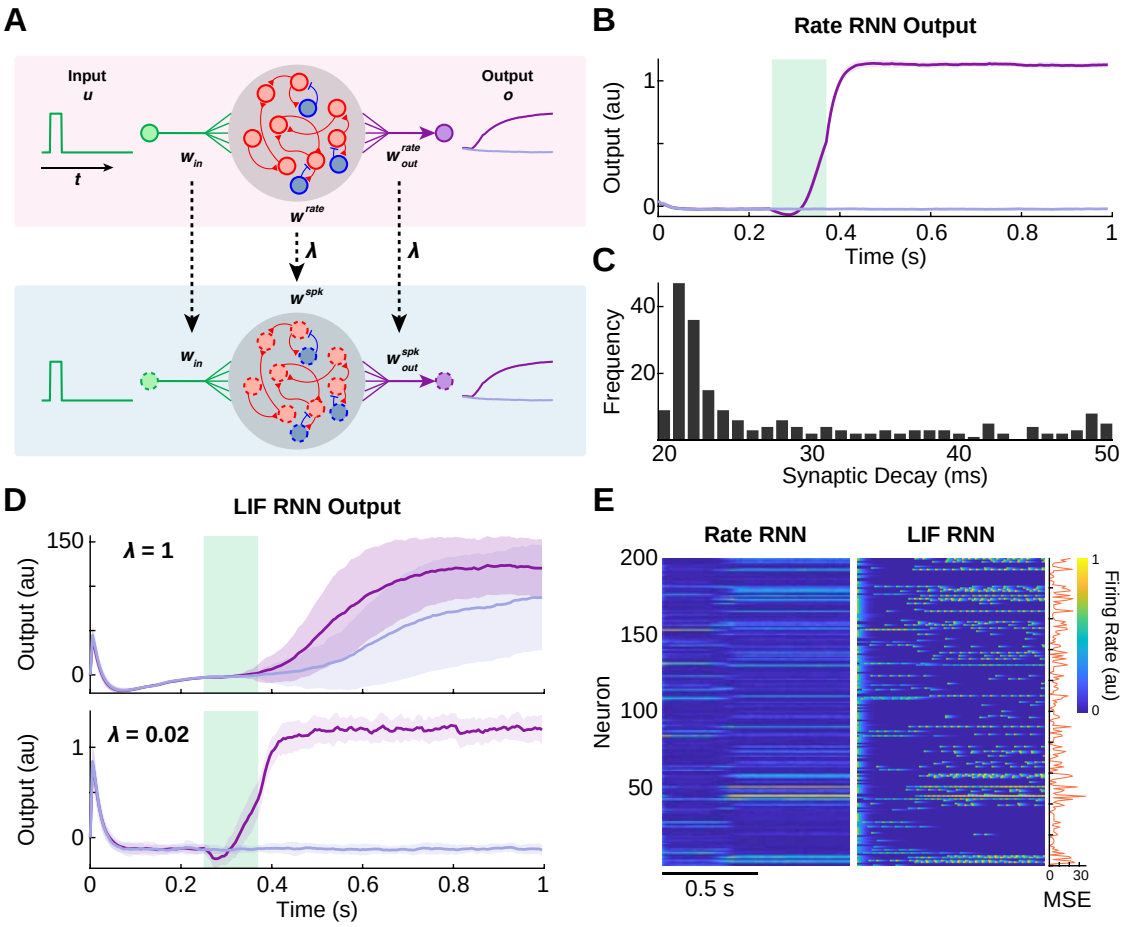

Fig. 1 | Transfer learning from rate RNNs to spiking RNNs. A. Schematic diagram illustrating transfer learning from a continuous rate RNN model (top) to a spiking RNN model (bottom). A rate network with excitatory (red circles with a solid outline) and inhibitory (blue circles with a solid outline) units was trained to perform a Go-NoGo task. The optimized synaptic decay time constants ($\boldsymbol{\tau}$) along with the weight parameters (W_{in}, W^{rate}, and W^{rate}_{out}) were transferred to a spiking network with LIF units (red and blue circles with a dashed outline). The connectivity and the readout weights were scaled by a constant factor, λ. **B.** Trained rate RNN performance on the Go-NoGo task. The mean ± SD output signals from 100 Go trials (dark purple) and from 100 NoGo trials (light purple) are shown. The green box represents the input stimulus given for the Go trials. **C.** Distribution of the tuned synaptic decay time constants ($\boldsymbol{\tau}$). Mean ± SD, 28.2 ± 9.4 ms. **D.** LIF RNN performance on the Go-NoGo task without scaling ($\lambda = 1$; top) and with appropriate scaling ($\lambda = 0.02$; bottom). Mean ± SD over 100 Go and 100 NoGo trials. **E.** Comparison of the time-varying rates from the trained rate network (left) and the spiking model (right). A single Go trial was used to extract the rates from the rate RNN and the firing rates scaled by λ from the LIF network. The mean squared error (MSE) was computed to quantify the difference in firing rate between the two network models (orange line).

142 dynamical properties after the weight matrices and the synaptic time constants were transferred.

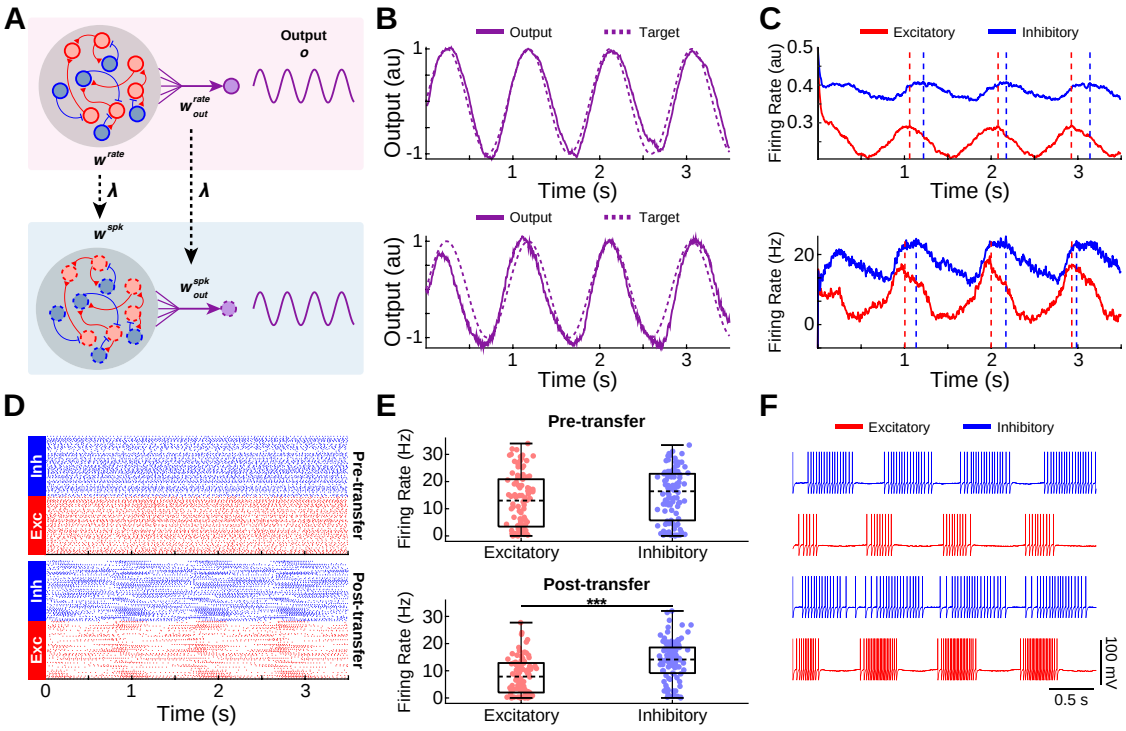

Fig. 2 | Autonomous oscillation task. A. Schematic outlining the autonomous oscillation task. A rate RNN was trained to produce a slow sinusoidal output signal without any external input signals (top). A network of LIF units was constructed to perform the same task using the transfer learning method (bottom). **B.** Comparison of the output signals from the trained rate model (top) and the LIF model (bottom). **C.** Comparison of the average excitatory (red) and inhibitory (blue) firing rates from the rate model (top) and the LIF model (bottom). The red and blue vertical dashed lines represent the local maxima for the mean excitatory and inhibitory firing rates, respectively. **D.** Raster plots of spiking activities from the LIF network before transfer learning (top) and after transfer learning (bottom). **E.** Average firing rates from the excitatory and inhibitory units from the LIF network before transfer learning (top) and after transfer learning (bottom). The average firing rates between the two populations did not differ significantly before transfer learning. The asterisks (***) indicate a significant difference at $p < 0.001$ (two-tailed Student's t-test). Box plot central lines, median; bottom and top edges, lower and upper quartiles. **F.** Example membrane voltage tracings from two excitatory and two inhibitory units in the LIF network model (post-transfer learning).

The spiking network produced and maintained the same sinusoidal target signal autonomously (Fig. 2B bottom).

In both networks (rate and LIF networks), inhibition closely tracked excitation with a temporal delay, as revealed in the average rate signals of the two populations in each network (Fig. 2C). These findings suggest that both networks operate in an excitation-inhibition balanced regime and are aligned with the previous experimental results in which excitation followed by inhibition was

shown to provide a narrow window for sensory integration [20–23].

Next, we investigated the effect of transfer learning on the network spiking activity. Prior to transfer of the weights, the spiking units connected via the initial random, sparse (10% sparsity) weights fired at high rates continuously (Fig. 2D top and 2E top). After transfer learning, the units fired in a more structured manner with the excitatory units and the inhibitory units firing on average at 8.2 Hz and 13.7 Hz, respectively (Fig. 2D bottom and 2E bottom). The post-transfer learning LIF units also exhibited diverse patterns of firing activities where many excitatory units fired during the windows provided by delayed inhibition (Fig. 2F).

Our framework can be also used to construct LIF networks to produce sinusoidal signals with faster frequencies and more complex signals (Supplementary Fig. 3).

Rate dynamics and mixed selectivity during context-dependent input integration. The tasks considered so far did not require complex cognitive computations. In this section, we consider a more complex task modeled after the context-dependent sensory integration task employed by Mante et al. [4]. Briefly, Mante et al. [4] trained rhesus monkeys to integrate inputs from one sensory modality (dominant color or dominant motion of randomly moving dots) while ignoring inputs from the other modality. A contextual cue was also given to instruct the monkeys which sensory modality they should attend to. The task required the monkeys to utilize flexible computations as the same modality can be either relevant or irrelevant depending on the contextual cue. Previous works have successfully trained continuous rate RNNs to perform a simplified version of the task and replicated the neural dynamics present in the experimental data [4, 10, 12]. Using our framework, we constructed the first spiking RNN model to our knowledge that can perform the task and capture the dynamics observed in the experimental data.

