A Neurocomputational Model of Basal Ganglia-Intralaminar Nuclei Interactions: Sense of Agency

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The Active Self
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Professorship Artificial Intelligence

The Basal Ganglia - Intralaminar Nuclei Prediction Circuit

Agency:
Agency is the feeling of control over our actions and their outcomes. Core requirement = predicting outcomes by internal states Probably cerebellum but the basal ganglia (BG) - intralaminar nuclei (ILN) circuit might also be a potential candidate.

BG-ILN connectivity:
Various BG and ILN regions are strongly interconnected forming loops [1]–[3]. Thalamo-striatal projections are relevant for behavioral switching, attentional shifting, and reinforcement [3]–[5]. Particularly the centromedian/parafascicular (CM/Pf) complex also projects to the subthalamic nucleus (STN), external globus pallidus (GPe), and internal globus pallidus/substantia nigra pars reticulata (GPi/SNr) [2], [6]–[8], with branched axons that simultaneously target both the STN and the GPi/SNr. Additionally, the ILN responds to attention-related salient stimuli, suggesting its role in adapting behavior based on prediction errors [3], [9].

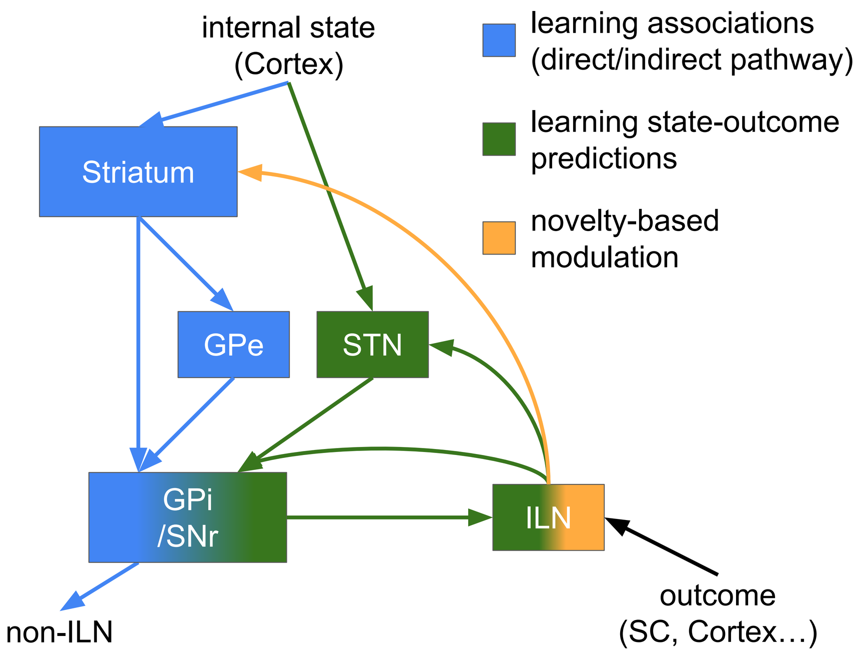

Figure 1: BG-ILN connectivity

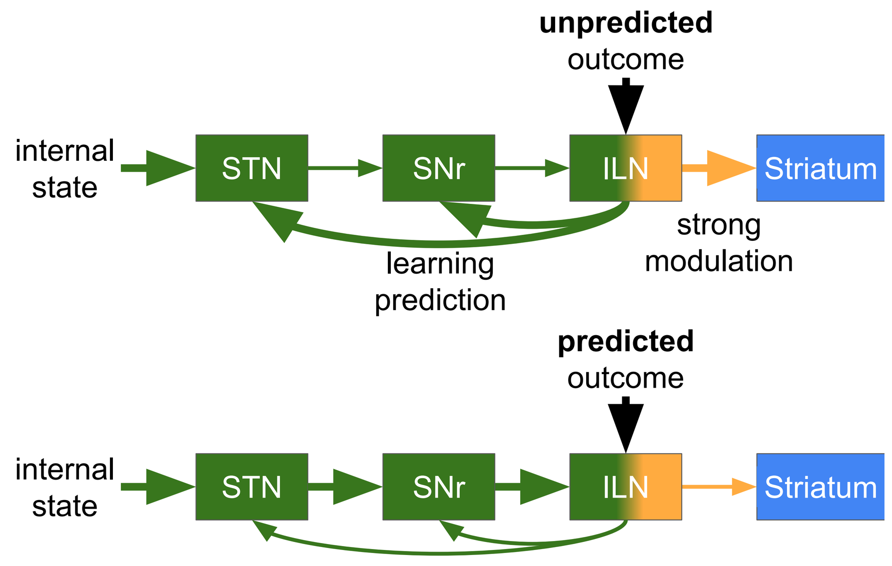

Figure 2: Prediction in the BG-ILN circuit

Hypotheses:
Functional
- Modulated by the branched axons from the ILN, the STN-SNr pathway learns to associate internal states with outcomes that may involve internal or external events.
