Context, Ambiguity, and Unlearning: Sources of Relapse after Behavioral Extinction

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There is now ample evidence that extinction, the loss of learned performance that occurs when a Pavlovian signal or an instrumental action is repeatedly presented without its reinforcer, does not reflect a destruction of the original learning. This article summarizes the evidence and extends and updates earlier reviews. The main alternative to "unlearning" is the idea that extinction (as well as other retroactive interference processes, including counterconditioning) involves new learning that is stored along with the old. One consequence is that the Pavlovian signal or instrumental action has two available "meanings" and thus has the properties of an ambiguous word: its current meaning (and the resulting behavioral output) depends on what the current context retrieves. Contexts can be provided by a variety of background stimuli, including the physical environment, internal drug state, and time. The second thing learned (e.g., extinction, counterconditioning) seems especially dependent on the context for retrieval. A variety of evidence is consistent with this analysis, which highlights several important sources of relapse after extinction. The article concludes with several issues for future research, among them the question of how we can optimize extinction and other putative "unlearning" treatments so as to prevent the various forms of relapse discussed here. Biol Psychiatry 2002;52: 976–986 © *2002 Society of Biological Psychiatry*

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Introduction

For at least the last decade, a consensus has been building that extinction, the loss of performance that occurs when a Pavlovian signal or an instrumental action is no longer paired with a reinforcer, does not destroy the original learning. The idea was actually present in Pavlov's writing (Pavlov 1927) and in that of others who followed him (e.g., Konorski 1948; Pearce and Hall 1980; Wagner 1981). Nonetheless, the notion that extinction causes unlearning is so pervasive that it itself seems difficult to unlearn. Thus, it is second nature to think that exposure therapies are effective at reducing anxiety disorders because they destroy the learning that led to them. Similarly, it seems natural to accept certain assumptions in theories of learning and memory that imply new learning will destroy the old (e.g., McClelland and Rumelhart 1985; Rescorla and Wagner 1972; see McCloskey and Cohen 1989).

Evidence that extinction is not unlearning has been reviewed in other places (e.g., Bouton 1988, 2000; Bouton and Swartzentruber 1991; Falls 1998; Rescorla 2001). In this article, I briefly summarize and update that evidence in a way that illustrates what I think is the most reasonable alternative to the destruction hypothesis. Extinction does not destroy the first-learned information but instead reflects new learning. The result is that the signal (or instrumental action) acquires a second "meaning" that is available along with the first. In this sense, the current meaning of the signal or action is ambiguous (Bouton 1984; Bouton and Bolles 1985). As with other ambiguous stimuli, such as ambiguous words, its current meaning is determined by the current context. Just as the word "Fire!" evokes different reactions in a movie theater and a shooting gallery, an extinguished Pavlovian signal will evoke different reactions in different contexts. The view rejects unlearning, accepts behavioral instability as a potential consequence of extinction, and highlights several possible sources of relapse.

Four Mechanisms of Relapse

Research on extinction has uncovered at least four phenomena indicating that it does not destroy the original learning. These are summarized in this section and in Table 1. All are potential mechanisms of relapse, and all indicate the importance of context in controlling performance after extinction has occurred. Most of the research has focused on extinction in Pavlovian conditioning, in which a conditional stimulus (CS, e.g., a tone) is first paired with a motivationally significant unconditional stimulus (US, e.g., foot shock), and the resulting reaction to the tone (e.g., fear) is eliminated ("extinguished") by

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Table 1. Four Context and Extinction Phenomena That May Provide Mechanisms of Relapse

Phenomenon	Description
Reinstatement	Recovery of behavior that occurs when the subject is exposed to the US after extinction. Strongly controlled by contextual conditioning produced when the US is presented, hence the phenomenon is strongest when the CS is tested in the context in which the US has occurred.
Renewal	Recovery of extinguished behavior that can occur when the context is changed after extinction. Most often observed when the subject is returned to the original context of conditioning, but it also depends in part on mere removal from the extinction context.
Spontaneous recovery	Recovery of responding that occurs when the CS is tested after time has passed following the conclusion of extinction.
Reaquisition	Recovery of responding that occurs when the CS is paired with the US (or reinforcer) again after extinction. Often rapid, especially when cues in the background renew conditioned performance (as above). Can be slow when the background cues continue to retrieve extinction.