For the task paradigm, we adopted a similar design as the one used by the previous modeling studies [4, 10, 12]. A network of recurrently connected units received two streams of noisy input signals along with a constant-valued signal that encoded the contextual cue (Fig. 3A; see Methods). To simulate a noisy sensory input signal, a random Gaussian time-series signal with zero mean and unit variance was first generated. Each input signal was then shifted by a positive or negative constant ("offset") to encode evidence toward the (+) or (-) choice, respectively (see Methods). Therefore, the offset value determined how much evidence for the specific choice was represented in the noisy input signal. The network was trained to produce an output signal approaching +1 (or -1) if the cued input signal had a positive (or negative) mean (Fig. 3A). For example, if the cued input signal was generated using a positive offset value, then the network should produce an

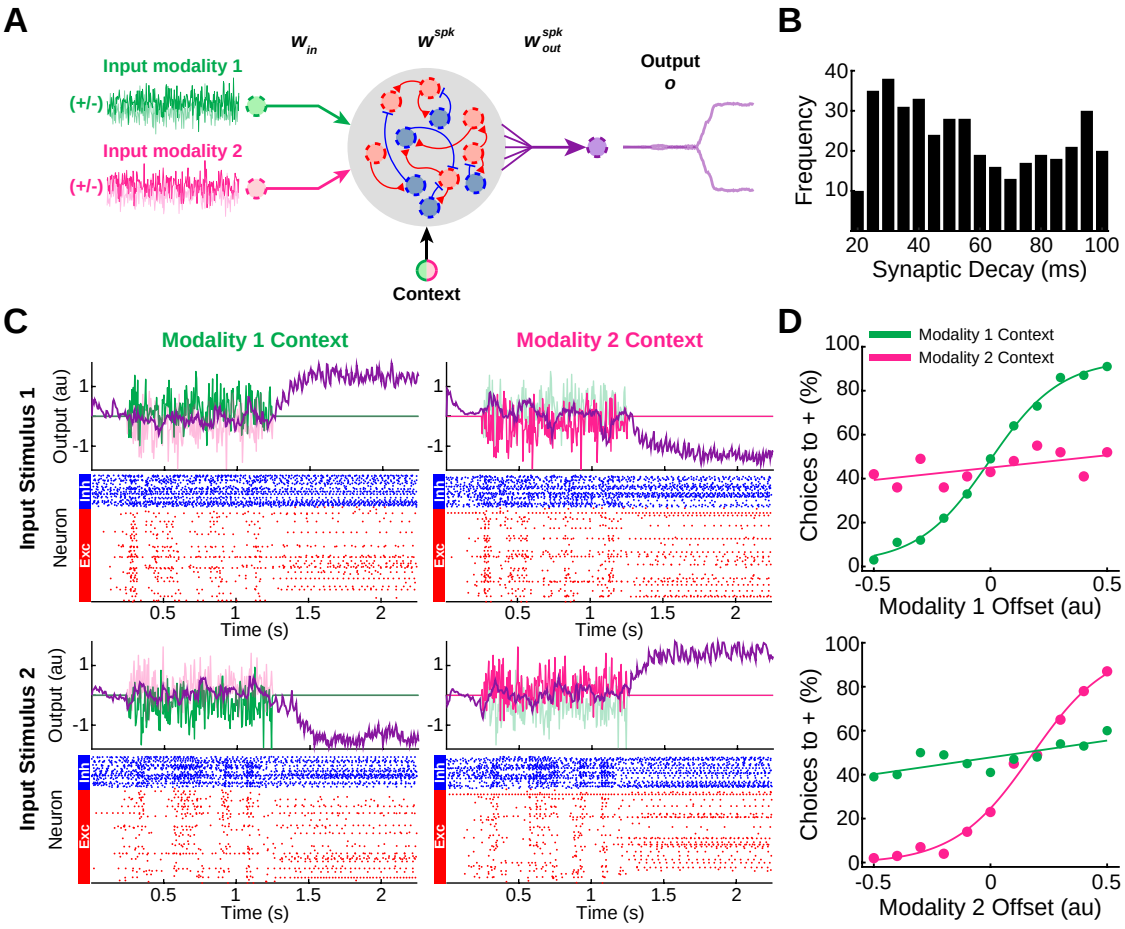

Fig. 3 | Biologically realistic spiking network performing a context-dependent input integration task. A. Diagram illustrating the task paradigm modeled after the context-dependent task used by Mante et al. [4]. Two streams of noisy input signals (green and magenta lines) along with a context signal were delivered to the LIF network. The network was trained to integrate and determine if the mean of the cued input signal (i.e. cued offset value) was positive ("+" choice) or negative ("-" choice). **B.** Distribution of the optimized synaptic decay time constants ($\boldsymbol{\tau}$). Mean ± SD, 51.0 ± 25.0 ms. **C.** Example output responses and spike raster plots from the LIF network model for two different input stimuli (rows) and two contexts (columns). The network successfully integrated and responded to the cued modality (dark green and dark magenta lines). The noisy input signals are scaled by 0.5 vertically for better visualization of the network responses (purple lines). **D.** Psychometric curves showing the percentage of trials where the LIF network indicated "+" choice as a function of the modality 1 offset values (top) and modality 2 offset values (bottom).

output that approaches +1 regardless of the mean of the irrelevant input signal.

A network of 400 rate units (299 excitatory and 101 inhibitory units) was successfully trained to perform the task. Unlike the simple Go-NoGo task (Fig. 1C), the integration task required

more units with slow synaptic decay time constants, as evidenced by the bimodal distribution of the optimized time constants (Fig. 3B). This is consistent with recent experimental results where neurons with long timescales played an important role in integration and processing of accumulated evidence [24].

Next, the dynamics of the trained rate RNN were transferred to a network of LIF units. The spiking network performed the same task equally well (Fig. 3C). The psychometric curves of the spiking network further confirmed that the network could indeed integrate the relevant input modality, while successfully ignoring the irrelevant modality (Fig. 3D). In other words, the network behavior was strongly dependent on the cued modality offset values, while the uncued modality offset values did not affect the network behavior. We also transferred the rate network dynamics to a network of QIF units and obtained similar results (Supplementary Fig. 4).

After verifying that the spiking RNN could perform the task reliably, we investigated whether the population dynamics underlying the spiking network were similar to the dynamics observed in the group of neurons recorded by Mante et al. [4]. Consistent with the experimental results, individual LIF units displayed mixed representation of the four task variables (modality 1, modality 2, network choice, and context; Fig. 4A). To further characterize the mixed representation, we performed the multivariate linear regression and the targeted dimensionality reduction techniques developed by Mante et al. [4] (see Methods). The de-noised regression correlation coefficients computed across all the units in the network revealed that the individual units encoded multiple task variables (Fig. 4B). More importantly, the network did not contain any distinct subgroups that specialized to represent the individual task variables, as indicated by the absence of clusters in the coefficients in Fig. 4B. This was also the case for the network of neurons recorded from the monkeys performing the task [4]. The targeted dimensionality reduction method applied to the binned spike data from the LIF network displayed the characteristic line attractor dynamics in the state space (Fig. 4C). The population responses formed arc-like trajectories along the choice axis, and the amplitude values of the trajectories were correlated with the offset values (compare Fig. 4C to Fig. 2 from Mante et al. [4]).

Working memory and neuronal synchronization modulated by inhibitory units. While the spiking network that we constructed in the previous section reproduced the rate dynamics manifested by recorded neurons, our spiking models provide additional information that can be explored. For instance, the spiking nature and the separate excitatory and inhibitory populations of our RNNs allow us to investigate the functional role of inhibitory units in governing local neu-

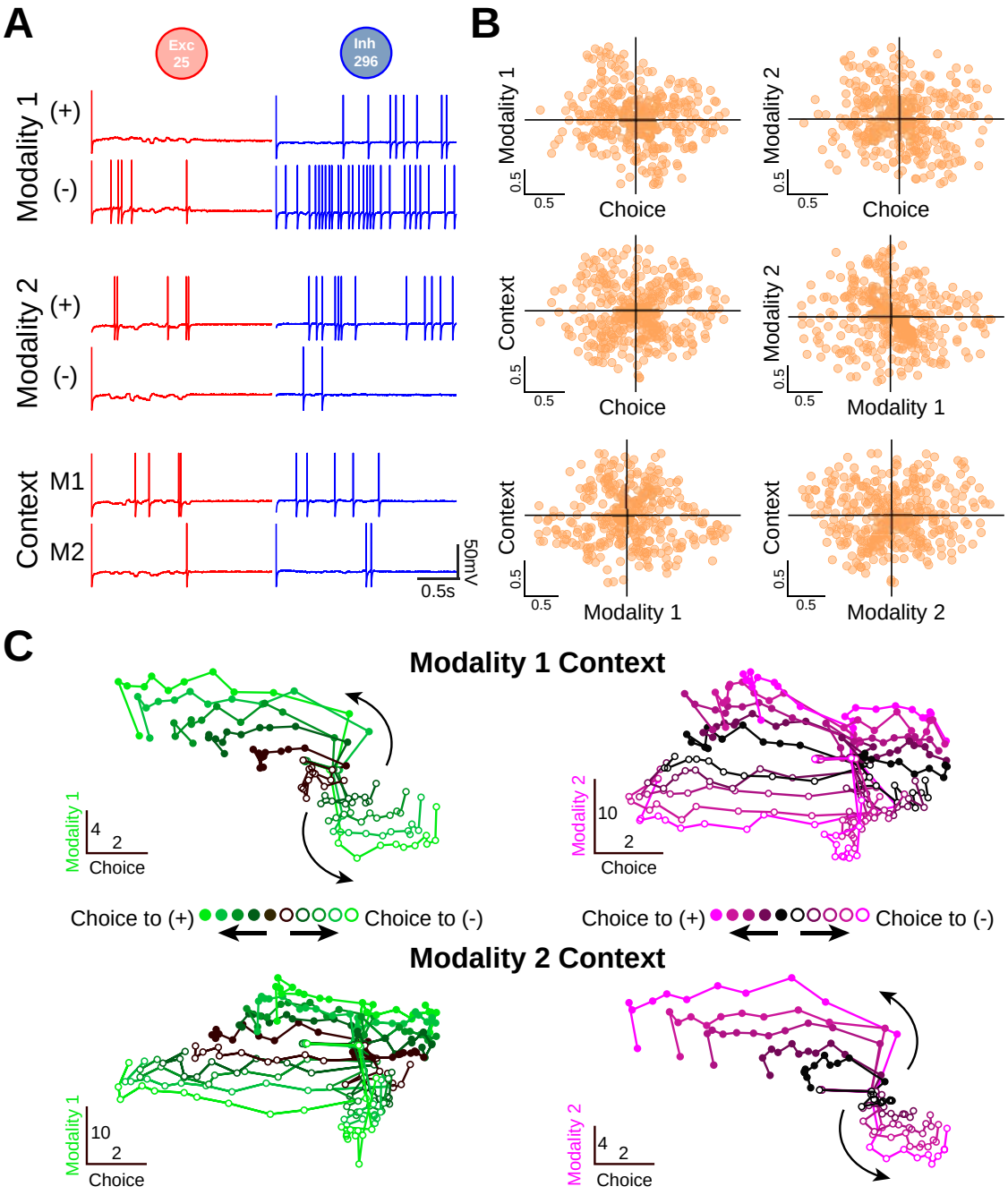

Fig. 4 | Caption on next page.

ronal synchrony and network behavior. Previous studies have shown that inhibitory interneurons, especially parvalbumin (PV)-positive interneurons, are critical for regulating neuronal synchrony [20, 23, 25]. Dysfunction and disruption of inhibitory signaling mediated by PV interneurons have been strongly associated with network dysfunctions along with behavioral impairment relevant to various neuropsychiatric disorders [26, 27]. Consistent with these findings, a recent study using a

Fig. 4 | (Previous page) **The LIF network model employs mixed representations of the task variables. A.** Mixed representation of the task variables at the level of single units. An excitatory unit (red) and an inhibitory unit (blue) with mixed representation of three task variables (modality 1, modality 2, and context) are shown as examples. The excitatory neuron preferred modality 1 input signals with negative offset values, modality 2 signals with positive offset values, and modality 1 context (left column). The inhibitory neuron also exhibited similar biases (right column). **B.** De-noised regression coefficients from all the units in the network. The coefficients were obtained from the multivariate linear regression analysis used by Mante et al. [4]. **C.** Average population responses projected to a low dimensional state space. The targeted dimensionality reduction technique (developed by Mante et al. [4]) was used to project the population activities to the state space spanned by the task-related axes (which were obtained using the regression analysis mentioned in **B**). For the modality 1 context (top row), the population responses from the trials with various modality 1 offset values were projected to the choice and modality 1 axes (left). The same trials were sorted by the irrelevant modality (modality 2) and shown on the right. Similar conventions used for the modality 2 context (bottom row). The offset magnitude (i.e. amount of evidence toward "+" or "-" choice) increases from dark to light. Filled and empty circles correspond to "+" choice and "-" choice trials, respectively.

mouse model of schizophrenia showed that decreased activity of PV neurons led to desynchronization of pyramidal neurons and working memory deficits often seen in schizophrenia [28].

