

Poster presentation

Open Access

Behavioral inhibition during reversal learning in the limbic system: a computational model

Adedoyin Maria Thompson*¹, Bernd Porr¹ and Florentin Wörgötter²

Address: ¹Department of Electronics and Electrical Engineering, University of Glasgow, Glasgow, G12 8LT, UK and ²Bernstein Center for Computational Neuroscience (BCCN), Bunsenstrasse 10, 37073 Göttingen, Germany

Email: Adedoyin Maria Thompson* - mariat@elec.gla.ac.uk

* Corresponding author

from Eighteenth Annual Computational Neuroscience Meeting: CNS*2009
Berlin, Germany. 18–23 July 2009

Published: 13 July 2009

BMC Neuroscience 2009, **10**(Suppl 1):P8 doi:10.1186/1471-2202-10-S1-P8

This abstract is available from: <http://www.biomedcentral.com/1471-2202/10/S1/P8>

© 2009 Thompson et al; licensee BioMed Central Ltd.

A biological model of the sub-cortical nuclei of the limbic system capable of performing reversal learning in a food-seeking task is presented. Contrary to other learning models, the reversal is modeled not by eliminating ("unlearning") learned behaviors, but rather by the inhibiting the previously learned behavior. This allows for the re-instatement of behavior to be quickly re-established in such a way that animal behavior is simulated. In this model, the role of dopamine is different to standard models. The activity of dopaminergic (DA) neurons required for successful conditioning has commonly been identified as an error signal whereby an increased DA activity codes a positive error i.e. long term potentiation (LTP) and a decrease in dopamine concentration codes a negative error. In this model learning is achieved by implementing a form of differential Hebbian learning known as Isotropic Sequence Order learning and a third factor (ISO3). This third factor enables learning to be triggered at relevant moments. It is modeled by dopaminergic neurons which can be activated via a direct excitatory glutamatergic pathway, and an indirect disinhibitory GABAergic pathway. While the former generates phasic DA release which during acquisition enables long term potentiation (LTP) to occur, the latter produces in an increased population of tonically active DA neurons which generates long term depression (LTD) when an adjustment in learned behavior is required. The nucleus accumbens is divided into two shell and core subunits and has been modeled to function

in distinct but complementary manners. Here, the core uses conditioned reinforcers to invigorate instrumental responding. The core has been modeled to learn to enable behavioral responding to reward predicting stimuli and as such undergoes minimal LTD so as not to eliminate these learned processes. On the other hand, LTD occurs in the shell that, through a shell-ventral pallido-medio dorsal pathway, influences the core and enables behavioral flexibility. A simple reversal-learning task will be used to demonstrate how this biophysically realistic pathway can be used to learn and reverse a simple food-seeking task.