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The Effectiveness of Theory-Driven Computer Models of Drug Addiction in Humans

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Abstract

Several people believe that neurocognitive dysfunctions are the cause of the maladaptive behaviour seen in drug addiction. Using computational tools to explore these dysfunctions in drug-dependent people has been more popular recently, in part because it offers a quantitative framework for inferring the psychological mechanisms that may have gone wrong in addiction. Thus, we set out to assess how well these theory-driven computational models have served this function in the field of addiction research.

Keywords: Drug addiction; Humans; Computational models

Introduction

Drug addiction is a serious type of substance use disease that is defined by dysfunctional drug use patterns that continue at the expense of the users' health and welfare. Drug addiction is now largely considered as a neuropsychiatric condition with obvious biological basis. It was previously thought of as a moral failing and conceptualised by a physical and psychological dependency on addictive chemicals. At its core, drug-dependent individuals frequently display a wide range of maladaptive addictive behaviours, such as a lack of control over drug use that persists despite unfavourable effects or a strong desire to use drugs rather than partake in other rewarding activities [1, 2].

With drug addiction, abnormal reinforcement learning occurs

The outcomes of adaptive activities frequently influence them [3]. People are more prone to repeat behaviours that result in favourable outcomes and refrain from behaviours that have unfavourable effects. Reward learning has been used to characterise this tendency, which uses previous outcomes to direct future behaviour towards maximising rewards and reducing penalties. Disruptions in reinforcement learning processes have been proposed as an explanation for why drug usage in addicted patients is not susceptible to negative outcomes because drug addiction is associated with maladaptive patterns of drug use that persist despite negative consequences [4].

Computational methods can be used to mathematically characterise reinforcement learning. These algorithms can break down reinforcement learning into its component parts, and the failure of any one of these parts can result in degraded learning.

These computational algorithms' free parameters contain a codification of these sub processes. For instance, the learning rate and inverse temperature (also known as the exploration/exploitation trade-off or reinforcement sensitivity) are two parameters that are present in the majority of reinforcement learning algorithms (such as Q-Learning). The learning rate represents how feedback affects decisions, whereas the inverse temperature parameter describes the propensity to make decisions based on learnt values. Also, it is possible to include parameters to the model of psychological processes that are considered important in the study of addiction. These characteristics, which reflect perseverative responding and disregard for alternative rewards, respectively, include "stickiness" (the propensity to repeat previous replies) and a counterfactual learning rate (the impact of current input on the unselected decision) [5, 6].

Discussion

Reward learning subprocesses are predominantly supported by dopaminergic neurons and frontostriatal networks, according to a significant body of research. The midbrain and the mesolimbic system are known sites of action for addictive substances, and long-term drug use is linked to dopaminergic downregulation and frontostriatal dysfunction. Thus, it's probable that these neuroadaptive modifications are reflected in the reduced reinforcement learning processes in drug-dependent patients [7, 8].

Conclusion

In recent studies, computer models have been used to quantitatively investigate changes to reinforcement learning processes in the brain and behaviour, revealing subtleties and insights that more traditional metrics (such summary or mean scores of behavioural responses) could not. Two distinct themes have so far emerged from the literature on addiction: altered prediction error signalling and the corresponding brain pathways, as well as compromised learning subprocesses [9, 10].

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Declaration of Competing Interest

None.

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