Motivated by this recent study, we constructed an excitatory-inhibitory spiking RNN to perform a task that required working memory and employed a spike-based synchrony measure to characterize how inhibitory signaling contributes to precise neuronal synchrony and working memory maintenance. We used a temporal exclusive-OR (XOR) task paradigm, where each trial began with two sequential stimuli separated by a brief delay period (Fig. 5A). A network of 200 LIF units (158 excitatory and 42 inhibitory units) with an average synaptic decay of 44.4 $\pm$ 28.0 ms could successfully perform the task (Fig. 5B). During a stimulus period, the input signal was held at either -1 or +1. If the two sequential stimuli had the same sign (+1/+1 or -1/-1), then the network was trained to produce an output signal approaching +1 (Fig. 5C top). If the stimuli had different signs (+1/-1 or -1/+1), the output of the network approached -1 (Fig. 5C bottom). This task is a classical example of working memory tasks as it requires the network to briefly retain and recall the first stimulus identity in order to make a correct decision during the response window.

The neural population trajectories projected to a low-dimensional space discovered by principal component analysis (PCA) revealed how the spiking network performed the working memory task (Fig. 5D; Supplementary Fig. 5; see Methods). During the fixation period before the presentation

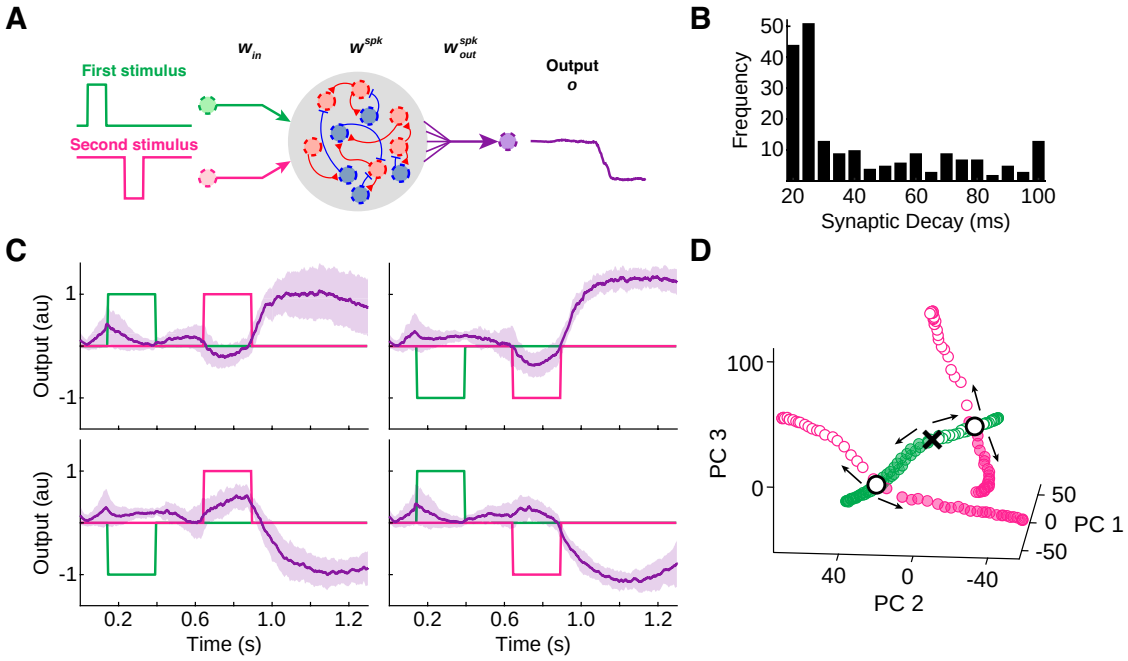

Fig. 5 | LIF network model performing a temporal XOR task. A. Schematic diagram showing the temporal XOR task paradigm. Two sequential stimuli (250 ms in duration each) separated by a 250 ms delay were given to the LIF network model. If the two stimuli did not match (shown here), the network output approached -1. For a matching case (+/+ or -/-), the output approached +1. **B.** Distribution of the optimized synaptic decay time constants (τ). Mean ± SD, 44.4 ± 28.0 ms. **C.** Network performance for each trial condition. Mean ± SD across 50 trials for each condition (dark purple). **D.** Low dimensional neural trajectory evolution during the XOR task. PCA was applied to the neural responses from the onset of the first stimulus period to the end of the second stimulus window. Black cross, first stimulus onset; black circles, second stimulus onset; green filled circles, "+1" first stimulus; green empty circles, "-1" first stimulus; magenta filled circles, "+1" second stimulus; magenta empty circles, "-1" second stimulus. See Supplementary Fig. 5 for different views.

of the first stimulus, all four trajectories corresponding to the four trial types stayed together as expected (data not shown). Then the trajectories diverged based on the identity of the first stimulus forming two stable "tunnels" traveling in the opposite directions: one for the "+1" first stimulus and the other for the "-1" first stimulus (green filled and empty circles in Fig. 5D). During the delay period, the dynamical landscape was maintained, and the two tunnels stayed well-separated. During the second stimulus period, these two tunnels bifurcated again to form four trajectories in a manner that allowed the network to preserve all three task variables (first stimulus, second stimulus, and response). The first principal component (PC) encoded the information related to the first stimulus: the trajectories with the "+1" first stimulus resided in the negative PC 1 region,

while the trajectories corresponding to the "-1" first stimulus stayed in the positive PC 1 area. The identity of the second stimulus was represented by the third PC, and the second PC encoded the network response variable. Therefore, the low-dimensional neural response trajectories revealed how short-term memories were represented in the spiking network performing the temporal XOR task.

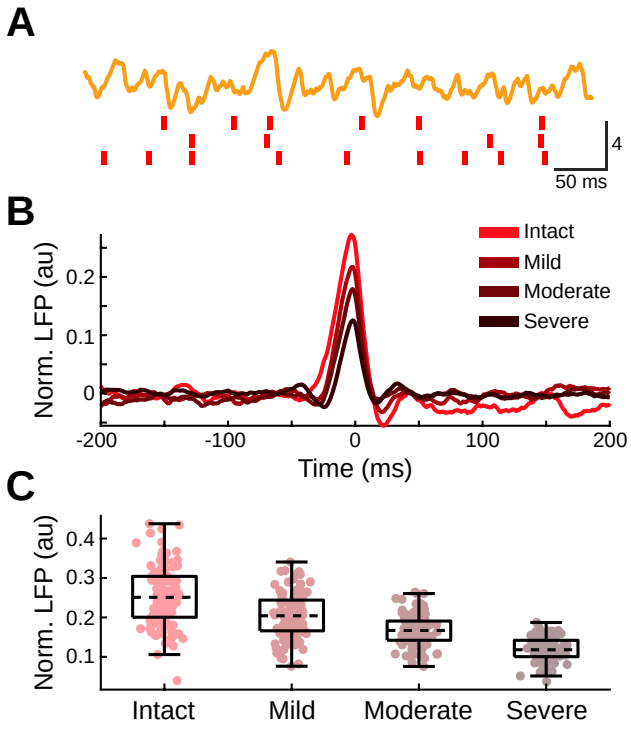

Fig. 6 | Inhibitory units tightly regulate network synchrony. A. Example LFP proxy signal (orange) with spikes from three randomly selected excitatory units (red). Spontaneous activities were extracted from the LIF network model constructed to perform the XOR task. The LFP signal was normalized using z-score transformation. **B.** STAs computed from the LIF network with different degrees of inhibitory signaling impairment. For each impairment condition, the average STA time-series over 100 trials is shown. Fraction of suppressed inhibitory units increases from light to dark. **C.** The STA amplitude values at spike times ($t = 0$) from all 100 trials are shown for each condition. Intact, no suppression of inhibitory units; mild, 24% inhibitory units suppressed; moderate, 48% inhibitory units suppressed; severe, 71% inhibitory units suppressed. Box plot central lines, median; bottom and top edges, lower and upper quartiles. All pairwise Student's t-tests were significant at $p < 0.001$.

Finally, we studied how attenuated inhibitory signaling altered the neuronal synchrony and the network dynamics. In order to model diminished PV interneuron-mediated signaling transmission, we suppressed random subpopulations of the inhibitory units in the trained model by delivering strong hyperpolarizing currents throughout the trial. The size of the subpopulations was varied

to simulate different degrees of inhibitory signaling attenuation. We considered three levels of inhibitory unit suppression: weak (24% of inhibitory units suppressed), moderate (48% inhibitory units suppressed), and severe (71% inhibitory units suppressed). We first characterized the spontaneous (no input stimuli) excitatory population synchrony for each of the three levels by computing spike-triggered average (STA) of local field potential (LFP) proxy signals (see Methods) [29]. If neurons fire in a synchronized manner with respect to the local population activity (as estimated by the LFP), the STA signal shows a prominent peak around each spike time [29, 30]. Here, the LFP signals were modeled as the average synaptic inputs into the excitatory units and normalized to the z-scores for each trial (Fig. 6A; see Methods). For the intact network, the excitatory units fired more often during synchronous excitatory synaptic input activities leading to a large positive peak in the average STA signal (Fig. 6B and 6C). As the fraction of the suppressed inhibitory units increased, the STA peak amplitude decreased indicating desynchronization of the excitatory units (Fig. 6B and 6C). In these impaired network models, the excitatory units fired more spontaneously (Supplementary Fig. 6). The increased spontaneous excitatory activities and disrupted network synchrony are in line with the recent findings where hypofunctioning PV interneurons led to desynchronized assemblies of pyramidal cells with increased spontaneous activities [28].

To assess the severity of working memory impairment in each of the models, we focused on encoding and maintenance of the first stimulus identity by the excitatory population using a cross-temporal pattern analysis method, which previous studies have successfully employed to probe dynamic working memory coding [12, 24, 31–33]. For each inhibitory suppression level, we obtained excitatory population responses for each trial type. Then these responses were grouped by the first stimulus identity only and were split into a training and a test dataset. A linear, maximum-correlation classifier was then trained to decode the identity of the first stimulus at each time point of the trial (see Methods). For the intact model, the excitatory units encoded the first stimulus robustly across the entire trial duration (Fig. 7 top left). This is consistent with the low dimensional trajectories shown in Fig. 5D along with the previous experimental findings where stable representations of stimuli persisted long after the presentation of the stimuli [24, 33]. The population coding of the first stimulus was disrupted as the inhibitory units were suppressed (Fig. 7). In the most severe case (71% of the inhibitory units suppressed), the identity of the first stimulus could only be decoded during the first stimulus period (Fig. 7 bottom right), suggesting that the loss of inhibitory signaling disrupted the stable working memory representation of the first stimulus identity. The neural responses projected to the first two PCs confirmed that the

