Spiking network model of the cerebellum as a reinforcement learning machine

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The cerebellum has been considered to perform error-based supervised learning via long-term depression (LTD) at synapses between parallel fibers and Purkinje cells (PCs). Since the discovery of multiple synaptic plasticities other than LTD, recent studies have suggested that synergistic plasticity mechanisms could enhance the learning capability of the cerebellum. We have proposed that the mechanisms allow the cerebellar circuit to perform reinforcement learning (RL). However, its detailed spike-based implementation is still missing. In this research, we implemented a cerebellar spiking network as an actor-critic model based on known anatomical properties of the cerebellum. We confirmed that our model successfully learned a state value and solved the mountain car task, a simple RL benchmark. Furthermore, our model demonstrated the ability to solve the delay eyeblink conditioning task using biologically plausible internal dynamics. The study presents the implementation of the first cerebellar spiking network model capable of performing RL.

Cerebellum | Spiking Neural Network | Reinforcement Learning | Simulation

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Introduction

Theories of learning in the cerebellum have historically attracted considerable interest. One of the most widely known theories, known as the Marr-Albus-Ito model (Marr, 1969; Ito, 1970; Albus, 1971), proposes that the cerebellum acts as an error-based supervised learning (SL) machine (Rosenblatt, 1958) to modify output and reduce the discrepancy between actual and desired outputs. In this theory, climbing fibers (CFs) deliver error or teacher signals that can trigger long-term depression (LTD) at parallel fiber (PF) and Purkinje cell (PC) synapses (Ito, 2001). Since the discovery of LTD in the early 1980s (Ito and Kano, 1982), many other forms of synaptic plasticity have been found in the cerebellum (D'Angelo, 2014). These findings have been extending the Marr-Albus-Ito theory gradually (Fujita, 2016; Raymond and Medina, 2018; Yamazaki, 2021). Furthermore, the learning theory has been examined by realistic spiking network models of the cerebellum (Medina et al., 2000; Gosui and Yamazaki, 2016; Hausknecht et al., 2017; Abadía et al., 2021; Kuriyama et al., 2021; Shinji et al., 2024; Geminiani et al., 2024). Especially, Hausknecht et al. (2017) examined the learning capability of the cerebellum through a cerebellar spiking network modeled in an SL context. The network successfully performed SL and control tasks but failed to solve RL and complex recognition tasks. Geminiani et al. (2024) implemented a spiking network-based cerebellar model featuring bidirectional plasticity at PF-PC and PF-molecular layer interneurons (MLIs) synapses across multiple microzones, with several microzones more likely depression and others more likely potentiation during motor control. Their results highlight the cerebellum's capacity for error correction and adaptive learning, particularly within the context of associative learning and motor control. Both computational studies modeled the cerebellum as a conventional associative learning machine and highlighted the cerebellar learning capability, especially in the SL context.

One of the other research directions is to adapt reinforcement learning (RL) theory (Sutton and Barto, 2018) as an alternative to conventional SL. In an RL context, there is an "agent" and an "environment". The agent takes an action affecting the state of the environment. In response to the action, the environment sends a "reward" to the agent which represents how good/bad the action was. The agent learns appropriate actions that maximize an expected future reward. One of the most notable differences between SL and RL is feedback information from the environment. An SL machine receives errors or teacher signals representing optimal actions, while an RL machine receives only reward signals.

Yamazaki and Lennon (2019) proposed the concept that the cerebellum might perform RL. Specifically, CFs deliver reward information, PCs select actions, and MLIs evaluate the current state. Synaptic weights at PF–MLI as well as PF–PC synapses are modulated by the CF signals. This structure was interpreted as an RL machine known as an actor-critic model(Sutton and Barto, 2018). However, there has been no detailed spike-based implementation for the cerebellar RL concept.

In this research, based on a continuous time actor-critic framework with spiking neurons (Frémaux et al., 2013), we implemented a cerebellar spiking network as an actor-critic model based on the known anatomical properties

of the cerebellum. In particular, we modified a weight-updating rule of the framework for the cerebellum. The implemented model was evaluated by a simple machine learning benchmark task known as the mountain car task (Moore, 1990). The behavior as a cerebellar model was also examined on delay eyeblink conditioning (Jirenhed et al., 2007), which is a standard cerebellum-dependent motor learning task. To our knowledge, the model presented here is the first cerebellar spiking network model that can act as an RL machine.

Results

A. Spike-based implementation of cerebellum-style RL

In this research, we implemented a cerebellar spiking network model as an RL machine while referring to the cerebellar RL concept (Yamazaki and Lennon, 2019) and the spiking actor-critic framework (Frémaux et al., 2013). A schematic of cell types and synaptic connections incorporated in the present model, along with a flow diagram of neural signaling, is shown in Figure 1. PFs excite stellate cells (SCs), basket cells (BCs), and PCs. SCs and BCs inhibit PCs. BCs and PCs inhibit each other, and also both have a self-inhibitory connection. More specifically, BCs and PCs are divided into multiple groups, which correspond to action choices as described in Section A.3. BCs inhibit PCs in the same group and BCs in other groups, whereas PCs inhibit BCs in the same group and PCs in other groups. PCs also inhibit neurons in the deep cerebellar nuclei (DCN), which are also divided into multiple groups. CFs provide excitatory inputs to SCs and PCs. We used CF signals to trigger synaptic plasticity. Therefore, we did not implement actual CF–SC and CF–PC connections.

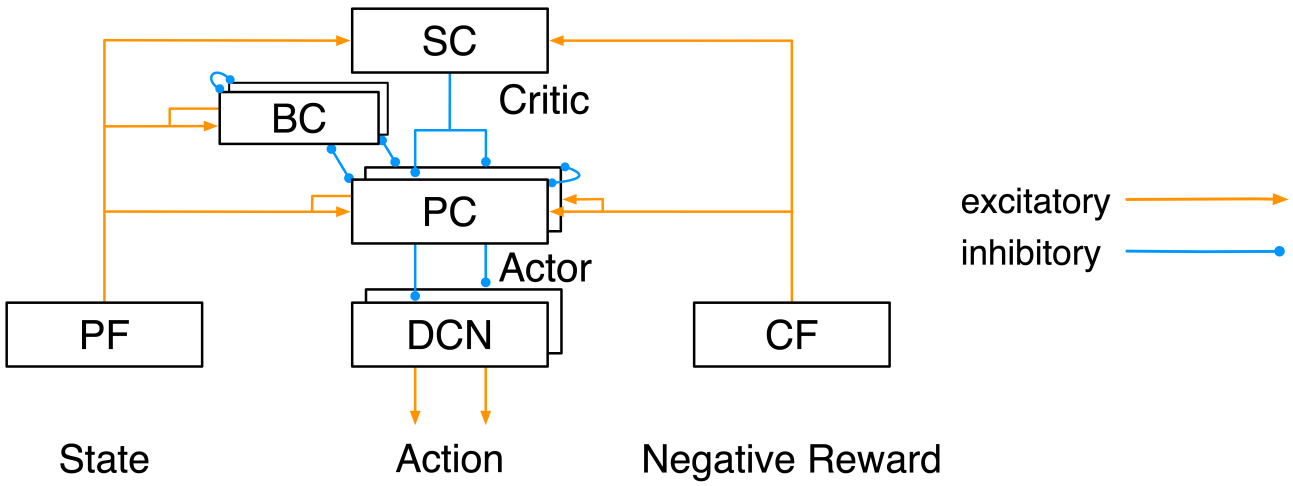

Figure 1. Schematic of the present cerebellar network implemented as an actor-critic model. PCs serve as the actor, and SCs serve as the critic. Groups of BCs, PCs, and DCN cells are organized based on the number of actions, with each action defining a corresponding group. Orange triangle-headed arrows and blue circle-headed arrows represent excitatory synapses and inhibitory synapses, respectively. Bidirectional circle-headed arrows represent cross inhibitions.

