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The Role of Habits in Anorexia Nervosa: Where We Are and Where to Go From Here?

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Abstract

Purpose of Review—The persistent maladaptive eating behavior characteristic of anorexia nervosa (AN) can be understood as a learned habit. This review describes the cognitive neuroscience background and the existing data from research in AN.

Recent Findings—Behavior is habitual after it is frequently repeated and becomes nearly automatic, relatively insensitive to outcome, and mediated by dorsal frontostriatal neural systems. There is evidence for such behavior in AN, in which restrictive intake has been related to dorsal frontostriatal systems. Other neural and neurocognitive data provide mixed findings, some of which suggest disturbances in habit systems in AN.

Summary—There are compelling behavioral and neural data to suggest that habit systems may underlie the persistence of AN. The habit model needs further research, via more direct behavioral hypothesis testing and probes of the development of habitual behavior. Investigation of the habit-centered model of AN may open avenues for the development of novel treatments.

Keywords

Anorexia nervosa; Habit; Reward; Cognitive neuroscience; Eating disorders; Frontostriatal

Introduction

Anorexia nervosa (AN) is a serious and debilitating illness with one of the highest mortality rates of any psychiatric disorder [1]. Defined by relentless restriction of food intake leading to an inappropriately low body weight, distortion of body image, and intense fear of weight gain [2], AN affects approximately 1% of women [3] across all socioeconomic classes [4]. Despite decades of research, the neurobiological mechanisms underlying AN are not well understood. Cognitive neuroscience has made significant strides in understanding the neural mechanisms underlying human behavior. These advances, when applied to AN, create a

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framework for probing the salient behavioral disturbances that characterize this disorder. In particular, increased understanding of how behaviors are learned and become almost automatic may shed light on the persistence of maladaptive food restriction in AN. In this article, we review these concepts from cognitive neuroscience and relevant data from recent research on AN.

The Cognitive Neuroscience of Persistent Behavior

Advances in the cognitive neuroscience of learning have implications for understanding the neural mechanisms of behavioral disorders. One important theoretical framework organizes behavior into goal-directed and habitual actions [5–7]. Goal-directed behavior is acquired via what is sometimes referred to as stimulus-response-outcome learning. In the presence of a particular cue, an action is followed by a reward (e.g., a mouse presses a lever to receive a pellet of food). This link between behavior and reward underlies the acquisition of new behaviors. By definition, stimulus-response-outcome learning is sensitive to the receipt of reward; thus, if a behavior is no longer rewarded (e.g., the mouse no longer receives a pellet of food after pressing the lever), engagement in the behavior will decrease. Goal-directed learning has been shown to engage neural pathways involved in reward, including the ventral striatum (nucleus accumbens), amygdala, and the orbitofrontal cortex (OFC).

Habits are characterized as learned, automatic behaviors that, once elicited by external or internal cues, proceed to completion with little conscious oversight. Habitual behaviors initially begin as goal-directed actions. With sufficient repetition, these goal-directed actions (i.e., stimulus-response-outcome behaviors) gradually become less sensitive to the outcome and more tightly linked to the circumstances (i.e., stimuli) surrounding the action (e.g., the mouse, having learned that pressing a lever in a specific cage often produces a rewarding pellet of food, will continue to press the lever even if the delivery of a pellet becomes increasingly rare). Less reliant on outcome, the behavior becomes increasingly automatic and linked to a cue, and is now termed stimulus-response learning. This process underlies the formation of habits [7–9]. By shifting to stimulus-response, this now habitual behavior becomes more cognitively efficient. That is, as behavior becomes more automatic, cognitive resources are freed up for other activities. For example, following the usual route home alleviates the need to focus on every turn and allows you to think about other things, such as what to make for dinner.

Extensive animal research has demonstrated that the neural pathways engaged in stimulus-response learning include the dorsolateral prefrontal cortex and the dorsal striatum [10–15]. These findings have been convincingly replicated in human studies involving participants with known striatal dysfunction from neurodegenerative disorders such as Parkinson’s disease and Huntington’s disease [16–19]. In psychiatric populations, disturbances in goal-directed and habitual systems have been implicated in disorders such as OCD [20, 21] and substance abuse [22, 23] in which affected individuals repetitively engage in maladaptive, habit-like behaviors. In substance abuse and OCD, theories have emphasized the shift from impulsive to compulsive behavior [22, 24] and this theory has been extended to eating disorders as well [25]. This perspective is consistent with habit development, as “compulsive” can be viewed as “habitual.” It may be that engagement of dorsal frontostriatal

circuits underlies the persistent and life-threatening dietary restriction seen in AN (see Fig. 1).

