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Flexible Learning Models Utilizing Different Neural Plasticities

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***Abstract*—The existing models for vestibulo-ocular reflex (VOR) and optokinetic response (OKR) learning utilize neural circuit structure and capture a few characteristics of these two learning systems. However, it remains unclear how the error signals guide these learning processes. Here, we propose novel dynamic learning models using error feedback in a flexible fashion to account for both VOR and OKR learning. We first used a feedback modulation model and found the error signals play an essential guiding role in gain compensation of wild-type mice. However, this feedback modulation model cannot accurately reproduce gain changes during the recovery period. Therefore, we propose a non-uniform feedback modulation model using flexible plasticity learning rules of different memory sites to take into account the effect of classical linearity models in both training and recovery periods. To further study learning characteristics of gain reduction, we introduce a reversal-phase feedback modulation model and explore the contribution of synaptic plasticity to adaptive learning, in which characteristics and bidirectional synaptic plasticity in the VOR-decrease learning mode can be fully recovered. Taken together, our results suggest that, to explain VOR and OKR learning systems, one needs dynamical models with flexible and multiple components at different or same sites of neuronal circuits.**

***Index Terms*—Vestibulo-ocular reflex (VOR), optokinetic response (OKR), synaptic plasticity, feedback modulation, learning**

I. Introduction

THE cerebellum contains roughly half of all neurons in the brain [1], yet it has a relatively simple circuit architecture with a few types of neurons, which makes it a tractable model for studying the dynamics and function of neurons and neural circuits [2]–[6]. Although the architecture of cerebellum is simple, it plays an important role in a variety of motor and cognitive functions [7], such as vestibulo-ocular reflex (VOR) [1] and optokinetic response (OKR) [8]. The VOR circuit uses head movement information and visual feedback information to continuously calibrate eye movements to reduce visual errors, which come from the retinal image slip caused by animal's head movement. The VOR gain [9] is the amplitude of eye movement in VOR, which is calculated as dividing the subject's eye movement rate by the head movement rate in experiments. VOR gain-adaptation is the process of continuous calibration of VOR gain by the VOR circuit with gain-increase and gain-decrease modes. When the head movement direction is opposite to the visual target movement direction, it causes an increase in VOR gain, otherwise there is a decrease in VOR gain [10]. Similar to VOR, the OKR gain is the ratio of visual screen movement rate to eye movement rate with the same change induction rules [11]. Experimental results suggested that the stabilization of retinal imaging needs to consider the effects of both VOR (spatial stability) and OKR (visual scene stability) [12], [13].

For the cerebellum, Purkinje cells (PCs), as the only output cells the cerebellum, receive excitatory inputs from granule cells (GCs) and inhibition inputs from molecular layer interneurons (MLIs) [14]–[18]. On the other hand, GCs, as the only input cells of the cerebellum receive sensory information via their synapses, named mossy fibers [4]. As a result, PCs receive sensory information from GCs via their synaptic connections, named parallel fibers [19], and also collect feedback information from vestibular nucleus (VN) via their synaptic connections, named climbing fibers (CFs) [20]. The interaction between these cells and synapses shape the learning process involved in the cerebellum [18].

It has been suggested that there are two branches of VOR loops [3], [21], in one loop vestibular information is through MFs to PFs and then to PCs projecting to the VN, in the end, the VN delivers information to extra-ocular motor neurons. In another loop the vestibular system directly translates information to VN producing the command to motor neurons. Studies have shown that the conjunction of CFs and PFs can induce long-term depression (LTD) [22], [23] or long-term potentiation (LTP) [10], [19] on the synapses between PFs and PCs, resulting in the change of VOR gain. A simple linearly filtered cerebellar model can explain some observations of VOR [24]. Although the existing VOR adaptive models can effectively describe the VOR learning, there are still remaining issues unclear. First, models reproduce the learning in the cerebellum well [14], [25], [26], but they do not have a feedback circuit revealing the teacher effects of CFs to the adaptability of learning [27], [28]. Then, models only consider a linear form for the consolidation of memory learning [24], [26]. However, experimental data show that this incremental approach is too simple to match observations well. Moreover, physiologic studies have shown that the increase and decrease in the VOR gain could be due to a bidirectional plastic mechanism at the same synaptic site [10], [29], which however,

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results in a divergence of VOR adaptive models [30], [31].

In this study, we propose a set of unified dynamic models to capture flexible learning in VOR and OKR. It is generally believed that the feedback loop in VOR from lower olive through CF to PC plays a role in error signals [27], [28], therefore, we propose a feedback modulation model to include the CF error signals that are considered to be balanced values of LTP on the PF-PC synapse and CF activity. We found adding the feedback loop to the existing VOR model can further optimize the VOR adaptive learning gain. We then propose a non-uniform feedback modulation model to take into account long-term learning with an incremental rate of synaptic plasticity, where the sensitivity of LTD between PF-PC and LTP between MF-VN obeys the normal distribution. Finally, we propose a reversal-phase feedback modulation model, in which we implemented the bidirectional plastic mechanism in MF-VN synapses and considered this to be the trigger of the intrinsic change in gain. We further deduced that the change of MF-VN synaptic weights in reversal-phase training mode was the equilibrium value of PF-PC synaptic weights and the synaptic weights of the intermediate molecular layer. With these models, we provide a dynamic yet flexible framework accounting for both VOR and OKR learning.

II. Methods

In this section, we first introduce a learning model of the cerebellum based on the anatomical and morphological results, including the feedback modulation model(FMM) and reversal-phase feedback modulation model(RFMM). Physiological studies have shown that the increase and decrease of the VOR gain is due to the stimulation of different learning mechanisms at the same synaptic site [10]. Therefore, we consider the plasticity mechanism of the PF-PC synapses and MF-VN synapses in FMM and RFMM. The model well describes the bidirectional plasticity mechanism in the reflective circuit, and can simulate the VOR reduction mode and the gain of eye movement changes in the large mode. Then, we propose a non-uniformity feedback modulation model(NFMM) to describe the non-linear change law of the synaptic plasticity LTD and LTP learning rate. Finally, we construct the error signal mediated by CF and intend to add it to the FMM to test whether it has a guiding effect on the VOR system.

A. Neural Model

Similar to the previous work [26], the neural activity $\nu_{VN}(t)$ of vestibular nuclei (VN) was modeled as

$$\nu_{VN}(t) = w_{MF-VN}(t)\nu_{MF}(t) - \nu_{PC}(t) + \nu_{VN,0} \quad (1)$$

where ν_{MF} is the input activity from mossy fibers (MFs), and ν_{PC} is the neural activity from Purkinje cells (PCs). $w_{MF-VN}(t)$ represents synaptic dynamics between MFs and VNs. $\nu_{VN,0}$ is the spontaneous activity of VN neurons.

PCs receive excitatory inputs from granule cells (GCs) and inhibitory inputs from molecular layer interneurons (MLIs) [32], [33], so the neural activity of PCs was modeled as

$$\nu_{PC}(t) = w_{PF-PC}(t)\nu_{GC}(t) - \nu_{MLI}(t) + \nu_{PC,0} \quad (2)$$

where ν_{GC} is the neural activity from GCs, and ν_{MLI} is the neural activity from MLIs. $w_{PF-PC}(t)$ represents synaptic dynamics between GCs and PCs. $\nu_{PC,0}$ is the spontaneous activity of PCs. As MLIs receive excitatory inputs from GCs directly, the neural activity of MLIs can be directly modulated by GCs with a static weight, and was modeled as $\nu_{MLI}(t) = w_{MLI}\nu_{GC}(t)$, where w_{MLI} represents the synaptic weights between GCs and MLIs.