In the cerebellar spiking network model, we implemented an RL algorithm known as the actor-critic model (Sutton and Barto, 2018) based on the previous spiking actor-critic framework (Frémaux et al., 2013).In RL, an agent in state $s(t)$ at time t takes action $a(t)$ following a policy π. In response to action $a(t)$, the environment changes its state and returns a reward $r(t)$ to the agent. The agent learns to find the optimal policy that maximizes the expected future reward. Specifically, the agent of the actor-critic model consists of two modules: an actor and a critic. The actor calculates preferences of actions $h_\alpha(t)$ and decides on action $a(t)$, while the critic computes the state value $V(t)$ which refers to the expected cumulative future reward. The temporal difference error (TD error) is calculated from the returned reward and the state value for updating the inner parameters of both the critic and the actor.

In our implementation, $s(t)$ was transmitted through PFs to PCs, BCs, and SCs. The actor and the critic were PCs and SCs, respectively. Practically, the activity of PCs represented the avoidance of actions $-h_\alpha(t)$, while the activity of SCs represented the additive inverse of the state value $-V(t)$ rather than TD error which represented by MLIs in the previous concept (Yamazaki and Lennon, 2019). BCs contributed to action selection by modulating the activity of PCs (Brown et al., 2019; Kim and Augustine, 2021). Then, the most activated DCN group decided on an actual action $a(t)$. In response to the action, reward information was transmitted through CFs to PCs and SCs.

A.1. Representation of states and rewards. States were represented by PFs. For simplicity, we arranged the PFs on a two-dimensional grid plane composed of a number of small tiles. We named this plane the PF plane. Each tile had an individual receptive field that determined the PF's firing rate based on the observed state, as described in Section D.

In the present study, rewards $r(t)$ were actually considered as punishments so that $r(t) \leq 0$. CFs delivered the information as excitatory signals by flipping the sign as follows:

$$\mathrm{CF}(t) = -r(t). \tag{1}$$

A.2. Critic. We designed the temporal activity of SCs to approximate the state value. Strictly speaking, the additive inverse of the state value was calculated by SCs as follows:

$$-V(t) = \nu \mathrm{SC}(t) - V_0, \tag{2}$$

where ν, $\mathrm{SC}(t)$, and V_0 represent a scaling factor, the temporal activity of SCs, and the baseline of the state value, respectively. The baseline V_0 was set at ν times the average value of $\mathrm{SC}(t)$, which was calculated during the latter half of an episode without learning. $\mathrm{SC}(t)$ was calculated as follows:

$$\mathrm{SC}(t) = \frac{1}{N_{\mathrm{SC}}} \sum_i \mathrm{SC}_i(t), \tag{3}$$

$$\mathrm{SC}_i(t) = \frac{\mathrm{SC}_{i,\mathrm{decay}}(t) - \mathrm{SC}_{i,\mathrm{rise}}(t)}{\tau_{\mathrm{SC,decay}} - \tau_{\mathrm{SC,rise}}}, \tag{4}$$

$$\frac{d\mathrm{SC}_{i,l}(t)}{dt} = -\frac{\mathrm{SC}_{i,l}(t)}{\tau_{\mathrm{SC},l}} + S_i(t), \tag{5}$$

where N_{SC} and $\mathrm{SC}_i(t)$ represent the number of SCs and the temporal activity of ith SC, respectively. l is a label in $\{\mathrm{rise}, \mathrm{decay}\}$. The temporal activity of ith SC rises with a time constant $\tau_{\mathrm{SC,rise}}$ and decays to 0 with a time constant $\tau_{\mathrm{SC,decay}}$. Spike train of the ith neuron is defined as $S_i(t) = \sum_f \delta_D(t - t_i^{(f)})$ with the Dirac delta function $\delta_D(t)$ and the fth spike of the ith neuron.

A.3. Actor and action selection. PCs performed the actor and determined the action taken. According to the classic behavioral research on PC activity during motor tasks (Ito, 1984; Jirenhed et al., 2007), we interpreted the temporal activity of PCs as expressing avoidance of certain actions. Specifically, we modeled that PCs compute the avoidance of an action $h_{\bar{\alpha}}(t)$ instead of the preference for an action $h_{\alpha}(t)$. Therefore, the taken action $a(t)$ was determined by the least-avoided action in action space $\mathcal{A}$ at time t.

$$a(t) = \underset{\alpha \in \mathcal{A}}{\mathrm{argmin}}\, h_{\bar{\alpha}}(t). \tag{6}$$

The avoidance of action $\alpha \in \mathcal{A}$ was represented by the activity of PCs in $\bar{\alpha}$ group as follows :

$$h_{\bar{\alpha}}(t) = \frac{1}{N_{\mathbf{PC}_{\bar{\alpha}}}} \sum_{i \in \mathbf{PC}_{\bar{\alpha}}} \mathbf{PC}_i(t), \tag{7}$$

where $N_{\mathbf{PC}_{\bar{\alpha}}}$ is the number of neurons in the group $\mathrm{PC}_{\bar{\alpha}}$. $\mathrm{PC}_i(t)$ was the temporal activity of ith PC defined as follows:

$$\mathrm{PC}_i(t) = \frac{\mathrm{PC}_{i,\mathrm{decay}}(t) - \mathrm{PC}_{i,\mathrm{rise}}(t)}{\tau_{\mathrm{PC,decay}} - \tau_{\mathrm{PC,rise}}}, \tag{8}$$

$$\tau_l \frac{d\mathrm{PC}_{i,l}(t)}{dt} = -\mathrm{PC}_{i,l}(t) + S_i(t), \tag{9}$$

where l, τ_l, and $S_i(t)$ represent the label $l \in \{\mathrm{decay}, \mathrm{rise}\}$, the time constant for l, and the spike train of the ith PC, respectively. Practically, the actual action was generated at the downstream DCN (Section D).

Those spike activities of PCs were used for updating synaptic weights to realize which action was selected. It was vital that one PC group pauses while the other PC groups activate. We referred to this pausing behavior as a “dent", while activation of neurons in a specific area was referred to as a “bump" by Frémaux et al. (2013). The inhibitory loop between BCs and PCs helped to pause the single group solely to make a dent. Although the original spiking actor-critic framework supports continuous action space, the present model only supports discrete action space with only two actions, because of the difficulty of making a single dent on PCs.

A.4. Weight-update rules. Our weight-update rule was based on TD long-term potentiation (LTP) which had been proposed in a previous spiking actor-critic model (Frémaux et al., 2013). However, unlike the previous model, which calculated the time derivative of the state value directly to compute the TD error, our model did not calculate it, as there was no component in the molecular layer of our model that explicitly handled the TD error.