Adapted and reproduced with permission from Bouton and Swartzentruber (1991).

CS, conditional stimulus; US, unconditional stimulus.

presenting the CS repeatedly alone. Similar principles apply to extinction in operant learning, however, in which an action (e.g., lever pressing) is first reinforced by a positive event (e.g., delivery of a food pellet) and then extinguished by removing the event (e.g., see Bouton and Swartzentruber 1991).

Reinstatement

If the significant event (US) is presented on its own after extinction, it can cause "reinstatement" of extinguished responding to the tone (CS; e.g., Delamater 1997; Pavlov 1927; Rescorla and Heth 1975). In a typical reinstatement experiment in Pavlovian fear conditioning, conditioning is first conducted by pairing a tone and a shock and then extinction is created by multiple exposures to the tone alone. Then, often in a separate session, the shock is presented on its own a few times. When the tone is tested again, often 24 hours later, it evokes fear again. Conditioned fear is not destroyed by extinction but can return again after mere exposure to the unconditional stimulus.

Research in my laboratory suggests that reinstatement depends on learning about the context, the constellation of cues that are in the background whenever learning occurs. In experiments with rats, the context is typically the apparatus or chamber in which the crucial events are presented. During reexposure to the shock US, the subject associates the shock with the current context, and the presence of this conditioning (or new expectation of the US) is what triggers fear of the CS when it is presented again. For example, we observe no reinstatement unless the shock is presented in the context in which the extinguished tone CS is tested (e.g., Bouton 1984; Bouton and Bolles 1979b; Bouton and King 1983, 1986; Bouton and Peck 1989; Frohardt et al 2000). The strength of reinstatement can also be predicted from the strength of contextual conditioning assessed independently (Bouton 1984; Bouton and King 1983). The phenomenon illustrates that performance after extinction depends on the organism's "knowledge" about the current context. Interestingly, the same background fear is far less likely to increase fear of a signal that has not been extinguished (Bouton 1984; Bouton and King 1986). Extinguished CSs are especially sensitive to this effect of context, perhaps because their meaning is ambiguous. Reinstatement also occurs when the reinforcer is presented after extinction of operant conditioning (e.g., de Wit and Stewart 1981; Reid 1958); a role for context conditioning has also been demonstrated there (Baker et al 1991).

Recent research suggests a second context-learning mechanism might also influence reinstatement (Westbrook et al 2002). During extinction, the organism can associate the CS (tone) with the context in which it is being presented. When footshock is then presented in the same context, the new context–shock association can influence responding to the tone through a combination of the context–shock and context–tone associations. In effect, if the context has become bad, anything associated with it is also treated as bad. Our previous work had emphasized that the testing must occur in the recently shocked context for reinstatement to occur. The newer mechanism can allow fear of an extinguished tone to recover in any context, provided that the context in which it was extinguished is newly associated with a traumatic event. Although more research is necessary on this latter mechanism, there are several reasons why an extinguished fear can be expected to relapse or return to behavior when a context is made dangerous by association with a stressor or traumatic event.

Renewal

Other research suggests that the reduction in fear that occurs in extinction itself depends on learning about the context. In the "renewal effect," the organism might receive conditioning in Context A and then extinction in a second context (Context B). Then, after responding has