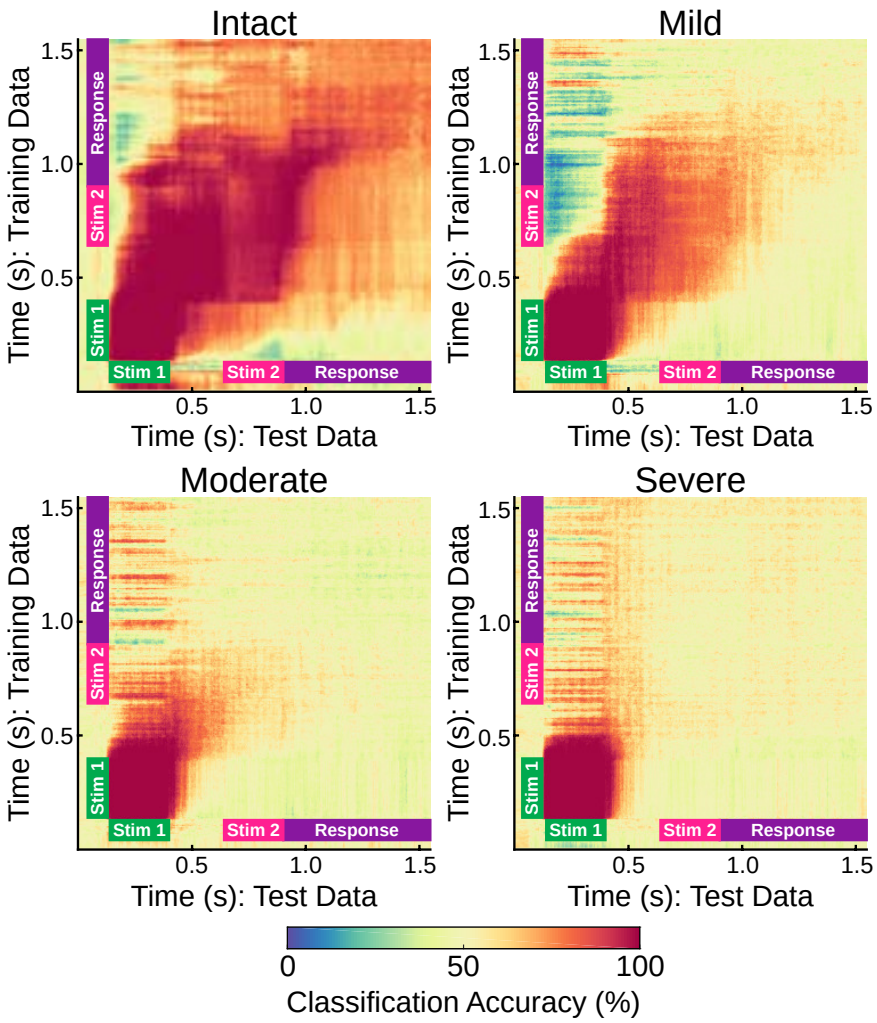

Fig. 7 | Suppression of the inhibitory units impairs working memory. Cross-temporal discriminability analysis of the first stimulus identity revealed reliable encoding that generalized across the trial (first stimulus, delay, second stimulus, and response epochs) for the intact network (top left). For a weak impairment condition (24% inhibitory units suppressed), the first stimulus could be decoded robustly until the beginning of the response epoch (top right). As the fraction of the suppressed inhibitory units increased further, the reliability of the stimulus encoding dropped markedly (bottom row).

memory of the first stimulus identity was indeed abolished in the moderate and severe models (Supplementary Fig. 7). On the other hand, suppressing a significant portion of the excitatory units (50% of the excitatory units suppressed) did not produce network desynchronization and working memory deficits (Supplementary Fig. 8). These findings indicate that the inhibitory units in our spiking model are critical for controlling network dynamics and carrying out important computations.

Discussion

In the current study, we presented a simple framework that harnesses the dynamics of trained continuous rate network models to produce functional spiking RNN models. This framework can flexibly incorporate functional connectivity constraints and heterogeneous synaptic time constants. The spiking RNNs were constructed to perform various cognitive task paradigms, including context-dependent input integration and working memory tasks; rate- and spike-based measures illuminated the neural dynamics underlying cognitive processes.

The type of approach used in this study (i.e. conversion of a rate network to a spiking network) has been previously employed in neuromorphic engineering to construct power-efficient deep spiking networks [34–36]. These studies mainly employed feedforward multi-layer networks or convolutional neural networks aimed to accurately classify input signals or images without placing too much emphasis on biophysical limitations. The overarching goal in these studies was to maximize task performance while minimizing power consumption and computational cost. On the other hand, the main aim of the present study was to construct spiking recurrent network models that abide by important biological constraints in order to relate emerging mechanisms and dynamics to experimentally observed findings. To this end, we have carefully designed our continuous rate RNNs to include several biological features. These include (1) non-negative firing rates (imposed by the sigmoid transfer function), (2) sparse connectivity that respects Dale's principle, and (3) heterogeneous synaptic decay time constants. Incorporating these biologically motivated details into our rate network model enabled us to utilize transfer learning to create a functional spiking model.

Recent studies have proposed methods that built on the FORCE method to train spiking RNNs [5, 15–17]. Conceptually, our work is most similar to the work by DePasquale et al. [16]. The method developed by DePasquale et al. [16] also relies on mapping a trained continuous-variable rate RNN to a spiking RNN model. However, the rate RNN model used in their study was designed to provide dynamically rich auxiliary basis functions meant to be distributed to overlapping populations of spiking units. Due to this reason, the relationship between their rate and spiking models is rather complex, and it is not straightforward to impose functional connectivity constraints on their spiking RNN model. An additional procedure was introduced to implement Dale's principle, but this led to more fragile spiking networks with considerably increased training time [16]. The one-to-one mapping between rate and spiking networks employed in our method solved these problems without sacrificing network stability and computational cost: biophysical constraints that we

wanted to incorporate into our spiking model were implemented in our rate network model first and then transferred to the spiking model.

The recurrent weight parametrization method proposed by Song et al. [10] to train continuous rate RNNs that satisfy Dale's principle was also employed in our study to constrain our rate models. Surprisingly, this constraint was transferable to spiking RNNs to produce separate excitatory and inhibitory populations. This biological feature allowed us to characterize the functional role of inhibitory units in governing neuronal synchrony and network dynamics (Fig. 6 and Fig. 7). Furthermore, other connectivity motifs motivated by biology can be enforced and transferred using our framework (Supplementary Fig. 1).

In addition to imposing specific connectivity patterns, we have also optimized synaptic decay time constants. Previous studies have investigated homogeneous models where all the units in a network shared the same time constant [16–18, 37]. However, Kim and Chow [5] underscored the importance of synaptic time scales in training spiking recurrent networks. If all the units in a network have slow synaptic time constants, they cannot track fast changes present in the target dynamics. On the other hand, if the synaptic time scale is too fast, the ability for spikes to encode continuous signals deteriorates resulting in a large "sampling" error. Instead of having all the units operate in the same time scale, we have included the synaptic time constants as another set of model parameters to be optimized via backpropagation. This modification allowed our spiking networks to exploit units with a diverse range of synaptic time scales. Diversity of neuronal and synaptic properties is found throughout the brain and may be a general principle.

Since our framework involves rate RNNs that operate in a rate coding scheme, the spiking RNNs that our framework produces also employ rate coding by nature. Previous studies have shown that spike-coding can improve spiking efficiency and enhance network stability [15, 21, 38], and it will be important to build on our current method to include spike-coding schemes. In addition, our framework does not model nonlinear dendritic processes which have been shown to play a significant role in efficient input integration and flexible information processing [17, 39, 40]. Incorporating nonlinear dendritic processes into our platform using the method proposed by Thalmeier et al. [17] will be an interesting next step to further investigate the role of dendritic computation in information processing. Lastly, the backpropagation method utilized in our framework to train rate RNNs in a supervised manner is not biologically plausible. However, previous studies have validated and uncovered neural mechanisms observed in experimental settings using RNN models trained with backpropagation [4, 10, 37]. Thus, a network model may be biologically plausible,

and improve our understanding of neural systems, even if it was constructed using non-biological means. Testing if our framework can be generalized to support more biologically realistic training methods, such as reinforcement learning methods, is also an important future direction.

In summary, we provide an easy-to-use platform that converts a continuous recurrent network model to a more biologically realistic, spiking model. The framework along with the findings presented in this study will be valuable for future experimental and theoretical studies aimed at uncovering neural computations underlying cognition.

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Author contributions

R.K. and T.J.S. designed the study and wrote the manuscript. R.K. and Y.L. performed model analyses and simulations.

Declaration of interests

The authors declare no competing interests.

Methods

Continuous rate network structure. The continuous rate RNN model contains N units recurrently connected to one another. The dynamics of the model is governed by

$$\boldsymbol{\tau}\frac{d\boldsymbol{x}}{dt} = -\boldsymbol{x} + W^{rate}\boldsymbol{r}^{rate} + \boldsymbol{I}_{ext} \tag{5}$$

where $\boldsymbol{\tau} \in \mathbb{R}^{1\times N}$ corresponds to the synaptic decay time constants for the N units in the network (see **Training details** on how these are initialized and optimized), $\boldsymbol{x} \in \mathbb{R}^{1\times N}$ is the synaptic current variable, $W^{rate} \in \mathbb{R}^{N\times N}$ is the synaptic connectivity matrix, and $\boldsymbol{r}^{rate} \in \mathbb{R}^{1\times N}$ is the output of the units. The output of each unit, which can be interpreted as the firing rate estimate, is obtained by applying a nonlinear transfer function to the synaptic current variable ($\boldsymbol{x}$) elementwise:

$$\boldsymbol{r}^{rate} = \phi(\boldsymbol{x})$$

We use a standard logistic sigmoid function for the transfer function to constrain the firing rates to be non-negative:

$$\phi(\boldsymbol{x}) = \frac{1}{1+\exp(-\boldsymbol{x})} \tag{6}$$

The connectivity weight matrix (W^{rate}) is initialized as a random, sparse matrix drawn from a normal distribution with zero mean and a standard deviation of $1.5/\sqrt{N \cdot P_c}$ where $P_c = 0.10$ is the initial connectivity probability.

The external currents ($\boldsymbol{I}_{ext}$) include task-specific input stimulus signals (see **Implementation of computational tasks and figure details**) along with a Gaussian white noise variable:

$$\boldsymbol{I}_{ext} = W_{in}\boldsymbol{u} + \boldsymbol{\mathcal{N}}(0, 0.01)$$

where the time-varying stimulus signals ($\boldsymbol{u} \in \mathbb{R}^{N_{in}\times 1}$) are fed to the network via $W_{in} \in \mathbb{R}^{N\times N_{in}}$, a Gaussian random matrix with zero mean and unit variance. N_{in} corresponds to the number of input signals associated with a specific task, and $\boldsymbol{\mathcal{N}}(0, 0.01) \in \mathbb{R}^{N\times 1}$ represents a Gaussian random noise with zero mean and variance of 0.01.

The output of the rate RNN at time t is computed as a linear readout of the population activity:

$$o^{rate}(t) = W_{out}^{rate}\boldsymbol{r}^{rate}(t)$$

where $W_{out}^{rate} \in \mathbb{R}^{1\times N}$ refers to the readout weights.

Eq. (5) is discretized using the first-order Euler approximation method:

$$\boldsymbol{x}_t = \left(1 - \frac{\Delta t}{\boldsymbol{\tau}}\right) \boldsymbol{x}_{t-1} + \frac{\Delta t}{\boldsymbol{\tau}}(W^{rate}\boldsymbol{r}^{rate}_{t-1} + W_{in}\boldsymbol{u}_{t-1}) + \boldsymbol{\mathcal{N}}(0, 0.01)$$

where $\Delta t = 5$ ms is the discretization time step size used throughout this study.

Spiking network structure. For our spiking RNN model, we considered a network of leaky integrate-and-fire (LIF) units governed by

$$\tau_m \frac{d\boldsymbol{v}}{dt} = -\boldsymbol{v} + W^{spk}\boldsymbol{r}^{spk} + \boldsymbol{I}_{ext} \tag{7}$$

In the above equation, $\tau_m = 10$ ms is the membrane time constant shared by all the LIF units, $\boldsymbol{v} \in \mathbb{R}^{1\times N}$ is the membrane voltage variable, $W^{spk} \in \mathbb{R}^{N\times N}$ is the recurrent connectivity matrix, and $\boldsymbol{r}^{spk} \in \mathbb{R}^{1\times N}$ represents the spike trains filtered by a synaptic filter. Throughout the study, the double exponential synaptic filter was used to filter the presynaptic spike trains:

$$\frac{dr_i^{spk}}{dt} = -\frac{r_i^{spk}}{\tau_i} + s_i$$
$$\frac{ds_i}{dt} = -\frac{s_i}{\tau_r} + \frac{1}{\tau_r \tau_i}\sum_{t_i^k < t} \delta(t - t_i^k)$$

where $\tau_r = 2$ ms and τ_i refer to the synaptic rise time and the synaptic decay time for unit i, respectively. The synaptic decay time constant values ($\tau_i \in \boldsymbol{\tau}$) are trained and transferred to our LIF RNN model (see **Training details**). The spike train produced by unit i is represented as a sum of Direc δ functions, and t_i^k refers to the k-th spike emitted by unit i.