To address this problem, we conducted an eligibility trace and transformed the weight-update rule. As a result, our weight-update rule for the state value was as follows:

$$\frac{d\Delta w_{i,j}^n}{dt} = \alpha\left(R(t)z_{i,j}(t) + \frac{\tau_r - \tau_z}{\tau_r\tau_z}V(t)z_{i,j}(t) - V(t)f_{i,j}(t)\right), \tag{10}$$

$$\frac{dz_{i,j}(t)}{dt} = -\frac{z_{i,j}(t)}{\tau_z} + f_{i,j}(t), \tag{11}$$

$$f_{i,j}(t) = S_i(t)\kappa_{i,j}(t), \tag{12}$$

$$\frac{d\kappa_{i,j}(t)}{dt} = -\frac{\kappa_{i,j}(t)}{\tau_\kappa} + S_j(t), \tag{13}$$

where $\Delta w_{i,j}^n$, α, $R(t)$, $z_{i,j}(t)$, τ_r, τ_z, $f_{i,j}(t)$, $S_i(t)$, $S_j(t)$, $\kappa_{i,j}(t)$, and τ_κ are the amount of synaptic change in nth episode, the learning rate, the negative reward, the eligibility trace for $w_{i,j}$, the reward discount time constant, the decay time constant of eligibility trace, the forcing term of eligibility trace, the postsynaptic spike train, the presynaptic spike train, the window function for spike timing dependent plasticity, and the decay time constant of the window function. According to the previous model (Frémaux et al., 2013), the eligibility term memorizes the history of inputs before the time of the last postsynaptic neuron spike, $\kappa_{i,j}(t)$ is reset to 0 after the postsynaptic neuron spike. The relationship of our weight-update rule to the original weight-update rule (Doya, 2000; Frémaux et al., 2013) is described in Section A of the Supplementary Information. The synaptic weight $w_{i,j}$ was updated at the end of every episode T_{end} by $w_{i,j}^{n+1} = w_{i,j}^n + \eta\Delta w_{i,j}^n(T_{\text{end}})$ with learning rate η, and the synaptic weight was bounded between $[0,1]$. Since SCs represented the additive inverse state value and we denoted the negative reward $r(t) = -\mathrm{CF}(t)$, the equation for the PF–SC synapses was expressed as follows:

$$\frac{d\Delta w_{i,j}^n}{dt} = \mathrm{CF}(t)z_{i,j}(t) - \frac{\tau_r - \tau_z}{\tau_r\tau_z}V(t)z_{i,j}(t) + V(t)f_{i,j}(t). \tag{14}$$

On the other hand, the weight-update rule for PF–PC synapses was expressed as follows:

$$\frac{d\Delta w_{i,j}^n}{dt} = -\mathrm{CF}(t)z_{i,j}(t) + \frac{\tau_r - \tau_z}{\tau_r\tau_z}V(t)z_{i,j}(t) - V(t)f_{i,j}(t). \tag{15}$$

The specific learning rates for PF–SC synapses and PF–PC synapses are denoted as η_{SC} and η_{PC}, respectively.

Specifically, given that the conjunctive activation of PFs and CFs induces LTP at PF–SC synapses (Jörntell and Ekerot, 2002, 2003; Jirenhed et al., 2013; Liu, 2013), our SCs learned the approximate additive inverse of the state-value. LTD at PF–SC synapses was due to PF firing, and was modulated by postsynaptic activation results in bidirectional synaptic plasticity (Jörntell and Ekerot, 2002, 2003; Jirenhed et al., 2013; Liu, 2013). At PF–PC synapses, the conjunctive activation of PFs and CFs induces LTD (D'Angelo, 2014), while PF activation induces LTP. These synaptic plasticities are also modulated by inhibitions of SCs (Rowan et al., 2018), and we assumed that PC activity contributes to eligibility trace and bidirectional synaptic plasticity (Bonnan et al., 2021; Silva et al., 2024).

Additionally, we confirmed that our critic is capable of learning state values through a Linear Track Task simulation (Section B of Supplementary Information), which is a simplified version of the water-maze task used to isolate learning by the critic from that by the actor (Frémaux et al., 2013).

B. Simulation of Mountain Car Task

In order to evaluate the learning capability of our model as an RL machine, we applied our model to the mountain car task (Moore, 1990), a classic RL challenge, and evaluated its performance over 10 trials. In this task, an agent controls a car situated between two hills (Figure 2A). The goal is to have the car climb up the mountain to reach a flag positioned at the peak of the right hill. Due to the car's limited engine power and the hill's steepness, direct ascent is not possible. An agent observes a position $p_x(t)$ and a horizontal element of the car velocity $v_x(t)$, and decides the direction (either left or right) to push the car with a constant force $F(t) = \bar{F}a(t),\ \ a(t) \in \{-1, 1\}$. In early episodes, the car was confined to moving at the bottom of the valley, and failed to reach the goal (Figure 2B). However, after hundreds of episodes, our model learned to utilize the slope for acceleration, and eventually reached the goal (Figure 2C). Specifically, we found that the trajectory involved the car ascending the right hill, then the left, and finally the right hill again. The additive inverse of the state value $-V(t)$, which was approximated by SC activity, was noisy but mostly stayed around 0 at every state before learning (Figure 2B). In a successful episode after learning (Figure 2C), the value was high at the start and decreased over time as the car approached the goal. The moving average of success rate over 10 trials progressively increased and stabilized at approximately over 80% after 600 episodes (Figure 2D). These results indicate that our model solved the mountain car task, and suggest that our model successfully acted as an RL machine.

