Multiple stages of automaticity

June 10, 2025

Introduction

We built the model shown in Fig. 1 and simulated it in a minimal forced-choice category learning task with just two stimuli, one per category. After each response, the model received correct/incorrect feedback. Dopamine-dependent, three-factor synaptic plasticity was implemented at all cortical-subcortical synapses (VIS-DMS and PM-DLS). Two-factor Hebbian learning governed all cortical-cortical synapses (VIS-PM and PM-M1). Each simulation ran for 2000 trials, with different simulations exploring the effects of lesioning either the DMS or DLS at various learning stages. Lesions were simulated by setting the membrane potential of the affected neurons to zero. We also froze synaptic weight updates after the lesion. Preliminary results show:

- Lesions early in learning – from trial 100 – to both the DMS and DLS prevent learning (Fig. 2 and fig. 3).
- Lesions at intermediate stages of learning – from trial 500 – to the DLS prevent learning (Fig. 5), but lesions to the DMS do not (Fig. 4). This reflects transfer from the first stage subcortical pathway to the first stage cortical pathway.
- Lesions at the latest stages of learning – from trial 1000 – do not impair learning regardless of whether the lesion is to the DMS (Fig. 6) or the DLS (Fig. 7). This reflects transfer from both subcortical pathways to both cortical pathways.

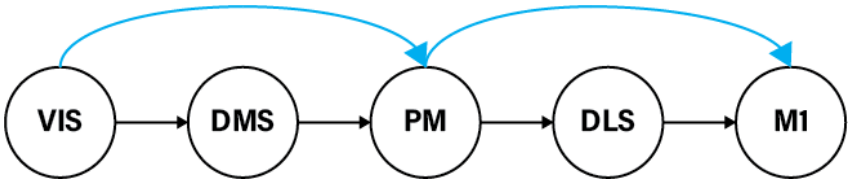

Figure 1: The model architecture. The model consists of two stages, each with a subcortical pathway (DMS and DLS) and a cortical pathway (VIS-PM and PM-M1). Dopamine-dependent, three-factor synaptic plasticity is implemented at all cortical-subcortical synapses (VIS-DMS and PM-DLS). Two-factor Hebbian learning governs all cortical-cortical synapses (VIS-PM and PM-M1).