- The prediction of an outcome is reflected in increased inhibition of the ILN by the SNr, which diminishes the ILN's response to the corresponding outcomes.

General
- The activity of the ILN represents a kind of prediction error.
- The BG - ILN circuits contribute to the sense of agency
- The BG - ILN circuits contribute to novelty detection
- The BG - ILN circuits contribute to information (novelty) seeking

Experimental Setup

Experimental paradigm [9]:
- Hold button is illuminated -> monkeys have to hold the illuminated button
- They have to fixate on a central LED throughout the trial
- After a random delay (500-1500 ms), one of two large LEDs (left or right) lights up as a cue
- At 100, 400, or 700 ms after the cue onset one of the two large LEDs lights up as a target
- If monkeys release the button within 500 ms after the target appearance they receive a reward
- Random intertrial interval (3-5s)

Experimental findings:
Neurons of the CM/PF complex responded to light flashes (the cues/targets) presented on the contralateral side. However, responses to target stimuli appearing on the contralateral side depended highly on the cue condition. Neurons responded to invalidly cued targets but responded less intensely to validly cued targets.

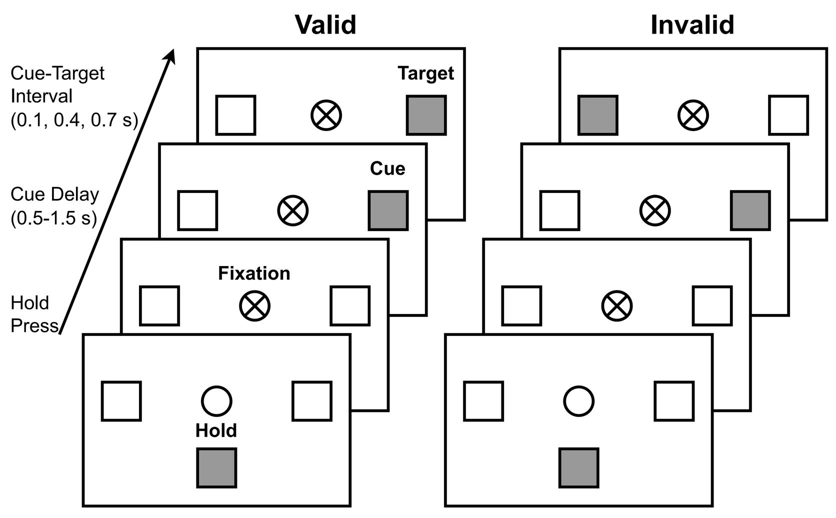

Figure 3: Experimental paradigm.
(Illustration created based on Figure 1 from [9])

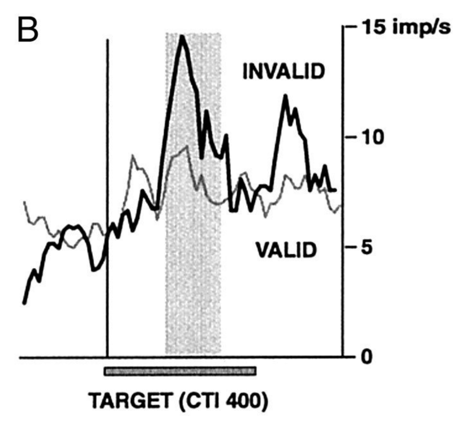

Figure 4: Experimental findings.
(Illustration is part of Figure 5 from [9])