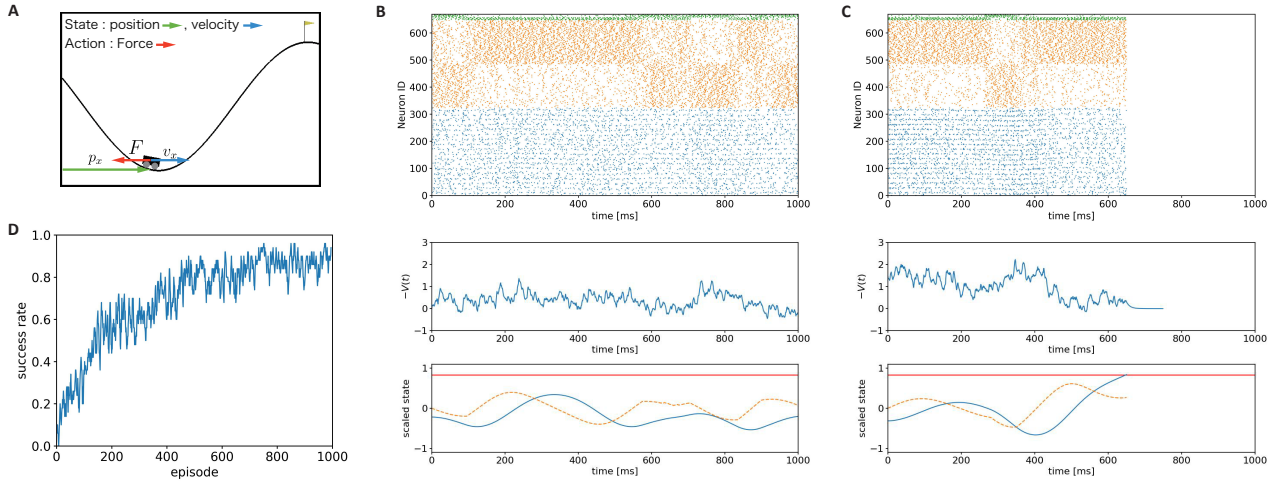

Figure 2. Simulation of the mountain car task. **A**) Schematic of the mountain car task. p_x is the horizontal position of the cart, v_x is the horizontal velocity, and F is the external force applied to the cart by the agent. The flag marks the goal position. For clarity in the image, we adjusted the x-axis to start at 0 on the left edge. **B,C**) Comparison of behaviors in the mountain car task. **B**) Example behavior in early episodes. **C**) Example behavior in later episodes. Top panels show raster plots of SCs (blue), BCs (orange) and PCs (green). The number of these neurons are 324, 324, and 20, respectively. Middle panels show trajectory of $-V(t)$ computed from the activity of SCs. Bottom panels show trajectory of scaled states. Blue line and orange dashed line represent the normalized value of a position and that of a velocity. Red line represents the goal position. When the position reaches the goal, an episode ends while a simulation lasts 100 ms (Section E.1). **D**) Success rate of mountain car task. The moving average of success rate across 10 trials with window size 5 episodes.

C. Simulation of Delay Eyeblink Conditioning Task

To examine whether our cerebellar model shows internal dynamics consistent with the biological cerebellum, we conducted simulation of a standard cerebellum-dependent motor learning task known as the delay eyeblink conditioning task (Jirenhed et al., 2007). In this task, an animal is presented with two types of stimuli: a conditioned stimulus (CS) and an unconditioned stimulus (US) (Figure 3A). The CS, a neutral stimulus such as a tone, initially does not trigger any response. On the other hand, the US, such as an air puff, naturally induces an unconditioned blink reflex in the animal. However, when the CS is repeatedly paired with the US, the animal begins to anticipate the air puff whenever the CS is presented. This anticipation leads the animal to blink in response to the CS alone, a learned behavior known as the conditioned response (CR). We modified the task to have two-dimensional state space: time t and eyelid position $p_e(t)$, along with two actions: opening and closing the eyelid (Figure 3B). The time axis ranges from 0 ms to 1,000 ms, corresponding to the duration of stimulus presentation, while the eyelid position is measured on a scale from fully closed (0) to fully open (1). The CS was presented from 0 ms to 1,000 ms as a time signal, and the US was introduced at 500 ms as a punishment if the eyelid was open, i.e., if $p_e(500)$ was greater than 0.1.

In early episodes, the agent preferred to open the eyelid (Figure 3C and 3B), so the agent received the US as a negative reward. As learning progressed, the anti-open PC group became active, whereas the anti-close PC group paused as if PCs expected the US onset. Thus, the eyelid started to close before the US onset. For every 10 trials, the agent successfully acquired the CR to the CS: closing its eyelid to avoid a punishment before the arrival of the US (Figure 3E). Note that we defined a successful episode as sufficiently closed the eyelid to avoid the punishment at the US onset. When focusing on the activity changes of a single PC in the anti-close group (Figure 3F), the neuron acquired a behavior to pause before US onset. These behaviors were consistent with behavioral experiments of the delayed eyeblink conditioning task (Jirenhed et al., 2007). We also analyzed the activity of SCs during the task (Figure 3G). In early episodes, since the agent received the punishment as the US, the SC activity as the additive inverse of the state value $-V(t)$ increased exponentially toward the arrival of the US. In later episodes when the agent successfully closed the eyelid more than 80% of the time, the SC activity returned to zero.

We also conducted a simulation of the same task without PF–MLI synaptic plasticity. The activity of the anti-close PC group decreased as it approached the US onset (Figure 4A). However, the agent was not able to elicit a stable closing response in our settings. In a later episode, the time to close the eye was delayed (Figure 4B) compared to the that in the intact simulation (Figure 3D), so the agent could not close the eye sufficiently. These results suggest that our model successfully solved the eyeblink task with biologically plausible internal dynamics.

Discussion

There has been growing interest in roles of MLIs in cerebellar learning theory. Traditionally, because MLIs are involved in controlling PC activities through MLI-PC inhibition, their primary role was considered as a supportive modulator for PC kinematic outcomes in motor coordination (Wulff et al., 2009; Liu, 2013; Jelitai et al., 2016; Astorga et al., 2017; Brown et al., 2019; Kim and Augustine, 2021). However, recent studies have shown that MLIs also