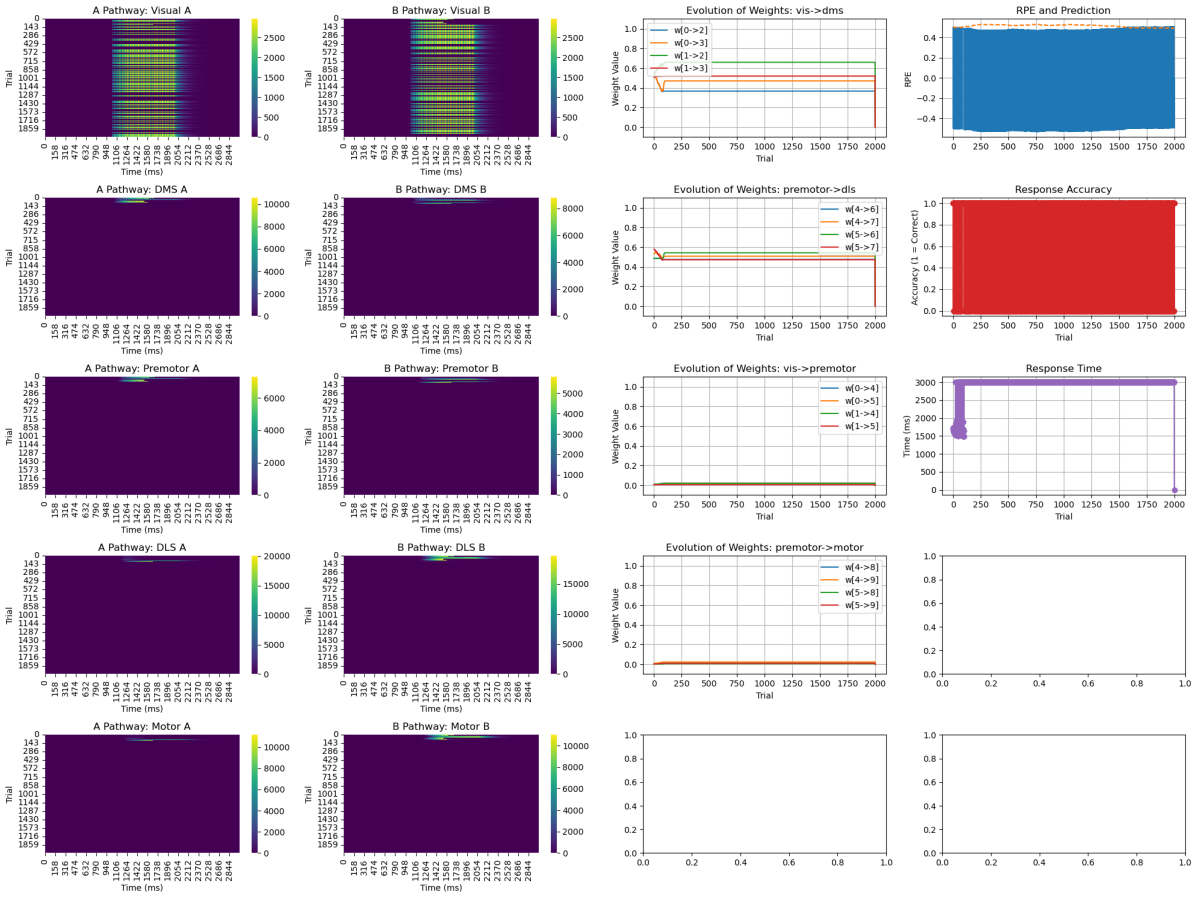

Figure 2: Lesions to the DMS at early stages of learning – trial 100 onward – prevent learning. The first two columns show activity (i.e., neurotransmitter release) across every time step and trial in every neuron. The third column shows synaptic weights across trials. The fourth column shows a variety of information pertaining the model's performance across trials. The first row of this column shows predicted rewards and RPE. The second row shows response accuracy (0 for incorrect and 1 for correct). The row column shows response time.

