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Current Opinion in Behavioral Sciences

What are habits and do they depend on the striatum? A view from the study of neuropsychological populations

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What are the neural substrates of habit learning in humans? Studies in neuropsychological populations have been central to answering the question, and for decades, research appeared to have provided a fairly consistent answer. However, developments in assays of habits in animals, as well as new approaches to dissecting habitual versus goal-directed control of behavior in humans, point to further complexities in human habit learning. This has raised new questions about the status of habits in neuropsychological populations and our understanding of how the brain supports habitual behavior. I review these emerging challenges and suggest a more nuanced approach to habit learning.

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Current Opinion in Behavioral Sciences 2018, **20**:17–24

This review comes from a themed issue on **Habits and skills**

Edited by **Barbara Knowlton** and **Jörn Diedrichsen**

http://dx.doi.org/10.1016/j.cobeha.2017.08.011

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Introduction

Habits are critical for adaptive behavior: They allow organisms to capitalize on invariance in the environment while directing valuable cognitive resources toward more demanding or exciting endeavors [1]. Yet, an over-reliance on habits renders behavior inflexible in the face of change, leading instead to inappropriate behaviors. Indeed, the concept of *habit* has been central to how we understand of a broad range of behaviors that are both adaptive and maladaptive (e.g., addiction [2], OCD [3], and eating disorders [4]).

But how coherent is the habit concept? Based on studies in neuropsychological patients, the neural bases of habit learning in humans were thought to be well established. Yet, as data from these populations accumulate, it is becoming clear that a single construct cannot explain the pattern of observed impairments. These findings may call into question whether habits are truly impaired in some neuropsychological populations. Alternatively, they suggest it may be necessary to distinguish between multiple different forms of habits. I consider these possibilities in light of emerging evidence from neuropsychology, highlight key challenges, and suggest some steps toward an updated integrative framework.

What are we studying when we study habits?

The habit construct is often defined by a collection of attributes: (a) habits are learned gradually rather than being innate, (b) learning and performance can proceed without full attention (i.e., under distraction), (c) learned behavior can be performed automatically, potentially without conscious awareness of what was learned, (d) sequences of simple behaviors may become routinized and performed as single units of behavior, and (e) habitual behavior is inflexible and becomes insensitive to the outcomes of behavior after extensive training [1,5,6]. Generally, stimulus–response (S–R) associations are thought to underlie habit formation [7,8]. However, the habit characteristics do not always cluster together; some may emerge without others [9]. For example, when habit acquisition is followed by outcome devaluation, continued responding for the devalued outcome (the mark of habitual behavior) may or may not be accompanied by awareness of correct response-outcome associations or the change in outcome value. This divergence between attributes results in a tension between efforts to precisely operationalize habits for study and to sufficiently capture the broad phenomenon.

Habit learning in neuropsychological populations — the human lesion model

Much of what we know about habit learning in humans comes from studies of patients with brain damage. Seminal discoveries revealed that damage to the hippocampus and medial temporal lobes (MTL) caused dense amnesia but spared a variety of learning capacities [7,10], some of which shared the features of habits described above. In addition, based on animal lesion studies, it was suggested that behavior is dependent on habit formation following hippocampal damage [7]. Thus, habits were initially defined in terms of capacities that functioned independently of the hippocampus.

The striatum was first described as the primary substrate of habits by Mishkin *et al.* [11] — a proposal that has been supported by a wealth of lesion studies in animals (rats and monkeys) [8,12–16]. Thus, to study habit learning in humans, researchers turned to patient populations with striatal damage. The most commonly studied patients are