For simplicity, the activity of GCs is assumed to the same as MF inputs, $\nu_{GC}(t) = \nu_{MF}(t)$. Experimental studies have shown that the MF activity is related to optokinetic stimulation, such that the peak modulation amplitude of the screen speed is as large as its average amplitude, and the peak of neuronal dynamics is a characteristic event excited at a certain time [34]. Thus the MF activity can be modeled as $as\nu_{MF}(t) = 1 + \sin(\frac{2\pi t}{T})$ during the head rotation, otherwise, it is the constant 1.

B. Feedback Modulation Model

The feedback modulation model (FMM) involves the impact of CF signals in the feedback loop on learning to establish the feedback regulation mechanism and more accurately represent the learning rule. We used a feedback signal, *i.e.*, the error signal from the inferior olivary nucleus backed to CFs targeting to PCs as in Fig. 1. In the neural circuit, CFs originate from the inferior olivary body passing through the cerebellar cortex and provide strong excitatory synapses for PCs and other inhibitory interneurons. The CF error signals act as a motion command and in turn act on a controlled target. It has been suggested that the retinal error signal is the net difference between the head rotation provided by the vestibular organ and the eyeball information that represents the perceived error of retinal modulation [35].

In order to study the guiding role of error signals, we propose a new model for error signals. In this model, the error signal is generated by the lower olive body and transmitted to the PCs through CFs, and finally to the VN. The error signal guides the PF-PC plasticity learning through the feedback loop. Therefore, the CF signal can be considered as proportionally related to the change of the LTP and the GC activity of the PF-PC plasticity site [25], as follows,

$$e(t) = \eta \times \nu_{MF}(t) - \varsigma \times w_{PF-PC}(t) \times \nu_{MF}(t) + \rho \times \nu_{CF} \quad (3)$$

where η, ς and ρ represent impact factors with values of 1.86, 0.75 and 0.5, respectively, to match the physiological data as previously [25]. $w_{PF-PC}(t)$ represents the influence factor of the MF activity on PCs. ν_{CF} is the CF activity following an exponential distribution $\frac{\lambda^n}{n!}e^{-\lambda}$ with $\lambda = 1$.

Recent studies suggest that multiple synapses in the cerebellar cortex are involved in memory formation, including the LTD on PF-PC synapses to explain short-term PC memory during 1 hour training, whereas the LTP on MF-VN synapses to explain the repeated VN long-term memory formed after 1 hour training, *i.e.*, learning after the transfer of the memory from cerebellar cortex to the brain stem [14]. Thus, we propose a learning model including two parts: the MF-VN and PF-PC synaptic plasticity.

We used a simplified formula for the MF-VN synaptic plasticity as

$$\frac{dw_{MF-VN}}{dt} = \frac{1}{\tau_{MF-VN}}(-w_{MF-VN}(t) + w_{MLI}) \quad (4)$$

For the PF-PC synaptic plasticity, physiological experiments show that in the recovery phase, because the observed object is in the dark, there is no visual information input, so no CF error signal in the feedback loop is generated. Thus, in the recovery phase, the PF-PC synaptic plasticity is only related to the spontaneous decay of the PF-PC synapse and the activated LTP of GCs. The complete PF-PC synaptic plasticity learning rule can be written as follows:

$$\frac{dw_{PF-PC}}{dt} = \begin{cases} \frac{1}{\tau_{learn}}(& -w_{PF-PC}(t) + w_0 \\ & -\varphi_{VOR}(t)) \quad \textit{During} \\ \frac{1}{\tau_{recov}}(& -w_{PF-PC}(t) + w_0) \\ & \textit{After} \end{cases} \quad (5)$$

where τ_{learn} and τ_{recov} are the time constants during and after training, $\tau_{learn} = 1/3$ hours and $\tau_{recov} = 2.5$ hours. w_0 represents the only activated LTP of GCs. $\varphi_{VOR}(t)$ represents the LTD caused by the combination of GCs and CFs, so we simplified $\varphi_{VOR}(t) = \theta_{MF}(t)\theta_{CF}(t)$. $\theta_{MF}(t)$ and $\theta_{CF}(t)$ describe the fluctuation of the MF activity and the CF activity around the mean, respectively, which can be calculated as $\theta_{MF}(t) = \nu_{MF}(t) - \overline{\nu}_{MF}$, and $\theta CF(t) = e(t) - \overline{e}$, where $\overline{\nu}_{MF} = 1$, $\overline{e} = 1$. Therefor, the learning rules of the MF-VN synaptic weight $w_{MF-VN}(t)$ and PF-PC synaptic weight $w_{PF-PC}(t)$ have been effectively expressed, setting $\nu_{VN,0} = 1$, $\nu_{PC,0} = 1$, $w_0 = 1$, $w_{MLI} = 1$ and $\tau_{MF-VN} = 5.5$ hours.

In the VOR-OKR neural system, the gain is the ratio of the output signal rate to the input signal rate. As the final output is the VN activity, following the notation as in [26], eye movement was defined to be in proportion to the modulatory activity of the VN(t) in response to the sinusoidally oscillating screen:

$$Eye(t) = g_{eye}\delta MF(t)(w_{MF-VN}(t) - w_{PF-PC}(t) + w_{MLI}), \quad (6)$$

where g_{eye} is a constant to translate the neuronal activity to eye movement. $\delta MF(t)$ is the fluctuation around the mean. The VOR-OKR gain is defined as the maximum amplitude of the eye movement with respect to the screen oscillation whose amplitude to 1 without loss of generality. Thus, the VOR-OKR gain can be defined as follows:

$$g(t) = g_{eye}2\|\delta MF(t)\|(w_{MF-VN}(t) - w_{PF-PC}(t) + w_{MLI}), \quad (7)$$

and can be further rewritten as

$$g(t) = g_0(w_{MF-VN}(t) - w_{PF-PC}(t) + w_{MLI}) \quad (8)$$

where $\|\delta MF(t)\|$ is the max of $\delta MF(t)$. $g_0 = g_{eye}2\|\delta MF(t)\|$ representing the scale constant and the size is determined by the initial gain value. $g_0 = 0.3$ was used here as in the experiment [26].

C. Non-uniform Feedback Modulation Model

Using the FMM above, it can be seen that, when the neural system is in the learning stage, the gain value increases at a constant speed with time, in other words, the synaptic plasticity increases or decreases linearly with time. However, experimental data show that the gain value grows non-uniformly with a growth rate increasing gradually in a certain period, and then decreasing gradually to zero [36], [37]. Therefore, we propose a non-uniform feedback modulation model (NFMM) assuming that the learning rates of synaptic plasticity at both PF-PC and MF-VN are not static but normally distributed, such that the non-uniformity of learning is considered, and experiment data are more reasonably simulated.

Specifically, the iterative formula of PF-PC synaptic weights is as follows:

$$\frac{dw_{PF-PC}}{dt} = \begin{cases} \frac{1}{\tau_{learn}}h(t)(& -w_{PF-PC}(t) + w_0 \\ & -\varphi_{VOR}(t)) \quad \textit{During} \\ \frac{1}{\tau_{recov}}h(t)(& -w_{PF-PC}(t) + w_0) \\ & \textit{After} \end{cases} \quad (9)$$

where $h(t)$ is a kernel function that adjusts the iterative rule of PF-PC synaptic updates as $h(t) = 19.09\tau\exp(-(t-\tau)^2/98\tau^2)$. $\tau = 24$ hours, the parameters 19.09 and 98 are the optimized values to give the best fitting.

Similarly, we also used $h(t)$ to adjust the LTP learning rate of MF-VN synaptic plasticity. The learning rule of MF-VN synaptic plasticity is

$$\frac{dw_{MF-VN}}{dt} = \frac{1}{\tau_{MF-VN}}h(t)(-w_{PF-PC}(t) + w_{MLI}) \quad (10)$$

The gain was computed in a similar way, except that the synaptic weight w exhibits nonlinear growth instead of linear growth.