decreased to zero, the CS might be returned to the original context (Context A) and tested again. The return to the original context typically "renews" fear of the CS (e.g., Bouton and Bolles 1979a; Bouton and King 1983; Bouton and Peck 1989; Harris et al 2000; Rauhut et al 2001). Although "ABA renewal" (in which conditioning, extinction, and testing occur in Contexts A, B, and A, respectively) is the most widely studied example of renewal, "ABC renewal" is also possible: if conditioning occurs in Context A, and extinction occurs in Context B, tests of the CS is a third context (Context C) can also renew fear of the CS (e.g., Bouton and Bolles 1979a; Bouton and Brooks 1993; Gunther et al 1998; Harris et al 2000). Although ABC renewal is not as strong as ABA renewal (e.g., Harris et al 2000), it is important because it indicates that mere removal of the CS from the extinction context can cause a recovery of fear. Thus, the loss of fear itself depends at least in part on learning that the CS is safe in the current context. "AAB renewal," in which testing occurs in a second context after conditioning and extinction have occurred in the first, has also been observed (Bouton and Ricker 1994; Tamai and Nakajima 2000), although it is more difficult to detect than either ABA or ABC renewal (e.g., Bouton and King 1983; see also Crombag and Shaham 2002; Nakajima et al 2000; Tamai and Nakajima 2000).

The renewal effect seems general and robust. It can occur after extensive extinction training. Bouton and Swartzentruber (1989) observed ABA renewal when 84 extinction trials followed eight conditioning trials. Conditioned fear was absent from behavior after approximately 20 of those extinction trials. Recent research indicates that ABA renewal (Rauhut et al 2001) and ABC renewal (Gunther et al 1998) can occur after more than 160 extinction trials, and 100 extinction trials produce no less ABA renewal than 20 trials (Rauhut et al 2001; see also Tamai and Nakajima 2000). Extensive extinction training does not destroy the original conditioning or the fundamental context-dependence of extinction.

Renewal has also been shown in a large number of methods. The phenomenon has been demonstrated in fear conditioning, in which the CS is associated with shock (as described earlier); in appetitive conditioning, in which the CS is associated with food (Bouton and Peck 1989; Brooks and Bouton 1994); and in flavor aversion learning, in which a taste CS is associated with illness (e.g., Chelonis et al 1999; Rosas and Bouton 1998). It has also been demonstrated in a causal judgment task in humans (Rosas et al 2001). In none of these cases does extinction destroy the original learning, which remains available in memory, ready to return to behavior depending on the context.

Like all of the context and relapse effects summarized here, renewal also occurs in operant conditioning (e.g., Nakajima et al 2000; for review see Bouton and Swartzentruber 1991). Crombag and Shaham (2002) recently reported an especially compelling demonstration. They first reinforced lever pressing in rats with delivery of an intravenous mixture of heroin and cocaine. After 10 sessions of training, they gave the rats 20 sessions of extinction, in which lever pressing no longer produced the drug. Some of the rats received extinction in the original training context, and others received it in a different context. As we typically see in Pavlovian conditioning, the original training transferred perfectly to the new context; there was no difference in the rate of lever pressing, or the rate of extinction, in animals extinguished in the two contexts (see also Nakajima et al 2000, Experiment 1). At the end of extinction, the rats were returned to the original context, in which responding once again occurred without reward. The rats that had been extinguished in the different context showed a powerful renewal; responding recovered to a rate that was roughly equivalent to the level at the beginning of extinction. In both operant and Pavlovian learning, the original learning remains after extinction, ready to be retrieved by the right manipulation of context.

Spontaneous Recovery

The most famous recovery effect known to occur after extinction is one that Pavlov (1927) discovered. He noted that if time elapses after extinction, the extinguished response can recover ("spontaneously") when the CS is tested again. This effect has been demonstrated in virtually every conditioning method (for a new demonstration, see Brooks et al 2001). In my laboratory, responding on the first test trial, although usually not the second, can be as strong as that observed at the end of conditioning (e.g., Brooks and Bouton 1993). Extinction does not have a permanent effect.

Although there are several possible explanations of spontaneous recovery (e.g., Devenport et al 1997; Robbins 1990), it can be seen as a natural implication of the ambiguity framework (e.g., Bouton 1988). According to memory theorists (e.g., Estes 1955; Spear 1978), the passage of time may naturally provide a gradually changing context. Given this possibility, just as extinction is specific to its physical context, it might be specific to the context of time. Viewed this way, spontaneous recovery is the renewal effect that happens when the CS or action is tested in a new temporal context (e.g., Bouton 1988, 1993).