The external current input ($\boldsymbol{I}_{ext}$) is similar to the one used in our continuous model (see **Continuous rate network structure**). The only difference is the addition of a constant background current set near the action potential threshold (see below).

The output of our spiking model at time t is given by

$$o^{spk}(t) = W^{spk}_{out}\boldsymbol{r}^{spk}(t)$$

Other LIF model parameters were set to the values used by Nicola and Clopath [18]. These include the action potential threshold (-40 mV), the reset potential (-65 mV), the absolute refractory period (2 ms), and the constant bias current (-40 pA). The parameter values for the LIF and the quadratic integrate-and-fire (QIF) models are listed in Supplementary Table 1.

Training details. In this study, we only considered supervised learning tasks. A task-specific target signal ($\boldsymbol{z}$) is used along with the rate RNN output ($\boldsymbol{o}^{rate}$) to define the loss function ($\mathcal{L}$), which our rate RNN model is trained to minimize. Throughout the study, we used the root mean squared error (RMSE) defined as

$$\mathcal{L} = \sqrt{\left(\sum_{t=1}^{T}(z(t) - o^{rate}(t))^2\right)} \tag{8}$$

where T is the total number of time points in a single trial.

In order to train the rate model to minimize the above loss function (Eq. 8), we employed ADaptive Moment Estimation (ADAM) stochastic gradient descent algorithm. The learning rate was set to 0.01, and the TensorFlow default values were used for the first and second moment decay rates. The gradient descent method was used to optimize the following parameters in the rate model: synaptic decay time constants ($\boldsymbol{\tau}$), recurrent connectivity matrix (W^{rate}), and readout weights (W^{rate}_{out}).

Here we describe the method to train synaptic decay time constants ($\boldsymbol{\tau}$) using backpropagation. First, the time constants are initialized with random values within the specified range:

$$\boldsymbol{\tau} = \sigma(\boldsymbol{\mathcal{N}}(0,1)) \cdot \tau_{step} + \tau_{min}$$

where $\sigma(\cdot)$ is the sigmoid function (identical to Eq. 6) used to constrain the time constants to be non-negative. The time constant values are also bounded by the minimum (τ_{min}) and the maximum ($\tau_{max} = \tau_{min} + \tau_{step}$) values. The error computed from the loss function (Eq. 8) is then backpropagated to update the time constants at each iteration:

$$\frac{\partial \mathcal{L}}{\partial \boldsymbol{\tau}} = \frac{\partial \mathcal{L}}{\partial \boldsymbol{r}} \cdot \frac{\partial \boldsymbol{r}}{\partial \boldsymbol{x}} \cdot \frac{\partial \boldsymbol{x}}{\partial \boldsymbol{\tau}}$$

The method proposed by Song et al. [10] was used to impose Dale's principle and create separate excitatory and inhibitory populations. Briefly, the recurrent connectivity matrix (W^{rate}) in the rate model is parametrized by

$$W^{rate} = [W^{rate}]_+ \cdot D \tag{9}$$

where the rectified linear operation ($[\cdot]_+$) is applied to the connectivity matrix at each update step. The diagonal matrix ($D \in \mathbb{R}^{N \times N}$) contains +1's for excitatory units and -1's for inhibitory units in the network. Each unit in the network is randomly assigned to one group (excitatory or inhibitory) before training, and the assignment does not change during training (i.e. D stays fixed).

To impose specific connectivity patterns, we apply a binary mask ($M \in \mathbb{R}^{N \times N}$) to Eq. 9:

$$W^{rate} = \left([W^{rate}]_+ \cdot D\right) \odot M$$

where $\odot$ refers to the Hadamard operation (elementwise multiplication). Similar to the diagonal matrix (D), the mask matrix stays fixed throughout training. For example, the following mask matrix can be used to create a subgroup of inhibitory units (Group A) that do not receive synaptic inputs from the rest of the inhibitory units (Group B) in the network (see Supplementary Fig. 1):

$$m_{ij} = \begin{cases} 0 & i \in \text{Group A}, j \in \text{Group B} \\ 1 & \text{otherwise} \end{cases}$$

where $m_{ij} \in M$ establishes (if $m_{ij} = 1$) or removes (if $m_{ij} = 0$) the connection from unit j to unit i.

Transfer learning from a rate model to a spiking model. In this section, we describe the method that we developed to perform transfer learning from a trained rate model to a LIF model. Once the rate RNN model is trained using the gradient descent method outlined in **Training details**, the rate model parameters are transferred to a LIF network in a one-to-one manner. First, the LIF network is initialized to have the same topology as the trained rate RNN. Next, the input weight matrix (W_{in}) and the synaptic decay time constants ($\boldsymbol{\tau}$) are transferred to the spiking RNN without any modification. Lastly, the recurrent connectivity matrix (W^{rate}) and the readout weights (W^{rate}_{out}) are scaled by a constant number, λ, and transferred to the spiking network.

If the recurrent connectivity weights from the trained rate model are transferred to a spiking network without any changes, the spiking model produces largely fluctuating signals (as illustrated in Fig. 1D), because the LIF firing rates are significantly larger than 1 (whereas the firing rates of the rate model are constrained to range between zero and one by the sigmoid transfer function).

To place the spiking RNN in the similar dynamic regime as the rate network, we first assume a linear relationship between the rate model connectivity weights and the spike model weights:

$$W^{spk} = \lambda \cdot W^{rate}$$

Using the above assumption, the synaptic drive (d) that unit i in the LIF RNN receives can be

553 expressed as

$$\begin{aligned} d_i^{spk}(t) &= \sum_{j=1}^{N} w_{ij}^{spk} \cdot r_j^{spk}(t) \\ &\approx \sum_{j=1}^{N} (\lambda \cdot w_{ij}^{rate}) \cdot r_j^{spk}(t) \\ &= \sum_{j=1}^{N} w_{ij}^{rate} \cdot (\lambda \cdot r_j^{spk}(t)) \end{aligned} \tag{10}$$

554 where $w_{ij}^{spk} \in W^{spk}$ is the synaptic weight from unit j to unit i.

555 Similarly, unit i in the rate RNN model receives the following synaptic drive at time t:

$$d_i^{rate}(t) = \sum_{j=1}^{N} w_{ij}^{rate} \cdot r_j^{rate}(t) \tag{11}$$

556 If we set the above two synaptic drives (Eq. 10 and Eq. 11) equal to each other, we have:

$$\begin{aligned} d_i^{spk}(t) &= d_i^{rate}(t) \\ \sum_{j=1}^{N} w_{ij}^{rate} \cdot (\lambda \cdot r_j^{spk}(t)) &= \sum_{j=1}^{N} w_{ij}^{rate} \cdot r_j^{rate}(t) \end{aligned} \tag{12}$$

557 Generalizing Eq. 12 to all the units in the network, we have

$$\boldsymbol{r}^{rate}(t) = \lambda \cdot \boldsymbol{r}^{spk}(t)$$

558 Therefore, if there exists a constant factor (λ) that can account for the firing rate scale difference
559 between the rate and the spiking models, the connectivity weights from the rate model (W^{rate})
560 can be scaled by the factor and transferred to the spiking model.

561 The readout weights from the rate model (W_{out}^{rate}) are also scaled by the same constant factor
562 (λ) to have the spiking network produce output signals similar to the ones from the trained rate
563 model:

$$\begin{aligned} o^{rate}(t) &= W_{out}^{rate} \cdot \boldsymbol{r}^{rate}(t) \\ &\approx W_{out}^{rate} \cdot (\lambda \cdot \boldsymbol{r}^{spk}(t)) \\ &= (\lambda \cdot W_{out}^{rate}) \cdot \boldsymbol{r}^{spk}(t) = o^{spk}(t) \end{aligned}$$

564 In order to find the optimal scaling factor, we developed a simple grid search algorithm. For a
565 given range of values for λ (ranged from 0.0125 to 0.10 with a step size of 0.0001), the algorithm
566 finds the optimal value that minimizes the RMSE between the rate network output and the spiking
567 model output signals.

Implementation of computational tasks and figure details. In this section, we describe the details of the parameters and methods used to generate all the main figures in the present study.

Fig. 1. A rate RNN of $N = 200$ units (162 excitatory and 38 inhibitory units) was trained to perform a Go-NoGo task. Each trial lasted for 1000 ms (200 time steps with 5 ms step size). The minimum and the maximum synaptic decay time constants were set to 20 ms and 50 ms, respectively. An input stimulus with a pulse 125 ms in duration was given for a Go trial, while no input stimulus was given for a NoGo trial. The network was trained to produce an output signal approaching +1 after the stimulus offset for a Go trial. For a NoGo trial, the network was trained to maintain its output at zero. A trial was considered correct if the maximum output signal during the response window was above 0.7 for the Go trial type. For a NoGo trial, if the maximum response value was less than 0.3, the trial was considered correct. For training, 6000 trials were randomly generated, and the model performance was evaluated after every 100 trials. Training was terminated when the loss function fell below 7 and the task performance reached at least 95%. The termination criteria were usually met at or before 2000 trials for this task. A scaling factor of 0.02 ($\lambda = 0.02$) was used to construct a LIF network model for this task.

Fig. 2. A rate RNN model with $N = 200$ units (98 excitatory and 102 inhibitory units) was trained to produce a sinusoidal signal (1 Hz) autonomously. The synaptic decay time constants were set to range from 20 ms to 50 ms. Each trial lasted for 3500 ms or 700 time steps with 5 ms step size, and the training was terminated when the loss function fell below 5. A scaling factor of 0.0286 ($\lambda = 0.0286$) was used to construct a LIF network model for this task.

Fig. 3. A network of $N = 400$ continuous-variable units (299 excitatory and 101 inhibitory units) were trained to perform the context-dependent input integration task. The input matrix ($\boldsymbol{u} \in \mathbb{R}^{4\times 750}$) contained four stimuli channels across time (750 time steps with 5 ms step size). The first two channels corresponded to the modality 1 and modality 2 noisy input signals. These signals were modeled as white-noise signals (sampled from the standard normal distribution) with constant offset terms. The sign of the offset term modeled the evidence toward (+) or (-) choices, while the magnitude of the offset determined the strength of the evidence. The noisy signals were only present during the stimulus window (250 ms – 2500 ms). Once the network was trained, the stimulus duration was shortened to 1000 ms (250 ms – 1250 ms). The last two channels of $\boldsymbol{u}$ represented the modality 1 and the modality 2 context signals. For instance, the third channel of $\boldsymbol{u}$ is set to one and the fourth channel is set to zero to model Modality 1 context.

For each trial used to train the rate model, the offset values for the two modality input signals

were randomly set to -0.5 or +0.5. The context signals were randomly set such that either modality 1 (third input channel is set to 1) or modality 2 (fourth input channel is set to 1) was cued for each trial. If the offset term of the cued modality was +0.5 (or -0.5) for a given trial, the network was instructed to produce an output signal approaching +1 (or -1) after the stimulus window. The model performance was assessed after every 100 training trials, and the training termination conditions were same as the ones used for *Figure 1.* A scaling factor of 0.0182 was used to construct a LIF network model for this task.

For the psychometric curves (Fig. 3D), the offset value was varied from -0.5 to +0.5 with a step size of 0.1.