D. Reversal-phase Feedback Modulation Model

Motor learning must be able to increase or decrease the amplitude of motion to accommodate the changes of the environment. Physiological studies have shown that the increase and decrease in gain is due to the different learning mechanisms that excite in the same synaptic site. In the gain-decrease training, MFs and PCs are activated at the same time, while in the gain-increase training, MFs and PCs have only one activated neuron [38]. Thus we propose a reversal-phase feedback modulation model (RFMM), such that the bidirectional plastic mechanism in MF-VN synapses was realized, and different learning mechanisms were excited at this site in both decrease and increase modes.

FMM and RFMM share the same loop of neural circuit, but inspire different LTD and LTP in different learning modes. Therefore, the expression of neural activity in RFMM is the same as that in the in-phase learning model except for the PCs and VN activity. We re-established the expression of PC and VN activity. PCs are still directly stimulated by PFs and MLIs, and they have spontaneous inhibitory activity as

$$\begin{aligned} \nu_{PC-rever}(t) =& w_{PF-PC}(t)\nu_{GC}(t) \\ &+ \nu_{MLI}(t) - \nu_{PC,0} \end{aligned} \quad (11)$$

where $w_{PF-PC}(t)$ represents the synaptic plasticity LTD of PF-PC synapses. $\nu_{PC,0}$ indicates the PC spontaneous activity. PCs receive excitatory inputs from PFs and inhibitory inputs from intermediate neurons. In the reversal-phase learning model, the effect of PCs on VN in the gain-decrease stage is exactly opposite with the gain-increase stage. The VN activity can be expressed as

$$\begin{aligned}\nu_{VN-rever}(t) =& w_{MF-VN}(t)\nu_{MF}(t) \\ &+ \nu_{MF}(t) + \nu_{VN,0}\end{aligned} \tag{12}$$

where $w_{MF-VN}(t)$ stands for LTP between MF-VN synapses. $\nu_{VN,0}$ indicates the activity of VN.

PF-PC synapses and MF-VN synapses are still responsible for motor learning, but the plasticity of excited synapses is very different. The learning rules of PF-PC synapse in the gain-decrease learning mode are as

$$\frac{dw_{PF-PC}}{dt} = \begin{cases} \frac{1}{\tau_{learn}}(& -w_{PF-PC}(t) + w_0 \\ & -\varphi_{VOR}(t)) \quad \textit{During} \\ \frac{1}{\tau_{recov}}(& -w_{PF-PC}(t) + w_0) \\ & \textit{After} \end{cases} \tag{13}$$

where $w_0 = 1$ is the spontaneous activity of MFs, $w_{PF-PC}(t)$ is the spontaneous decay of synapses, and $\varphi_{VOR}(t)$ represents the LTD excited by GCs and CFs. It is known from experiments that, when MF and PC are simultaneously activated or resting at the same time, synaptic weights from MFs to VNs decrease; when only one of the two is activated, synaptic weights from MFs to VNs increases. Therefore, the expression of learning plasticity at MF-VN synapses in the gain-increase learning mode is

$$\frac{dw_{MF-VN}}{dt} = \frac{1}{\tau_{MF-VN}}(w_{PF-PC}(t) - w_{MLI}) \tag{14}$$

According to the definition of gain, the expression of decreased gain in the reversal-phase learning mode is

$$g_d(t) = g_{d0}(w_{MF-VN}(t) - w_{PF-PC}(t) + w_{MLI}) \tag{15}$$

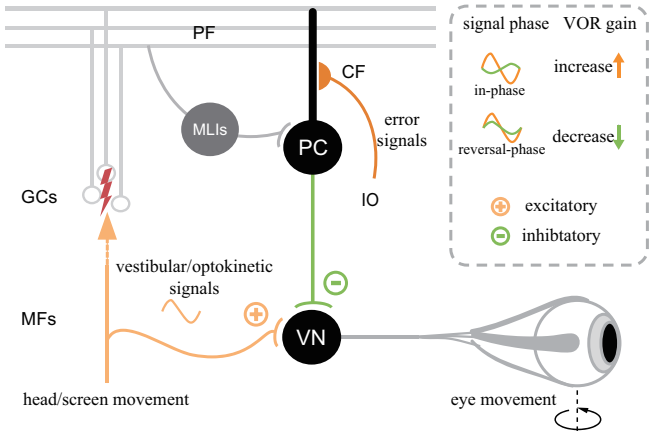
where $g_{d0} = 0.35$ was used in simulations.

III. Results

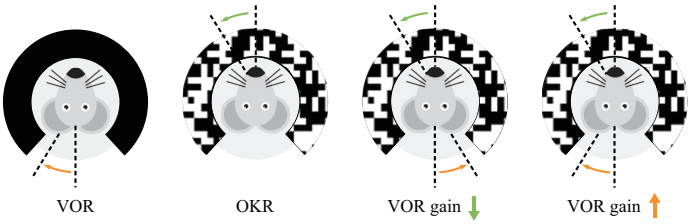
A. Neural Circuit of the VOR and OKR Learning

The neural circuitry involving the VOR and OKR learning is illustrated in Fig. 1, where the cells and fibers play an indispensable role. MFs, as the input systems, penetrate the granular layer of the Cerebellum and convey information from the vestibular system to the cerebellar cortex. PFs transmit the information of the granular layer to PCs, which are the important information transfer units. The axons of PCs are the only output of the cerebellar cortex and form inhibitory synapses with cerebellar nucleus cells. VN receives inhibitory signals from PCs and excitatory signals of MFs, then processes the information to send the resulting information to external motor neurons, which is the central processor in the VOR/OKR system.

VOR learning modes can be divided into the VOR-increase and VOR-decrease modes according to the relative direction of head movement and visual target movement. In the VOR-increase mode, the direction of the subject's head is opposite to the target, which will cause an increase in VOR gain; in the VOR-decrease mode, the direction of the subject's head and the direction of target's are the same, which will cause the VOR gain to decrease (phase reversal) [26]. Here, the VOR gain is defined as the ratio of the eye movement rate of the observer to the head movement rate, reflecting the strength of VOR adaptive learning [Fig.1(b)]. VOR and OKR are operated in a similar way, except that MFs mediate optokinetic signals and vestibular signals, respectively.



(a) Neural circuitry of VOR and OKR learning



(b) Schematic representation of VOR gain

Fig. 1. Illustration of neural circuitry of VOR and OKR learning. (a) During the VOR-increase period, the decrease in the excitation rate of PCs causes the VN to receive the inhibitory signal from the PC, which is out of phase with the excitation signal from the MF, and leads to an increase in VN response and VOR gain. On the contrary, CF increases the excitation rate of PCs during the VOR-decrease period, which caused the inhibitory signal from PC and the vestibular excitation signal from MF in phase, resulting in a smaller VN response and a decrease in VOR gain. Other elements of VOR gain adaptation and OKR gain adaptation are the same except that MFs mediate vestibular signals and optomotor signals, respectively. Therefore, the VOR gain adaptation model is also suitable for OKR gain adaptation with modifying the signal mediated by MFs. (b) Schematic representation of VOR gain. In the experiment, turn the mouse's head in the dark can cause VOR (1st from left); Fix the mouse's head and swipe the screen in front of the mouse can cause OKR (2nd from left). When the direction of screen movement and head movement are the same, VOR gain will increase, otherwise will decrease.