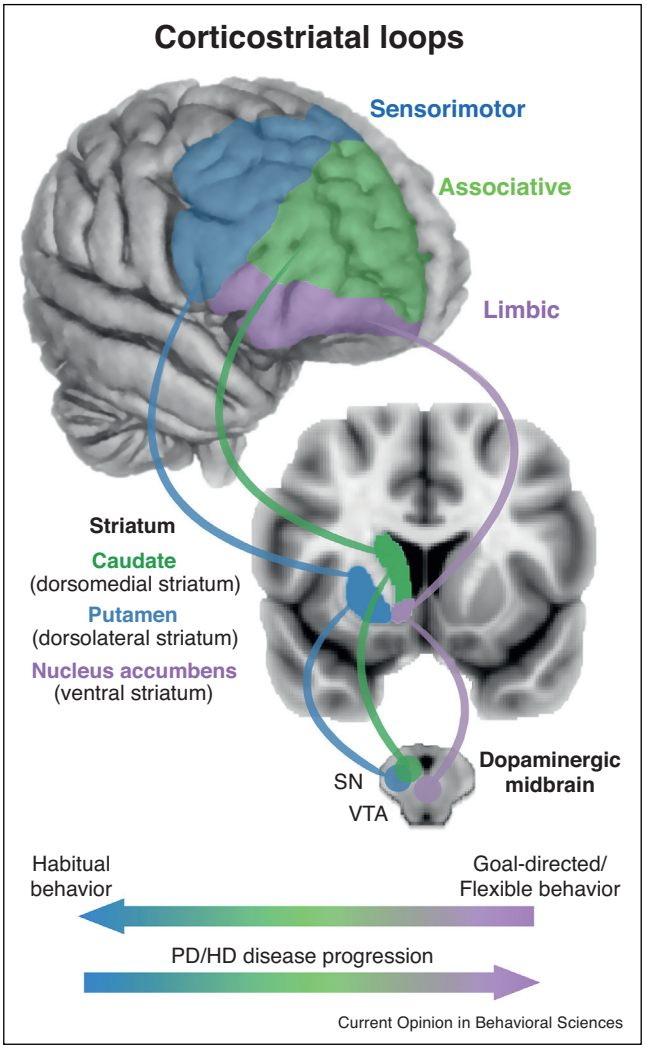
individuals with Parkinson's disease (PD) or Huntington's disease (HD). PD patients have striatal dysfunction due to loss of dopaminergic input from the midbrain to the striatum [17–19], with detrimental effects on learning, memory, and cognition, as well as motivation and affect [20–22]. Dopamine in the striatum conveys critical learning signals [23,24] and underlies learning-dependent plasticity [25]. In HD, striatal dysfunction is the direct result of atrophy and neuronal loss in the striatum [26] rather than loss of dopamine. PD and HD patients are typically studied during early disease stages when dysfunction is relatively restricted to dorsal striatal regions (the putamen in PD; putamen and caudate in HD) as opposed to ventral regions (nucleus accumbens) [17–19,26–28], providing a reasonable analog to lesion studies in animals (Figure 1).

Investigating habit learning in humans

Having identified an appropriate human lesion model, the next significant challenge was to devise appropriate tests of habit learning. A simple solution might be to directly adapt paradigms used in animals, but the risk is that humans and non-human animals may not learn tasks using the same cognitive and neural mechanisms [16,29,30]. Consider the *concurrent discrimination task*: animals learn the correct responses gradually from feedback over hundreds of trials, but healthy adults learn the same responses in just a few trials, and have flexible, declarative knowledge about their learning. Interestingly, Bayley *et al.* [31•] tested two amnesic patients with no declarative memory and found that they learned the task, but did so in a gradual, rote, and inflexible manner. This suggested that while humans can learn S–R habits gradually, like rodents and monkeys, they do so only when declarative memory abilities are inactivated. Such findings reveal the need for tasks that capture gradual S–R habit learning while circumventing rapid, declarative (and MTL dependent) memory.

Perhaps the most commonly-used task thought to achieve this goal is the probabilistic classification task (PCT) [32], which is thought to capture the incremental learning from response-contingent feedback characteristic of tasks used in animals. The PCT requires participants to make binary predictions based on a complex set of cues that are probabilistically associated with outcomes (see Figure 2a, b and caption for detailed task description). The complexity of the task structure is intended to overwhelm the ability to rapidly memorize cue-outcome associations. Indeed, Collins *et al.* recently showed that when working memory was challenged by an increased number of stimuli, participants relied more on gradual S–R learning mechanisms [33•,34]. As expected, studies showed that whereas MTL damage did not prevent learning of the PCT [32,35,36], PD and HD patients were impaired at learning despite having intact declarative task knowledge [35,37–39]. Thus, studies using the PCT in neuropsychological populations confirmed the prediction from animal studies that human habit learning depends on the striatum and not the MTL.