Maladaptive Eating Behavior in AN

AN usually begins with a reduction in caloric intake below metabolic needs. The restriction of food intake is commonly called “dieting” but may not start as intent to lose weight (e.g., in some patients, the initial weight loss is secondary to a medical problem or a change in eating for reasons other than achieving weight loss). Although the precipitating events leading to the decision to restrict dietary intake vary, once established, disturbances in eating behavior among patients with AN are remarkably stereotyped [26–29]. Almost uniformly, affected individuals consume a low calorie diet with limited variety and low energy density, with particular emphasis on avoidance of dietary fat [26–28]. These restrictive food intake behaviors often persist even after intensive treatment leads to normalization of weight [26, 28, 30, 31]. Studies using videotaped meals have also demonstrated disturbances in mealtime behaviors, such as delayed onset of eating and manipulation of food (e.g., tearing, nibbling, and picking), which reliably distinguish the behavior of individuals with AN from that of healthy controls [32, 33]. Furthermore, dietary intake patterns indicate that decreased diet variety and energy density are associated with poorer outcome and risk of relapse over time [34, 35].

The Role of Habit in AN

The maladaptive eating behaviors characteristic of AN are consistent with cognitive neuroscience definitions of habit [36]. The dieting behavior, which commonly begins in adolescence, is experienced as rewarding for reasons specific to the individual. For some, it may be compliments on physical appearance or the steadfast adherence to an apparently “healthy” diet. For others, it may be relief of anxiety or increased sense of accomplishment and self-control. This reinforcement is sufficiently rewarding to support continuing the dieting behavior, which is then repeated multiple times daily over weeks to months. What began initially as a goal-directed behavior responsive to reinforcement gradually becomes less dependent on outcome and increasingly resistant to change [28, 30]. Food restriction becomes tightly linked to a range of external and internal cues, and less sensitive to the original reward or outcome [36]. This model implies that neural systems associated with habitual behavior, specifically, fronto-dorsal-striatal circuits, underlie the persistent food restriction of AN.

Other neurobiological models of AN have also suggested the involvement of habit-related circuits. Godier and Park [37] emphasized the role of dopamine and stress in catalyzing the development of habitual, “compulsive” behaviors that promote and maintain weight loss. O’Hara’s [38] model discusses cognitive and behavioral habits in AN, and proposes that illness is mediated by striatal circuits. Lloyd and colleagues [39] also describe behaviors in AN as habitual and propose that these habits develop because of anxiety and the anxiolytic properties of starvation.

A habit-centered framework provides a way to understand many of the perplexing behaviors exhibited by individuals with AN. Particular cues (i.e., stimuli), such as sitting down in front of a meal, elicit ritualized behaviors (i.e., responses), such as cutting food into small pieces or eating foods in a particular order, which serve to reduce caloric intake. Other common disorder-related behaviors, such as body checking, binge eating, and exercise, also demonstrate patterns suggestive of a cue-behavior relationship in that they tend to occur at predictable times of day and are frequently preceded by changes in mood [40, 41]. These may also be examples of persistent stimulus-response behavior in AN.

Tests of a Habit-Centered Model in AN: Neuroimaging

A key prediction of the habit hypothesis posits that restrictive food intake in AN is associated with activity in neural structures such as the dorsal striatum and dorsolateral prefrontal cortex (dlPFC), as these are known to be engaged during habitual behavior [36]. Our group tested this hypothesis using a food choice task which examined neural activity via fMRI while patients made decisions about what to eat (with real life consequences) [42••]. In the food choice task, participants are asked to choose between a food they rated as "Neutral" on both healthiness and tastiness and 75 other food items. Choice of a range of foods is then measured by asking patients to indicate their preference between the reference item and a series of images of high and low fat foods. At the conclusion of the experiment, one of their choices is randomly chosen and the individual is asked to eat it, helping to assure that patients' choices reflect their actual preferences. The validity of the task in AN has been established through demonstrating that patients choose fewer high fat foods than do healthy controls, and that selection of high fat foods is correlated with actual caloric intake on the following day [42••, 43]. Neural activity during food choice of individuals with AN was compared to that of controls. Both groups showed the expected, and similar, activity in mesolimbic reward regions, including the medial prefrontal cortex. In contrast, the groups did differ in that there was significantly greater activity in the dorsal striatum among the individuals with AN during food choice. Furthermore, in the AN group, the level of connectivity between the dorsal striatum and the dorsolateral prefrontal cortex was significantly correlated with food intake on the day following the experiment [42••].