In this study, we first simulated the gain change of the eye movement of wild-types mice in the VOR-increase mode through the feedback modulation model (FMM). Compared to the previous model [denoted as Yamazaki model, [26]] without feedback modulation, our results demonstrate that the CF has a significant guiding effect on the VOR leaning. However, neither the Yamazaki model nor the FMM can accurately fit the experimental results of the recovery period. Therefore, we developed the non-uniform feedback modulation model(NFMM), which can reconstruct the staged non-uniform learning mechanism of PF-PC and MF-VN synapses. The

NFMM can better explain the effects of interval training and concentrated training on the formation of long-term memory. The NFMM is comparably with the Yamazaki model to describe experimental results of different training protocols. Finally, we simulated the gain changes of eye movements in the VOR-decrease mode through reversal-phase feedback modulation model (RFMM) and achieved the two-way plasticity mechanism between MF-VN synapses, that is, different learning mechanisms are stimulated at the same site with different modes.

B. The Guiding Effect of the CF Error Signal on the OKR Learning

The FMM model combines the LTD of PF-PC synapses and the LTP of MF-VN synapses. The MF-VN synapses update synaptic weights according to the correlation between the pre-synaptic MF activity and post-synaptic VN activity. Compared to the Yamazaki model, the FMM innovatively considers the effect of the CF signals in the feedback loop on learning, establishes a feedback adjustment mechanism, and more accurately represents the VOR learning rule. The VOR learning and OKR learning have the similar circuit structure, cell activity, and synaptic learning rules. The difference is that the input signal of OKR is the optokinetic signal instead of the vestibular signal. Therefore, the proposed VOR adaptive learning model, the FMM, is also applicable to the OKR. The experimental data used in this section come from the data used in the Yamazaki model, which is derived from the average value of the OKR experimental gain of a group of mice (12 mice) [26].

We simulated the OKR learning of this group of mice through the FMM model, and the results show that the initial gain is 0.3, the 1 hour training on the first day cause that the gain rapidly increase to 0.12. After the training, the gain gradually decrease and reaches a stable value within 23 hours. These results show that the 1 hour training can form a short-term memory that disappears within 1 day. In other words, the OKR gain does not immediately return to the level before training after each training session. After 5 days of repeated training, the OKR gain gradually increased to 0.55, indicating that the period of 5-day training form a long-term memory [Fig. 2(a)]. The results from the feedback model simulation here perfectly fit physiological experiment data. Furthermore, the feedback adjustment mechanism more accurately fits the OKR learning during training. It can be seen that after the third day, the model does not reach the experimental data after training, and the FMM is closer to the experimental data, with a linear correlation coefficient as 0.98 and the summed square of error as only 0.0019.

Then we observed the changes in the weights of PF-PC and MF-VN synapses [it's change trend is similar to Fig. 2(b)]. The PC-PF synaptic weight is repeatedly reduced five times and can be completely recovered. Instead, the weights between MF and VN synapses increase day by day, mainly during the recovery period. The MF-VN synaptic weights only increase a little during training. After that, the weight continue to increase to a larger value, indicating that the weight change

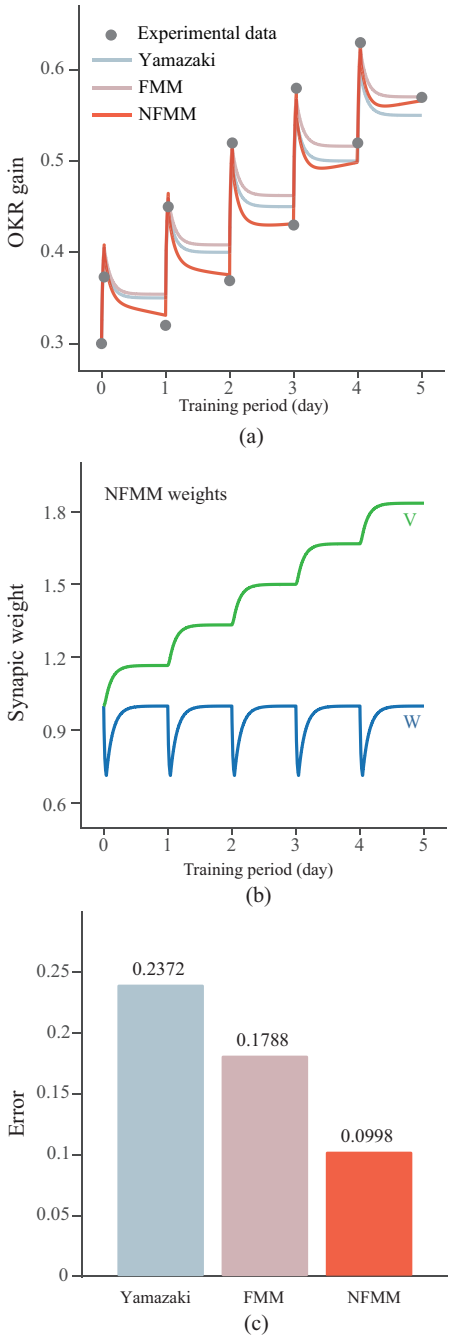


Fig. 2. Experimental data of OKR training explained better by NFMM. (a) Time course of experimental data fitted by the Yamazaki model, FMM, and NFMM. (b) Time course of synapse weight in NFMM. v is the weight of MF-VN and w is the weight of PF-PC. (c) Errors indicated by the Manhattan distance between experimental data (gray data points in (a)) and different modeling data (colored data in (a).

mainly occurs after training. These results indicate that the PF-PC synapse stores short-term memory, which is formed during the 1-hour training and decays within 1 day; on the contrary, the MF-VN synapse stores long-term memory that passes the repeated training and can last up to several days. It is worth noting that the MF-VN synaptic weights did not deviate to infinity or decay spontaneously to 0 after training. The continuous increase of MF-VN weights indicates the formation of long-term memory.

These results suggest that the simulation of the FMM in

the VOR-increase learning stage has reached a very precise effect. In order to illustrate the superiority of the FMM, the error, quantified by the Manhattan distance, of the Yamazaki model and the FMM model are calculated separately, and the correlation between the simulation results of the two models and physiological experimental data [Fig. 2(c)] are compared. The difference between the FMM and the Yamazaki model is significantly. The error distance is significantly reduced, indicating that the model proposed in this paper is superior to the Yamazaki model, and the CF error signal has a clear guiding effect. The VOR learning model FMM established the feedback adjustment mechanism that can discribe the VOR learning more accurately.

It is not difficult to find that, from the simulation results of the FMM, although the OKR gain of FMM can reach the peak after the training period, it can not fit the learning of the recovery period very accurately, which means the synaptic plasticity of the recovery period is insufficiently decayed. Therefore, we propose the NFMM based on the FMM. The NMMM assumes that the learning rate of the synaptic plasticity of the LTD between PF-PC synapses and the LTP between the MF-VN synapses are not uniformed, but following a normal distribution. We found this heterogeneous learning rates can further improve the fitting of physiological experimental data.

From the experimental results obtained by the NFMM [Fig. 2(a)], it can be seen that through the first day's training, the average VOR gain of wild-type mice increase to 0.38 after the training period, then gradually decrease to 0.32 after a 23-hour recovery period; through the second day of training, the gain reaches to 0.47 after the training period, and then gradually decrease to 0.36 after 23 hours; a five-day cycle produces a process in which the gain first increase and then gradually decrease. It can be clearly found that in both the training period and the recovery period, the NFMM shows a good fit to physiological experiment data, particularly capturing the dynamical time course of neural activity. The recovery period is obviously closer to the actual situation.

Fig. 2(b) shows the changes of PF-PC and MF-VN synaptic weights in the NFMM. The PC-PF synaptic weights are repeatedly reduced five times and completely recovered, but the degree of daily decrease is not the same, that is, the daily learning rate changes. The daily increase in synaptic weights between MF-VNs is different, which means that the effectiveness of memory consolidation after training changes every day as the training progresses. Such changes are obviously closer to real learning than linear changes. And the non-uniform learning mechanism in the NFMM predicts that, when the training days are long enough, the weights of the PF-PC synapses and MF-VN synapses can reach a convergent extreme value. Thus, the plasticity of the synapse reaches the upper limit of saturation and cannot strengthen though training.