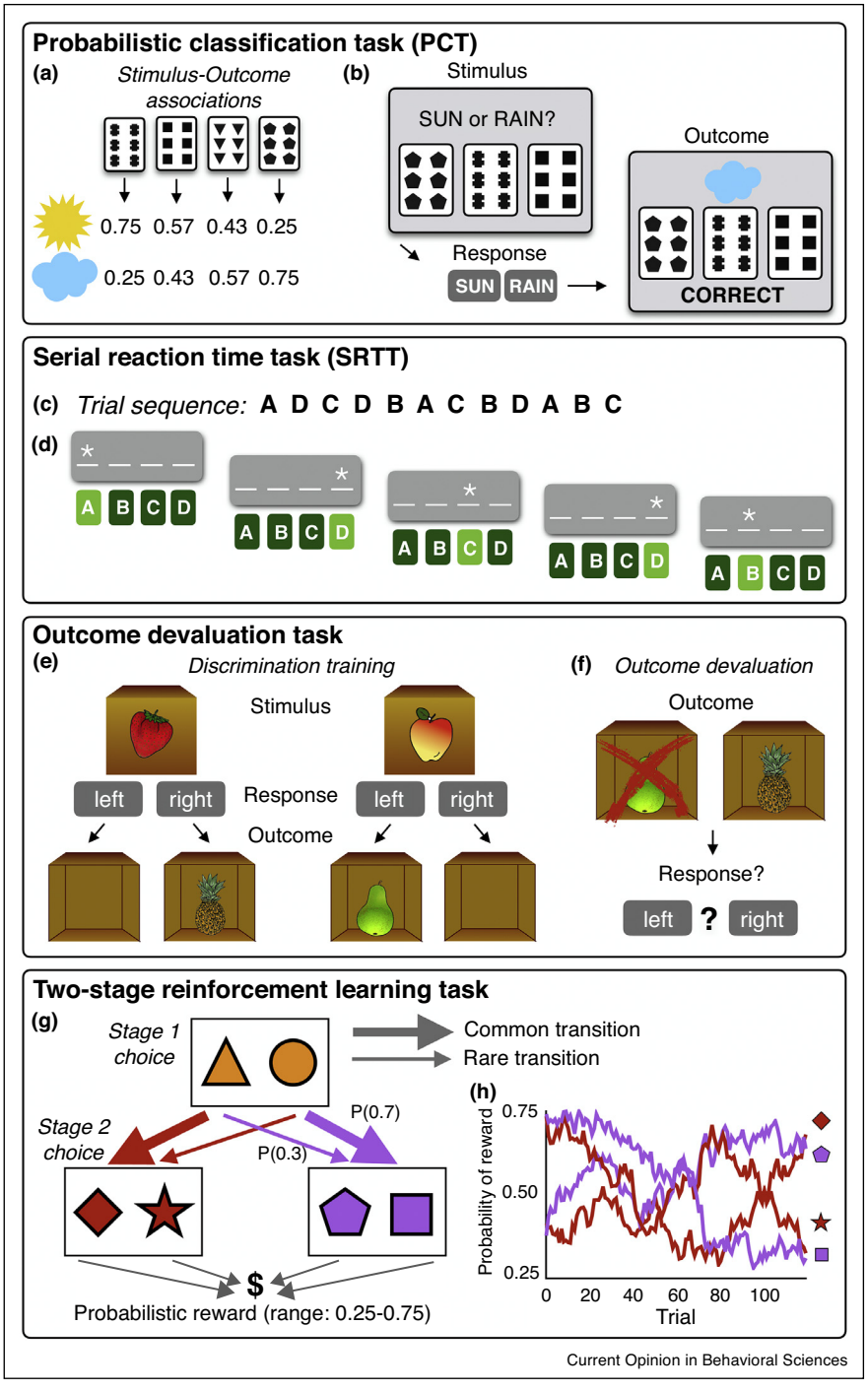
Figure 1



Simplified schematic of three corticostriatal loops important for habitual and goal-directed behavior. (Bottom) Hypothesized progression of involvement of corticostriatal loops as behavior develops from goal-directed to habitual and progression of striatal dysfunction in neuropsychological populations. VTA, ventral tegmental area; SN, substantia nigra; PD, Parkinson's disease; HD, Huntington disease.

