

Beyond a Cognitive Dichotomy: Can Multiple Decision Systems Prove Useful to Distinguish Compulsive and Impulsive Symptom Dimensions?

To the Editor:

The human mind likes simplicity, and so do scientists. This explains the popularity of dual system theories such as Daniel Kahneman's fast and slow thinking. A dual systems theory that has a great following in cognitive neuroscience and computational psychiatry is the dichotomy between model-based and model-free learning and decision making (1). The model-based system suggests a model of the world incorporating hidden states and details how one can transition between these states. This allows goal-directed and flexible planning, but it is computationally demanding. The model-free system, on the other hand, does not entail such a model and suggests learning of stimulus–outcome associations only through lived experiences. This allows fast and simple computing but is often constrained to simplistic and habit-like learning. Human behavior is found to be influenced by both systems, and ventral striatum and dopamine transmission seems to play a role for a relative weighting of both systems (1,2).

A plethora of studies has investigated the relative contributions of these two systems to various psychiatric symptoms. Under particular investigation are symptoms of obsessive-compulsive disorder (OCD) and substance use disorder (SUD). While the symptoms and diagnostic criteria of these disorders are quite different, both are signified by repeating harmful behaviors despite negative consequences (3), such as compulsive chronic drug intake that is no longer rewarding. Therefore, compulsions in both conditions may be related to an imbalance of model-free and model-based control. Indeed, reduced model-based control was found in SUD, OCD, and other disorders across the impulsivity–compulsivity spectrum (4,5). Impaired model-basedness has been interpreted as a transdiagnostic cognitive deficit, supporting criticism of psychiatric categories. This has led to the notion that compulsivity and impulsivity might be only partially independent dimensions and that patients with SUD transition from impulsivity to compulsivity (3). However, clinicians may sometimes raise an eyebrow in questioning the clinical utility of a phenotype that cannot differentiate between individuals who appear distinct in clinical observation.

The standard answer from cognitive neuroscience so far has been that transdiagnostic compulsions can share common cognitive and neural substrates. While this may be plausible under certain circumstances, recent findings suggest that focusing on a dichotomy may be an oversimplification and that, by accounting for additional cognitive biases, compulsivity- and impulsivity-related disorders can be disentangled. Prototypical is the recent study by Shahar *et al.* (6) that reveals a third learning component besides the well-established model-based and model-free systems. This additional motor-spatial model-free system learns the value of (in this task completely irrelevant) motor responses (e.g., left vs. right button presses). Importantly, the expression of this

motor-spatial model-free learning system is also negatively associated with model-based control—the metric that was found to be reduced in various impulsive-compulsive psychiatric conditions.

This demands a reinterpretation of existing findings and raises exciting possibilities to dissociate impulsive and compulsive symptoms. Namely, a reduced model-basedness as jointly seen in impulsive and compulsive conditions could arise from either 1) a relatively increased contribution of the traditional stimulus-specific model-free system or 2) an excessive contribution of the newly identified motor-spatial model-free system. In addition, it is conceivable that impulsive and compulsive symptoms express distinct contribution profiles of stimulus-specific versus motor-spatial model-free system.

Excitingly, this could also match clinical observations. Excessive repetition of behavioral patterns (compulsions) in OCD is often linked to avoidance of some potential or even hypothetical negative outcome but is also frequently executed in the absence of any specific stimuli. Furthermore, there is a clinical overlap with motor stereotypies and tics. This suggests that the motor-spatial system may be more relevant for OCD. In our simple example of binary choice, we postulate that choosing left or right per se is likely to matter to patients with OCD (motor perseveration) (Figure 1C). On the other hand, patients with SUD also show repetitive behaviors (compulsive drug intake), but these behaviors are usually referred to as being highly stimulus specific. In our simplistic example of binary choice, choosing the desired drink with left or right does not matter (stimulus perseveration) (Figure 1C). This dissociation could eventually be of great clinical relevance, for example, as a potential vulnerability marker in younger individuals such as adolescents.

In line with such accounts, it is likely that further cognitive biases help to distinguish impulsive and compulsive symptoms from each other. For example, the influence of drug-associated value may affect model-based control in a highly context-specific manner, potentially reflecting Pavlovian influences. This resonates well with an altered Pavlovian-to-instrumental transfer in relapse in SUD (7) and with a link between expectations about alcohol intake and the expression of model-based control in relapse (8). Pavlovian influences therefore may play an important role in explaining the somewhat mixed findings with regard to model-based control and symptoms of addiction (4,8–11). Model-free and model-based facets of Pavlovian learning have received empirical evidence recently in humans in the context of goal- and sign-tracking behavior (12)—a phenotype implicated in addiction vulnerability (13).

