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An extinction cue reduces appetitive Pavlovian reinstatement in rats

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ABSTRACT

A Pavlovian appetitive conditioning preparation with rats was used to assess the effect of an extinction cue on reinstatement after extinction. Reinstatement provides an animal analog to relapses following treatment in humans; it occurs when a conditioned stimulus elicits strong conditioned responding following extinction and presentation of the unconditioned stimulus. An extinction cue is a stimulus presented during extinction of behavior controlled by the conditioned stimulus and is also presented later when the behavior would be expected to return/relapse following extinction (i.e., when reinstatement occurs). An extinction cue has been shown previously to reduce and prevent other instances of relapse analogs (spontaneous recovery and renewal). The authors tested whether an extinction cue would also reduce reinstatement, and included controls for reinstatement and for potential alternative accounts of an extinction cue's effect on reinstatement. The extinction cue reduced reinstatement, but a cue not presented during extinction did not affect reinstatement, bearing on several alternative explanations of the reduction effect. The authors suggest the extinction cue reduces reinstatement by helping to retrieve a memory encoded during extinction, and that reinstatement is due at least in part to a failure to retrieve that extinction memory.

1. Introduction

In Pavlovian conditioning preparations, reinstatement is the return of a conditioned response (CR) elicited by a conditioned stimulus (CS) that is observed during testing that follows extinction and subsequent presentation of the unconditioned stimulus (US; e.g., Bouton, 1984; Rescorla & Heth, 1975). The US presentations are responsible for the recovery of the CR during testing. Reinstatement is among several animal analogs of the relapse of unwanted behaviors in humans. Other analogs also represent an increase in the CR elicited by the CS following extinction, either when the physical/background or internal state context is changed (renewal; e.g., Bouton & Bolles, 1979; Brooks & Bouton, 1994), or when time passes following extinction (spontaneous recovery; e.g., Brooks & Bouton, 1993). Considerable animal learning and memory research has investigated the possible causes of reinstatement and other relapse analogs. Several theories have been proposed (e.g., Bouton, 1993; Holland, 1990; McLaren & Mackintosh, 2002;

Abbreviations: CR, conditioned response; CS, conditioned stimulus; A, used to represent the specific CS in each study; EC, extinction cue; X, used to represent the specific EC in each study; US, unconditioned stimulus; +, used to indicate the presence of the unconditioned stimulus following the CS, A, or X; −, used to indicate the absence of the unconditioned stimulus following the CS, A, or X; Rein, abbreviation for "reinstatement", used in group names; Cntrl, abbreviation for "control", used in group names; Ext, abbreviation for "extinction", used in group names; Neut, abbreviation for "neutral", used in group names; VT, variable time, referring to the schedule of reinforcement; ITI, inter-trial interval, referring to the duration between presentations of the stimuli

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Rescorla, 1979; Pearce & Hall, 1980; Wagner, 1981; Westbrook, Iordanova, McNally, Richardson, & Harris, 2002; and others). A full analysis of reinstatement theories is beyond the scope of this article. There are likely multiple mechanisms that contribute to reinstatement, and reliance on one mechanism alone is apt to yield an insufficient accounting for all instances of such relapse effects (for an excellent review, see McConnell & Miller, 2014).

We focus on the possible contribution of just a small set of those accounts that give weight to memory retrieval processes. Such accounts of reinstatement include acknowledgement that an inhibitory (CS-no US) association, or memory, forms during extinction. Some emphasize that by various mechanisms, the USs following extinction produce a direct and/or indirect increase in activation or retrieval of the CS-US conditioning association/memory (e.g., Bouton, 1984; Westbrook et al., 2002). Others incorporate memory retrieval processes involving extinguished responses, emphasizing the role forgetting of stimulus attributes of the context plays in reinstatement experiments (e.g., MacArdy & Riccio, 1995).

Other accounts can be taken to imply an alternative or additional mechanism whereby the USs that follow extinction produce a decrease in retrieval of the CS-no US extinction memory that contributes to reinstatement (e.g., Bouton, 1993; Ledgerwood, Richardson, & Cranney, 2004; Westbrook et al., 2002), though this component of those theories is not always directly stated. While many existing reinstatement experiments report results that can be taken to be consistent with the decreased retrieval of extinction mechanism, none reports direct evidence from a design focusing on the role of a stimulus different from the CS or US intended to serve the specific purpose of potentially activating an otherwise poorly-retrieved extinction memory. Such a manipulation could begin to investigate whether reinstating USs contribute to a failure to activate the CS extinction memory.