In addition, the errors between experimental data and models is quantified as the Manhattan distance computing between individual experimental data points and those corresponding model data from the two learning models of NFMM and FMM [Fig. 2(c)]. It shows that the NFMM combined with the non-uniform synaptic learning mechanism has a significantly better result for reproducing physiological experiments than the linear FMM and Yamazaki model. Taken together, these results shows that the NFMM is able to optimize the performance of the learning model very well and explain the missing learning dynamics over time.

C. Spacing Effect for the VOR Gain Adaptation

Experiments have shown that the spaced training scheme interspersed with the effective recovery period provide a stronger long-term memory after 24 hours of the 1-hour centralized training interval [39]. It shows that interval training is more effective than concentrated training in the formation of long-term memory, and this phenomenon is known as the spacing effect in general [40]. We reproduced the spacing effect using our model. Fig. 3 shows the simulated VOR gain obtained in different interval training schemes, including the following four spaced training modes [26]: 1) 1-h concentrated training; 2) 15 mins training rest for 1 hour, repeat 4 times in 1 day; 3) training for 15 mins every day for 4 days; 4) training for 7.5 mins again or for 8 days.

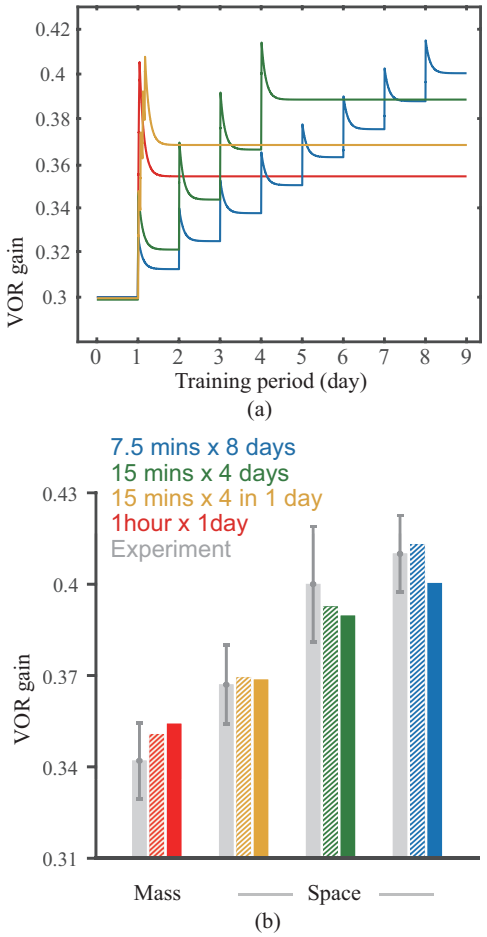


Fig. 3. VOR gain using different training protocols. (a) VOR gain over the training time course using massed training as 1-h training in a day (red), spaced training as 15-min training spaced by 1 h, four times in a day (yellow), spaced training as 15-min training each day for 4 d and total time was 1 h (green), and spaced training as 7.5-min training each day for 8 d and total time was 1 h (blue). (b) Comparison of simulated VOR gain from NFMM (colored) and Yamazaki model (slashed) with experimental data (gray). Colors for simulation data represent the training paradigms as in (a).

In these training modes, intensive training shows the largest instantaneous VOR gain at the moment after the end of

training [Fig. 3 (a)]. The VOR instantaneous gain of the interval training after each training is relatively flat, but the gain increases gradually throughout all training periods. After training, the gain of interval training exceeds the gain of concentrated training [Fig. 3(b)]. The result shows that a proper scheme of interval training can promote the formation of long-term memory, which is consistent with the results given in experiments. The NFMM has a comparable performance for spaced training, compared to the Yamazaki model. The VOR gains of NFMM in different training protocols are slightly better or worse than the Yamazaki model. However, both models are able to reveal the final outcome of training protocols. Instead, our NFMM is more powerful to recover the underlying missing learning dynamics over time, which is important for understanding the learning process.

D. Bidirectional Synaptic Plasticity

The research on the generalization index of monkeys at different training frequencies, i.e., representing the generalization ability of the VOR gain at different test frequencies, shows that the increase and decrease of the VOR gain is not exact inversion on the loop [31]. Therefore, we constructed a reversal-phase feedback modulation model (RFMM) to study the synaptic plasticity learning mechanism in the VOR-increase mode. Some studies [10], [42] have found that the VOR-decrease mode and VOR-increase mode share the same VOR circuit, but stimulate LTD and LTP in different learning modes respectively. As a result, the activity of other cells in the VOR-decrease mode has the same expression as that in VOR-increase mode, except PC and VN activity.

We tested the predictive effect of the RFMM on the $\times 0$ training of wild-type mice in the VOR-decrease mode, in which experimental data were the average value for sample individuals [41]. We computed the Manhattan distance using the data and model results [41], then compare them with the RFMM. Fig. 4(a) describes changes in wild-type mice for the VOR gain, where the gain reduces from 1 to 0.5 and then returns to 0.6 after the first day of training, and the total gain reduction of the first day is 0.4. On the second day, the gain drops sharply from 0.6 to 0.38 after 1-hour training and recovers to 0.52 after 23 hours, the training gain reduction is 0.24. On the third day, the gain decreases from 0.52 to 0.39 after 1-hour training and recovers to 0.52 after 23 hours, the total training gain reduction is 0. On the fourth day, the gain decreases from 0.52 to 0.38 after training for 1 hour and reaches to 0.7 after 23 hours, the total training gain reduction is -0.18. One can easily see that training of the fourth day does not make the gain value decreased but larger, which is consistent with experiments. However, currently there is no theory to explain this phenomenon. Although the VOR gain has been repeated locally, the overall VOR gain has been decreasing during the four days of training.

We analyze the changes of the PF-PC and MF-VN synaptic weights at the same time [Fig. 4(b)]. PC-PF synaptic weights are repeatedly reduced five times and fully recovered. Unlike the VOR-increase, PF-PC synaptic weights of VOR-decrease are at the degree of decrease every day during training. We

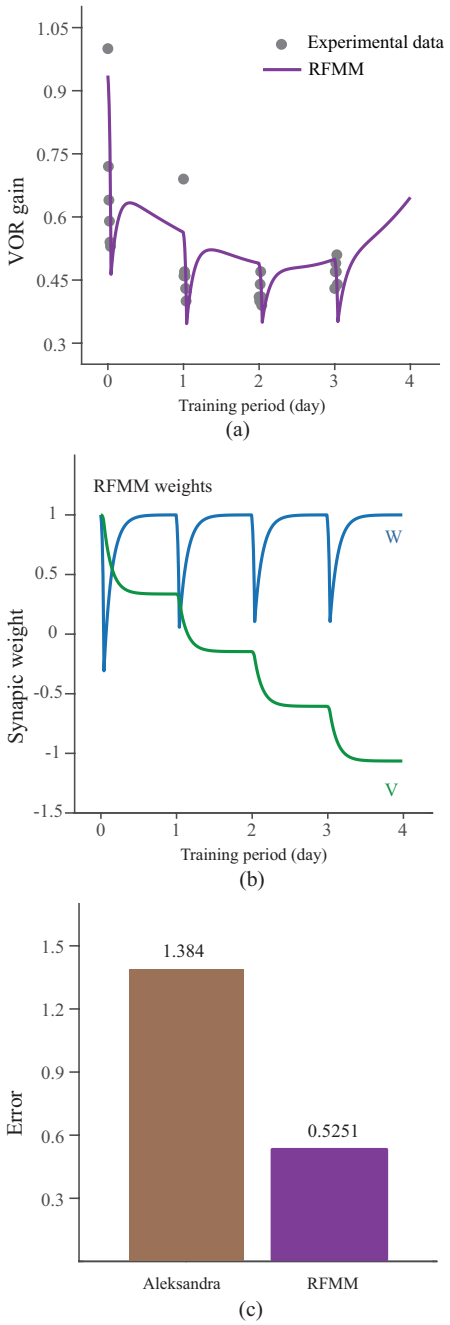


Fig. 4. Experimental data of VOR-decrease training explained better by RFMM. (a) Experimental data is derived from the average gain of a group of mice in the VOR-decrease experiment. The curve is simulated data generated by RFMM. (b) Synapse weight of RFMM. v is the weight of MF-VN and w is the weight of PF-PC. (c) Errors indicated by the Manhattan distance computed by the Aleksandra model [41] and RFMM.