Another task used extensively to investigate habits and skills is the serial reaction time task (SRTT; [40]), which involves making rapid motor responses to cues that, unbeknownst to the participant, follow a repeating sequence (see Figure 2c,d and caption for detailed task description). The SRTT captures a different aspect of habits than the PCT — the SRTT assesses the development of chunked representations of series of behaviors that can be executed automatically [1,12]. SRTT type tasks have been translated more literally between use in humans

Figure 2



Tasks commonly used to study habit learning in humans. **(a)** In the probabilistic classification task (PCT) four cues are independently and probabilistically associated with SUN and RAIN outcomes. One, two, or three cards are shown on each trial, combining to form 14 possible stimuli. The complex task structure is intended to circumvent declarative memory for stimulus-outcome associations, and probabilistic feedback requires gradual updating of stimulus–response associations across multiple trials. **(b)** Example trial: a stimulus consisting of three cue cards is shown, requiring a response of SUN or RAIN, followed by feedback indicating the actual outcome on the trial and including the verbal feedback 'correct' or 'incorrect'. Performance is assessed in terms of making optimal responses: a response is considered correct if the more likely outcome for a given stimulus is selected, regardless of whether the response matches the feedback given on that trial. Perfect performance is possible, although feedback will continue to be mixed due to the probabilistic stimulus-outcome associations. **(c)** In the serial reaction time task (SRTT) stimuli are, unbeknownst to participants, presented in a repeating sequence. Typically the sequence used is a 12-item second-order conditional sequence (as the one depicted) in which all first-order transitions (e.g., AB, AC, AD) are equally likely. Thus some form of higher level

and animals (e.g., monkeys [41] and rodents [42]) than has the PCT. A key difference between the SRTT and the PCT may be the role of feedback in learning. On the PCT, internally generated choice responses to stimuli are followed by feedback. By contrast, on the most commonly used SRTT (in humans), responses are prompted by externally signaled stimuli and are not followed by feedback. Perhaps related to this distinction, the SRTT may rely more on the putamen than on the caudate, relative to the PCT. Notably, despite significant differences between the tasks, the PCT and SRTT share the key habit characteristic that learning can occur with limited attention, as when a distracting secondary task must be performed concurrently ([40,43]; see also Table 1). Numerous studies in PD and HD patients have shown that, in contrast to patients with MTL damage [36,40,44], learning on the SRTT is impaired or attenuated following striatal dysfunction [45–48] (but see [49]).

Taken together, a strength of PCT and SRTT studies across multiple neuropsychological populations is that they reveal a double dissociation in MTL versus striatal contributions to habit learning. Importantly, the putatively habitual (S–R and sequence learning) and non-habitual (flexible and declarative knowledge) aspects of behavior are both sensitive to distinct brain lesions. Moreover, the pattern of impairments is consistent with observations and predictions from lesion studies in animals [8,15]. However, while these findings link habits to striatal function vis-à-vis the hippocampus, they do not provide a precise assessment of the specific striatal regions implicated in habits.

In animals, findings point to a specific role for the dorsolateral (putamen in primates) rather than the dorsomedial (caudate in primates) striatum in habit learning [50–52] (Figure 1). However, in humans, the use of dopamine replacement medication in PD raises questions about whether learning impairments result from dysfunction of dorsolateral striatum per se or instead from dopamine 'overdosing' of intact dorsomedial and ventral regions, as suggested by one prominent proposal accounting for the mixed effects of dopamine medication on cognition [53]. It should be noted that effects of dopamine replacement therapy are complex, a topic beyond the scope of this review. Most early studies in PD tested patients while on medication, but a study comparing PCT performance in patients on and off medication found impairments only when on medication [38] (although for some S–R learning tasks, PD patients are impaired on and off medication, e.g., [54,55]). The presence of deficits in HD, which in its early stages implicates the caudate in addition to the putamen, but not the ventral striatum, could also suggest that the caudate rather than the putamen plays a prominent role in this type of learning. Moreover, a patient with lesion damage restricted to the dorsal striatum showed profound impairment on a PCT [56], suggesting that learning deficits are not related to overdosing of the ventral striatum, but consistent with a role for the caudate (also implicated in several fMRI studies, e.g., [57]). Thus, open questions remain about striatal subregion contributions to habit learning in humans.