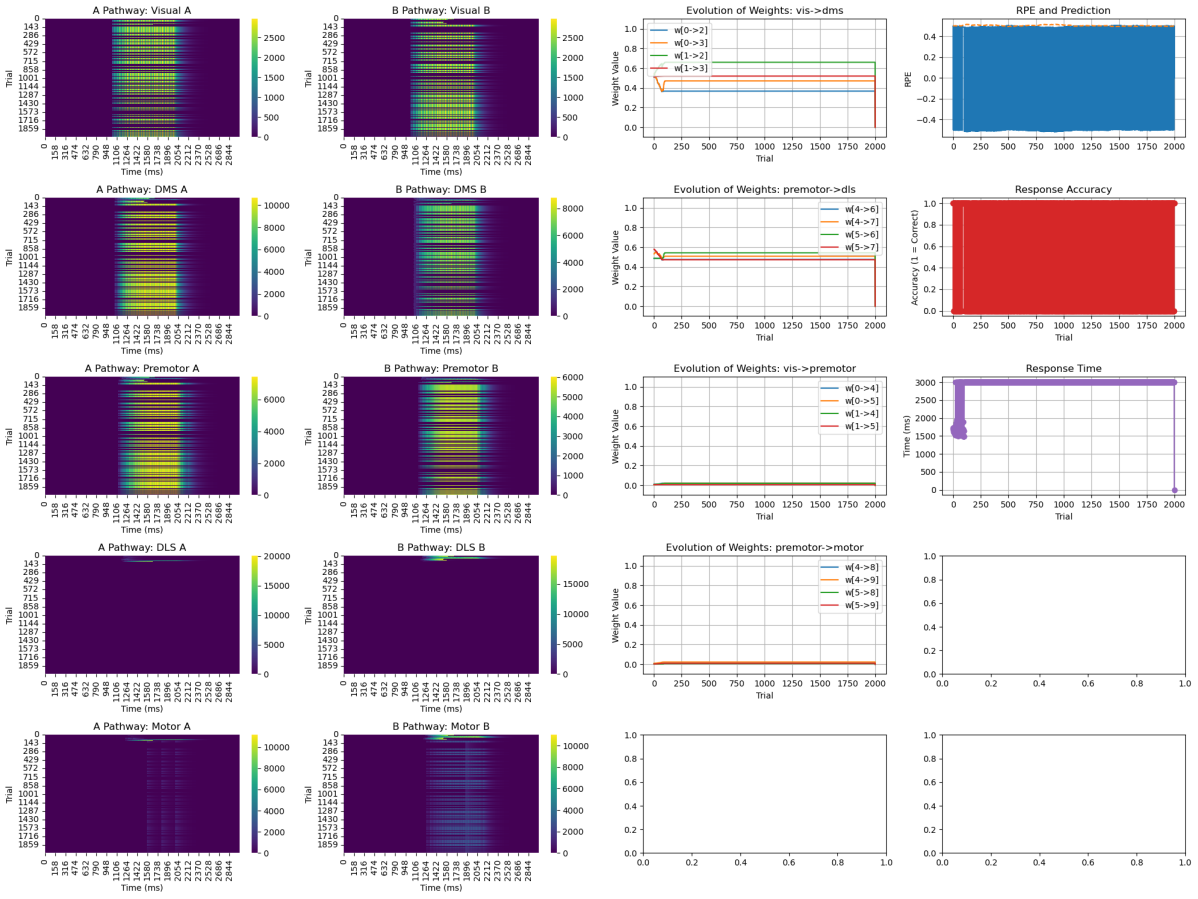

Figure 3: Lesions to the DLS at early stages of learning – trial 100 onward – prevent learning. The first two columns show activity (i.e., neurotransmitter release) across every time step and trial in every neuron. The third column shows synaptic weights across trials. The fourth column shows a variety of information pertaining the model's performance across trials. The first row of this column shows predicted rewards and RPE. The second row shows response accuracy (0 for incorrect and 1 for correct). The row column shows response time.