This idea suggests that both renewal and spontaneous recovery come about because the organism fails to retrieve extinction outside the extinction context. If true, then

either effect should be attenuated if a retrieval cue that reminds the subject of extinction is presented just before the recovery test. Such an effect has now been demonstrated repeatedly by Cody Brooks (Brooks 2000; Brooks and Bouton 1993; Brooks and Bowker 2001; Brooks et al 1999). He has shown that if a brief cue that has been presented at various times within an extinction session is presented again just before the spontaneous recovery test, it reduces spontaneous recovery. Thus, recovery seems to result from a failure to retrieve extinction after a delay. Importantly, the retrieval cue also attenuates the renewal effect. That is, when conditioning, extinction, and testing occur in physical contexts A, B, and A, then presenting a cue from extinction during the final test reduces the renewal that is otherwise observed (Brooks and Bouton 1994). The results suggest that spontaneous recovery and renewal are caused by similar failures to retrieve extinction outside the extinction context. They also suggest that either form of relapse can be reduced with cues that remind the organism of extinction.

Reacquisition

Of course, a straightforward way to cause a return of extinguished responding is to pair again the extinguished signal or action with the reinforcer. The reacquisition of responding that results can be rapid, as if the original association has been "saved." For instance, Napier et al (1992) found that reconditioning of an eye-blink response in rabbits was significantly faster after conditioning and extinction than when the CS was novel. Certain details of Napier et al's experiments allowed them to rule out the possibility that rapid reacquisition was merely due to spontaneous recovery or reinstatement (see Bouton 1986). Instead, new pairings of an extinguished CS with the US can cause a rapid return of responding that appears to be a mechanism of relapse in its own right (e.g., Kehoe and Macrae 1997).

The idea that reacquisition is rapid after extinction is consistent with all the other evidence that extinction is not unlearning. However, reacquisition is not always fast. In fact, it can be significantly slower than the original learning in fear conditioning (Bouton 1986; Bouton and Swartzentruber 1989) and taste aversion learning (Calton et al 1996; Hart et al 1995), especially after a large number of extinction trials. It is also slow in appetitive conditioning when many extinction trials follow relatively few initial conditioning trials (Ricker and Bouton 1996). Interestingly, reacquisition can be slow even in the presence of evidence confirming that the original learning has not been destroyed (Bouton and Swartzentruber 1989).

How can fast and slow reacquisition be reconciled? The key may be ambiguity: Extinction leaves the CS with two meanings, either of which can be translated into behavior depending on the prevailing context. Slow reacquisition is caused by the continued retrieval of extinction. When cues that retrieve extinction are removed, for example, by switching the context, reacquisition is not as slow (Bouton and Swartzentruber 1989). If some other cue is present during reconditioning that promotes retrieval of acquisition, reconditioning is rapid. Rapid reacquisition may be another renewal effect, wherein acquisition is retrieved by a cue generated by reconditioning.

A good candidate for that cue is recent conditioning trials (Ricker and Bouton 1996). Consider a closer analysis of conditioning and extinction. During conditioning, the subject might learn that conditioning trials (CS–US pairings) are connected with other conditioning trials (Capaldi 1994). Recent CS–US pairings are thus a part of the context of acquisition. By contrast, when extinction is conducted over many trials, the subject can also learn that extinction trials are connected with other extinction trials. When CS–US pairings are then resumed, the first few may introduce a feature of the conditioning context and thus cause a renewal of responding. Consistent with this analysis, when reacquisition is conducted with a mixture of CS–US and CS-alone presentations, high responding is observed on trials that follow a CS–US pairing, but relatively low (extinctionlike) responding is observed on trials that follow a CS alone (Ricker and Bouton 1996). Rapid reacquisition also requires a large number of acquisition trials, which presumably permit learning that CS–US trials are part of the context of acquisition (Ricker and Bouton 1996). The amount of responding one observes thus depends on a subtle interaction between various types of contextual cues.