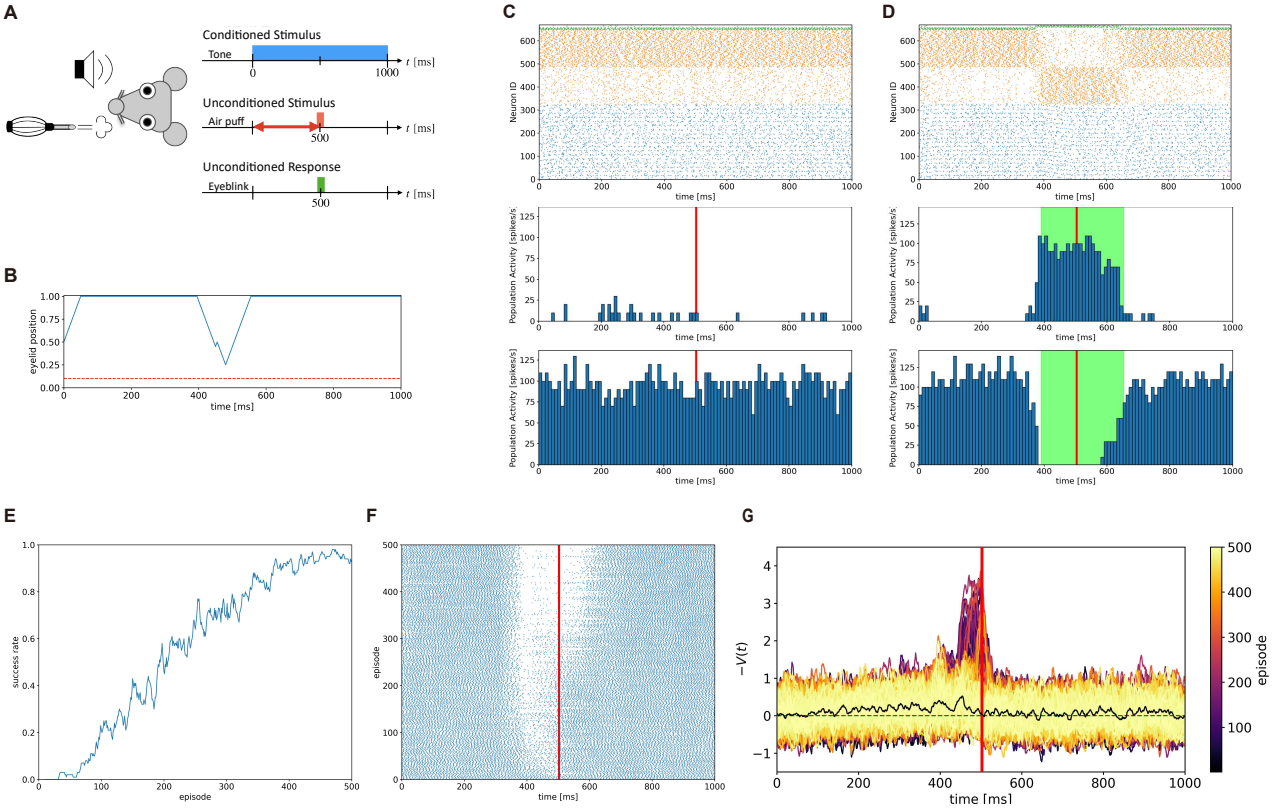

Figure 3. Simulation of the delay eyeblink conditioning task. **A**) Schematic of eyeblink conditioning task. In this task, a neutral stimulus (CS) is presented to an agent, and during the presentation, an aversive stimulus (US) is also fed. Here, the time interval from the onset of the CS to the onset of the US is fixed. After a number of paired presentations, the agent closes their eyes (CR) in response to the CS without US. **B**) Example of a state trajectory on the state space of eyeblink conditioning task. The blue line shows an example of state trajectory. The horizontal axis represents the duration from CS onset, and the vertical axis represents the eyelid position. Dashed line represents a threshold. When the eyelid position is above the threshold at the onset of the US ($t = 500$ ms), the agent receives a punishment. **C,D**) Results of the delay eyeblink conditioning task in a failure episode (C) and a success episode (D). Top panels show raster plots of SCs (blue), BCs (orange) and PCs (green). The number of these neurons are 324, 324, and 20, respectively. Middle and bottom panels show peristimulus time histograms of anti-open PC population (middle) and those of anti-close PC population (bottom). Bin width is 10 ms. Red line represents the time of the US onset, while green shaded intervals show when the agent close its eyelid around the US onset. **E**) The moving average of success rate over 10 trials. **F**) The activity changes of a single PC in the anti-close group. Dots represent spikes of the PC, and red line represents the time of the US onset. **G**) Additive inverse of the state value during the eyeblink conditioning task. Each colored trace shows the additive inverse of the state value represented by the stellate cell neurons activity against time in 500 episodes (from black purple in the 1st trial to light yellow in the 500th trial, transitioning through shades of red, orange, and yellow). The black line shows the average of over last 10 trials. The green dashed line shows $-V(t) = 0$. The red thick line represents the time of the US onset.

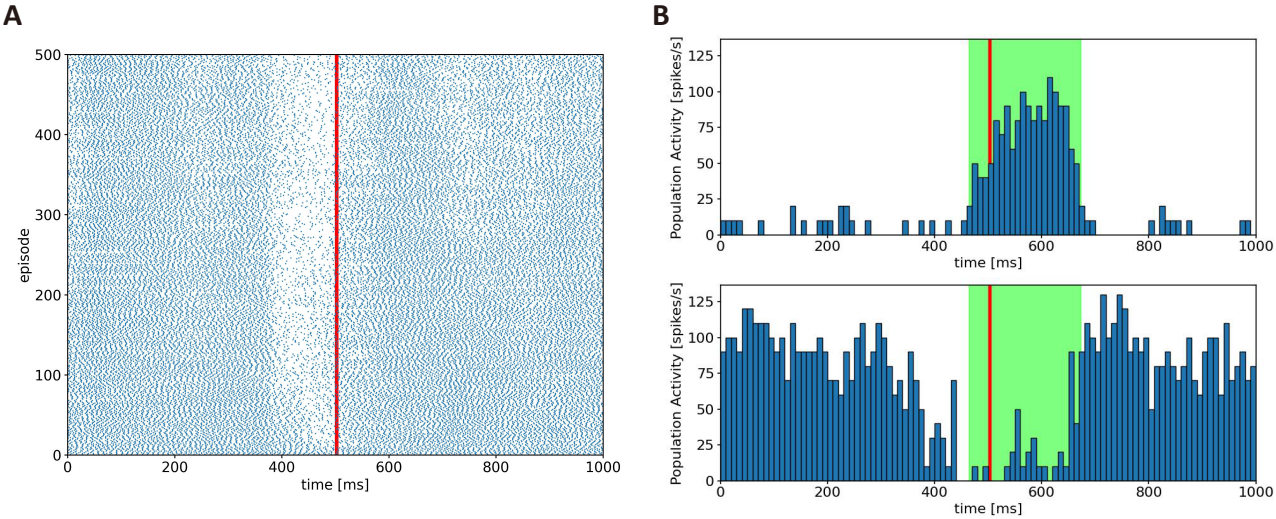

Figure 4. Results of the delay eyeblink conditioning task without PF–SC synaptic plasticity. **A**) The activity changes of a single PC in the anti-close group. Dots represent spikes of the PC, and red line represents the time of the US onset. **B**) Example peristimulus time histograms of anti-open PC population (top) and those of anti-close PC population (bottom) in the later episode. Bin width is 10 ms. Red lines represent the time of the US onset, while the green shaded intervals show when the agent closes its eyelid around the US onset.

contribute to learning in the cerebellum (Wulff et al., 2009; Liu, 2013; Johansson et al., 2014; ten Brinke et al., 2015; Lee et al., 2015; Hirano et al., 2016; Boele et al., 2018; Rowan et al., 2018; Ma et al., 2020; Kim and Augustine, 2021).

In this study, SCs represented the state value as a critic and contributed to modulating PF–PC synaptic plasticity. Practically, since CFs conveyed negative rewards and the conjunctive activation of a CF and a PF induced LTP (Jörntell and Ekerot, 2002, 2003; Jirenhed et al., 2013; Liu, 2013), the activity of SCs reflected the additive inverse of the state value $-V(t)$ (Figure S1B,2C, and 3G). These results are not inconsistent with biological experiments (ten Brinke et al., 2015; Ma et al., 2020), which show increases in MLIs' firing frequency from the time of the CS onset to the time of the US onset. Besides, in our delay eyeblink conditioning task simulation, SC activity increased toward the US onset in the early episodes, then SC lost their activation and became closer to baseline activity in the late episodes (Figure 3G). This behavior resulted from the disappearance of CF signals due to the successful avoidance of punishments. Additionally, the pausing behavior of PCs was established even without PF–SC synaptic plasticity, i.e., the agent could respond to CS instead of US (Figures 3F and 4A). However, the amplitude of eye closing was insufficient to avoid the US (Figure 4B). These results are also consistent with experimental results where MLI–PC inhibitions were impaired (Boele et al., 2018), and suggest that changes in SCs activity play a critical role in modulating PF–PC synaptic plasticity to achieve precise timing. Further investigations into the activity of SCs during learning are necessary to fully understand their functional role in cerebellar learning theory.

Recent studies have shown that CFs might deliver not only error signals (Ito, 1984) but also multiplex information including motor information and reward-related signals (Ohmae and Medina, 2015; Heffley and Hull, 2019; Kostadinov et al., 2019; Hoang et al., 2023a). Ohmae and Medina (2015) observed the activity of individual CFs during cerebellum-dependent eyeblink conditioning in mice. They found that CFs conveyed information about both unexpected deliveries and unexpected omissions of the US. They proposed that these patterns of CF activity might perform as a TD error. Heffley and Hull (2019); Kostadinov et al. (2019) show that CFs signaled reward expectation, delivery, and omission, whereas Ikezoe et al. (2023) suggested that CFs convey behavioral information on multiplex variables including reward signals during a voluntary movement task in mice. Furthermore, Hoang et al. (2023b) employed a table-type Q-learning model as the cerebellum and showed that their model was able to reproduce the learning course of the licking responses of mice in a Go/No-go auditory-discrimination task.