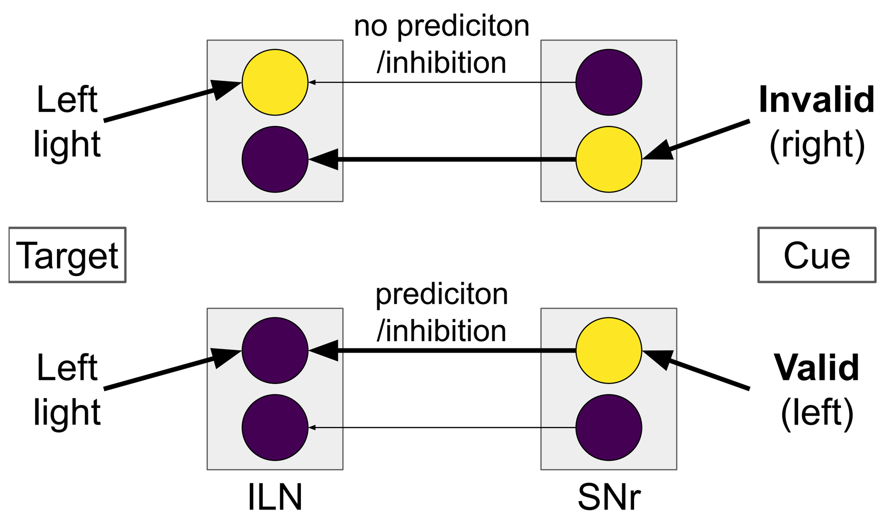

Figure 5: Cues predict targets

Interpretation with BG-ILN prediction:
We propose that the presentation of a cue elicits an internal state, which, through task training, becomes associated with the appearance of a target on the same side via learning within the Cortex->STN->SNr->CM/Pf pathway. Consequently, after training and following valid cues, CM/Pf neurons that would typically respond to the target on the cued side receive increased inhibitory input from the SNr, leading to a diminished response. In contrast, while invalid cues similarly elicit an internal state predicting a target on the cued side, the target appears on the opposite side, and the neurons responding to that side do not experience an increase in inhibition.

Model

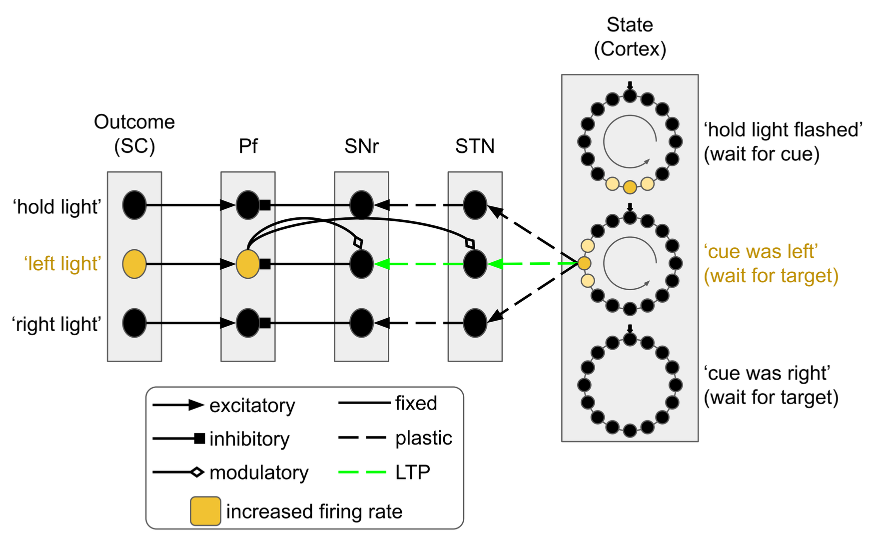

Figure 6: Model overview

- rate-coded neurons
- neurosimulator ANNarchy [10]
- inputs to the model:
  - at hold, cue, and target light onsets: increase the firing rate of corresponding SC neurons for 50 ms
  - after hold and cue lights: increase the firing rate of a single cortical neuron belonging to the elicited state for 200 ms (held active as response sequence)
- Synaptic plasticity in Cortex->STN->SNr:
Learning Rule Equation