found that, as gain decreases, the magnitude becomes smaller. The weights of MF-VN synapses gradually decreases day by day mainly during training, and are basically unchanged in the recovery period. MF-VN synaptic weights are only reduced 0.15 during the 1-hour training of the first day. After that, the weight continues decreasing to 0.4, which shows that the weight change mainly occurs in the recovery period: gain quickly decreases to -0.19 in 1-hour training, and slowly returns to the initial value in the recovery period. These results indicate that short-term memory is formed during 1 hour of

training and decays within 1 day. In contrast, the MF-VN synapses store long-term memory, which can be obtained from repeated training and last several days.

Although the VOR-decrease mode has the same loop structure as the VOR-increase mode, learning rules are not the same. Therefore, we proposed RFMM based on FMM to reproduce the learning mode when the VOR gain is reduced, and achieved VOR through FMM and RFMM via systems bidirectional synaptic plasticity. In order to illustrate the superiority of RFMM, a previous model was selected for comparison [41]. On the one hand, the correlation coefficient between the simulation data generated by RFMM and the experimental data is 0.86 while the Aleksandra model is 0.68. On the other hand, errors measured by the Manhattan distance between RFMM and experimental data is 0.5251, while the Aleksandra model is 1.384 [Fig. 4(c)]. These results suggest that bidirectional modulation on synapse can capture the learning dynamics of short-term and long-term memory formation.

IV. Conclusion and Discussion

In this study, we presented the FMM to reflect the CF signal's guidance on the VOR gain learning using experimental data of wild-type mice to test the gain and synaptic weight changes in the VOR-increase mode. It can be seen that the FMM approximately reproduced the VOR learning and its effectiveness, with a correlation coefficient of 0.98 for experiment data and the fitting result better than the previous model. These results indicate that the error signal from inferior olivary nucleus transmitted to the PCs via CFs is compared to the signal from the vestibule to guide plasticity learning achieving the calibration of the VOR system. These results are consistent with the previous research on the function of the CF error signals [43].

We then proposed the NFMM on the basis of the FMM to further improve the fitting accuracy for the physiological data of wild-type mice. The simulation results shows that the NFMM with a staged non-uniform synapse learning mechanism reproduced the experimental data of the VOR learning better, in particular, it can overcome the problem that FMM cannot achieve the effective attenuation in the recovery period. This indicates that the synaptic plasticity of the LTD and LTP between PF-PC and MF-VN synapses follows a nonlinear model rather than a uniformly growing model.

Finally, we further proposed the RFMM, using experiment data of wild mice in the VOR-decrease mode, in order to test the bidirectional plasticity mechanism of PF-PC and MF-VN synapses in the VOR learning. The agreement between the simulation results and experiment data confirmed the view that different training protocols stimulate different learning mechanisms in the same memory location so that the direction of the VOR gain changes is different.

A. Feedback Effect

It is generally believed that the feedback loop in VOR is from lower olive through CFs to PCs for adaptive control, and receives the error signal to recalculate the VOR adaptive learning gain. The error signal originates from the lower olive [27] and is transmitted through the CFs acting as a teacher signal [28]. Recent studies found that the CF activity can be stimulated when a difference direction or speed appears between the expected movement and the actual movement of the subject's eyeball [44], [45]. In addition, the CF signal is weakly modulated by head movement in a dark environment. Both suggested that CFs provide PCs with visual error signals and weak head movement signals as another information entry of the VOR neural circuit [25]. The existing models include the synaptic memory site between PCs and PCs, as well as the synaptic memory site between MFs and VN. However, they are can not accurately reproduce the performance of the VOR learning. Therefore, we simulated the CF error signal that is considered to be the balanced value of LTP on the PF-PC synapses, activity of the granular cells and climbing fiber activity. Based on the CF signal model, we added the feedback loop to the existing VOR model and further optimized the VOR adaptive learning gain.

B. Dynamical Learning Mechanisms

The motor learning in the cerebellar cortex is mainly responsible for short-term learning, after which learning is transferred to the cerebellum in a form of memory consolidation, and then form a long-term learning [8]. Long-term learning is constantly gained in the repetitive learning process and forms a memory [46], [47]. The studies on consolidation and transfer of memory in the cerebellum indicate that PC activity guides the plasticity of the target neurons in VN, and the polarity of the PC activity may be calculated by accurately calculating the time of the simple peak relative to the input of the climbing. A simplified learning model [26] was to implement both LTD and LTP at PF–PC and MF–VN synapses so that long-term memory formation occurs after training but not during training, indicating that the memory consolidation occurs during post-training periods [30]. However, the incremental gain per day is a fixed value, which means that the consolidation of memory is an ideal linear model. Similarly, another model [24] was proposed to use synaptic plasticity mechanisms in which either the activity of neurons or the plasticity of synapses were represented by linear equations [48]. However, the data show that the gain value is non-uniformly increasing [36], [37], and its growth rate gradually increases in a certain period, and then gradually reduces. Thus, this fixed incremental approach is too simple and idealized to match the phenomenon observed in the physiological experiment.

Instead, we assumed that the LTD between PF-PC and LTP between MF-VN follow a normal distribution, which refers to the dynamic rate of synaptic plasticity of LTD/LTP. This suggests that in the early (2-3 days) training, the increment of the VOR gain increases and the incremental extremes can be reached in the middle of training period. At the end of the training period, the increase in gain gradually decreases due to the limits of organism, but the gain still presents an increasing trend, which explains why a weakness period can occur in the late of learning. Therefore, our model is able to describe earning dynamics over a time course, instead of a fixed final output of animal performance.

C. Bidirectional Mechanisms at the Same Synapses

Physiologic studies have shown that the increase and decrease in the VOR gain is due to different learning mechanisms at the same synaptic site [10]. The research on generalization index suggests that the learning change of motion amplitude at the loop level is not completely reversible. Two hypotheses have been proposed. One assumes that different training modes provide different neurons, and the other assumes that different training modes can trigger different synaptic sites [31]. Data show that MFs and PCs are activated simultaneously in the training of VOR gain reduction, whereas in the training with increased VOR gain, only MFs or PCs can be activated [33]. Thus, it is necessary to study the VOR adaptive learning in different modes regardless of the reason leading to the difference between two models. Here we implemented the bidirectional plastic mechanism with MF-VN synapses and considered this to cause intrinsic change in VOR gain. During reversal-phase training, the PF-PC synaptic plasticity is the equilibrium value of the MF spontaneous LTD, and synaptic spontaneous attenuation and LTD excited by the co-stimulation of PF and PC. For MF-VN plasticity, the synaptic weights from MF to VN are reduced when MF and PC are simultaneously activated or simultaneously resting. We also further analyzed and deduced that the change of synaptic weights of MF-VN in reversal-phase training mode was the equilibrium value of the synaptic weight of PF-PC and the synaptic weights of intermediate molecular layer. Thus, our model provides a more flexible framework installing multiple mechanisms on the same synapses, in line with the studies combining different types of synaptic plasticity on same synapses [49]–[51], which enhance the capability of neural computation.