In the present study, our primary focus was on implementing the cerebellar spiking network model as an RL machine. Therefore we assumed CFs to convey negative rewards as a punishment based on our previous cerebellar RL concept (Yamazaki and Lennon, 2019). Even if CFs signaled positive reward-related value, our SCs could learn and represent the positive state value using the same synaptic plasticity mechanisms. While the interpretation of individual cell functions might require some adjustments for different CF representations, the core of our model implementation could likely remain applicable.

While historically the cerebellum has been considered to perform SL, the basal ganglia has been considered to perform RL (Doya et al., 2001), since dopaminergic neurons in the substantia nigra pars compacta (SNpc) and the ventral tegmental area broadcast a signal corresponding to TD error (Schultz et al., 1997; Doya et al., 2001; Sutton and Barto, 2018). Recent studies revealed that the basal ganglia and the cerebellum are interconnected at the subcortical level (Bostan et al., 2010; Fermin et al., 2016; Bostan and Strick, 2018; Washburn et al., 2024) and cooperate to learn and acquire an action selection strategy. Specifically, Washburn et al. (2024) showed that the cerebellum directly modulates SNpc neuron activity so that the cerebellum might affect learning mechanisms in the basal ganglia. If the cerebellum performs RL as we suggest, what does the link between the cerebellum and the basal ganglia mean for learning? One possible interpretation is hierarchical RL (HRL) (Sutton and Barto, 2018) as discussed in our previous research (Yamazaki and Lennon, 2019). The instance of HRL consists of two hierarchical RL machines. The higher RL machine breaks down a complex task into simpler, smaller subtasks. The lower RL machine tries to solve these subtasks one by one. Eventually, HRL involves creating a hierarchy of policies that makes the learning process more efficient and scalable (Morimoto and Doya, 2001; Kulkarni et al., 2016; Sutton and Barto, 2018). If the basal ganglia and the cerebellum cooperate to perform HRL, the resulting network would demonstrate powerful learning capabilities.

We successfully implemented the first cerebellar spiking network model capable of performing RL, which solved a simple RL benchmark task known as the mountain car task. Furthermore, our cerebellar model demonstrated the ability to solve the delay eyeblink conditioning task using biologically plausible internal dynamics. These results support the theory that the cerebellum can perform RL in addition to conventional SL. Future research could explore the functional role of the cerebellum in the cooperation of subcortical systems for learning.

Methods

D. Implementations

First, we discretized a state space and mapped it on the PF plane whose grid size is $N_x \times N_y$. A tile positioned at (i, j) on the PF plane had its own responsible state $\mathbf{o}_{i,j}$ as follows:

$$\mathbf{o}_{i,j} = [i\Delta s_x, j\Delta s_y]^T, \tag{16}$$

$$\Delta s_x = \frac{s_{x,\text{max}} - s_{y,\text{min}}}{N_x}, \quad \Delta s_y = \frac{s_{y,\text{max}} - s_{y,\text{min}}}{N_y} \tag{17}$$

where x and y are the axes of the PF plane, and $(s_{x,\text{min}}, s_{x,\text{max}})$ and $(s_{y,\text{min}}, s_{y,\text{max}})$ represent the ranges of state x and y, respectively. When the agent observed state $\mathbf{o}(t) = [x(t), y(t)]^T$, PFs on a tile at (i, j) fired as an inhomogeneous Poisson spike generator with a firing rate $\rho_{i,j}(\mathbf{o}(t))$ defined as follows :

$$\rho_{i,j}(\mathbf{o}(t)) = \frac{\rho_{\text{PF}}}{\sqrt{2\pi\sigma^2}} \exp\left(-\frac{||\mathbf{o}(t) - \mathbf{o}_{i,j}||^2}{2\sigma^2}\right), \tag{18}$$

where $\rho_{\text{PF}} = 50$ Hz is a scaling factor of firing rate, and $\sigma = 0.5$ is a standard deviation. To maintain the total activity of all PFs when the agent is at the edge of a state space, we added “margin" tiles as an outer frame.

We utilized a Leaky Integrate-and-Fire model (LIF) to SCs, BCs, and PCs. A LIF model was implemented as follows.

$$C_m \frac{du_i}{dt} = -g_{\text{L}}(u_i - E_{\text{L}}) + I_{i,\text{syn}}(t) + I_{\text{ext}} + I_{i,\text{noise}}(t), \tag{19}$$

$$t_i^{(f)} : u_i\left(t_i^{(f)}\right) = U_{\text{th}}, \tag{20}$$

$$\lim_{t_i^{(f)} \to t,\ t > t_i^{(f)}} u_i(t) = U_{\text{reset}}, \tag{21}$$

where C_m, u_i, t, g_{L}, E_{L}, $I_{i,\text{syn}}$, I_{ext}, and $I_{i,\text{noise}}$ represent the membrane capacitance, the membrane potential of the ith neuron, time, the leak conductance, the resting potential, the synaptic current to the ith neuron, the external input current, and the noise current to the ith neuron, respectively. When the membrane potential of the ith neuron reaches the threshold potential U_{th}, that time is denoted as $t_i^{(f)}$, and the neuron fires the fth spike and resets its membrane potential to the reset potential U_{reset}. A synaptic current was defined as follows:

$$I_{i,\text{syn}}(t) = \sum_c s_c w_{i,j} I_{i,j}(t), \tag{22}$$

$$\frac{dI_{i,j}(t)}{dt} = -\frac{I_{i,j}(t)}{\tau_c} + S_j(t), \tag{23}$$

where c is a synapse label, s_c, $w_{i,j}$, τ_c, and $S_j(t)$ are the scaling factor, synaptic weight between the jth presynaptic neuron and the ith postsynaptic neuron, decay time constant, and spike train of the jth presynaptic neuron, respectively. Whether it is an inhibitory or excitatory current is determined by the sign of the scaling factor. Noise current $I_{i,\text{noise}}(t)$ was modeled as follows:

$$\frac{dI_{i,\text{noise}}}{dt} = -\frac{I_{i,\text{noise}}}{\tau_{\text{noise}}} + \zeta n(t), \tag{24}$$

where τ_{noise} is a time constant, and ζ is an amplitude factor. $n(t)$ is a uniform random variable in the range $[-1, 1]$. Parameters of all neurons were set as listed in Table 1, and parameters of all synapses were set as listed in Table 2.

As mentioned above, PFs were distributed on the two-dimensional grid plane, and the grid size was determined by each task. Each tile contained 100 PFs. SCs were prepared 1 per tile. Each SC positioned at $(i\Delta s_x, j\Delta s_y)$ could receive excitatory inputs from PFs located in $s_{x,\min} \leq x \leq s_{x,\max}$ and $(j-1)\Delta s_y \leq y \leq (j+1)\Delta s_y$. The number of BCs was the same as that of SCs, and the number of PCs was 20. Both BCs and PCs could receive excitatory PF inputs from all tiles. The BC–BC, BC–PC, PC–BC, and PC–PC connections were constructed based on the grouping manner mentioned in Section A. The actual synapses were formed stochastically (Table 2). In this study, we divided BCs and PCs into two groups for all tasks. Additionally, for the sake of simplicity, we assumed that all CFs synchronized their activity so that our model had a single CF.