What does all this learning theory have to do with relapse of anxiety disorders? Imagine a child who is repeatedly teased by groups of children because of his body weight. The teasing continues to the point that he feels self-conscious and anxious with any group of kids on the playground, in the locker room, and so forth. After therapy, and perhaps a move to a different school, the boy becomes more comfortable with himself and with social situations. But then a minor tease from a new friend might signal another repeated round of teasing, stimulating the full-blown fear and social anxiety again. The mechanism might contribute to any disorder or habit that results from repeated bouts or binges of conditioning trials. Smokers may smoke many cigarettes in rapid succession; consequently, smoking a cigarette may become a contextual cue for smoking more cigarettes. An initial lapse (perhaps brought on by renewal or spontaneous recovery, say) can thus spiral into relapse (Bouton 2000).

Research in my laboratory has begun to confirm an interesting implication of this analysis. If recent condition-

ing trials are a cue that renews responding, then we should be able to reduce their impact in a special extinction procedure that includes an occasional CS–US pairing among a string of extinction (CS-only) trials. Such a procedure would now connect CS–US pairings with *extinction*, rather than conditioning. In appetitive conditioning experiments, we found that a "lean" partial reinforcement schedule (e.g., a schedule in which one eighth of the "extinction" trials were paired with food) weakened behavior relative to conditioning, although it was not as effective as normal extinction in this respect. More important, the procedure slowed down rapid reacquisition and abolished the tendency for CS–US pairings to cue conditioning performance on the next trial. In the long run, although it does not eliminate behavior as completely, occasional exposures to conditioning trials in extinction may create a change in behavior that is less vulnerable to this particular mechanism of relapse.

Converging Evidence against Unlearning

Other Forms of Retroactive Interference

The idea that extinction does not destroy the original learning is compatible with a great deal of other research. For example, extinction is just one example of several retroactive interference paradigms in which new learning is introduced to replace old learning (Bouton 1993). Interestingly, the ideas just described may generalize to all examples of interference (Bouton 1993). For example, in "counterconditioning," a rat might first receive tone–shock pairings and then pairings of the tone with a different US, such as food. In this case, the original fear response decreases and is replaced by a second (food) response. Experiments on counterconditioning provided the original rationale for systematic desensitization (Wolpe 1958), in which fear cues are not just extinguished, but deliberately associated with a new response (relaxation) to eliminate anxiety and fear.

Although it is once again tempting to assume a role for unlearning, counterconditioning is another place where we find ambiguity: In the end, the signal might have two available meanings (tone–shock vs. tone–food) that can pivot on what the current context retrieves. Rats that have received tone-shock pairings followed by tone–food pairings show reinstatement of fear if they are exposed to the shock again (Brooks et al 1995). As in extinction, reinstatement here is a context conditioning effect that depends on receiving the reinstating shocks in the context in which testing is to occur. Other experiments have demonstrated the renewal effect in counterconditioning (Peck and Bouton 1990). When tone-shock pairings occurred in Context A and then tone-food pairings occurred in Context B, a return to A caused a recovery of fear (and a disappearance of food performance). (When tone–food pairings preceded tone–shock, a return to the original context correspondingly renewed food performance.) Still other experiments have demonstrated spontaneous recovery (Bouton and Peck 1992). Here, conditioned fear eliminated by tone–food pairings recovered when the signal was tested 28 days later. (When tone–food preceded tone–shock, the original food response likewise returned.) Just like extinction, counterconditioning may leave the CS susceptible to the effects of context and time. As reviewed by Bouton (1993), retroactive interference designs do not create unlearning; instead, the new meaning creates a state of ambiguity, which the current context resolves. Rescorla (e.g., Rescorla 2001) recently argued that retroactive interference paradigms might even leave the original meaning perfectly preserved (e.g., Rescorla 1993, 1996). The relapse effects for extinction summarized earlier are probably relevant to any therapy in which new learning is used to eliminate psychopathology based on the old.