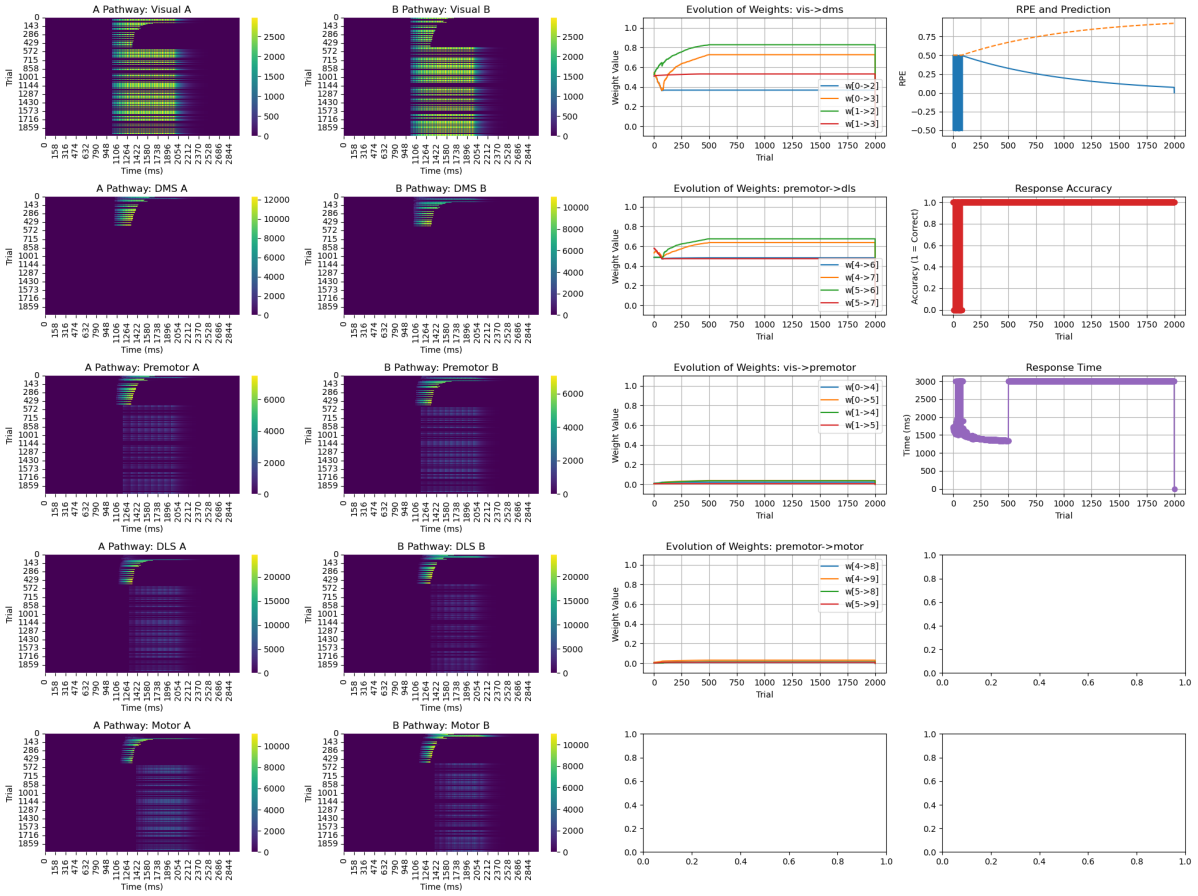

Figure 4: Lesions to the DMS at intermediate stages of learning – trial 500 onward – do not impair learning, reflecting transfer from the first stage subcortical pathway to the first stage cortical pathway. The first two columns show activity (i.e., neurotransmitter release) across every time step and trial in every neuron. The third column shows synaptic weights across trials. The fourth column shows a variety of information pertaining the model's performance across trials. The first row of this column shows predicted rewards and RPE. The second row shows response accuracy (0 for incorrect and 1 for correct). The row column shows response time.