The present experiment assessed this possibility by testing for retrieval of the CS-no US extinction memory when a reinstatement effect is customarily observed. We used an *extinction cue* (EC) correlated with extinction of the CS, which bears some similarities to a contextual stimulus or extinction context (but cf. Brooks & Bowker, 2001), and is programmed for a brief duration (30s) every few minutes. A similarly- or identically-utilized cue has been previously employed in investigations of spontaneous recovery and renewal (e.g., Brooks, 2000; Brooks & Bouton, 1993, 1994; Brooks & Bowker, 2001). For example, Brooks and Bowker (2001) reported that spontaneous recovery can be reduced and even eliminated by an EC that was presented during testing and previously during sessions of extinction of the CR elicited by a CS. CR reduction by an EC has been shown in the case of spontaneous recovery using various Pavlovian preparations (e.g., Brooks, Palmatier, Johnson, & Garcia, 1999; Brooks, Vaughn, Freeman, & Woods, 2004). Brooks and Bouton (1994) showed a similar CR reduction by an EC in Pavlovian renewal. The similar ability of the EC to reduce spontaneous recovery and renewal prompted Brooks and Bouton (1994) to suggest that those effects may involve a similar mechanism: failure to activate the extinction association during testing. At least in part, reinstatement may be caused by a similar mechanism (e.g., Bouton, 1993). If so, presentation of an EC during reinstatement testing should reduce reinstatement. The current experiment employed such an EC and also included a control to constrain interpretations of any cue-reduction effect on reinstatement.

The design of the experiment is shown in Table 1. Four groups of food-restricted rats were given conditioning, extinction, and reinstatement testing with CS A. Conditioning involved multiple pairings of CS A with a food-pellet US. Extinction for Groups Rein, Cntrl, and Ext Cue involved a cued-extinction procedure in which the EC (X) was intermixed with nonreinforced A trials; the cued-extinction procedure is designed to encourage encoding of X as a feature of extinction so that X may later facilitate retrieval of the CS-no US extinction memory during reinstatement testing. Following extinction, Groups Rein and Ext Cue received unsignaled US presentations followed by testing for reinstatement with A. Group Ext Cue was tested with X presented just prior to A. Group Rein was tested the same way but with no cue. If X can reduce reinstatement of responding elicited by A, there should be less test responding in Group Ext Cue compared with Group Rein. Group Cntrl was treated similarly to Group Rein but did not receive reinstating USs after extinction; comparison of Group Rein to Cntrl demonstrates the basic reinstatement effect in Group Rein.

If the EC reduces the CR elicited by A in reinstatement testing, the reduction might be due to possibilities other than extinction memory retrieval and that instead rely on simply presenting X again just before A at test. For example, X might simply disrupt the rats' response to the subsequent CS during testing, or X might perceptually distract the rats from attending to the CS (e.g., akin to external inhibition; Pavlov, 1927). Or X at test might result in a failure to generalize responding to A (a stimulus generalization decrement effect) because of differences in how X and A were programmed in extinction and in testing (specified below). However, this possibility seems unlikely because results from prior experiments show the X – A interval is not an influence on test responding with experimental parameters such as those used here (Brooks, 2000). Nevertheless, these possibilities might account for reduced reinstatement by appeal to processes apart from those in which X activates memory retrieval processes.

In spontaneous recovery and renewal experiments, those accounts of reduced CS responding by ECs added at test have been investigated and no evidence for their role has been found (e.g., Brooks & Bouton, 1993, 1994). However, in a reinstatement

Table 1
Design of Experiment.

Group	Pre	Conditioning	Extinction	Rein	Test
Rein	—	A+	X → A−,A−	+	A−
Cntrl	—	A+	X → A−,A−	—	A−
Ext Cue	—	A+	X → A−,A−	+	X → A−
Neut Cue	X−	A+	A−,A−	+	X → A−

Note: A and X were the CS and EC, respectively, and were tone and darkness stimuli, counterbalanced; "+" = food USs; "−" = no US; " → " indicates the serial relation of X and A; "—" indicates exposure to the apparatus.