Neurobiology

Perhaps not surprisingly, a growing literature on the neurobiology of extinction further supports the idea that extinction depends on new learning rather than unlearning (see Davis, this issue). For example, activation of N-methyl-D-aspartate (NMDA) glutamate receptors in the amygdala appears necessary for both fear conditioning (e.g., Campeau et al 1992; Miserendino et al 1990) and fear extinction (Falls et al 1992; Lee and Kim 1998; see also Santini et al 2001). Such receptors are involved in long-term potentiation, a form of brain plasticity that is presumed to underlie many forms of learning. Finding a role for them in both acquisition and extinction, particularly at the same brain site, suggests that the two processes might have a similar substrate (see also Lu et al 2001). Interestingly, facilitation of NMDA receptor function might also facilitate extinction (Walker et al 2002). Although the molecular mechanisms behind acquisition and extinction are still uncertain (e.g., Lattal and Abel 2001), the evidence suggests that extinction involves new brain plasticity, rather than a destruction of the original.

Clinical Evidence

The idea that therapy might create a context-dependent form of new learning is also consistent with clinical research. For instance, in anxiety disorders, Rachman (1979, 1989) documented a "return of fear" after exposure therapy that seems reminiscent of spontaneous recovery. In addition, human fears that have been reduced by exposure in one context may be renewed when they are tested in a different context (Mineka et al 1999; Mystkowski et al 2002; see also Rodriguez et al 1999). For

example, Mystkowski et al (2002) gave undergraduates who reported spider fear a single session of graded exposure to a tarantula in a context provided by either a room or a patio outdoors. Seven days later, the participants were tested for their reactions to the spider in the same context and the different context in a counterbalanced order. Subjective ratings of distress were higher in the new context, suggesting a renewal of spider fear. The strength of renewed anxiety demonstrated in humans thus far does not seem as large as that suggested by animal research. However, as various authors have noted, the human designs invoke ABC renewal, which in animals is typically weaker than ABA renewal. The human experiments usually involve the experimenter meeting participants in a common room before moving them to the treatment rooms, perhaps supporting generalization between them. Despite the inherent difficulties of this kind of research, Mystkowski et al's results suggest that about 30% of the distress that had been reduced during treatment was recovered when the spider was tested in the different context. (Only about 7.5% returned in the same context.) Thus, therapy for an anxiety disorder might indeed cause new learning that is at least partly specific to the context in which it is learned.

A renewal effect has also been reported in humans given extinction exposure to alcohol cues. Collins and Brandon (2002) gave social drinkers 7–10 exposures to the sight and odor of beer in a distinctive room. The exposures reduced both salivation and the self-reported urge to drink that otherwise occurred in the presence of these cues. However, when participants were then tested in a different room, a substantial recovery of both responses was observed; 69%–92% of what had been reduced returned. Equally important, the presence of a cue during testing that had also been present during extinction (a salient and distinctive pencil and clipboard on which the urge ratings were completed) reduced these renewal effects (see Brooks and Bouton 1994). Thus, in both animals and humans, the effects of extinction may be reduced with a change of context but can be at least partly recovered by an extinction reminder cue.

Current Issues

What Kinds of Cues Are Contexts?

When one translates animal research into clinical practice, it is natural to ask about the boundaries of the concept or definition of context. As I suggested earlier, it is useful to think that a variety of different background cues might play the role of context in extinction. In fact, research on context effects in both animal and human learning and memory suggests that a wide variety of stimuli might potentially function this way (Table 2). Drugs, for example, can provide salient internal contexts that may be especially relevant to the treatment of anxiety. When rats are given fear extinction while under the influence of a benzodiazepine tranquilizer, fear is renewed when the animal is tested outside the drug state (Bouton et al 1990). The implications of such "state-dependent" fear extinction (see also Cunningham 1979) seem apparent; the use of drugs in combination with therapy may theoretically backfire when the individual is taken off the drug. Another implication connects with the known links between anxiety disorders and substance abuse (e.g., Kushner et al 2000). If a person takes a drug to reduce anxiety, state-dependent fear extinction might preserve the original anxiety that might otherwise extinguish with natural exposure trials. Thus, anxiety could lead to drug use, and drug use could further preserve the anxiety. A self-perpetuating cycle could ensue.