Though not a direct test of the habit-centered model, this neuroimaging study demonstrates that individuals with AN engage different neural circuits than do controls when choosing what to eat and provides suggestive data supporting a role for habits in the perpetuation of AN as well as information about potentially key neural circuits for intervention.

Tests of a Habit-Centered Model of AN: Behavior

The fact that these studies of neural activity are consistent with predictions based on a habit-centered model of AN is compelling, but, since neuroimaging research is inherently correlational, such evidence must be viewed as circumstantial. Testing the habit-centered model of behavior requires behavioral experiments. A central behavioral prediction of the habit model is that the individual's actions are less dependent on the outcome, and are more tightly linked to the cue. One way to test the cue-response relationship is to attempt to break the connection and measure the effect on behavior. Our group designed a behavioral

intervention, developed from habit-reversal techniques, that focuses specifically on antecedent cues for maladaptive behavior [44•]. In a small, proof-of-concept RCT, participants received either 12 sessions of a manualized treatment consisting of cue-awareness, creation of new behavioral routines, suppression of maladaptive habits, and emotion regulation or standard supportive psychotherapy. Habit strength, or the likelihood a behavior will be elicited by a particular stimulus or context, was measured via the Self-Report Habit Index (SRHI). The SRHI is a self-report measure developed in the social sciences literature which assesses a combination of behavior frequency and automaticity [45]. Compared to those treated with supportive psychotherapy, individuals who received the habit-reversal intervention showed a significantly greater reduction in SRHI score and on measures of eating disorder-specific psychopathology, as well as a trend-level increase in food intake in a laboratory test meal. Though preliminary, these results support the habit model of AN by demonstrating that the maladaptive behaviors characteristic of AN are cue-dependent. These results are also in line with those of Coniglio and colleagues [46] who found that habit strength for dietary restriction (as measured by the SRHI) was associated with the severity of food restriction and eating disorder-related functional impairment in a sample of individuals with AN and atypical AN. Furthermore, of several tested variables, including diagnosis (atypical AN vs AN), illness duration, and cognitive restraint, only habit strength was found to be a significant predictor of variance in reported food restriction.

Other efforts to examine habit in AN have focused on habit learning and neurocognitive tasks administered in a laboratory setting. One approach focuses on outcome sensitivity and devaluation of monetary rewards, and tests how behavior in response to cues changes as the reward value changes. For example, the slips-of-action task measures participants' ability to refrain from responding to cues when the rewards associated with them are devalued [47]. After first learning that stimuli are associated with particular rewards, the reward values change and participants must learn to select the valuable stimuli while avoiding those no longer associated with reward. Persistent responding to devalued outcomes is interpreted as reliance on habitual associations (as observed in experiments in rodents for which the procedure was established). Although reduced sensitivity to outcome devaluation has been described in numerous psychiatric populations, including OCD [20], recent experiments with this task did not identify differences between AN and healthy participants [48]. Thus, in this laboratory-based, non-food-related task, individuals with AN exhibited no greater reliance on habit-centered mechanisms than did healthy individuals. It may be that a task using eating-relevant stimuli would yield different findings, but this has yet to be examined.

Efforts to better understand the role of habit in AN have also included administration of tasks which examine how learning from reward and feedback shape behavior [49, 50]. These tasks have known underlying neural substrates, and can be used to probe relevant neural systems [51]. Our group administered a task with known striatal-dependent incremental learning and hippocampal-dependent transfer of learning components (acquired equivalence task [49]). Individuals with AN, while underweight and after weight restoration, showed decreased learning in the striatal-dependent learning component, with no group differences in the hippocampal-dependent transfer of learning [52]. These findings suggest that individuals with AN seem to show slower learning from feedback. As reinforcement learning shapes interactions with the environment, this task suggests an impairment among

individuals with AN that could interfere with efforts to learn new behaviors, which is often a goal of treatment. Using a two-step reinforcement learning task designed to measure the relative influence of goal-directed ("model-based") learning versus habitual ("model-free") learning [50], Gillan and colleagues [53] demonstrated in a large, internet-based study that eating disorder-related symptomatology was associated with deficits in goal-directed control. Although these studies do not directly test habitual behavior in AN, their findings suggest deficits in learning and altered response to feedback which may contribute to the formation and persistence of habit.

