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Habit formation: Implications for alcoholism research

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ABSTRACT

Characteristics of individuals with severe alcohol use disorders include heightened cue sensitivity, compulsive seeking, craving, and continued alcohol use in the face of negative consequences. Animal models are useful for understanding behavioral and neurological mechanisms underlying problematic alcohol use. Seeking of operant reinforcers including alcohol is processed by two mechanisms, commonly referred to as "goal-directed" (action-outcome) and "habitual" (stimulus-response). As substance use disorders are characterized by continued use regardless of unfavorable outcomes, it is plausible that drug use causes an unnatural disruption of these mechanisms. We present a critical analysis of literature pertaining to behavioral neuroscience alcoholism research involving habit formation.

Traditionally, when operant behavior is unaffected by a loss of subjective value of a reinforcer (devaluation), the behavior is considered habitual. Acquisition of instrumental behavior requires corticostriatal mechanisms that depend heavily on the prefrontal cortex and ventral striatum, whereas practiced behavior is more predominantly controlled by the dorsal striatum. Dopaminergic signaling is necessary for the neurological adaptations involved in stimulus-response action, and drugs of abuse appear to facilitate habitual behavior through high levels of dopamine release. Evidence suggests that the use of alcohol as a reinforcer expedites habit formation, and that a history of alcohol use produces alterations in striatal morphology, aids habit learning for non-psychoactive reinforcers, and promotes alcohol drinking despite aversive adulterants.

In this review, we suggest directions for future alcoholism research that seeks to measure action made despite a devalued outcome, including procedural modifications and genotypic, pharmacological, or neurological manipulations. Most alcoholism models currently in use fail to reach substantial blood ethanol concentrations, a shortcoming that may be alleviated through the use of high-drinking rodent lines. Additionally, satiety, one common mechanism of devaluing reinforcers, is not recommended for alcohol research because the psychoactive effects of alcohol depress response rates, mimicking devaluation effects. Overall, further research of habit formation and potentially related perseverative behaviors could be invaluable in discovering genetic variance, traits that correlate with persistent alcohol seeking, implicated neural structures and processes of alcohol use, and eventually novel pharmacological treatment for alcoholism.

Introduction

Alcoholism is a chronic disease of uncontrollable alcohol use. Alcoholics usually oscillate between abstinence and relapsed heavy use; accordingly, abstinence "survival" curves demonstrate that the percentage of successfully treated alcoholics in a given sample decreases over time (Kirshenbaum, Olsen, & Bickel, 2009). Prolonged use of alcohol is correlated with greater resistance to treatment, including therapy and community-based interventions, prompting recent experimentation with procedures using medication and/or deep-brain stimulation (e.g., Müller et al., 2009; Pastor, Jones, & Currie, 2012). Consistent with this idea, naltrexone, an opiate antagonist used to treat some alcohol use disorders (AUDs), is relatively ineffective in chronic, severe alcoholics (Krystal et al., 2001), a finding suggesting that prolonged use of alcohol may alter the neural substrates affected by its use. However, the mechanisms by which treatment-resistant alcoholism differs from less persistent AUDs are unclear. Elucidation of these mechanisms could lead to treatments that are more successful.

Substance use is linked to craving and positive expectancies about outcomes, associating it with intentional behavior (Robinson & Berridge, 2003). However, the inability to quit despite an

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intention to do so is a criterion for clinical diagnosis of substance dependence (American Psychiatric Association, 2013), implicating use as a process that persists in spite of a desire for other outcomes. These conflicting but not mutually exclusive notions suggest activation of two processes, called goal-directed and habitual behavior (Hogarth & Chase, 2011). Goal-directed behavior is defined as an action (e.g., an instrumental response) mediated by its association with a desirable outcome (Dickinson, 1985). Habitual behavior, in contrast, is defined by responding with equal strength to an associated desired or aversive outcome alike, indicating indifference to the present value of reinforcement. Habitual responses are quickly elicited by a stimulus linked to a lengthy reinforcement history, also known as a "trigger" or "cue" in the environment (Schulte et al., 2012). It is suggested that drug exposure subverts natural habit learning toward an automatized drug-driven condition, creating ingrained stimulus-response habits that are resistant to attempts at behavioral change (Belin, Jonkman, Dickinson, Robbins, & Everitt, 2009; Everitt & Robbins, 2005; Robinson & Berridge, 2003).

The susceptibility of a dependent individual to a cue that instills craving for and seeking of a substance that causes adverse consequences is phenomenologically similar to a stimulus provoking responding for a devalued outcome in an animal model. Habitual behavior in animal models is commonly measured using devalued outcomes through conditioned aversion or specific satiety, as described in detail below. Recently, there has been a great interest in habit formation using alcohol as a reinforcer, or facilitation of habitual responding for another reinforcer during concurrent alcohol consumption. Both of these are exciting prospects, given recent findings regarding the analogous behavior of human alcoholics, particularly cue sensitivity predicting relapse susceptibility and lack of planning and/or goal-directed perseveration (Coskunpinar, Dir, & Cyders, 2013; Schulte et al., 2012). Traditional habit formation and other manifestations of perseverative behavior may be critical mechanisms for the understanding and treatment of chronic drug abuse. An in-depth examination of how these concepts relate to alcoholism may prove valuable to the goal of developing improved interventions. The intention of this manuscript is to review human and animal literature in support of this pursuit.