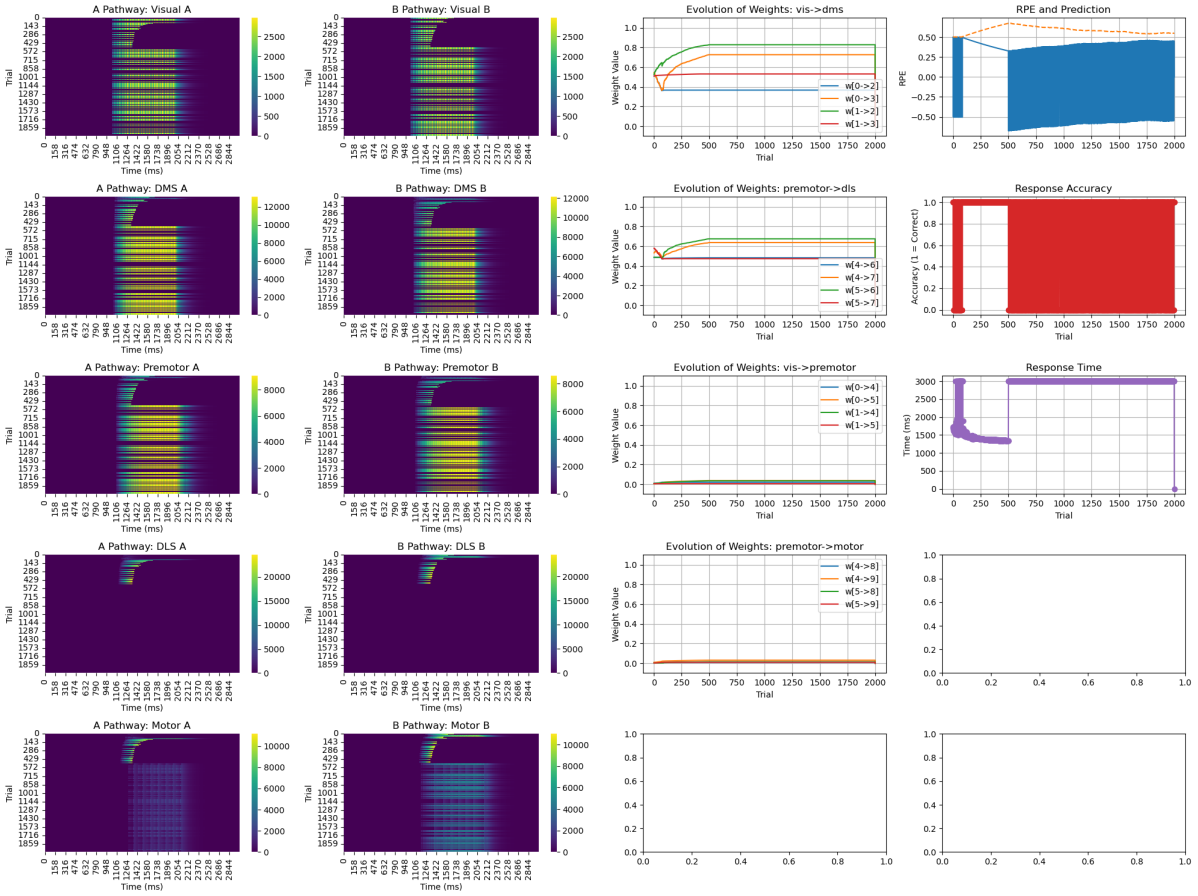

Figure 5: Lesions to the DLS at intermediate stages of learning – trial 500 onward – continue to impair learning, reflecting the lack of transfer from the second stage subcortical pathway to the second stage cortical pathway. The first two columns show activity (i.e., neurotransmitter release) across every time step and trial in every neuron. The third column shows synaptic weights across trials. The fourth column shows a variety of information pertaining the model's performance across trials. The first row of this column shows predicted rewards and RPE. The second row shows response accuracy (0 for incorrect and 1 for correct). The row column shows response time.

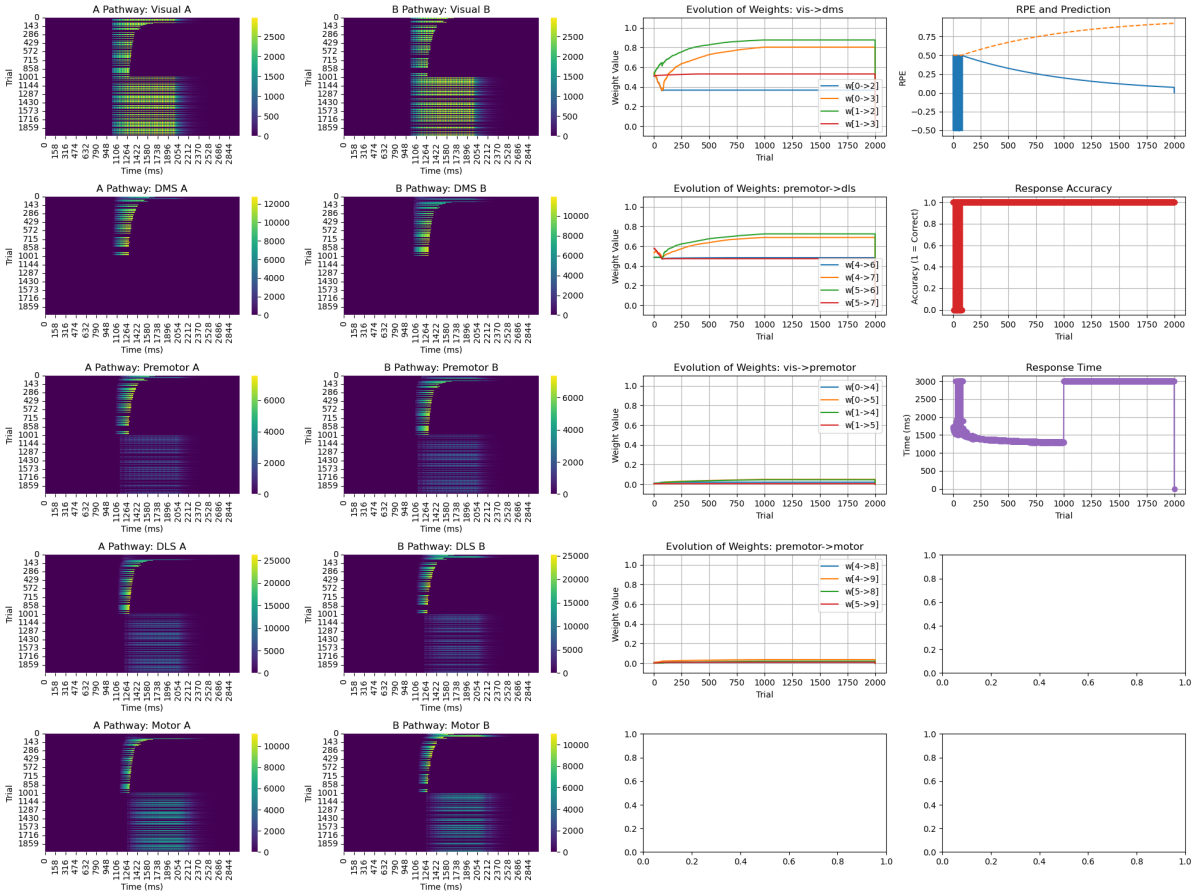

Figure 6: Lesions to the DMS at the latest stages of learning – trial 1000 onward – do not impair learning, reflecting transfer from the first stage subcortical pathway to the first stage cortical pathways. This is just a contunation of the results in Fig. 4. The first two columns show activity (i.e., neurotransmitter release) across every time step and trial in every neuron. The third column shows synaptic weights across trials. The fourth column shows a variety of information pertaining the model's performance across trials. The first row of this column shows predicted rewards and RPE. The second row shows response accuracy (0 for incorrect and 1 for correct). The row column shows response time.