Are habits affected by striatal dysfunction in humans? A focus on habitual versus goal-directed control

More recently-developed tasks designed to dissect habitual versus goal-directed control of behavior highlight further complexities in human habit learning. For example, the *Outcome devaluation task*, an adaptation of habit assays developed for animal studies, focuses on sensitivity versus insensitivity to outcome devaluation as the hallmark characteristic of goal-directed versus habitual behavior. This distinction arose from animal learning [5,6], and studies employing such assays have contributed to remarkable progress in delineating more precisely the neural circuits underlying habitual and goal-directed behavior in rats. Translation of this approach to humans has, in some cases, been very direct, by using primary food rewards as outcomes and devaluing these outcomes through satiation (e.g., [58,59]). A more common approach has been to use verbal or monetary outcomes and verbally instructed devaluation (Figure 2e,f; see also Watson and de Wit, this issue). Another frequently used task is a sequential decision task, which aims to capture a distinction from computational neuroscience between 'model-free' and 'model-based' learning, with the former thought to underlie habitual S–R behavior and the latter goal-directed behavior [60] (Figure 2g,h). Both tasks have been used extensively in populations with psychiatric conditions thought to involve corticostriatal dysfunction [3,61,62]. By comparison, these tasks have been used

(Figure 2 Legend Continued) chunking of elements is required. **(d)** Participants are instructed to press a key corresponding to the spatial location of the onscreen cue. Learning is primarily indicated by increased differences in reaction times for sequence stimuli compared to stimuli presented in random order. **(e)** During discrimination training in the Outcome devaluation task, participants must learn whether a left or right key press to a stimulus leads to an empty box (and no points) or a filled box (and points). **(f)** During the outcome devaluation test, participants are shown which outcomes no longer are worth points and must continue making responses to accrue points. Here, the correct response is a right key press, as that leads to the still valuable pineapple outcome. **(g)** In the Two-stage learning task, the choice at Stage 1 determines which state is more likely to occur in Stage 2. Each Stage 1 stimulus leads to one of two Stage 2 states with a fixed probability — 70% chance of one state (common transition) and 30% chance of the other state (rare transition). At each Stage 2 state participants choose between two stimuli and discover whether they receive a reward or not. **(h)** Each Stage 2 stimulus is (independently) associated with a gradually shifting probability of reward ranging from 0.25 to 0.75. Habitual behavior is characterized by repeated choices of Stage 1 stimuli that led to reward, regardless of whether the Stage 1 choice resulted in a common or rare transition to Stage 2. By contrast, goal-directed behavior takes into account the transition structure of the task (e.g., a reward would only lead to a repeated choice of the Stage 1 stimulus following a common transition).

Table 1

Characteristics of main tasks used to study habit learning in humans.

Main outcome measures[a]	Patient performance[b]		Role of attention	Tests of behavioral flexibility[d]
	PD	AMN	(dual task sensitivity)[c]	
PROBABILISTIC CLASSIFICATION TASK (PCT)				*Contingency degradation:*
Habits Optimal responses to stimuli	↓	✓	No	Insensitive
Goals/Flexible Knowledge of stimulus-outcome association probabilities. Selecting the most predictive stimulus combinations given specific outcomes.	✓	↓	Yes	Sensitive
SERIAL REACTION TIME TASK (SRTT)				*Process dissociation procedure:*
Habits Decreased RT to sequence trials. Increased RT and errors to random trials.	↓	✓	No	Inclusion = exclusion performance
Goals/Flexible Verbal report of sequence order. Generation of subsequent trial location given initial cued location.	?	↓	Yes	Inclusion > exclusion performance
OUTCOME DEVALUATION TASK				*Slips of action test:*
Habits Sustained responding for devalued outcomes	✓	—	—	Increased slips
Goals/Flexible Decreased responding for devalued outcomes	?	—	—	Decreased slips
TWO-STAGE REINFORCEMENT LEARNING TASK				
Habits Reward sensitivity without sensitivity to transition probabilities	✓	—	No	
Goals/Flexible Sensitivity to transition probabilities between stage 1 and stage 2	↓(PD -) ✓(PD+)	—	Yes	

