Beyond the reward prediction error: achieving reversal learning with Hebbian cortical plasticity and serotonergic modulation

Bernd Porr, bernd.porr@glasgow.ac.uk

University of Glasgow | School of Engineering

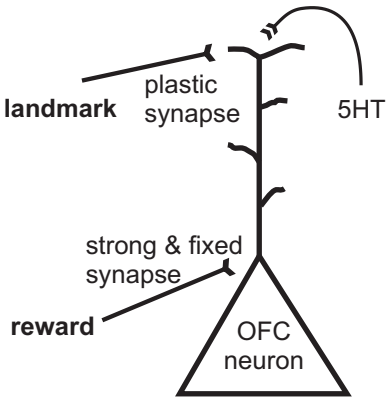
Introduction

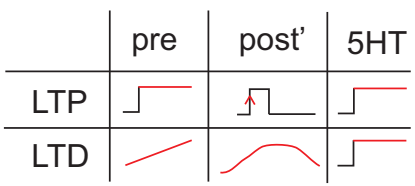
Theoretical models of reversal learning usually use the dopaminergic reward prediction error generated in the sub-cortical structures. However, it has long been argued (Rolls, 2008) that the reward related behavioural flexibility is rather cortical than sub-cortical and not dopamine driven. We present a model of reversal learning which won't require a reward prediction error.

Methods

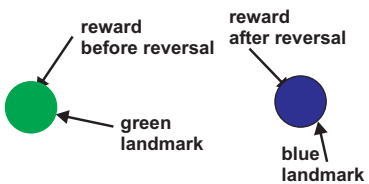
We utilise the structure and functional differentiation of cortical pyramidal neurons, here of orbitofrontal cortex neurons.

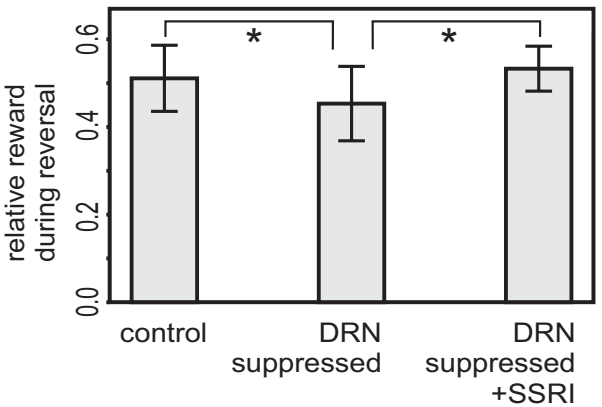
The primary reward drives non-plastic synapses close to the soma and causes strong postsynaptic responses which in turn cause a large calcium influx. New associations are established with Hebbian plasticity via plastic synapses in the dendritic tree. LTP is triggered when strong somatic reward inputs drive a cortical neuron into spiking. LTD is triggered when the primary reward is not driving the OFC pyramidal neurons causing less and slower changing postsynaptic activity. Serotonin acts as an accelerator for both LTP and LTD being a rectified reward prediction error.

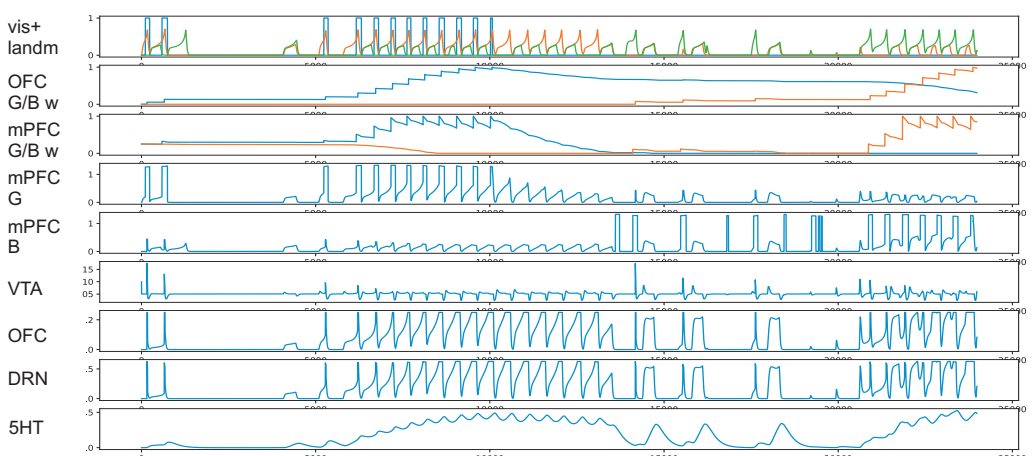




Experiments of a simulated animal performing a reversal task were run for a control scenario, for one with less serotonin (DRN suppresssed) and one with a simulated application of serotonin reuptake inhibitors (SSRI).







Results and conclusions

We show that Hebbian plasticity is able to model reversal learning and that serotonin controls the rate of plasticity which in turn regulates the animal's flexibility. Reduced serotonin increases the number of non-rewarding actions during reversal. SSRIs can help to overcome this deficiency, but we'd predict that any drug boosting plasticity will have this effect.