Table 2. Some Examples of Contextual Stimuli Studied in Animal and Human Laboratories

Exteroceptive context:
Room, place, environment, other external background stimuli (e.g., Bouton 1993; Smith 1988; Spear 1978)
Interoceptive context:
Drug state (e.g., Bouton et al 1990; Cunningham 1979; Overton 1985)
Hormonal state (e.g., Ahlers and Richardson 1985)
Mood state (e.g., Bower 1981; Eich 1995)
Deprivation state (e.g., Davidson 1993)
Recent events (e.g., Bouton et al 1993; Ricker and Bouton 1996)
Expectation of events (e.g., Bouton et al 1993)
Passage of time (e.g., Bouton 1993; Rosas and Bouton 1998)

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One can imagine all of the contexts sketched in Table 2 fluctuating in the normal course of life. Sometimes they might change alone, but sometimes they might also change in combination with other cues. Recent work that has simultaneously manipulated both time and physical context suggests that the two types of cues can have additive effects (e.g., Rosas and Bouton 1998; Rosas et al 2001; Westbrook et al 2000). That is, combining a physical context change and a retention interval functionally can create an even bigger context change. However, it has been useful to recognize that physical contexts are themselves embedded in the context provided by the passage of time (Bouton et al 1999). Although there is good reason to think that many stimuli can play the role of context, we need more basic information about how they might combine and interact.

When Are Contexts Actually Important?

One of the most interesting discoveries in research on context effects in animal learning is that different kinds of learning are not equally affected by context. For example,

although extinction is relatively context-specific, conditioning is usually less so. Regardless of the conditioning preparation, we often find surprisingly little effect on behavior of switching the context after conditioning (e.g., Bouton and King 1983; Bouton and Peck 1989; Harris et al 2000; Westbrook et al 2000). Conditioning is also more stable than extinction over time (Bouton 1993). The inherent asymmetry in the context-dependence of conditioning and extinction may be a reason that behavior disorders may seem so persistent.

One possibility is that the context specificity of extinction, an inhibitory process, might reflect the more general principle that inhibition is context-specific; however, research suggests this is not the case. Conditioned inhibition, like simple conditioning, generalizes nicely across contexts (Bouton and Nelson 1994; Nelson 2002; Nelson and Bouton 1997). Instead, extinction appears to be context-specific because it is the second thing learned about the CS (Nelson 2002; Swartzentruber and Bouton 1992; see also Harris et al 2000; Westbrook et al 2000). It is as if the learning and memory system encodes the second thing learned about a stimulus as a conditional, context-specific exception to the rule.

The asymmetry in the context-specificity of conditioning and extinction is evident in the ABC renewal design. That is, the fact that organisms renew their extinguished responding in Context C suggests that conditioning must generalize more from A than extinction does from B. (Spontaneous recovery likewise suggests that conditioning generalizes better over temporal contexts than does extinction.) However, Harris et al (2000) have emphasized the fact that ABC renewal is weaker than ABA (see also Bouton and Brooks 1993). Such a finding begins to suggest that once extinction has occurred, conditioning is stronger in the context in which it was learned. Harris et al showed that conditioning, which was context-free before extinction, became more context-dependent after extinction. Perhaps the extinguished signal's new ambiguity renders both its first and second meanings context-dependent. Nonetheless, it is still clear that conditioning generalizes better across contexts than extinction. Bouton (1994) discussed several functional and mechanistic reasons this pattern might be true.

How Can We Prevent Relapse?