Synaptic plasticity was applied to PF–SC and PF–PC synapses. The details of the weight-update rules are described in Equations Eq. (14) and Eq. (15).

Table 1. Neuron-specific parameters. These parameters were adjusted from those used in our previous model (Kuriyama et al., 2021).

Type	SC	BC	PC
C_m [pF]	107	107	107
g_{L} [mS]	2.32	2.32	2.32
E_L [mV]	-68.0	-68.0	-65.0
U_{th} [mV]	-55.0	-55.0	-55.0
U_{r} [mV]	-70.0	-70.0	-66.0
I_{ext} [pA]	30.0	30.0	30.0
ζ [pA]	5.0	1.0	5.0

Table 2. Synaptic parameters for each connection type. A hyphen in a weight cell means it will be modified because of synaptic plasticity.

Connection Type	scaling factor	weight	τ_c [ms]	probability
PF–SC	200.0	-	8.3	0.1
PF–BC	10.0	1.0	8.3	0.5
PF–PC	5.0	-	8.3	0.5
SC–PC	–0.05	0.03	10.0	0.5
BC–BC	–2.0	1.0	10.0	0.3
BC–PC	–1.0	1.0	10.0	0.3
PC–BC	–2.0	1.0	10.0	0.8
PC–PC	–19.8	1.0	5.0	1.0

Finally, the outcome of actions were generated by DCN cells prepared in an equal number of PCs. Each DCN cell was inhibited by its counterpart PC. This configuration means that the activity of each DCN cell, denoted as DCN_i, was modulated by the corresponding PC activity $\mathrm{PC}_i(t)$ defined by Equation 8, as follows:

$$\tau_{\mathrm{DCN}} \frac{d\mathrm{DCN}_i(t)}{dt} = -\left(\mathrm{DCN}_i(t) - \mathrm{DCN}_0\right) - \nu \mathrm{PC}_i(t), \tag{25}$$

where τ_{DCN}, DCN_0, and ν represent the decay time constant, baseline activity of DCN, and a scaling factor, respectively.

Since PCs in the group $\mathrm{PC}_{\bar{\alpha}}$ represented the avoidance of action α and inhibited DCN cells, the activity of DCN group DCN_α represented the preference of actions $h_\alpha(t)$ as follows:

$$h_\alpha(t) = \frac{1}{N_{\mathrm{DCN}_\alpha}} \sum_{i \in \mathrm{DCN}_\alpha} \mathrm{DCN}_i(t), \tag{26}$$

where N_{DCN_α} is the number of neurons in the group DCN_α, and $\mathrm{DCN}_i(t)$ is the activity of ith DCN in the group DCN_α corresponding to action α. Therefore, the most activated DCN group represented the most preferred action so that the final outcome of actions was chosen as follows:

$$a(t) = \underset{\alpha \in \mathcal{A}}{\operatorname{argmax}}\, h_\alpha(t). \tag{27}$$

E. Task Definitions

E.1. Pre- and post-episode processes. As a pre-episode process, at the start of each episode, a free run for 200 ms was carried out to discard initial transient activity that could affect learning. Then, at the end of each episode, a simulation continued for 100 ms while stopping the firing of PFs to update synaptic weights. These pre- and post-episode processes were performed in all tasks.

E.2. Mountain Car Task. The dynamics of the car are described by the following differential equations :

$$\frac{dv_x(t)}{dt} = v_x(t) + \bar{F}a(t) - g\cos(3p_x(t)) \tag{28}$$

$$\frac{dp_x(t)}{dt} = v_x(t), \tag{29}$$

where $\bar{F} = 0.001$ is a force amplitude, $a(t) \in \{-1, 1\}$ represents an action as the force direction, $g = 0.0025$ represents the gravity. Observation spaces of the velocity and position are bounded by $[-1.2, 0.6]$ and $[-0.07, 0.07]$, respectively. When the car collides with the wall, its velocity is immediately reset to 0 without any negative reward associated with the collision. An agent receives constant negative reward every step in the original settings. However, the maximum firing frequency of CF, which is approximately 10 Hz (Zeeuw et al., 1998), was not enough to represent constant n. Therefore, in this study, we modeled a new negative reward function for CF. CF fired when the car was far from the goal and the speed was slow. Thus, the firing rate $\nu(t)$ was defined as follows:

$$\nu(t) = \bar{\nu}\left(1 - \frac{p_{\mathrm{goal}} - p_x(t)}{p_{\mathrm{goal}} - p_{\mathrm{wall}}}\right)\left(1 - \frac{|v_x(t)|}{v_{\max}}\right), \tag{30}$$

where $\bar{\nu}$, p_{goal}, p_{wall}, and $v_{\max}$ are max firing rate of 5 Hz, goal position, left side wall position, and limit of speed respectively. Then a negative reward function is defined as follows:

$$R(t) = \begin{cases} -5 & \text{when CF fires at time } t, \\ 0 & \text{otherwise.} \end{cases} \tag{31}$$

The car starts from the bottom of the valley with a velocity of 0 in every episode. The action $a(t)$ represents the direction in which the agent pushes the car. An episode ends when the car reaches the goal or when the elapsed time exceeds 1,000 ms, with no special negative reward given.

The grid size of the PF plane was 16×16. The parameters of the state value function, defined in Equation Eq. (2), were $\nu = 200$ and $V_0 = 2.48$. The reward discount time constant τ_r, the decay time constant of eligibility trace τ_z, and the decay time constant of the window function τ_κ were set to 100 ms, 20 ms, and 20 ms, respectively. Initial synaptic weights of PF–SC and PF–PC were 0.05 and 0.8. Both learning rates for the critic and the agent were 0.4.

We conducted 10 trials of 1,000 episodes each. We calculated the changes in success rate for each trial using a moving average of results (success=1, failure=0) with a window size of 10 episodes, and then calculated the average across trials.

E.3. Delay Eyeblink Conditioning Task. The dynamics of the eyelid is described as follows:

$$\frac{dp_e(t)}{dt} = 0.01a(t), \tag{32}$$

where $a(t) \in \{-1, 1\}$ represents the action the agent takes at time t. Also, negative reward is defined as follows:

$$r(t) = \begin{cases} -5 & \text{when } t = 500 \text{ and } p_e(t) > 0.1, \\ 0 & \text{otherwise.} \end{cases} \tag{33}$$

The grid size of the PF plane was 16×16. The parameters of the state value function, which defined in Equation Eq. (2), were $\nu = 200$ and $V_0 = 2.48$. The reward discount time constant τ_r, the decay time constant of eligibility trace τ_z, and the decay time constant of the window function τ_κ were set to 100 ms, 20 ms, and 20 ms, respectively. To prioritize the 'open' action, we initially set synaptic weights for PF–PC$_{\text{anti close}}$ to 0.8, while PF–PC$_{\text{anti open}}$ was set to 0.7. We conducted the simulation for 500 episodes with learning rates $\eta_{\text{SC}} = 0.05$ and $\eta_{\text{PC}} = 0.3$.

The whole code of our model and all environments were written in C++. All ordinary differential equations were solved numerically with a forward Euler method with a temporal resolution of 1.0 ms.

Conflict of interest statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author contributions

TY conceived and designed the research. RK and HY contributed the formulation and the implementation of the algorithms. RK wrote the code, performed simulation, and analyzed the data. RK and TY wrote the original draft. RK, HY, and TY discussed on the draft and revised for the final version. All authors contributed to the article and approved the submitted version.