Conclusions and Future Directions

Though an increasing body of evidence supports the role of habit in the persistence of weight loss behaviors characteristic of AN, challenges remain in constructing a definitive test of this model. One salient issue has been the challenge of translating experimental approaches from animals to humans. For example, while animals may require hundreds of trials to learn the correct response during a feedback learning task, healthy humans are able to respond correctly after only a few trials [54]. Another challenge facing translational research is discerning whether reduced sensitivity to outcome devaluation represents robust habit formation, weak goal-directed behavior, or both [55•].

Paradigms to test response to outcome devaluation in humans have been very challenging. The NIMH Research Domain Criteria (RDoC) Council Workgroup recently published a critical review of the measures in the RDoC matrix [56]. They included three tasks that address the construct of habit at the behavioral level using a mixture of reinforcement learning and devaluation components, and none of these has been used extensively [57–59]. Nonetheless, a few promising paradigms for probing the habitual nature of behavior do exist. One widely used approach is the two-stage decision task (or Two-Step Task [50]) which measures participants' ability to make choices based upon the predicted likely outcomes of their actions on a trial-by-trial basis [60•, 61]. Individuals with OCD, as well as other psychiatric illnesses, have shown a reduction in model-based (i.e., goal-directed) learning on this task [61]. However, some studies have found a relationship between model-based (but not model-free) learning and devaluation sensitivity [60•, 62], which suggests that model-free learning may not effectively capture the stimulus-response learning driving habit formation [55•].

A second approach is to create a dual-task condition [63]. In this experimental design, while completing one task (e.g., a decision-making task), participants are simultaneously asked to engage working memory in a separate task. The secondary task depletes available declarative memory and biases the participant to utilize more automated, habit-learning processes in the decision-making component. In healthy individuals, the addition of a cognitive task leads to more automated behavior [63]. For example, participants could be asked to make choices between highly palatable or highly healthy food items while simultaneously conducting a math task. A related approach to investigating the influence of habit on behavior builds on the resource depletion model which proposes that increased reliance on habitual behavior in the setting of serial decision-making effectively "depletes" self-control resources [64]. The applicability of this approach to examining habit in eating

behavior was recently demonstrated in a study which demonstrated that healthy volunteers were more likely to select a healthier, habit-trained food option (carrots) than the more preferred unhealthy item (M&M's) after completing a resource depletion task [65]. Finally, the Pavlovian-to-instrumental transfer (PIT) task is a learning paradigm that tests whether a learned (conditioned) association leads to outcome-independent behavior within the task (test phase) [66]. These tasks have been administered among patients with substance-use disorders and obesity to examine the influence of cues on behavior [67–73]. All of these approaches that probe behavior may be helpful in understanding the role of habit in AN.

Viewing the persistent restriction of food intake in AN from the perspective of habit formation suggests a number of hypotheses to be examined. For example, early in the development of AN, habits should be less well established, and engagement of dorsal-striatal circuits should be less robust. In contrast, the strength of habits, measured both behaviorally and via neuroimagining, should be related to chronicity and to refractoriness to treatment. On the other hand, long-standing recovery should be associated with reduced reliance of food choice on habit mechanisms.

A habit-driven model of AN presents some new directions for treatment development, as well. The preliminary yet promising results from our pilot study suggest that a behavioral intervention built upon habit-reversal techniques can improve eating disorder psychopathology, including restrictive dietary intake [44•]. Advances in brain-based technologies and neuromodulation, such as repetitive transcranial magnetic stimulation (rTMS), may be able to target habit circuitry [42••]. Though rTMS is already a well-established therapeutic tool in some psychiatric disorders [74], work has only recently begun to investigate its use in AN. Early results are promising, with recent proof-of-concept studies showing an effect of rTMS on AN symptomatology [75]. Treatment development research in AN could emphasize behavioral approaches to habit, neuromodulatory approaches to the dorsal frontostriatal circuits that underlie habits, and/or dopaminergic pharmacologic interventions that may impact the development or persistence of habits.

As described above, existing data provide compelling evidence for habits and engagement of dorsal frontostriatal circuits in the salient behavior of maladaptive food intake in AN [42••, 44•, 46]. These findings open new questions regarding the development of AN. When does restrictive food intake become habitual? Is behavior governed by different neural circuitry from the outset (i.e., a predisposing vulnerability)? Or does this emerge with the progression of illness, either with time or with severity? It will also be useful to understand whether these neural systems change with existing treatments. If not, these may be useful targets for novel approaches. Better understanding of habits in AN holds promise for improving outcomes of this devastating illness.