Fig. 4. To investigate how task variables and offset values affected the network dynamics, we used the spiking network constructed in Fig. 3 to generate neural responses for different trial conditions. We considered 11 levels of offset ranging from -0.5 to +0.5 with a step size of 0.1. Therefore, there were a total of 242 trial conditions: (11 offsets for modality 1) $\times$ (11 offsets for modality 2) $\times$ (2 contexts). For each condition, we generated 50 trials and extracted spike trains from all the units. The spike data was preprocessed in a similar manner as done by Mante et al. [4]. Briefly, time-varying firing rates were first estimated by counting spikes in a non-overlapping, sliding window (50 ms in duration). Next, the firing rates from all the trials ($242 \times 50 = 12100$ trials) were concatenated, resulting in a large matrix with 400 rows (one for each unit) and $12100 \times T$ columns (where $T = 44$ is the number of time points in each trial). The firing rates of each unit (i.e. each row of the large matrix) were normalized by z-score transformation using the mean and the standard deviation across all the trials (i.e. across the columns of the matrix). The z-scored neural responses were then used for the multi-variable linear regression and the targeted dimensionality reduction analyses (implemented using the details outlined in Mante et al. [4]).

Fig. 5. A rate RNN network composed of $N = 200$ units (158 excitatory and 42 inhibitory units) was trained to perform a temporal exclusive OR (XOR) task. The input matrix ($\boldsymbol{u} \in \mathbb{R}^{2\times 300}$) contained two input channels for two sequential stimuli (over 300 time steps with 5 ms step size). The first channel delivered the first stimulus (250 ms in duration), while the second channel modeled the second stimulus (250 ms in duration) which began 50 ms after the offset of the first stimulus. The short delay (50 ms) allowed the model to learn the task efficiently, and the delay was increased to 250 ms after training without affecting the model performance. During each stimulus window, the corresponding input channel was set to either -1 or +1. If the two sequential stimuli had the same sign (-1/-1 or +1/+1), the network was trained to produce an output signal approaching +1

after the offset of the second stimulus. If the stimuli had opposite signs (-1/+1 or +1/-1), then the network produced an output signal approaching -1. Training was stopped when the loss function fell below 7, and the task performance was greater than 95%. A scaling factor of 0.0167 was used to construct a LIF network model for this task.

Principal component analysis (PCA) was performed on the instantaneous firing rates obtained from the LIF network. The firing rates were estimated by applying the double synaptic filter shown in **Spiking network structure**. For each trial condition, neural responses were extracted from 50 trials. There were a total of 200 trials (4 trial conditions and 50 trials per condition). PCA was then applied to the neural responses (concatenated across all the trials), and the top three principal components were used to represent the low dimensional network activities (Fig. 5D).

Fig. 6. To compute spike-triggered averages (STAs) of local field potential (LFP) proxy signals, spontaneous spike trains were first extracted from the LIF model: for each "spontaneous" trial (i.e. no stimulus input given to the network), spike trains from the excitatory units were extracted. For each trial, the LFP proxy signal was modeled as z-scored average synaptic inputs into the excitatory units over time. A 400-ms window, centered at each spike time in the extracted spike trains, was used to extract spike-triggered LFP segments. These segments were then averaged to obtain the STA for the trial. For each inhibitory suppression condition (intact, mild, moderate, and severe), STAs were computed from 100 trials and averaged across the trials.

Fig. 7. A cross-temporal decoding method similar to the ones used by Miconi [12], Meyers et al. [31] was employed to assess the encoding stability of the LIF network. More specifically, we studied the stability of the first stimulus encoding by the excitatory units for each inhibitory suppression condition. For each trial condition (-1/-1, -1/+1, +1/-1, +1/+1), population activities (time-varying firing rates) from 50 trials were extracted. These trials were then separated by the identity of the first stimulus leading to two groups of neural responses from the "-1" condition (100 trials with the "-1" first stimulus) and the "+1" condition (100 trials with the "+1" first stimulus). The first half of the neural activities from each condition was chosen as a training dataset, while the second half was used for testing. A maximal-correlation classifier (identical to the one used by Miconi [12]) was then trained on the training dataset and tested on the test data.

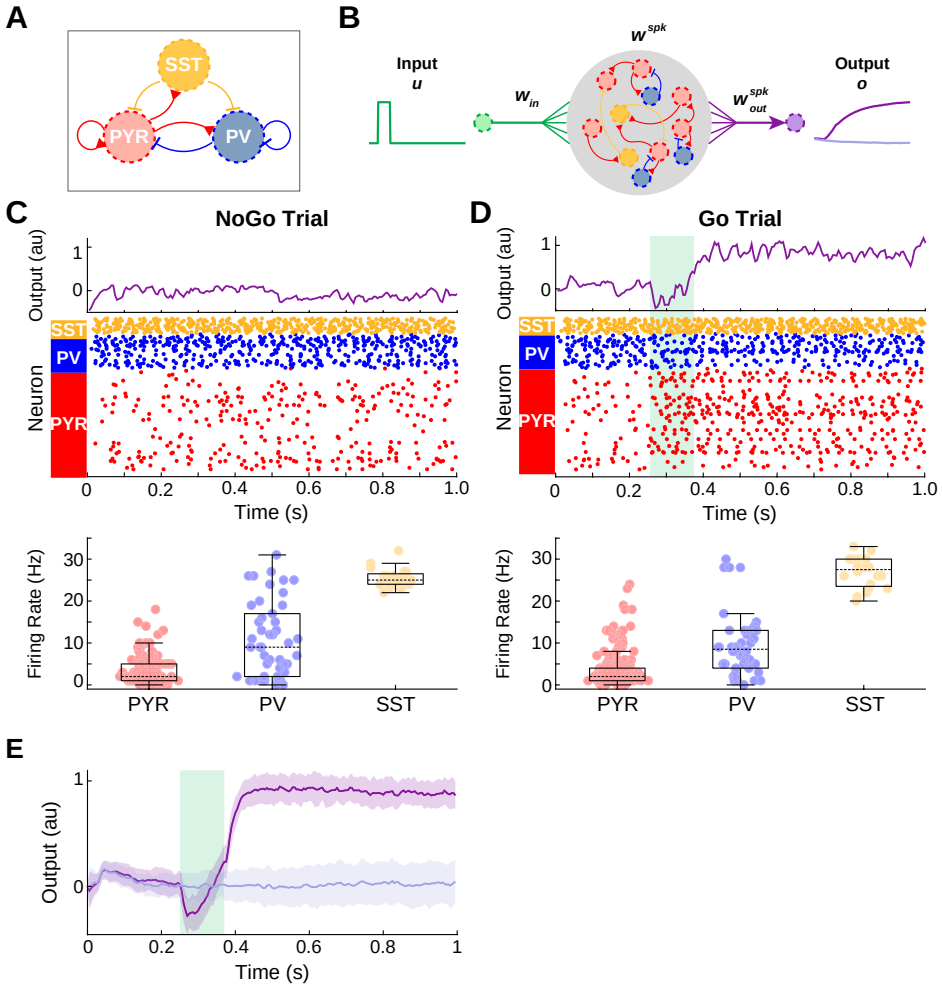
Code availability

The implementation of our framework and the codes to generate all the figures in this work are available at `https://github.com/rkim35/spikeRNN`.

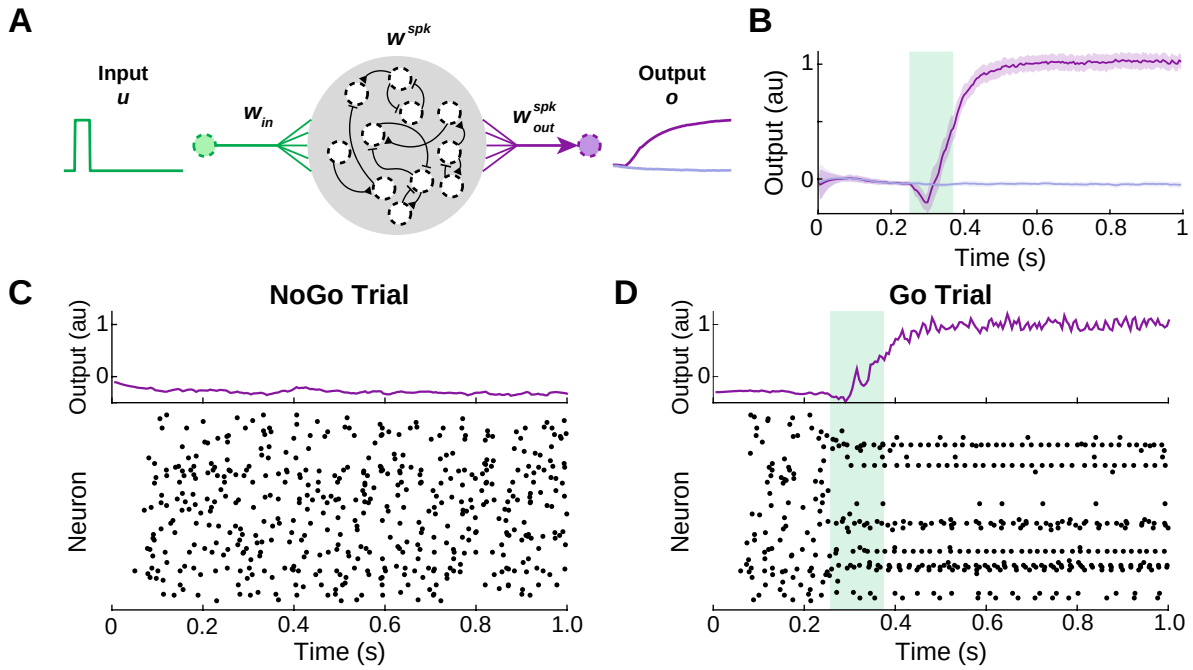
Data availability

The trained models used in the present study are available as MATLAB-formatted data at `https://github.com/rkim35/spikeRNN`.