[a]Main measures of habitual and goal-directed/flexible behavior on the PCT, SRTT, Outcome devaluation task, and Two-stage reinforcement learning task.

[b]Patterns of impairment in neuropsychological populations. PD: Parkinson's disease; PD—: PD off medication; PD+: PD on medication; AMN: amnesic; **✓:** similar to healthy groups; **↓**: impaired relative to healthy control participants; ?: mixed results; —: not tested.

[c]Sensitivity to dual task interference is a classic test of whether a cognitive process relies on attentional resources, or instead can proceed relatively automatically. Dual task performance requires participants to perform the primary task while concurrently performing a secondary task (e.g., keeping a running count of the number of tones played at random times or holding a string of digits in mind during the primary task). As seen in the table, diminished need for attention is a unifying characteristic of habit learning across tasks.

[d]Behavioral flexibility can be tested in some form in multiple tasks. In the PCT, measures of declarative knowledge require some flexibility in the representation of stimulus-outcome associations. However, *contingency degradation*, used to assess habitual and goal directed behavior in animal studies, could potentially be applied to the main performance outcome measure by degrading the contingencies between stimuli and RAIN/SUN outcomes. In the SRTT, the *process dissociation procedure* (PDP) has been used to assess whether sequence knowledge is automatic or under flexible control. The PDP involves responding to or observing one or more cues from the trained sequence and then generating a response. Under *inclusion* instructions, the response should be the next likely location in the sequence. Under *exclusion* instructions, the response should not overlap with the trained sequence. If sequence knowledge is mainly automatic and not under flexible control, sequence locations will be generated despite instructions to avoid such responses. The Outcome devaluation task often includes a *slips-of-action test* in which the balance between habitual and goal-directed control is tested. As in the devaluation phase of the task (**Figure 2f**), a verbal/graphic instruction indicates which outcomes are no longer valuable. Participants are then required to respond rapidly to presentation of individual stimuli and make a response (Go) to still valuable stimuli and withhold a response (No go) to no-longer valuable stimuli.

much less frequently in neuropsychological populations in which underlying neuropathology is better characterized. In virtually all cases, these studies have demonstrated deficits in goal-directed control of behavior with concurrent sparing of habits.

In terms of understanding human habits, an obvious approach would be to use these tasks in patients with known striatal lesions. Particularly PD, which in its early stages mainly affects the putamen, should be most instructive. However, in the two existing studies that tested patients with PD (or any other neuropsychological disorders known to affect the striatum), habit learning was intact and, instead, goal-directed behavior was impaired [63,64•]. Notably, in the Sharp *et al.* study [64•], the same PD patients were tested both off (after overnight withdrawal) and on dopaminergic medication, and habit learning was intact in both medication states, whereas goal-directed behavior was impaired off medication and restored to the level of healthy controls on medication.

These results were puzzling in light of previous work, and also due to the strong expectation for a critical role for the putamen and dopamine in habit learning based on numerous animal [50] and human fMRI studies [43,59,65]. Importantly, the recent results in PD patients question whether previous studies in humans truly were testing habit learning. In some cases there appears to be direct conflict between outcomes. Patients with Tourette syndrome, which is thought to involve structural and functional dysfunction of corticostriatal circuits, exhibited enhanced habit learning on an outcome devaluation task [66], whereas a study using the PCT found impaired habit learning [67]. In both studies, the results were most prominent in unmedicated patients. However, cross-study comparisons may be complicated by the heterogeneity of disease presentation and variety of medications used (e.g., neuroleptics, antidepressants, benzodiazepines), and both factors may relate to comorbidities. Nonetheless, such mixed results highlight the need for greater clarity in how habits are defined and tested — particularly when behavioral tasks are used as tools to examine the underlying neuropathology in populations in which it is less well understood.