Modeling Results

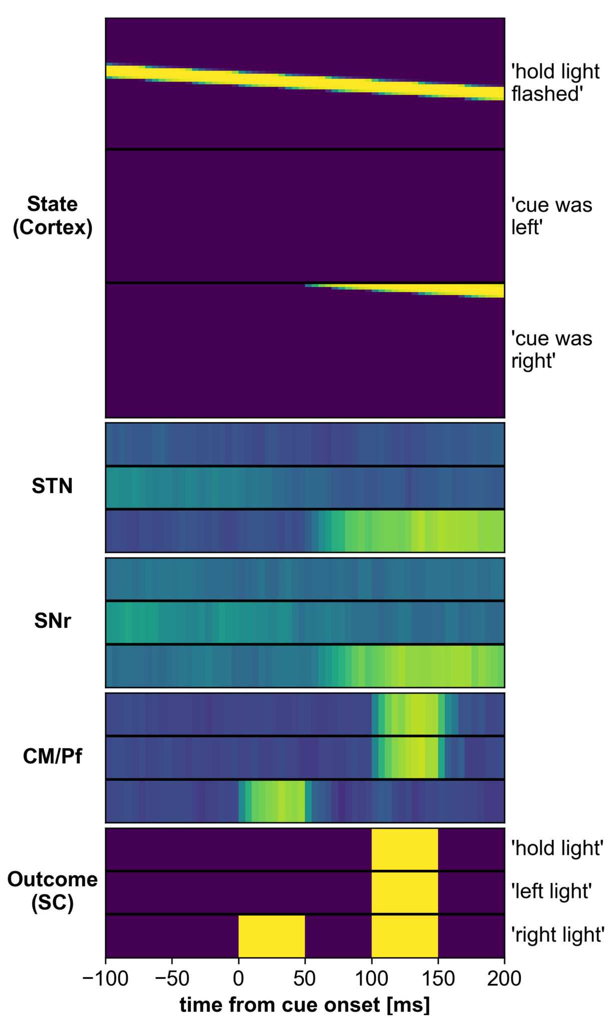

Figure 7: Firing rates single trial

Firing rates of the model's neurons during the cue and target presentation (cue-target interval = 100 ms) in a single trial after learning are shown. At target onset, all three outcomes are activated in this trial. The cue 'right light' elicits the state 'cue was right' in the cortex, and in the STN and SNr, neurons associated with the outcome 'right light' exhibit increased firing rates. This illustrates the learned association 'cue was right' -> 'right light'. The Pf responds to excitatory input from the SC, but only when the inhibitory input from the SNr is not increased.

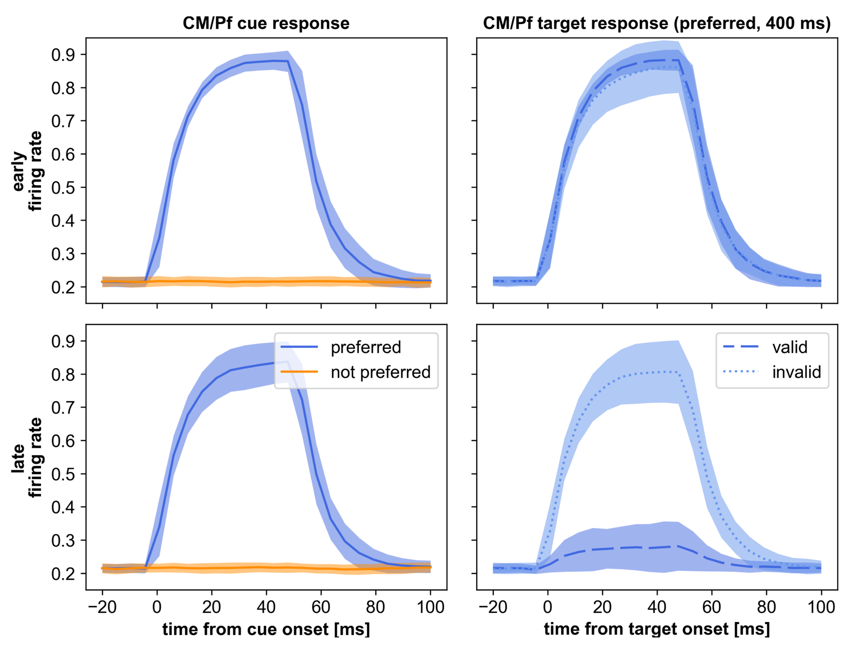

Figure 8: Firing rates single trial

Responses of Pf neurons to cues and targets averaged over the first and last 100 trials (before and after training) of each respective category. The response to the cues does not change significantly after training. In contrast, the response to the targets depends on the cue validity after task training. The response to invalidly cued targets does not change but the response to validly cued targets decreases as in [9].