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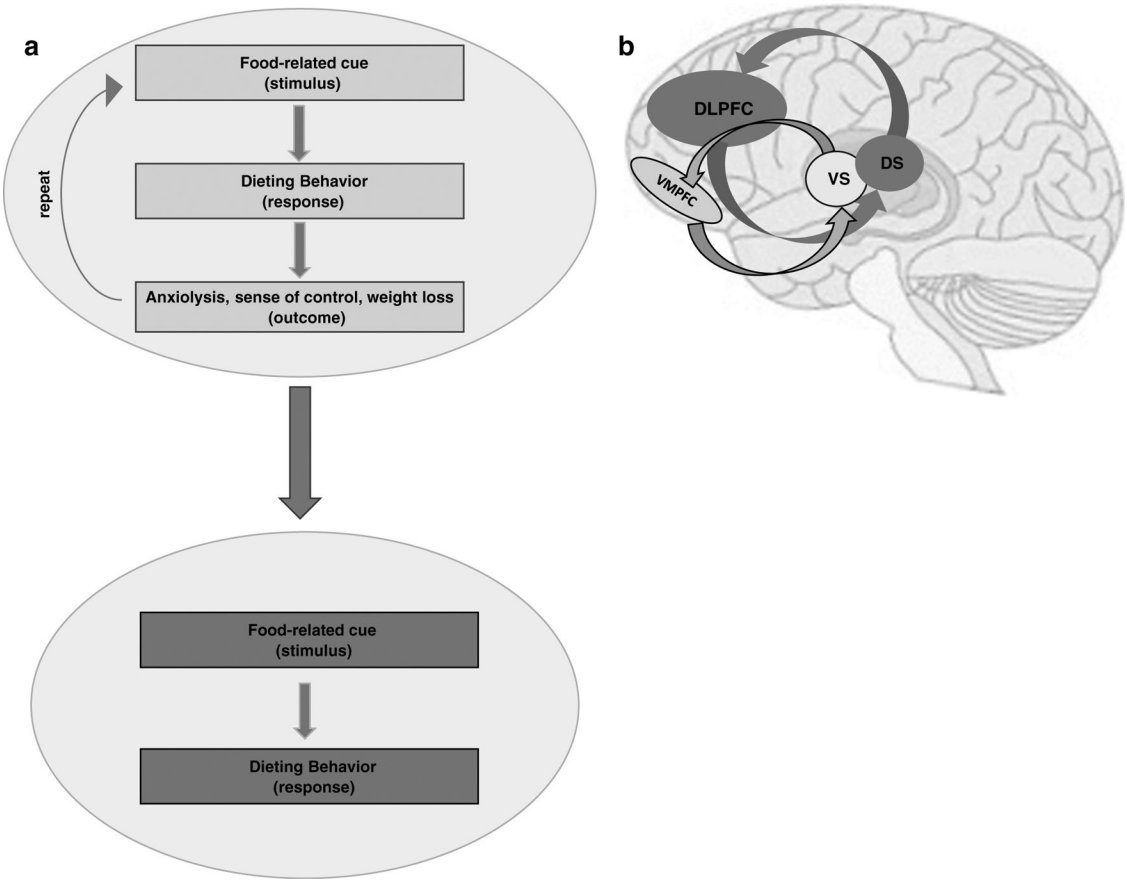

Fig. 1.

a Habit-centered model of AN. Early in illness, the characteristic dieting behavior of AN involves stimulus-response-outcome learning. In this model, dieting behavior begins as a goal-directed action that is taken in response to a stimulus (e.g., mealtime) and is sensitive to outcome (i.e., reward). Repeated multiple times daily over weeks to months, the behavior gradually becomes less insensitive to outcome and occurs almost automatically (or habitually) after exposure to a food-related stimulus. **b** Neural representation of reward and habit circuitry. During stimulus-response-outcome learning, dieting behavior is sensitive to the receipt of reward and engages key reward-related circuits including the ventral striatum (VS) and ventromedial prefrontal cortex (vmpFC). As behavior becomes less reliant on reward and more automated, habit-associated structures are engaged, including the dorsal striatum (DS) and dorsolateral prefrontal cortex (dlPFC)