D. Limitations

The gain was defined as the ratio of the eyeball output signal to the head input signal [24]. In the model, the corresponding definition is the ratio of the system input and output. In behaviors, when the target is fixed and the head rotation rate is 1 unit, the eye movement is opposite to the head movement and the speed is equal, the gain is 1. When the target speed is 1 unit and the head speed is 1 unit, the object can be seen clearly when the eye is stationary, the gain is 0. When the target moves in 2 units, the head moves in 1 unit, the eye must compensate 1 unit motion rate in direction of the target movement to clear the imaging of the target on the retina, the gain is -1. When the gain changes from 1 to -1, the eye should appear a process of adaptation, that is gradually from the opposite direction of the target movement into the same direction with the target movement. When the target moves at 1 unit in the opposite direction to the head, the eye must adjust the movement in the direction of the target movement. In terms of the inertia law of kinematics, the eyeball moves in 1 unit rate with the head movement, but in terms of relative motion, the eyeball moves in the direction of the target movement toward the head in 2 units. So when the cerebellum fully adapts to this condition (that is, learns a certain memory), the eye must move to the target movement at a rate of 2 units to completely catch the movement of the target, and the movement rate is opposite to the head movement as the gain of 2. Therefore, the positive or negative of gain is not the absolute size, but rather represents the direction of change.

Although the FMM reproduces the learning of in-phase OKR, different learning requires different kernel functions to adapt. Therefore, we need a more dynamic model to train or choose the appropriate kernel function actively. We could not test the validity of our model with more data due to limited experimental data, so the robustness of the model remains to be seen using more data in the future studies. While the reversal-phase VOR learning model reproduces data from a set of VOR reversal phase training, the correlation coefficient between the simulated data and the experimental data may be not ideal, so one can also find a more appropriate kernel function to adjust learning. It has suggested that the learning mechanism of VOR-decrease mode and VOR-increase mode could not be accurately reversed [41]. Although the model in this study reflects this view to some extent, more in-depth research is still needed to explore the essential differences between two modes of the VOR learning.

When the brain receives different stimuli to implement tasks, one needs to switch between different computational rules accounting for flexible working schemes. Specifically, the neural system needs to implement different learning mechanisms on the same memory site to adapt environmental changes. Thus, one needs to have models for dynamic switching or routing information as seen in other brain areas [52] or using different machnisems, such as neural synchrony [53]. One of the limitations in our models is that they can not accommodate the LTD and LTP in the same network loop. For now, our FMM, NFMM, and RFMM use different learning mechanisms for different simulations. The future work is needed to implement a routing function for different tasks.

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References

[1] W. Lange, "Cell number and cell density in the cerebellar cortex of man and some other mammals," *Cell & Tissue Research*, vol. 157, no. 1, pp. 115–124, 1975.

[2] L. Koziol, D. Budding, N. Andreasen, S. D'Arrigo, and H. Imamizu, "Consensus paper: The cerebellum's role in movement and cognition," *The Cerebellum*, vol. 13, pp. 151–177, 2013.

[3] C. De Zeeuw and M. ten Brinke, "Motor learning and the cerebellum," *Cold Spring Harbor perspectives in biology*, vol. 7, Sep. 2015.

[4] V. Zampini, J. K. Liu, M. A. Diana, P. P. Maldonado, N. Brunel, and S. Dieudonné, "Mechanisms and functional roles of glutamatergic synapse diversity in a cerebellar circuit," *eLife*, vol. 5, e15872, 2016.

[5] L. An, Y. Tang, Q. Wang, *et al.*, "Coding capacity of purkinje cells with different schemes of morphological reduction," *Frontiers in Computational Neuroscience*, vol. 13, May 2019. DOI: 10.3389/fncom.2019.00029.

[6] L. An, Y. Tang, D. Wang, *et al.*, "Intrinsic and synaptic properties shaping diverse behaviors of neural dynamics," *Frontiers in Computational Neuroscience*, vol. 14, Apr. 2020. DOI: 10.3389/fncom.2020.00026.

[7] S. Armano, P. Rossi, V. Taglietti, and E. D'Angelo, "Long-term potentiation of intrinsic excitability at the mossy fiber-granule cell synapse of rat cerebellum," *Journal of Neuroscience the Official Journal of the Society for Neuroscience*, vol. 20, no. 14, p. 5208, 2000.

[8] F. Shutoh, M. Ohki, H. Kitazawa, S. Itohara, and S. Nagao, "Memory trace of motor learning shifts transsynaptically from cerebellar cortex to nuclei for consolidation," *Neuroscience*, vol. 139, no. 2, pp. 767–777,

[9] Sebastiaan, Koekkoek, Arjan, *et al.*, "Gain adaptation and phase dynamics of compensatory eye movements in mice," *Genes & Function*, 1997.

[10] E. S. Boyden and J. L. Raymond, "Active reversal of motor memories reveals rules governing memory encoding," *Neuron*, vol. 39, pp. 1031–1042, 2003.

[11] M. Kawato and H. Gomi, "The cerebellum and vor/okr learning models," *Trends in Neurosciences*, vol. 15, no. 11, pp. 445–453, 1992.

[12] Robinson and D. A, "The use of control systems analysis in the neurophysiology of eye movements," *Ann Rev Neurosci*, vol. 4, no. 1, pp. 463–503, 1981.

[13] G. Schweigart, T. Mergner, I. Evdokimidis, S. Morand, and W. Becker, "Gaze stabilization by optokinetic reflex (okr) and vestibulo-ocular reflex (vor) during active head rotation in man," *Vision Research*, vol. 37, no. 12, 1997.

[14] P. Wulff, M. Schonewille, M. Renzi, *et al.*, "Synaptic inhibition of purkinje cells mediates consolidation of vestibulo-cerebellar motor learning," *Nature neuroscience*, vol. 12, pp. 1042–9, Sep. 2009.

[15] M. Jelitai, P. Puggioni, T. Ishikawa, A. Rinaldi, and I. Duguid, "Dendritic excitation–inhibition balance shapes cerebellar output during motor behaviour," *Nature Communications*, vol. 7, no. 1, p. 27 976 716, Dec. 2016. DOI: 10.1038/ncomms13722.

[16] Y. Tang, L. An, Y. Yuan, Q. Pei, Q. Wang, and J. K. Liu, "Modulation of the dynamics of cerebellar Purkinje cells through the interaction of excitatory and inhibitory feedforward pathways," *PLOS Computational Biology*, vol. 17, no. 2, A. C. Gajic, Ed., e1008670, Feb. 2021, Publisher: Public Library of Science. DOI: 10.1371/journal.pcbi.1008670.

[17] Y. Tang, L. An, Q. Wang, and J. K. Liu, "Regulating synchronous oscillations of cerebellar granule cells by different types of inhibition," *PLOS Computational Biology*, vol. 17, no. 6, M. Migliore, Ed., e1009163, Jun. 2021, Publisher: Public Library of Science. DOI: 10.1371/journal.pcbi.1009163.

[18] C. I. D. Zeeuw, S. G. Lisberger, and J. L. Raymond, "Diversity and dynamism in the cerebellum," *Nature Neuroscience*, Dec. 2020, Publisher: Springer Science and Business Media LLC. DOI: 10.1038/s41593-020-00754-9.

[19] C. Hansel, D. J. Linden, and E. D. D'Angelo, "Beyond parallel fiber ltd: The diversity of synaptic and non-synaptic plasticity in the cerebellum," *Nature Neuroscience*, vol. 4, no. 5, pp. 467–475, 2001.