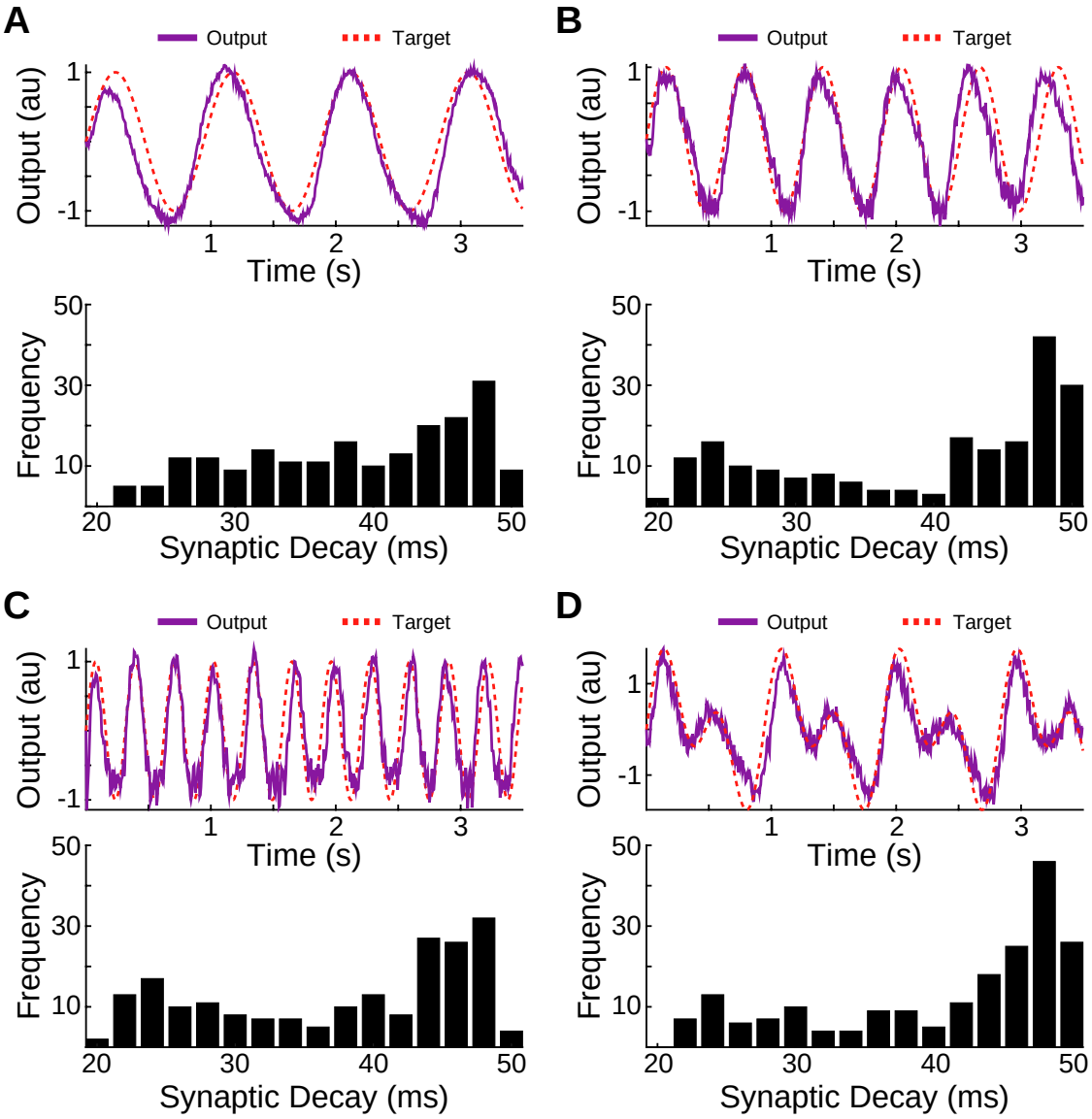
Supplementary Figures



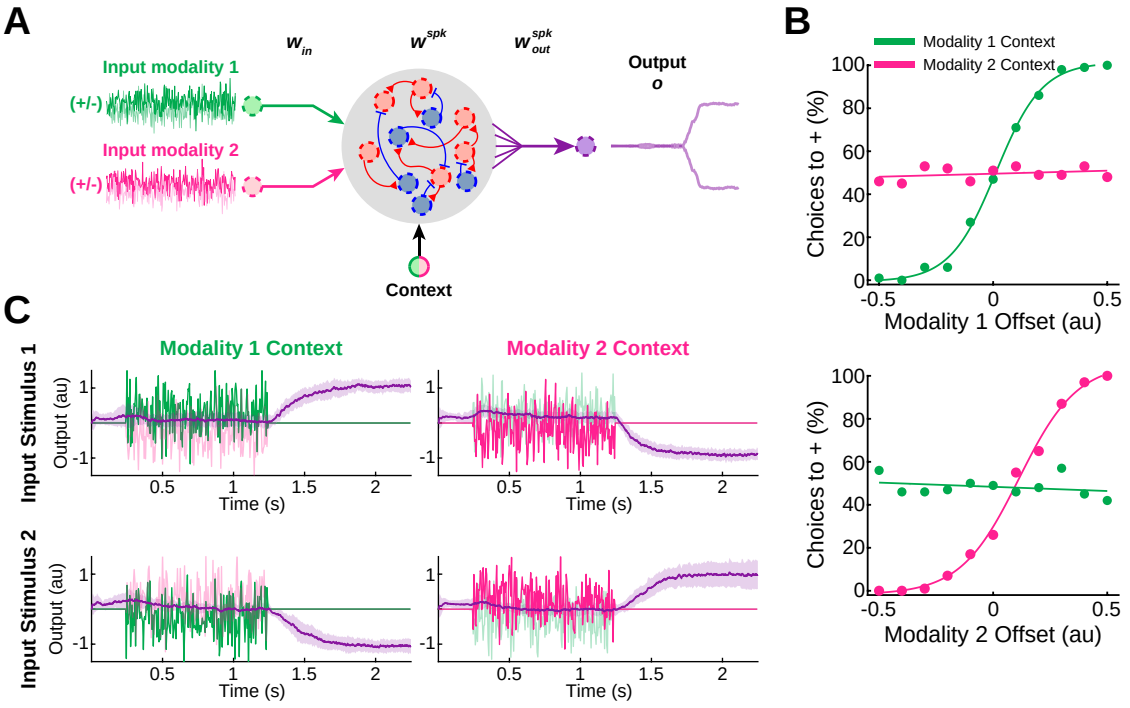
Supplementary Fig. 1 | Incorporation of additional functional connectivity constraints. A. Common cortical microcircuit motif where somatostatin-expressing interneurons (SST; yellow circle) inhibit both pyramidal (PYR; red circle) and parvalbumin-expressing (PV; blue circle) neurons. **B.** Schematic illustrating the incorporation of the connectivity motif shown in **A** into a LIF network model. The connectivity pattern was imposed during training of a rate network model ($N = 200$) to perform the Go-NoGo task. There were 134 PYR, 46 PV, and 20 SST units. A spiking model was constructed using the trained rate model with $\lambda = 0.02$. **C.** Example output response and spikes from the LIF network model for a single NoGo trial. Mean ± SD firing rate for each population is also shown (PYR, 3.08 ± 3.29 Hz; PV, 10.80 ± 8.94 Hz; SST, 25.50 ± 2.33 Hz). **D.** Example output response and spikes from the LIF network model for a single Go trial. Mean ± SD firing rate for each population is also shown (PYR, 4.72 ± 5.89 Hz; PV, 9.30 ± 8.16 Hz; SST, 27.05 ± 3.98 Hz). Box plot central lines, median; bottom and top edges, lower and upper quartiles. **E.** LIF network model performance on 50 NoGo trials (light purple) and 50 Go trials (dark purple). Mean ± SD shown.



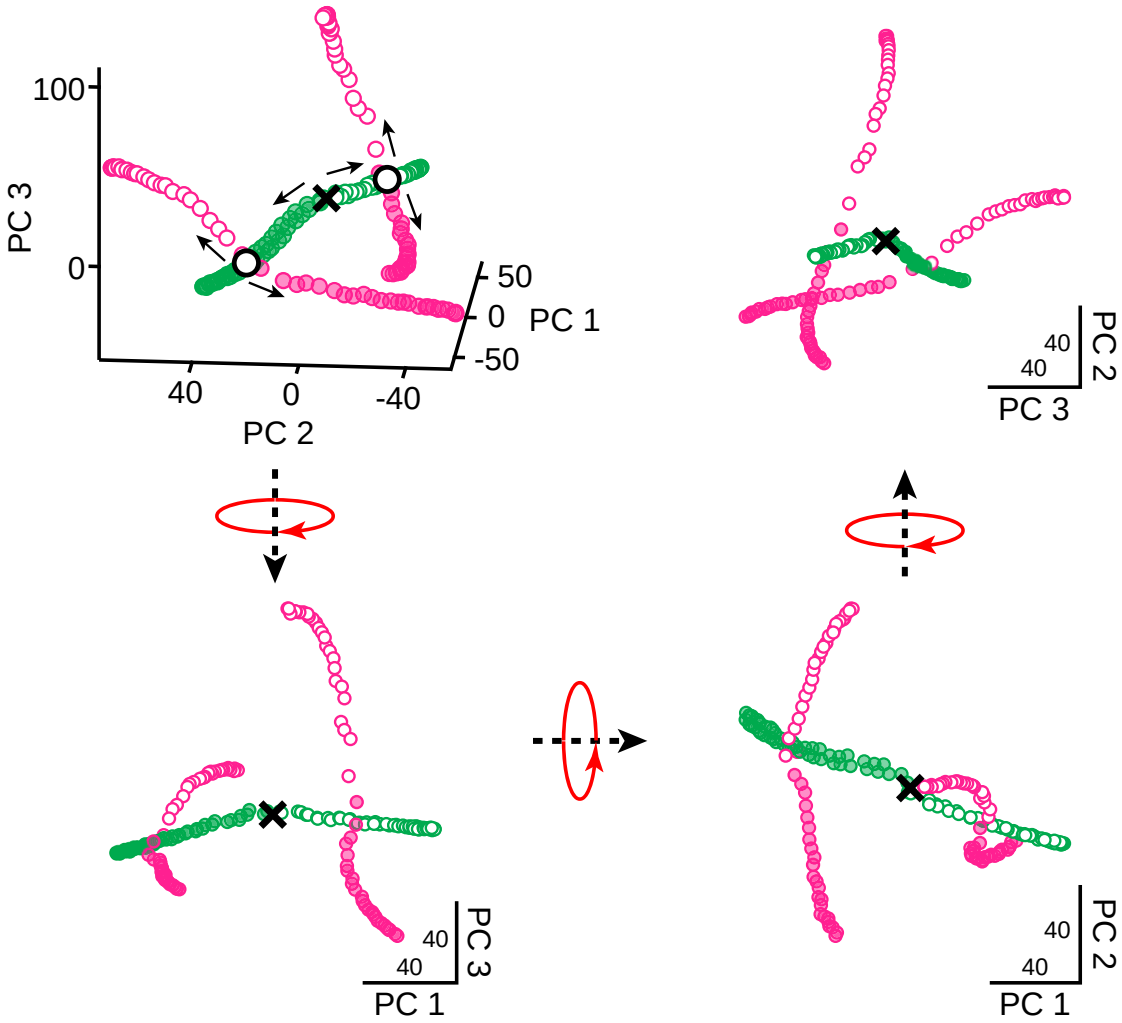
Supplementary Fig. 2 | Dale's principle constraint can be relaxed. A. Schematic diagram showing a LIF network model without Dale's principle. A rate RNN model ($N = 200$) without Dale's principle was first trained to perform the Go-NoGo task. The scaling factor (λ) was set to 0.02. Note that each unit (black dotted circles) can exert both excitatory and inhibitory effects. **B.** LIF network model performance on 50 NoGo trials (light purple) and 50 Go trials (dark purple). Mean ± SD shown. **C.** Example output response (top) and spikes (bottom) from the LIF network model for a single NoGo trial. **D.** Example output response (top) and spikes (bottom) from the LIF network model for a single Go trial.



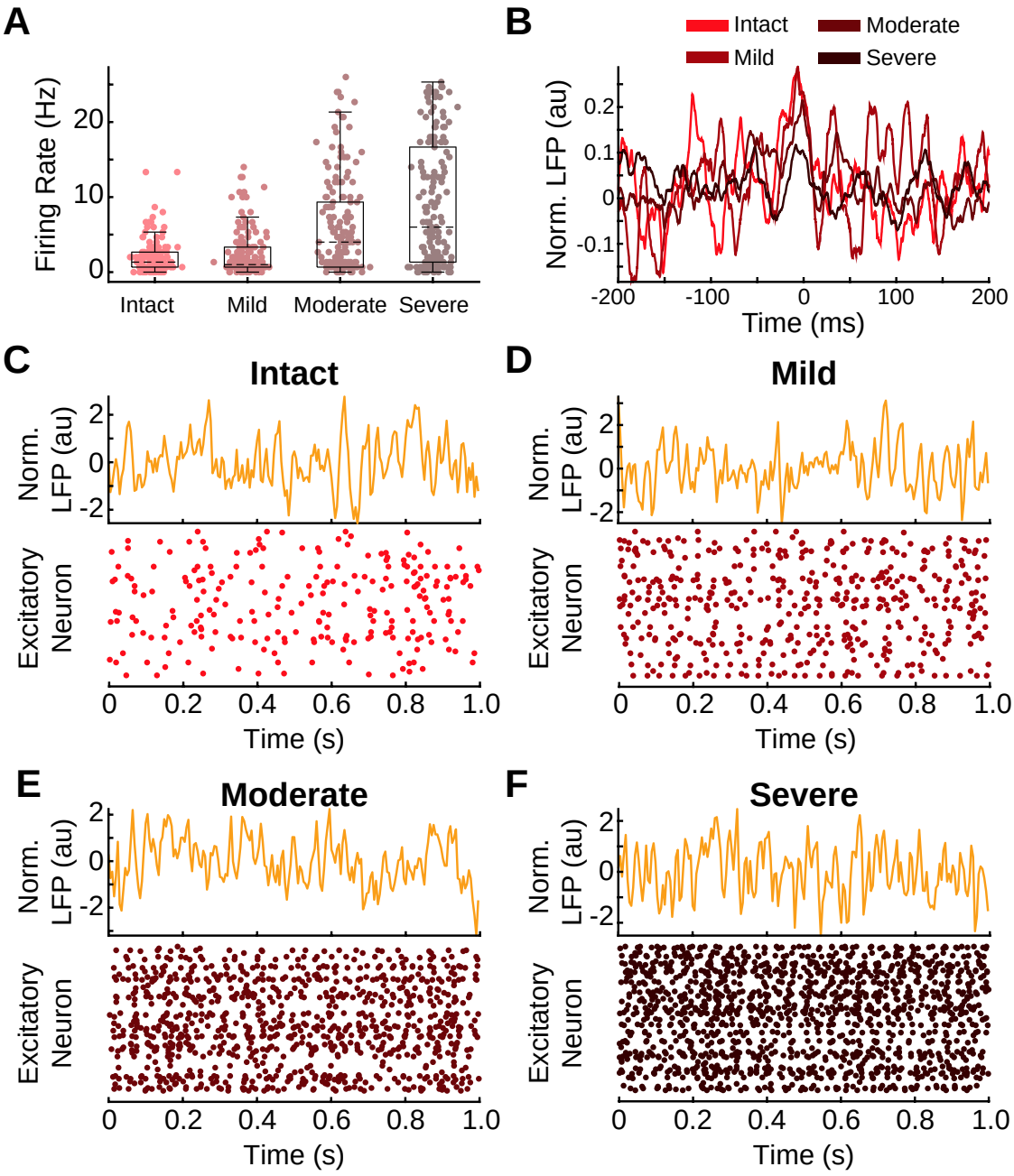
Supplementary Fig. 3 | Autonomous oscillation tasks with different target signals. A. Output signal (solid purple line) from a spiking model constructed to produce a 1 Hz sine signal (dotted magenta line) along with the distribution of the trained synaptic time constants (bottom). The spiking model used here is identical to the one used for Fig. 2. **B.** Output signal (solid purple line) from a spiking model constructed to produce a 1.5 Hz sine signal (dotted magenta line). The optimized synaptic decay time constants are also shown (bottom). **C.** Output signal (solid purple line) from a spiking model constructed to produce a 3 Hz sine signal (dotted magenta line) along with the distribution of the trained synaptic time constants (bottom). **D.** Output signal (solid purple line) from a spiking model constructed to produce a target signal obtained by combining the two target signals from **A** and **B**, and the tuned synaptic decay time constants (bottom).



