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ORAL PRESENTATION

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Drug-dominated dopamine circuits spiral addicts down to a cognitive/behavioral conflict: a neurocomputational theory

Mehdi Keramati*, Boris Gutkin

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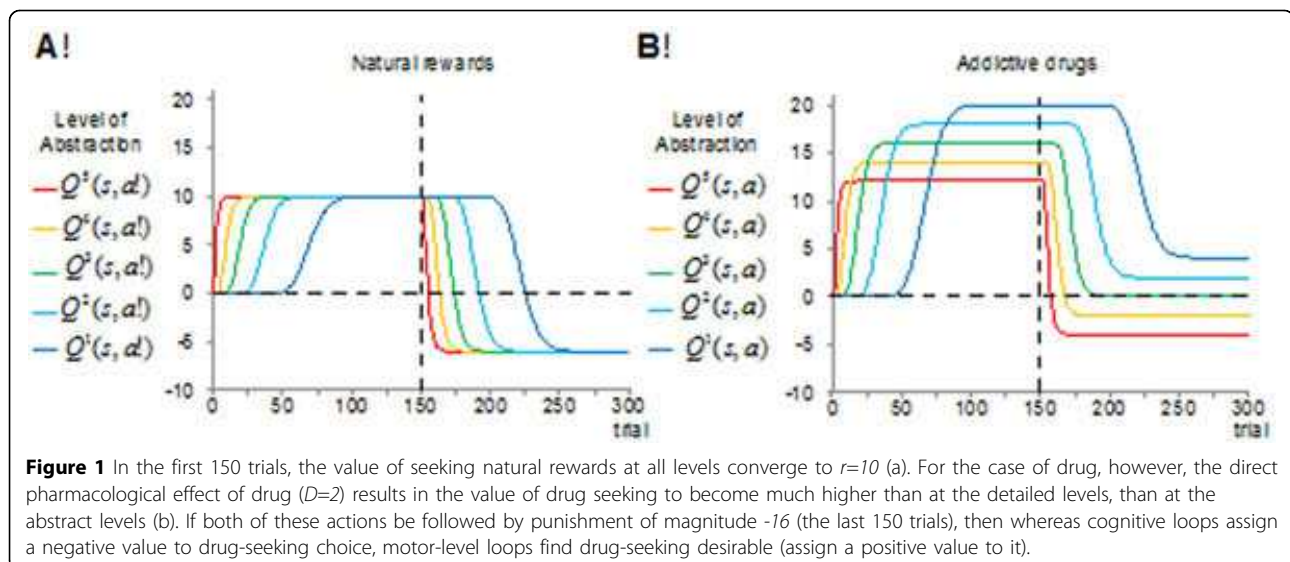
Long-term addicts find themselves powerless to resist drugs, despite knowing that drug-taking may be a harmful course of action, and an explicit motivation to quit. In controlled experiments, human addicts show a self-described mistake characterized by an inconsistency between drug-seeking response and their reported subjective value. We provide a unified computational theory for this inconsistency by showing how addictive drugs gradually produce a motivational bias toward drug-seeking at low-level habitual decision processes, despite the low abstract cognitive values. This pathology emerges within

the hierarchical reinforcement learning (HRL) framework when chronic drug-exposure pharmacologically hijacks the dopaminergic spirals that cascade reinforcement signal down the ventro-dorsal cortico-striatal hierarchy.

$$\delta_t^n = [r_t + V^{n+1}(s_{t+1}) - Q^n(s_t, a_t)] + D \quad (1)$$

$$Q^n(s_t, a_t) \leftarrow Q^n(s_t, a_t) + \alpha \delta_t^n$$

Here, r_t is the rewarding value of the outcome, be it natural rewards or addictive drugs. These equations show that in order to compute the prediction error



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signal for updating the value (Q) of state-action pairs at the n -th level of decision hierarchy, the value of the temporally-advanced state (s_{t+1}) comes from one higher level of abstraction ($n+1$). This captures the role of dopamine-dependent serial connectivity linking the ventral to the dorsal striatum (known as dopamine spirals), which is suggested to integrate information across the segregated cortico-basal ganglia loops, thereby allowing more abstract levels to tune the reinforcement signal used at more detailed levels [1]. The pharmacological effect of addictive drugs on increasing the extracellular concentration of dopamine within the striatum is incorporated into this model by adding a positive term D to the prediction error signal. Simulation results (Figure 1) show that drug-induced dopamine-release puts a bias on the transfer of reinforcement signal from one level of abstraction to the next. The accumulation of these biases along the rostral-caudal axis progressively induces a significant discrepancy in the value of drug-seeking behaviors at the top and bottom extremes of the hierarchy, thereby, an inconsistency between cognitive plans and motor-level habits.

Beside this central phenomenon, our model also accounts for several behavioral and neurobiological aspects of addiction, such as the gradual insensitivity of drug-seeking to drug-associated punishments (compulsivity), the delayed development of cue-elicited dopamine efflux in addicts' dorsal striatum, and the occurrence of blocking effect for drug rewards. It also suggests key testable predictions and beyond that, sets the stage for a view of addiction as a pathology of hierarchical decision making processes.

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Reference

1. Haruno M, Kawato M: Heterarchical reinforcement-learning model for integration of multiple cortico-striatal loops: fMRI examination in stimulus-action-reward association learning. *Neural Networks* 2006, **19**:1242-1254.

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