[20] Y. Zang and E. D. Schutter, "Climbing Fibers Provide Graded Error Signals in Cerebellar Learning," *Frontiers in Systems Neuroscience*, vol. 13, Sep. 2019, Publisher: Frontiers Media SA. DOI: 10.3389/fnsys.2019.00046.

[21] M. Ito, *The Cerebellum and Neural Control.* Raven Press, 1984.

[22] T. V. P. Bliss and T. Lømo, "Long-lasting potentiation of synaptic transmission in the dentate area of the anaesthetized rabbit following stimulation of the perforant path," *Journal of Physiology*, vol. 232, no. 2, 1973.

[23] Ito and M, "Cerebellar control of the vestibulo-ocular reflex–around the flocculus hypothesis.," *Annual Review of Neuroscience*, vol. 5, no. 1, p. 275, 1982.

[24] J. L. Raymond and S. G. Lisberger, "Neural learning rules for the vestibulo-ocular reflex," *Journal of Neuroscience*, vol. 18, no. 21, pp. 9112–9129, 1998.

[25] Clopath, Claudia, Badura, *et al.*, "A cerebellar learning model of vestibulo-ocular reflex adaptation in wild-type and mutant mice.," *Journal of Neuroscience*, 2014.

[26] T. Yamazaki, S. Nagao, W. Lennon, and S. Tanaka, "Modeling memory consolidation during posttraining periods in cerebellovestibular learning," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 112, no. 11, pp. 3541–3546, 2015.

[27] M. Ito, "Cerebellar learning in the vestibulo-ocular reflex," *Trends in Cognitive Sciences*, vol. 2, no. 9, pp. 313–21, 1998.

[28] M. Ito, "Ito, m.: Cerebellar circuitry as a neuronal machine [j]. prog. neurobiol. 78(3-5), 272-303," *Progress in Neurobiology*, vol. 78, no. 3-5, pp. 272–303, 2006.

[29] M. Coesmans, J. Weber, C. De Zeeuw, and C. Hansel, "Bidirectional parallel fiber plasticity in the cerebellum under climbing fiber control," *Neuron*, vol. 44, pp. 691–700, Nov. 2004.

[30] V. Lev-Ram, S. Mehta, D. Kleinfeld, and R. Tsien, "Reversing cerebellar long-term depression," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 100, pp. 15 989–93, Jan. 2004.

[31] R. R. Kimpo, E. S. Boyden, A. Katoh, M. C. Ke, and J. L. Raymond, "Distinct patterns of stimulus generalization of increases and decreases in vor gain," *Journal of Neurophysiology*, vol. 94, no. 5, pp. 3092–3100, 2005.

[32] P. Seja, M. Schonewille, G. Spitzmaul, A. Badura, and T. J. Jentsch, "Raising cytosolic cl - in cerebellar granule cells affects their excitability and vestibulo-ocular learning," *Embo Journal*, vol. 31, no. 5, pp. 1217–1230, 2012.

[33] E. Galliano, Z. Gao, M. Schonewille, *et al.*, "Silencing the majority of cerebellar granule cells uncovers their essential role in motor learning and consolidation," *Cell reports*, vol. 3, Apr. 2013.

[34] W. Gerstner, "Spiking neuron models," in Jan. 2010, pp. 277–280.

[35] M. Ito, "Error detection and representation in the olivo-cerebellar system," *Frontiers in neural circuits*, vol. 7, p. 1, Feb. 2013.

[36] R. Schmid, A. Buizza, and D. Zambarbieri, "A non-linear model for visual-vestibular interaction during body rotation in man," *Biological Cybernetics*, vol. 36, no. 3, pp. 143–151, 1980.

[37] E. Koenig, J. H. J. Allum, and J. Dichgans, "Visual-vestibular interaction upon nystagmus slow phase velocity in man," *Acta Oto-Laryngologica*, vol. 85, no. 1-6, pp. 397–410, 1978.

[38] J. Menzies, J. Porrill, M. Dutia, and P. Dean, "Synaptic plasticity in medial vestibular nucleus neurons: Comparison with computational requirements of vor adaptation," *PloS one*, vol. 5, 2010.

[39] T. Okamoto, S. Endo, T. Shirao, and S. Nagao, "Role of cerebellar cortical protein synthesis in transfer of memory trace of cerebellum-dependent motor learning," *Journal of Neuroence*, vol. 31, no. 24, pp. 8958–8966, 2011.

[40] Ebbinghaus and Hermann, "Memory: A contribution to experimental psychology," *Annals of Neurosciences*, vol. 20, no. 4, 2013.

[41] Aleksandra, Badura, Claudia, *et al.*, "Modeled changes of cerebellar activity in mutant mice are predictive of their learning impairments.," *Scientific Reports*, 2016.

[42] C. D. Aizenman, P. B. Manis, and D. J. Linden, "Polarity of long-term synaptic gain change is related to postsynaptic spike firing at a cerebellar inhibitory synapse," *Neuron*, vol. 21, no. 4, p. 827, 1998.

[43] R. R. Kimpo, J. M. Rinaldi, C. K. Kim, H. L. Payne, and J. L. Raymond, "Gating of neural error signals during motor learning," *Elife*, 2014.

[44] J. I. Simpson, D. R. W. Wylie, and C. I. De Zeeuw, "More on climbing fiber signals and their consequence(s)," *Behav & Brain Sci*, vol. 19, no. 03, pp. 496–498, 1996.

[45] K. Voges, B. Wu, L. Post, M. Schonewille, and C. De Zeeuw, "Mechanisms underlying vestibulo-cerebellar motor learning in mice depend on movement direction," *The Journal of physiology*, vol. 595, Jun. 2017.

[46] Kassardjian and D. C., "The site of a motor memory shifts with consolidation," *Journal of Neuroscience*, vol. 25, no. 35, pp. 7979–7985, 2005.

[47] T. Okamoto, T. Shirao, F. Shutoh, T. Suzuki, and S. Nagao, "Post-training cerebellar cortical activity plays an important role for consolidation of memory of cerebellum-dependent motor learning," *Neuroscience Letters*, vol. 504, no. 1, pp. 53–56, 2011.

[48] Y. Yang and S. G. Lisberger, "Role of plasticity at different sites across the time course of cerebellar motor learning," *Journal of Neuroscience the Official Journal of the Society for Neuroscience*, vol. 34, no. 21, pp. 7077–7090, 2014.

[49] J. K. Liu, "Learning rule of homeostatic synaptic scaling: Presynaptic dependent or not," *Neural computation*, vol. 23, no. 12, pp. 3145–3161, 2011.

[50] J. K. Liu and D. V. Buonomano, "Embedding multiple trajectories in simulated recurrent neural networks in a self-organizing manner," *Journal of Neuroscience*, vol. 29, no. 42, pp. 13 172–81, 2009.

[51] F. Zenke and W. Gerstner, "Hebbian plasticity requires compensatory processes on multiple timescales," *Philosophical Transactions of the Royal Society B: Biological Sciences*, vol. 372, no. 1715, p. 20 160 259, Mar. 2017. DOI: 10.1098/rstb.2016.0259.

[52] C. Donahue and D. Lee, "Dynamic routing of task-relevant signals for decision making in dorsolateral prefrontal cortex," *Nature Neuroscience*, vol. 18, no. 2, pp. 295–301, Jan. 12, 2015. DOI: 10.1038/nn.3918.

[53] A. Palmigiano, T. Geisel, F. Wolf, and D. Battaglia, "Flexible information routing by transient synchrony," *Nature Neuroscience*, vol. 20, no. 7, pp. 1014–1022, May 22, 2017. DOI: 10.1038/nn.4569.

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