References

[1] K. K. Cover and B. N. Mathur, "Rostral Intralaminar Thalamus Engagement in Cognition and Behavior," English, *Frontiers in Behavioral Neuroscience*, vol. 15, Apr. 2021, Publisher: Frontiers, ISSN: 1662-5153. DOI: 10.3389/fnbeh.2021.652764.

[2] E. Gonzalo-Martín, C. Alonso-Martínez, L. P. Sepúlveda, and F. Clasca, "Micropopulation mapping of the mouse parafascicular nucleus connections reveals diverse input–output motifs," English, *Frontiers in Neuroanatomy*, vol. 17, Jan. 2024, Publisher: Frontiers, ISSN: 1662-5129. DOI: 10.3389/fnana.2023.1305500.

[3] Y. Smith, A. Galvan, T. J. Ellender, *et al.*, "The thalamostriatal system in normal and diseased states," English, *Frontiers in Systems Neuroscience*, vol. 8, Jan. 2014, Publisher: Frontiers, ISSN: 1662-5137. DOI: 10.3389/fnsys.2014.00005.

[4] K. K. Cover, U. Gyawali, W. G. Kerkhoff, *et al.*, "Activation of the Rostral Intralaminar Thalamus Drives Reinforcement through Striatal Dopamine Release," English, *Cell Reports*, vol. 26, no. 6, 1389–1398.e3, Feb. 2019, Publisher: Elsevier, ISSN: 2211-1247. DOI: 10.1016/j.celrep.2019.01.044.

[5] K. K. Cover, A. G. Lieberman, M. M. Heckman, and B. N. Mathur, "The rostral intralaminar nuclear complex of the thalamus supports striatally mediated action reinforcement," *eLife*, vol. 12, J. Ding and K. M. Wassum, Eds., e83627, Apr. 2023, Publisher: eLife Sciences Publications, Ltd, ISSN: 2050-084X. DOI: 10.7554/eLife.83627.

[6] M. Castle, M. S. Aymerich, C. Sanchez-Escobar, N. Gonzalo, J. A. Obeso, and J. L. Lanciego, "Thalamic innervation of the direct and indirect basal ganglia pathways in the rat: Ipsi- and contralateral projections," en, *Journal of Comparative Neurology*, vol. 483, no. 2, pp. 143–153, 2005, _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1002/cne.20421, ISSN: 1096-9861. DOI: 10.1002/cne.20421.

[7] T. Kita, N. Shigematsu, and H. Kita, "Intralaminar and tectal projections to the subthalamus in the rat," en, *European Journal of Neuroscience*, vol. 44, no. 11, pp. 2899–2908, 2016, _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/ejn.13413, ISSN: 1460-9568. DOI: 10.1111/ejn.13413.

[8] M. Hanini-Daoud, F. Jaouen, P. Salin, L. Kerkerian-Le Goff, and N. Maurice, "Processing of information from the parafascicular nucleus of the thalamus through the basal ganglia," en, *Journal of Neuroscience Research*, vol. 100, no. 6, pp. 1370–1385, 2022, _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1002/jnr.25046, ISSN: 1097-4547. DOI: 10.1002/jnr.25046.

[9] T. Minamimoto and M. Kimura, "Participation of the Thalamic CM-Pf Complex in Attentional Orienting," *Journal of Neurophysiology*, vol. 87, no. 6, pp. 3090–3101, Jun. 2002, Publisher: American Physiological Society, ISSN: 0022-3077. DOI: 10.1152/jn.2002.87.6.3090.

[10] J. Vitay, H. Dinkelbach, and F. Hamker, "ANNarchy: A code generation approach to neural simulations on parallel hardware," *Frontiers in Neuroinformatics*, vol. 9, no. 19, pp. 1–20, 2015. DOI: 10.3389/fninf.2015.00019.

Acknowledgements

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