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Contextual control of human fear associations in a renewal paradigm

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Abstract

The original model of behavior change suggests that extinction is context dependent whereas fear acquisition is context independent [Bouton, M. E. & Ricker, S. T. (1994). Renewal of extinguished responding in a second context. *Animal Learning and Behavior, 22,* 317–324]. Supportive evidence stems mainly from animal studies, showing that after acquisition (conditioned stimulus–unconditioned stimulus (CS–US)) in Context A and extinction in Context B, fear is renewed by presenting the CS in acquisition Context A (ABA *renewal*) or in a novel Context C (ABC *renewal*). By implication, the model predicts equal ABA and ABC renewal. However, there is also evidence to suggest that the context dependency of extinction and the context independency of acquisition may be less stringent than originally proposed. The present study investigated renewal in humans using a differential fear conditioning paradigm with a shock US and online shock expectancy ratings and electrodermal responses as dependent variables. Experiment 1 compared an ABA condition with an AAA condition. Experiment 2 compared three conditions: ABA, ABC, and AAA. Both experiments demonstrated ABA renewal. Most importantly, Experiment 2 showed larger ABA than ABC renewal. Overall, results for expectancy ratings were more convincing than for electrodermal responses. In line with the extinction model, the present findings support the context dependency of extinction in humans. In contrast to the model, the findings suggest that in humans not only extinction learning, but also fear acquisition is controlled by its current context.

Keywords: ABA and ABC renewal; Human fear conditioning; Extinction; Skin conductance; Expectancy ratings; Exposure therapy

Introduction

Simple Pavlovian conditioning in animals (Pavlov, 1927) has provided major clinical applications for humans. Conditioning refers to the process of pairing an initially neutral conditioned stimulus (CS) (e.g., a tone) with an intrinsically aversive unconditioned stimulus (US) (e.g., an electric shock). The learned association between the CS and the occurrence of the aversive event generates a robust conditioned fear response to the CS on its own. Pavlovian fear conditioning has been considered as an important experimental model for the etiology of anxiety disorders for at least 80 years (Mowrer, 1939; Watson & Rayner, 1920). Of even greater clinical relevance is that extinction, that is, attenuation of Pavlovian conditioned fear by repeated

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presentations to the CS in absence of the US, has been the explicit model for behavior therapy (Eysenck, 1981; Marks, 1978).

Although CS alone presentations may extinguish conditioned fear responses, it is well established that an extinction procedure does not erase the original learned fear association (Bouton, 2000; Pearce & Hall, 1980; Rescorla, 2001). Retention of the original CS–US association has been uncovered following extinction by a variety of factors including a context change (*renewal*), presenting unsignaled USs (*reinstatement*), or simply the passage of time (*spontaneous recovery*) (Bouton & Bolles, 1979; Bouton & King, 1983; Pavlov, 1927). Instead of erasure, Bouton and colleagues assert that extinction involves new learning that competes with the original learning (see for reviews Bouton, 2000, 2002). The implication is that, once an aversive association has been learned, the memory trace for this association will be forever.

Assuming that the original association (CS–US) remains intact, extinction is thought to provide the CS with a second association (CS–noUS) that is available along with the first. A central tenet of the theory of Bouton is that these conflicting ''meanings'' supply the CS with ambiguity and that the context determines which meaning or association prevails in a certain situation (Bouton, 2002). This is represented in the original model of extinction learning (Bouton & Ricker, 1994). In this model it is hypothesized that merely elicitation of the CS–noUS association depends on the context whereas elicitation of the CS–US association is context *in*dependent. That is, in all contexts the original learned association is activated unless the extinction context turns it off. This hypothesis is derived from findings that a context change after simple acquisition has little impact on conditioned behavior (e.g., Bouton & King, 1983; Swartzentruber & Bouton, 1992), while the same context change after extinction often produces a loss of the new learned behavior (i.e., renewal) (e.g., Bouton & Bolles, 1979; Bouton & Peck, 1989). By implication, the model predicts that ''renewed responding should occur whenever an extinguished CS is tested in a context that differs from the extinction context'' (Bouton & Ricker, 1994, p. 317). Extrapolating to clinical practice, someone may acquire a spider phobia at home (Context A), which is treated by exposure in a therapeutical setting (Context B). Outside the treatment context, either at home (Context A) or in a new situation (Context C), the patient might suffer relapse when confronted with a spider (CS) due to a context change following exposure.

Evidence of the context dependency of extinction has been provided by studies on renewal in both animals and humans. When conditioned responding is acquired in Context A and extinguished in Context B, rodents demonstrate robust recovery of conditioned responding upon testing the CS in the original Context A, that is, ABA renewal (Bouton & King, 1983; Harris, Jones, Bailey, & Westbrook, 2000; Thomas, Larsen, & Ayres, 2003), or in a new Context C, that is, ABC renewal (Bouton & Bolles, 1979; Bouton & Brooks, 1993; Gunther, Denniston, & Miller, 1998). Renewal is also found when acquisition and extinction take place in the same context and testing occurs in a different context, that is, AAB renewal (Bouton & Ricker, 1994; Tamai & Nakajima, 2000). In humans, however, evidence of the renewal effect is relatively scarce. Most studies have been conducted in anxious individuals who were treated for their pre-existing fear by a short exposure treatment. Such studies show that fear returns when these individuals are tested in a context different from the treatment context (e.g., Mineka, Mystkowski, Hladek, & Rodriguez, 1999; Mystkowski, Craske, & Echiverri, 2002; Mystkowski, Mineka, Vernon, & Zinbarg, 2003). Only recently, renewal in humans has been investigated in which fear was experimentally induced (Milad, Orr, Pitman, & Rauch, 2005; Vansteenwegen et al., 2005). For instance, in a differential fear conditioning paradigm, Vansteenwegen et al. (2005) showed participants during acquisition two neutral slides of pictorial faces of which one (CS+) was paired with a loud noise (US), whereas the other (CS−) was never paired with the US. Extinction of conditioned fear was established by presenting both slides without the US in a different context. Conditioned fear responding to the CS+ renewed when returning to the acquisition context (i.e., ABA renewal). The abovementioned findings strengthen the idea that effects of exposure therapy may be lost when the previously feared object is encountered outside the treatment context.

Despite convincing evidence of the renewal effect, also several anomalies in the literature are available suggesting that the proposed model on extinction learning might be unnecessarily defeatist. First of all, the frequently reported absence of AAB renewal in animal laboratory studies is difficult to reconcile with the current model of extinction (Bouton & King, 1983; but see Bouton & Ricker, 1994; Bouton & Swartzentruber, 1989; Goddard, 1999). Furthermore, the observation by Denniston, Chang, and Miller (2003) that massive relative to moderate extinction training can attenuate the ABA renewal effect in rodents cannot be explained