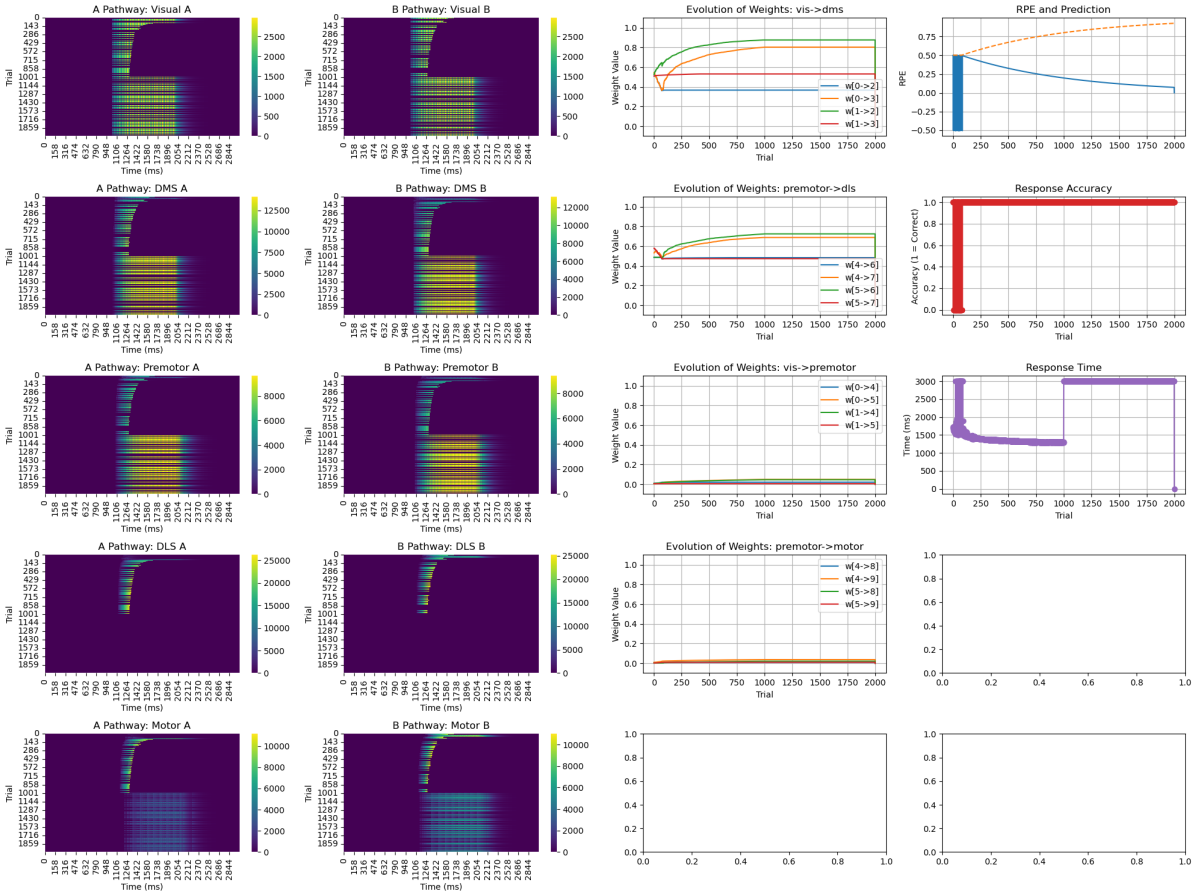

Figure 7: Lesions to the DLS at the latest stages of learning – trial 1000 onward – do not impair learning, reflecting transfer from the second stage subcortical pathway to the second stage cortical pathways. The first two columns show activity (i.e., neurotransmitter release) across every time step and trial in every neuron. The third column shows synaptic weights across trials. The fourth column shows a variety of information pertaining the model's performance across trials. The first row of this column shows predicted rewards and RPE. The second row shows response accuracy (0 for incorrect and 1 for correct). The row column shows response time.