Behavioral classifications using conventional reinforcement

The principle that a trained animal will perform an action while in a motivated state to achieve a desired outcome, such as a food or fluid reinforcer, has been described for over 100 years (Thorndike, 1911). Seminal research investigating the effects of post-operant conditioning manipulations of subjective value of appetitive reinforcers was conducted using rodents in the early 1980s in order to better understand the associative structure underlying instrumental learning (Adams, 1980). Pairing a gustatory reinforcer with lithium chloride (LiCl)-induced illness creates an aversion to that specific reinforcer, as demonstrated both by conditioned aversion to the taste of the reward and avoidance of the location where the LiCl was presented (Adams, 1982; Chen & Amsel, 1980). Alternatively, unlimited access to the reinforcer is used to instill satiety prior to operant testing (e.g., Coutureau & Killcross, 2003). A third, less common, process of manipulating the value of a reinforcer is through classically conditioned pairing with high-speed rotation, inducing mechanical rather than chemical illness (Holland & Rescorla, 1975). These procedures are commonly referred to as reinforcer devaluation. In the operant setting, motivation to respond would logically be greatly reduced once that reinforcer is devalued. However, Adams (1980) first reported that LiCl pairing had no effect on responding during extinction conditions (i.e., without reinforcer delivery, a measure of isolated motivation). That is, when animals were not directly exposed to the reinforcer during instrumental behavior, response rates for a devalued expected outcome were the same as for one whose appetitive qualities remained intact. These experimental data are consistent with behavior that corresponds to a mechanistic stimulus-response model, in which an action is either an innate or an acquired habit that is triggered by a particular stimulus (Dickinson, 1985).

In contrast, behaviors that are sensitive to reinforcer devaluation are goal-directed, manifested according to a teleological (i.e., purposeful, or outcome-driven) model of animal behavior (Dickinson, 1985). Operant behaviors are initially more goal-directed in nature, but they tend to become primarily driven by habit following extended practice. This shift is regarded as a change in the dominant force behind behavior from response-outcome associations to stimulus-response associations (Balleine & Dickinson, 1998; Rescorla, 1994). Unlike habitual behavior, goal-directed behavior is dependent upon specific motivation for an outcome; for instance, if given a choice of two actions under extinction conditions, animals preferentially respond for a non-satiated reinforcer as opposed to a satiated one because of a higher motivational state (Balleine & Dickinson, 1998). Goal-directed behavior is also responsive to a variety of contingency manipulations. Extinction generally causes responding to quickly cease (Bouton, Winterbauer, & Todd, 2012; Milad, Rauch, Pitman, & Quirk, 2006), as do contingency degradation and omission, a condition in which a previously reinforced response is now only reinforced when it is withheld (Dickinson, Squire, Varga, & Smith, 1998). Pavlovian-to-instrumental transfer (PIT), the capacity of a Pavlovian stimulus that predicts a reinforcer to elicit or increase instrumental responses for the same reinforcer, also affects goal-directed behavior (Crombag, Galarce, & Holland, 2008; see Holmes, Marchand, & Coutureau, 2010 for a comprehensive review of PIT).

Actions that are goal-directed and habitual alike are self-initiated, motivated behaviors that are made with some knowledge of a distinct outcome, e.g., an operant reinforcer (Dickinson, 1985). However, in the case of habitual actions, the present value of a reinforcer and its contingency with a response do not control the nature or strength of the action. Thus, behaviors that fully persist in spite of post-training instrumental contingency manipulations are considered habitual (Adams, 1982; Dickinson, 1985). The defining feature of initiation of habitual responding is exposure to a conditioned stimulus associated with the response through prior contiguity and a prolonged reinforcement history. Reinforcer devaluation, then, does not abolish performance during subsequent extinction testing (Adams, 1982), relative to a non-devalued control also subjected to extinction conditions. Furthermore, contingency degradation is ineffective at reducing the expression of behavior (Dickinson et al., 1998). However, habitual behavior may actually be more sensitive to PIT than goal-directed behavior. Holland (2004) demonstrated that as overtrained rats became less sensitive to reinforcer devaluation, they showed a corresponding increase in the influence of outcome-associated stimuli on response rates. This finding supports the notion that outcome representation is involved even in trained habitual behavior, and suggests that even when a reward is devalued, Pavlovian cues associated with that reward maintain their motivational properties.

Differing training procedures can profoundly affect the development of habitual behavior. Dickinson, Nicholas, and Adams (1983) tested rats for their susceptibility to reinforcer devaluation following equivalent amounts of training with ratio vs. interval schedules. Ratio schedules are defined by a close link between a particular number of responses and the reinforcer (e.g., a fixed ratio, or FR, 3 schedule provides a reward every 3 responses; variable ratio, or VR, 10 is a reward every 10 responses on average), while interval schedules are reinforced based on responses that occur