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Comment on *Maggi* et al., *"Cognitive correlates of prospective memory in dystonia"*

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Dear Editor,

We would like to commend the interesting study of Maggi et al., 2019 [1], who demonstrated a prospective memory impairment which correlated with impaired set shifting in a group of patients with focal dystonia. We recently demonstrated a reversal learning impairment in patients with cervical dystonia using a reinforcement learning task [2]. Furthermore, this behaviour was replicated in a computational model of the striatum when the post-synaptic signalling of a dopamine dependant error signal (negative prediction error signal) was selectively impaired. Although direct comparison of our task and the MIST (Memory for Intentions Screening Test) is not straightforward, there is significant overlap in the prefrontal and basal ganglia circuity which are thought to implement both reversal learning and set-shifting [3]. This suggests a common mechanistic link between our studies.

Maggi et al. propose that an impairment of the anterior (rostral) prefrontal cortex could be the neurobiological sub-strait for this impairment. This is a sound interpretation based on evidence that this region is a principle site for prospective memory encoding. An alternative and potentially unifying explanation though, is that impaired set-shifting reflects a subcortical impairment in the striatal integration of dopamine based teaching signals. Although not an archetypal reinforcement learning task the MIST implicitly involves reward learning, as the outcome from each of the 8 "trials" is incentivised by participant feedback via a points based system. Under conditions of impaired negative prediction error signalling, this feedback would not generate a robust striatal value signal [4]. In turn, this would lead to attentional perseveration in the distractor task, and corresponding impairment in the patient's ability to switch their attention to the cognitive demands of prospective memory recall.

This would be analogous to the perseveration seen in our task which is a consequence of being unable extinguish the previously learnt stimulus response relationship. This interpretation is consistent with the exiting literature demonstrating the role of the basal ganglia thalamocortical circuitry in working memory [5]. Notably, it also supports a unifying subcortical basis for both the motor and non-motor symptoms of dystonia.

Declarations of interest

None.

Funding sources for study

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