One possibility is that, contrary to assumptions, earlier studies using the PCT captured a deficit in goal-directed learning — a form of learning necessary for the initial acquisition of habits. The acquisition phase may involve dorsomedial circuits preceding transition to a reliance on dorsolateral circuits [50] (see Figure 1). Indeed, the critical role for the caudate may be learning from feedback [68,69]. Another possibility is that more recent tasks lack the sensitivity to assess habit learning (or may in fact tap learning that is not strongly related to habits [70•]). I am unaware of research that has found selective deficits in habit learning while sparing goal-directed behavior in psychiatric or neuropsychological populations on these recent tasks. Similarly, experimental manipulations in healthy individuals have been shown to interfere with goal-directed behavior while sparing habits, with no interventions leading to the reverse pattern. Furthermore, no studies have tested populations with memory impairments. Although this may appear less critical, a shared characteristic of habit learning across animals and humans has been independence from the MTL, which has not been established for these recent tasks in humans.

Thus, despite the general appearance of a solid understanding of habits and their neural substrates as a result of decades of work in animals and humans, the current state of affairs is in fact much less clear-cut. What is needed is a reassessment of the evidence, the scientific goals, and the means of studying habit learning in humans.

Improving the research agenda

In this section, I suggest steps that may guide a future research agenda toward greater clarity and true convergence across approaches to habits.

A multifaceted definition of habits. The current broad definition of habit is an overarching problem, as consensus that a particular task targets habits per se may not exist. To aid crosstalk between research communities and avoid clustering around particular paradigms, more precision on which attribute of habits one is studying (e.g., insensitivity to outcome devaluation vs. chunking action sequences) and, importantly, how it is being operationalized will be useful. Moreover, some attributes are fairly general but are labeled differently depending on the task. For example, 'inflexibility' overlaps substantially across most definitions of habit. Although inflexibility is in itself a vague term, it has been or could be operationalized and tested in most paradigms (see Table 1).

More precisely operationalized tests. Tasks must have both specificity and sensitivity. Specific outcome measures are needed within tasks to distinguish between habitual and goal-directed behavior, but both measures should be sensitive to interference of some kind, as is the case in animal work. While tasks that allow translation between studies in animals and humans are ideal, such translation is not without challenges. For example, outcome devaluation has been applied successfully across animals and humans. Yet the difficulty capturing habit circuits in humans using this approach suggests that translation of tasks from animals to humans is less direct than hoped. At a minimum, the assumption that tasks are functioning similarly across species should be tested explicitly. Conversely, for the PCT, in which habit learning measures show sensitivity to brain damage in humans, most measures of flexible, declarative knowledge lack a clear analog in animal learning.

Importance of human lesion models. Human lesion models are far from perfect; they lack the precision of animal

models, a particular shortcoming given the important differential roles of subregions within the striatum. Additionally, human lesions represent a static intervention, which is problematic given that habit learning is dynamic and proceeds gradually. Nonetheless, as of now, neuropsychological populations provide the strongest available test of the necessary role of the striatum in habit learning. Studying neuropsychological populations in conjunction with fMRI studies in healthy individuals may be particularly helpful to address the latter issue.

Conclusion

Habits are central to human behavior and cognition, and a clear understanding of their operations and neural basis is crucial for understanding behavior in both health and disease. Yet it is now clear that classic approaches to defining habits and assessing them with experimental tasks require further development. To this end, improvements on the current research agenda are needed at multiple levels. The use of neuropsychological populations is critical to this effort, and used in conjunction with neuroimaging and carefully designed behavioral paradigms, hold the most promise for elucidating the neural bases of habits.

Conflict of interest statement

Nothing declared.

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The authors discuss problems with the distinction between model-based and model-free reinforcement learning and highlight some problems accounting for habitual behaviors within this framework. They forward a proposal that integrates ideas about sequential learning and model-based reinforcement learning to account for goal-directed and habitual behavior.