

## CHAPTER 5

# **Integrated learning and modulatory effects of dopamine in cortico-basal ganglia networks: temporal difference learning of MSN receptive fields for a memory task**

### **5.1. Overview**

As shown in the preceding chapter, it was shown that dopamine induced bistability of striatal medium spiny neurons locks the activity in the basal ganglia; this modulation helps preserve cortical activity encoding for salient stimuli that trigger dopamine release and thus protects the working memory of these stimuli from corruption by noise and distraction. The locking of the gate in the basal ganglia follows from the dual enhancing/suppressing effect of dopamine discussed in Chapter 2; dopamine induced bistability increases the activation threshold for reaching the active up state, but once the threshold is exceeded, MSNs display enhanced firing frequency and duration. As shown in fig 2.10, dopamine controls the input threshold for reaching the up state, but it is the level of cortical input to each MSN that determines whether the unit is enhanced or suppressed. In this chapter, I demonstrate that dopamine mediated plasticity of cortico-striatal connections can control the excitatory input to MSNs so as to implement the gating properties demonstrated in Chapter 4. In this chapter, dopamine release is controlled by a variant of the temporal difference (TD) algorithm that reasonably approximates the response of dopamine neurons. Dopamine acts as a third factor in a Hebbian-like learning rule for plasticity of the striatal afferents that is suggested by experimental evidence. The preliminary results described in this chapter demonstrate that this learning rule in combination with the TD learning signal is sufficient for the network to learn to perform spatial memory tasks; the MSNs develop Gaussian-like receptive fields similar to those built into the network model in Chapter 4, and an appropriate gating function is realized in the basal ganglia.

### **5.2. Introduction**

The involvement of dopamine in learning is demonstrated by the deficiencies in learning new tasks exhibited by human subject with pathologies of the dopamine system. For instance, Parkinson's subjects are less able to extract probabilistic relationships in simple prediction games (Knowlton et al., 1996). Behavioral experiments in animal models confirm this involvement: unilateral dopamine depletion retards the acquisition of new arm-reaching tasks on the contralateral side, but does not impede learning on the ipsilateral side (Miyashita et al., 1995). At the cellular level, dopamine has been demonstrated to be involved in synaptic plasticity of the afferent inputs to medium spiny neurons (Calabresi et al., 1997; Reynolds and Wickens, 2000; Reynolds and Wickens, 2002). The precise nature of the learning at these synapses is still under debate. However, convincing evidence indicates that, in general, dopamine may act as a third factor in a correlational learning scheme (Reynolds and Wickens, 2002): synaptic weights increase when elevation of both pre- and post-synaptic activity is accompanied by an increase in dopamine, while synaptic weights decrease when elevation of both pre- and post-synaptic activity is accompanied by a decrease in dopamine.

The response properties of dopamine releasing neurons have been reviewed in Chapter 1; these neurons exhibit a burst response to the delivery of unexpected rewards and to the presentation of

stimuli which reliably precede rewards, and they pause when an expected reward is not delivered. These properties have led to the proposal (Barto, 1995; Montague et al., 1996) that the release of dopamine is a neural equivalent of the reward value prediction signal in temporal difference (TD) algorithms. These algorithms, which implement reinforcement learning, TD have been successfully applied to a range of difficult learning problems such as pole balancing, backgammon, and robot control (see Sutton and Barto (1998)), and have been explored in biologically inspired networks developed to understand the neural substrates of reward-based learning (Montague et al., 1995; Suri et al., 2001) and motor skill acquisition (Nakahara et al., 2001). In this chapter, I evaluate whether the TD learning signal can be utilized so as to establish the appropriate input connectivity to medium spiny neurons in the striatum, in order to correctly perform the memory guided saccade task presented in Chapter 4.