So what now—do we need to start again from the beginning and collect new data? Yes and no. No, because Shahar *et al.* (6) described the motor-spatial model-free component using the same task that most previous studies have used. We can thus reanalyze the existing data using the novel computational models. This also provides a unique opportunity to pool data across multiple research centers, similar to common efforts in

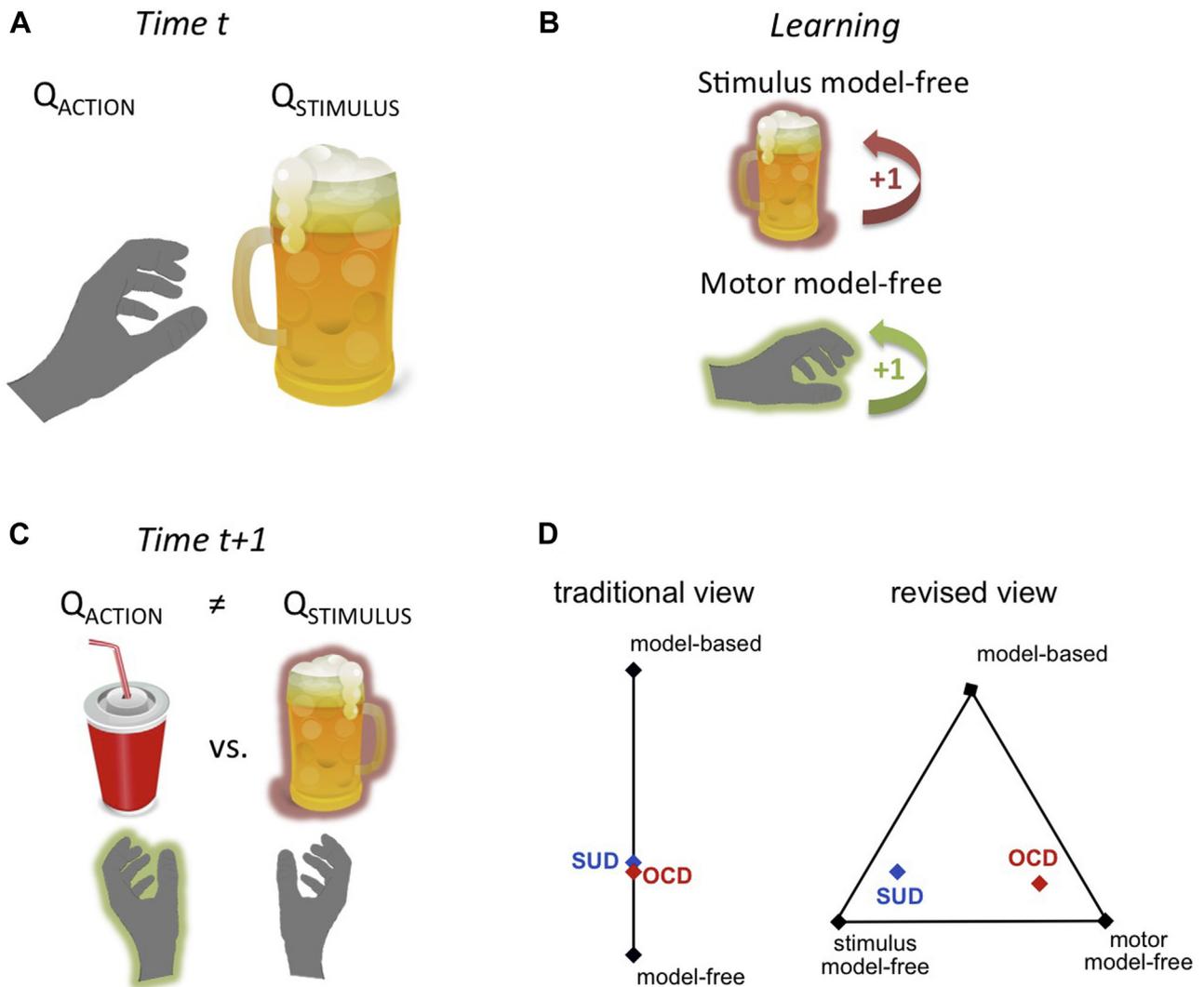


Figure 1. Illustration of the stimulus and motor model-free learning systems exemplified by drinking behavior. **(A)** Left-hand choice for drinking a beer. The chosen stimulus, a pint of beer, and the performed left-hand motor action can be assigned values denoted as Q_{STIMULUS} and Q_{ACTION} , respectively. **(B)** Stimulus- and motor-based learning systems both assign credit (taking beer drinking as a reinforcer) but do so either to the beer stimulus (red) or to the left-hand action (green). **(C)** Sometime later, imagine that there is a choice between two drinks—the previously chosen beer and a soft drink. Predictions of each learning system are in conflict in the case that it would require different motor actions to reach these stimuli. Stimulus-based model-free learning has assigned credit to the beer stimulus and thus would predict a right-hand action for the beer. In contrast, a motor model-free system would increase the tendency to perform a left-hand action, which in this example would result in a choice of the soft drink. **(D)** Schematic summary of the traditional view that both obsessive-compulsive disorder (OCD) and substance use disorder (SUD) show reduced reliance on model-based control over decision making and thus are characterized by the same cognitive alteration. We propose a revised view by suggesting that reduced model-based behavior in both conditions may result from distinct reliance on stimulus versus motor model-free learning.

neuroimaging and genetics. Moreover, exploiting the benefit of large patient groups also allows assessing whether there are characteristic subgroups within a disorder that express impaired model-based control. Yes, because new experimental work will allow us to systematically probe different systems and thus empower us to more decisively draw conclusions in terms of symptom relationships. Taken together, both lines of investigation can allow us to test whether the contributions of multiple systems differ in impulsive versus compulsive disorders and whether those differences are reflected in separate neural systems associated with compulsivity and impulsivity (14,15). In the long run, a multifaceted

differentiation of patients may be of clinical importance to design treatments, for example, of exposure therapies that focus on the specific impairments of the patient.

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Article Information

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