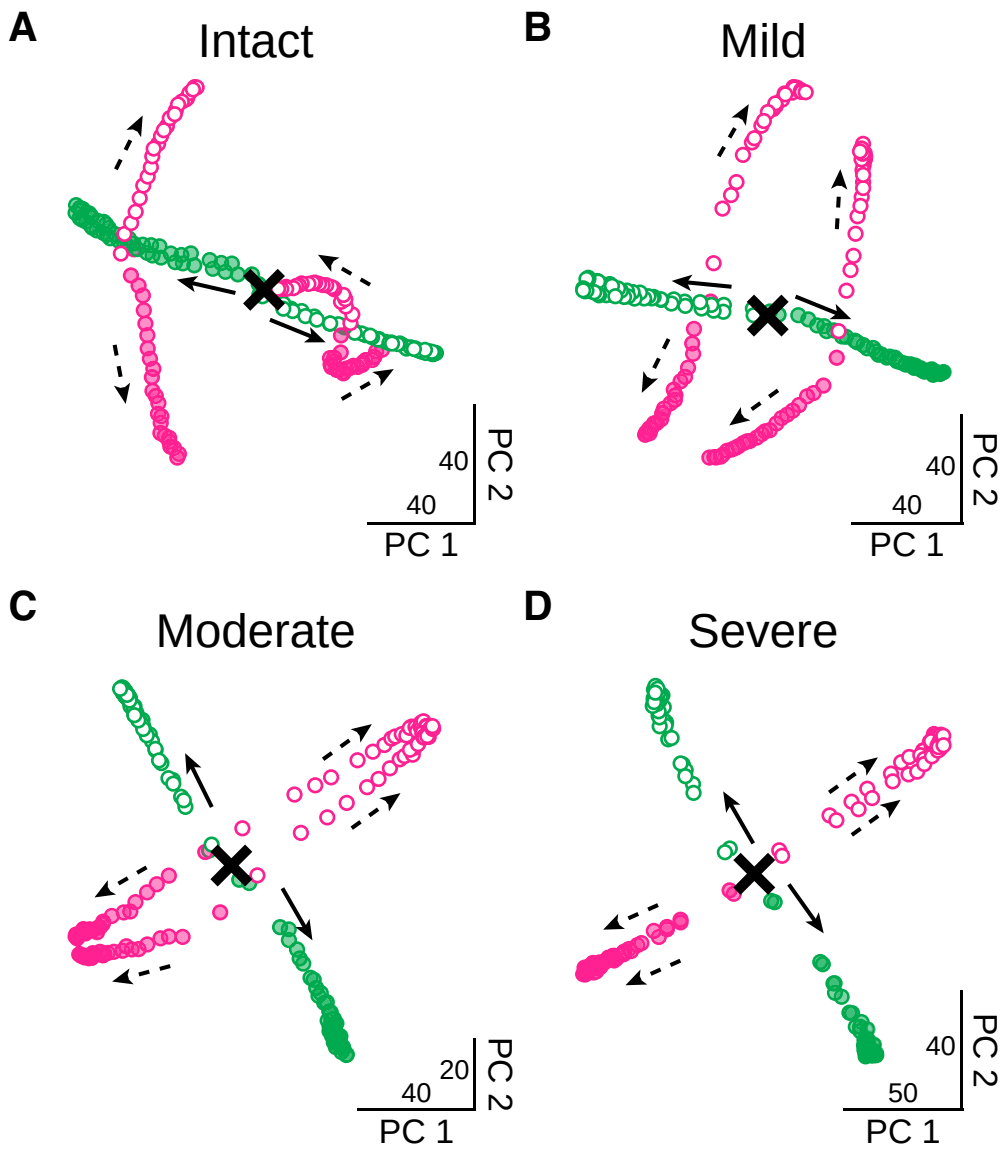
Supplementary Fig. 4 | Quadratic integrate-and-fire (QIF) model constructed to perform the context-dependent input integration task. A. The task paradigm and the trained rate network model used for Fig. 3 were employed to build a QIF model. The QIF model parameter values are listed in Supplementary Table 1. **B.** Psychometric curves from the QIF network model. The percentage of trials where the QIF network indicated "+" choice as a function of the modality 1 offset values (top) and modality 2 offsets (bottom). **C.** The QIF model successfully performed the task by integrating cued modality input signals. Example noisy input signals (scaled by 0.5 vertically for visualization; green and magenta lines) from a single trial are shown. Mean ± SD response signals (purple lines) across 50 trials for each trial type.



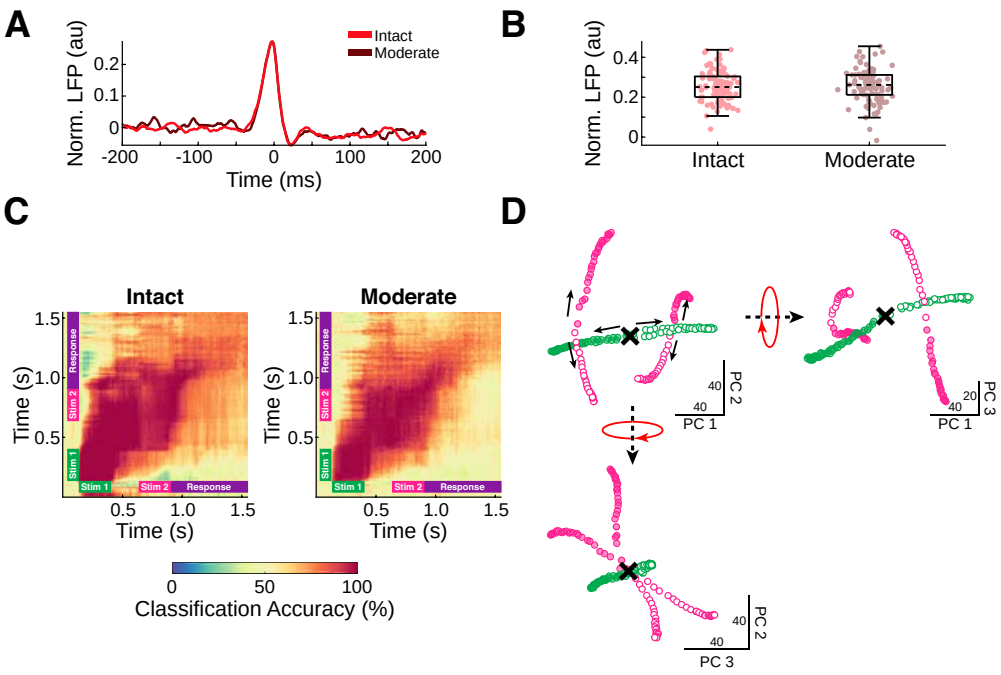
Supplementary Fig. 5 | Low dimensional neural response trajectories of the intact network model during the sequential XOR task. Different views of the three-dimensional PCA plot shown in Fig. 5D (reproduced here for reference).



Supplementary Fig. 6 | Increased spontaneous excitatory unit activities from network models with impaired inhibitory units. A. As the fraction of the suppressed inhibitory units increased, the spontaneous firing rates of the excitatory units also increased. For each condition (intact, mild, moderate, and severe), the excitatory firing rates from a single trial are shown. Box plot central lines, median; bottom and top edges, lower and upper quartiles. Same color scheme as Fig. 6. **B.** Single-trial STA signals from the four conditions. The LFP signal and the excitatory spike raster plot for the intact model (**C**), the mild model (**D**), the moderate model (**E**), and the severe model (**F**) are also shown.



Supplementary Fig. 7 | Low dimensional trajectories reveal impaired working memory computations in networks with compromised inhibitory units. Neural population trajectories projected to the first two principal components (PCs) for the intact model (**A**), the mild model (**B**), the moderate model (**C**), and the severe model (**D**). As the fraction of the suppressed inhibitory units increased, the network began to lose the memory of the the first stimulus identity and retained only the second stimulus identity as shown by the two opposite "tunnels" formed during the second stimulus epoch (magenta empty and filled circles) in **D**. The trajectories evolve temporally in the following order: black cross (first stimulus onset), solid arrows (first stimulus epoch), dashed arrows (second stimulus epoch).



Supplementary Fig. 8 | Suppression of excitatory units does not lead to significant neuronal desynchrony and working memory impairment. A. STAs computed from the intact LIF network (red line) and the network with 50% of the excitatory units suppressed (brown line). For each condition, the average STA time-series over 100 trials is shown. **B.** The STA amplitude values at spike times ($t = 0$) from the two conditions (intact and moderate) were not significantly different. Box plot central lines, median; bottom and top edges, lower and upper quartiles. **C.** The network model with impaired excitatory units was able to encode the first stimulus identity reliably across the trial epochs. The cross-temporal decoding analysis (same as the one used for Fig. 7) was performed. **D.** Neural response trajectories of the "moderate" network model projected to the first three principal components (PCs). Even with the suppressed excitatory units, the network was able to preserve the three task-related variables: first stimulus identity, second stimulus identity, and response.

Supplementary Notes

For the quadratic integrate-and-fire (QIF) model (Supplementary Fig. 4), we considered a network of units governed by

$$\tau_m \frac{d\boldsymbol{v}}{dt} = \boldsymbol{v}^2 + W^{spk}\boldsymbol{r}^{spk} + \boldsymbol{I}_{ext}$$

The definitions of the variables are identical to the ones used for the LIF network model.

Supplementary Table

	LIF	QIF
Membrane time constant (τ_m)	10 ms	10 ms
Absolute refractory period	2 ms	2 ms
Synaptic rise time (τ_r)	2 ms	2 ms
Constant bias current	-40 pA	0 pA
Spike threshold	-40 mV	30 mV
Spike reset voltage	-65 mV	-65 mV

Supplementary Table 1 | Parameter values used to construct LIF and QIF networks.