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Supplementary Information

A. Relationship between our weight-update rule and the previous weight-update rule

The weight-update rule and eligibility trace in our model are defined as follows:

$$\frac{d\Delta w^n_{i,j}(t)}{dt} = R(t)z_{i,j}(t) + \frac{\tau_r - \tau_z}{\tau_r \tau_z} V(t) z_{i,j}(t) - V(t) f_{i,j}(t), \tag{S1}$$

$$\frac{dz_{i,j}(t)}{dt} = -\frac{z_{i,j}(t)}{\tau_z} + f_{i,j}(t). \tag{S2}$$

By incorporating the definitions, $\frac{d\Delta w_{i,j}(t)}{dt}$ can be transformed as follows (Equations):

$$\frac{d\Delta w^n_{i,j}(t)}{dt} = R(t)z_{i,j}(t) + \frac{\tau_r - \tau_z}{\tau_r \tau_z} V(t) z_{i,j}(t) - V(t) f_{i,j}(t) \tag{S3}$$

$$= R(t)z_{i,j}(t) - \frac{1}{\tau_r} V(t) z_{i,j}(t) + \frac{1}{\tau_z} V(t) z_{i,j}(t) - V(t) f_{i,j}(t) \tag{S4}$$

$$= R(t)z_{i,j}(t) - \frac{1}{\tau_r} V(t) z_{i,j}(t) + V(t) \left\{ \frac{1}{\tau_z} z_{i,j}(t) - f_{i,j}(t) \right\} \tag{S5}$$

$$= R(t)z_{i,j}(t) - \frac{1}{\tau_r} V(t) z_{i,j}(t) - V(t) \frac{dz_{i,j}(t)}{dt}. \tag{S6}$$

The total weight change over an episode $\Delta w_{i,j}(T_{\text{end}})$ is expressed as follows:

$$\Delta w_{i,j}(T_{\text{end}}) = \Delta w_{i,j}(0) + \int_0^{T_{\text{end}}} \frac{d\Delta w^n_{i,j}(t)}{dt} dt \tag{S7}$$

$$= \Delta w_{i,j}(0) + \int_0^{T_{\text{end}}} \left\{ R(t)z_{i,j}(t) - \frac{1}{\tau_r} V(t) z_{i,j}(t) - V(t) \frac{dz_{i,j}(t)}{dt} \right\} dt \tag{S8}$$

$$= \Delta w_{i,j}(0) - [V(t)z(t)]_0^{T_{\text{end}}} + \int_0^{T_{\text{end}}} \left\{ R(t)z_{i,j}(t) - \frac{1}{\tau_r} V(t) z_{i,j}(t) + \dot{V}(t) z_{i,j}(t) \right\} dt \tag{S9}$$

$$= \Delta w_{i,j}(0) - [V(t)z(t)]_0^{T_{\text{end}}} + \int_0^{T_{\text{end}}} \left\{ R(t) - \frac{1}{\tau_r} V(t) + \dot{V}(t) \right\} z_{i,j}(t) dt. \tag{S10}$$

Here, by assuming $\Delta w_{i,j}(0) = 0$, $z(0) = 0$, and $V(T_{\text{end}}) = 0$, the following equation is obtained:

$$\Delta w_{i,j}(T_{\text{end}}) = \int_0^{T_{\text{end}}} \left\{ R(t) - \frac{1}{\tau_r} V(t) + \dot{V}(t) \right\} z_{i,j}(t) dt. \tag{S11}$$

The expression within the curly brackets is equivalent to the continuous time TD error defined by Doya (2000), which was used by the previous spiking actor-critic framework (Frémaux et al., 2013).

B. Simulation of Linear Track Task

In order to evaluate whether our critic could approximate $-V(t)$, we conducted a simulation of the linear track task (Frémaux et al., 2013). In this task, the environment is a narrow rectangular plane (Figure S1A), on which the agent consistently moves from the leftmost side (start) to the rightmost side (goal) at a fixed speed. Thus, the agent always reaches the goal at the same time, and receives the same negative reward across all episodes. Once the time constant of the reward discount τ_r is determined, the theoretical value can be solved (Figure S1A).

In this task, the plane size was 40×1, and a fixed starting position was $s = 5$. The agent ran with a fixed speed of 0.05 per ms toward a goal area $(s > 39)$. When the agent reached the goal, a negative reward of -5 was given. The parameters of the state value function, which defined in Equation Eq. (2), were $\nu = 300$ and $V_0 = 12.4$. The learning rate and the initial weight of PF–SC were 0.04, and 0.05. The reward discount time constant τ_r, the decay time constant of eligibility trace τ_z, and the decay time constant of the window function τ_κ were set to 100 ms, 20 ms, and 20 ms, respectively.

On the first run, the SC represented a noisy, around zero value function (Figure S1B). Over 100 trials, the SCs learned the value function progressively. Then the SC activity increased exponentially toward the goal, as if it expected the given negative reward (Figure S1B). The averaged value in 20 late trials, as plotted with a black line, nicely matches the theoretical value function. These results were consistent with previous research (Frémaux et al., 2013), and suggest that our weight-update rules were successful and that the SCs were able to approximate the additive inverse of the state value $-V(t)$.

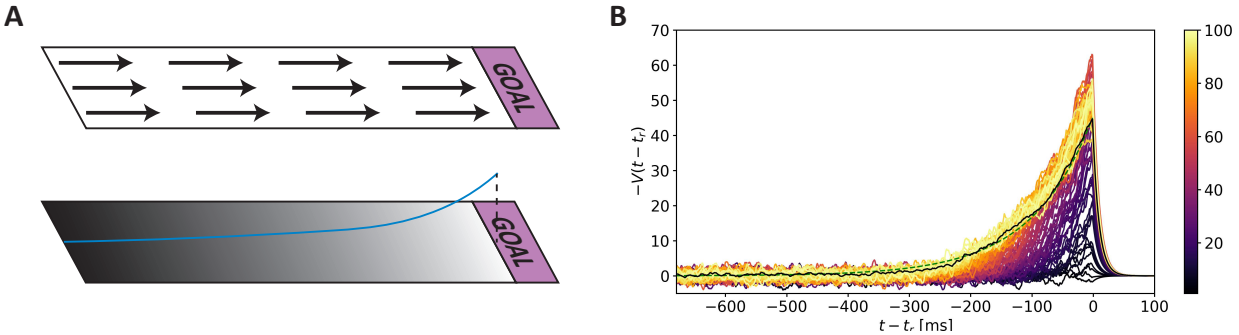

Figure S1. Simulation of Linear Track Task **A**) Environment of the linear track task. Top panel shows the environment and fixed policy. The agent starts at the left edge, and moves straight to the goal shown as a pink area. Bottom panel shows theoretical state value. When the agent follows a fixed policy, the theoretical state value is uniquely determined. **B**) Additive inverse of the state value learned by the critic. Each colored trace shows the value represented by the stellate cell neurons activity against time in 100 first simulation trials (from dark purple in the 1st trial to light yellow in the 100th trial, transitioning through shades of red, orange, and yellow). The plots are aligned with the timing of the negative reward delivery t_r. The black line shows the average of over last 10 trials. The green dashed line shows the theoretical value function.

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