### 5.3. Methods

The network properties described in Chapter 4 were explored under the assumption in which the memory task had already been learned; the network connections were set so as to achieve perfect performance. I now explore whether appropriate connections from input units to striatal units can be established through dopamine mediated learning so as to achieve good performance of the memory task. The network model used in this investigation is similar to the one presented in Chapter 4, except for some significant changes in connectivity (see fig 5.1). First, direct connections from input units to PFC units are excluded, as these connections could implement good network performance without the need to learn the input-striatal connections. It is possible that connections from input to PFC units could be learned simultaneously with (or slightly follow) learning the input to striatal connections. Second, the input-striatal weights are initially random; these connections are then learned with a TD signal coupled with a dopamine controlled learning rule. Third, uniform inhibitory collaterals between striatal units are included so as to reduce the amount of activity in the striatum that results from the initially random afferent connections to striatal spiny neurons; the resulting sparse activity in the striatum facilitates the formation of a bump state in the cortex, which is critical for the robustness of the type of unsupervised learning explored in this chapter (if the network does not establish a bump state following input presentation, correct output will not be achieved and learning cannot take place). Fourth, the release of dopamine is now controlled by the TD algorithm, as formulated in previous studies of dopamine-assisted learning (Montague et al., 1996).

Figure 5.1 shows a schematic illustration of the resulting network and the components of the TD algorithm. The unit  $D$  represents a small group of dopamine releasing neurons. These neurons collect inputs  $r(\tau)$  representing reward related and/or salient information from the environment and within the organism. Convergent information from cortical representations of the state  $X(i, \tau)$  of unit  $i$  at time  $\tau$  is collected by the unit  $Q$ , which in turn signals to the dopamine releasing neurons the temporal derivative  $\dot{V}(\tau)$  of the value of the cortical state. This derivative is computed as an increment from time  $\tau - 1$  to time  $\tau$ :  $\dot{V}(\tau) = V(\tau) - V(\tau - 1)$ , where  $V(\tau) = \sum_i W^{QX}(i, \tau)X(i, \tau)$  is a weighted sum of the cortical representation at time  $\tau$ . This cortical representation of the input is a "tapped-delay-line" in which an initial pattern of cortical activity propagates through the time-labeled cortical arrays like a traveling wave;  $\bar{X}(0) = \bar{X}_T$  is copied sequentially to consecutive arrays with increasing time indices (i.e. at time  $\tau = 3$ ,  $\bar{X}(\tau = 3) = \bar{X}_T$  and  $\bar{X}(\tau \neq 3) = \bar{0}$ ).

The output  $\delta(\tau)$  of the dopamine unit is the sum of its input and some basal activity  $b(\tau)$ :

$$(5.1) \quad \delta(\tau) = r(\tau) + V(\tau) - V(\tau - 1) + b(\tau).$$

The goal of temporal difference methods is to learn weights  $W^{QX}$  so that the current state value  $V(\tau)$  is an estimate of the sum of future rewards. The role of  $\delta(\tau)$  is to report inconsistencies in the prediction of reward; the objective of learning is to achieve  $\delta(\tau) = 0$ . The connections  $W^{QX}$



$\delta(\tau)$  is a prediction error that indicates whether the future forecasted reward is better than expected [ $\delta(\tau) > b(\tau)$ ] or worse than expected [ $\delta(\tau) < b(\tau)$ ]. In the early trials of a conditioning task, the delivery of unexpected reward ( $r(\tau) > 0$ ) results in a positive spike in  $\delta(\tau)$ . After learning has occurred, an unexpected presentation of the conditioned cue elicits an increase in  $\delta(\tau)$ , but the subsequent delivery of the reward does not elicit a prediction error because it is anticipated. The omission of an anticipated reward leads to a negative spike in  $\delta(\tau)$ . The response properties represented by  $\delta(\tau)$  are qualitatively similar to the properties of dopamine responses (see Chapter 1).

In this model, the prediction error  $\delta(\tau)$  represents the concentration of dopamine in the network, which has modulatory and learning effects. The modulatory effects are implemented by triggering  $\gamma(t)$  whenever  $\delta(\tau)$  is positive. The modulatory gain factor  $\gamma(t)$  represents the dopamine mediated modulation of striatal membrane properties and has the same time dependence as presented in Chapter 2. The value of  $\delta(\tau)$  affects learning of the TD connections  $w^{QX}$ , as described in eq 5.2. The value of  $\delta(\tau)$  also affects learning of the input to striatal connections  $W^{ST}$ : dopamine acts as a third factor in a Hebbian correlational learning rule:

$$(5.3) \quad \Delta W^{ST}(i, j) = \eta^{ST} Z^S(i, \tau) Z^T(j, \tau),$$

where  $\eta^{ST}$  is a learning rate, and  $Z^S(i, \tau)$  and  $Z^T(j, \tau)$  are traces of the activity of striatal and input units, respectively. This expression is consistent with experimental evidence for the role of dopamine in mediating striatal plasticity (Reynolds and Wickens, 2002). The connection weights  $W^{ST}(i, j)$  and the activity traces are updated every time step  $\tau$ . The input activity trace  $Z^T(j, \tau)$  tracks the maximal activation of each input unit  $j$ :  $Z^T(j, \tau) = \max\{r^T(j, \tau - 1), r^T(j, \tau)\}$ , where  $r^T(j, \tau)$  is the firing rate activity of the  $j$ th input unit at time  $\tau$ . The trace of the striatal units is an integrator:  $\dot{Z}^S(i) = \eta^S[r^S(i) - Z^S(i)]$ . This term is necessary because the competition in the striatal network, implemented by the inhibitory collaterals  $W^{SS}$ , allows for transient activation of striatal units that do not correspond to the 'winning' units. It is these winning units that control the switching of the cortical network; therefore, the input to these units should be modified by the learning algorithm, while the input to transiently active striatal units should not be modified. The low-pass filtering properties of the striatal trace  $Z^S(i, \tau)$  ensures that learning is directed toward the units that exhibit sustained activity and not the units that are briefly activated during the dynamic settling of the competitive striatal network.

The parameters that characterize the learning process and the underlying network are listed in table 5.1.

The dynamics of the input, striatal, and cortical units follows from integration with small time steps ( $\Delta t = 0.01 - 5.0$  ms), to achieve the same error tolerance as in Chapter 4. The TD algorithm operates on a relatively coarse time scale; the resolution of the learning algorithm is determined by a time bin of size  $\Delta\tau=20$  ms. The learning rates for the TD algorithm  $\eta^{QX} = 0.3$  and the striatal units  $\eta^{ST} = 0.4$  were chosen so that learning took place during a reasonable number of trials. A finer time resolution (more bins of smaller duration) would change the dynamics of learning but would not alter the general behavior of the network. The change in dynamics can be compensated by a modification of the learning rates; as long as the product  $\eta$  times  $\delta(\tau)$  is constant, similar learning dynamics can be achieved for time steps of different size. Methods also exist to implement the learning algorithm in continuous time; these typically involve some sort of trace signal that provides a mechanism for solving the problems of spatial and temporal credit assignment. The exploration of these possibilities is outside of the scope of the current project, but it remains an interesting direction for future investigation.

Table 5.1. Learning and network parameters

|             |     |
|-------------|-----|
| $W^{ET}$    | 0   |
| $W^{SS}$    | 0.1 |
| $\eta^{QX}$ | 0.3 |
| $\eta^{ST}$ | 0.4 |
| $\eta^S$    | 4   |

#### 5.4. Results

The simulations reported here demonstrate the capacity of the network to learn the appropriate memory encoding for spatially-guided delayed response tasks. The initialization of the connections  $W^{ST}$  between input units and striatal units is drawn from a uniform distribution in the interval [0.021, 0.063]; this interval was chosen so that the summed activation for each MSN unit was above activation threshold. If MSNs are not activated by the input activity, learning cannot take place. Four equidistant angular positions are chosen for target locations. In each learning trial, a target is randomly chosen from these four positions and presented to the network by activating the corresponding group of input units for 400 ms. The angle encoded by the bump in the PFC network is read out 500 ms after target offset. This relatively short delay was used in order to reduce simulation time; since no noise was included in the PFC network, there is no noise-induced memory drift and the delay duration has no effect on performance. If the encoded memory is within 0.1 radians of the target position, a reward  $r(t) = 1$  is given; otherwise there is no reward and  $r(t) = 0$ . A technique called optimistic initialization, in which the network initially expects every trial to be rewarded, is used in order to generate negative reward predictions  $\delta(\tau) < b$  and promote exploration. This technique is frequently used in unsupervised learning algorithms in which action selection is initially highly stochastic. Figure 5.2 shows the initial random input to striatal connections, and the values of these connections after 1000 trials. The connectivity matrix after learning shows that a few striatal units develop receptive fields similar to the Gaussian connectivity assumed in Chapter 4. I suspect that the graded tails of the receptive fields follow from the graded Gaussian form of the input activation that encodes the corresponding target locations. After training, the network is able to form four bump states; one for each target location. Other target positions cannot be encoded without additional training. Preliminary simulations indicate that in a single training period of up to 10,000 trials, the network is not able to learn appropriate Gaussian receptive fields of all striatal units such that any angle can be encoded by a single set of connectivities. This could be due to an insufficiently small number of trials, or sub-optimal values for the learning parameters. In principle, it seems reasonable to expect that the network could learn appropriate receptive fields for all striatal units such that any arbitrary target location can be encoded, as in Chapter 4.

#### 5.5. Conclusion

The results presented in this section indicate that the proposed role of dopamine in mediating learning of cortico-striatal connections in combination with a reasonable approximation of the response properties of dopamine neurons implemented by the TD signal is likely to be sufficient to learn appropriate receptive fields of MSNs for the spatial memory task. The coordinated role of dopamine as an agent for both learning and modulation in the striatum remains understudied; many interesting questions are yet to be addressed. For instance, can dopamine mediated learning control the cortical input to striatal units appropriately to exploit the enhanced threshold for reaching the active up state in high dopamine conditions so as to selectively enhance those features of the converging input that are most salient? For example, one would imagine that the subjects in the saccade

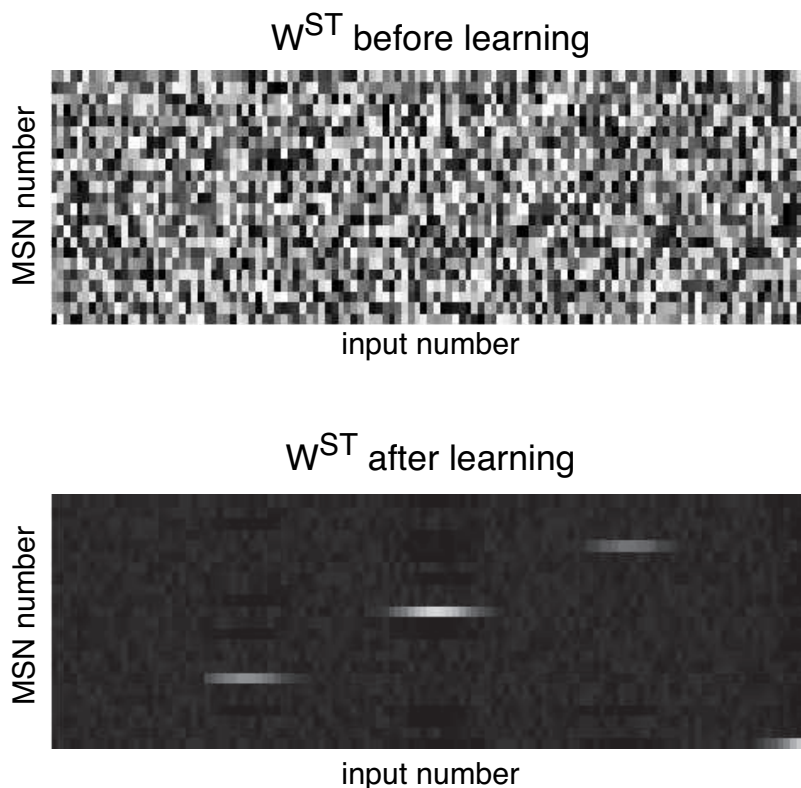


Figure 5.2. Striatal afferent connections before and after learning. The values of the connections from input units (arranged on the ordinate by increasing preferred direction) to striatal units (arranged on the abscissa by increasing preferred direction) are shown in greyscale, where black indicates small values.

experiment conducted by Kawagoe et al (Kawagoe et al., 1998) do process many aspects of the target (e.g. position, color, intensity, duration). However, only the position of the target is relevant for the successful completion of the task. It could be advantageous to enhance the neural representation of the position, while suppressing neural signals related to other features of the stimulus and other information about the environment (sounds of the experimenters and equipment) that could interfere with or corrupt the representation of the target position and thus compromise performance and reward acquisition. The ability of dopamine to control both the level of excitatory drive and the thresholds for activation in striatal units indicates that it can provide the necessary components to implement signal extraction related to reward. Further computational and experimental work is needed to bear light on these interesting issues.

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