If the original learning is not destroyed by a retroactive interference treatment, and if relapse is therefore always possible, we need to know how to optimize the new learning so as to prevent the phenomena of relapse. At this point in time, we know more about how to produce the relapse effects reviewed here than we know how to prevent them. Nonetheless, it is possible to make some initial claims and point to some directions for future research.

If extinction does not depend on unlearning but instead on context-dependent new learning, then extinction (and other therapeutic retroactive interference treatments) would clearly be most beneficial if it were conducted in the contexts in which the disorder is most problematic to the client. "Booster" trials would similarly be effective after time has elapsed (and the temporal context has changed). Furthermore, as reviewed earlier, retrieval cues are also helpful in renewal and spontaneous recovery effects. Therapists might therefore build them into treatment and the posttherapy period, for example, by using reminder cards or reminder phone calls from the therapist (e.g., Hiss et al 1994). Research on human memory also suggests that damaging effects of context change can be ameliorated if the subject is instructed to remember the learning context (Smith 1979). Although retrieval cues and retrieval strategies will be helpful, it would be nice to have more information about them. For example, it is not yet known how successful retrieval cues will be at attenuating reinstatement (but see Rescorla and Cunningham 1972) or rapid reacquisition.

From the ambiguity perspective, the key to successful treatment may be designing therapy so that the new learning it inevitably involves will generalize effectively to new contexts. Contexts are collections of many cues, some of which are unique but many of which are presumably shared with other contexts. From this perspective, a therapeutic goal might be to connect therapy with as many shared cues as possible. Conducting extinction in several different contexts is one way to increase the number of shared cues. There is evidence that extinction in multiple physical contexts reduces renewal in rats (Chelonis et al 1999; Gunther et al 1998). For example, Gunther et al found that extinction in Contexts B, C, and D (after fear conditioning in Context A) reduced, but probably did not eliminate, renewal that was observed when the CS was tested in Context E. However, in a perceptive second experiment, they also showed that extinction in three contexts was not as effective at reducing renewal when conditioning had also been conducted in three (other) contexts. That result reminds us that even extinction in multiple contexts does not erase the original learning. Instead, preventing renewal will depend on a kind of balance between the generalization of both extinction and conditioning. Given the high probability that anxiety disorders (and other forms of psychopathology) develop from experiences in many contexts, the challenge for treatment seems clear. Even extinction delivered in multiple contexts might not be a magic bullet.

A related hypothesis is that extinction in multiple temporal contexts (i.e., when trials or sessions are distrib-

uted widely over time) might have similarly beneficial effects. I am not aware of any research on conditioning in animals that addresses the effects of spaced extinctions on relapse. Nonetheless, human memory research suggests that material learned over spaced trials is better retained over longer intervals (e.g., Glenberg 1976). Based on this sort of finding, Craske and her collaborators tested different ways of scheduling exposure therapy sessions over time (e.g., Rowe and Craske 1998; Tsao and Craske 2000). Their results suggest that exposure sessions distributed in time are more effective than massed exposures at preventing a return of fear at a 1-month follow-up. We need more basic information about the effects of this kind of variable. For example, do spaced extinction exposures eliminate spontaneous recovery, or do they merely extend the interval required to observe it? Do extinction trials in multiple temporal contexts prevent renewal that occurs with a change of physical context, and vice versa? The idea that learning in multiple contexts merely increases the number of shared contextual cues does not necessarily predict that kind of benefit (what do temporal and physical contexts have in common?). But the fact that context switches during extinction will inevitably produce retrieval failures or response recoveries during extinction might itself increase the durability of extinction (see Lang et al 1999; Rescorla 2001). These ideas need to be tested in further research.

Recent experiments with rats suggest still another possibility. Rauhut et al (2001) tested the impact of several fear extinction treatments on the ABA renewal effect. Extinction procedures that additionally involved exposures to the shock US (e.g., unpaired presentations of the CS and US) were best at eliminating renewal and also interfering with reacquisition in the original context. Remarkably, a group that only received US exposures in Context B did just as well as a group that had received CS presentations there. Although both groups showed fear during the renewal test in Context A, they were both better off than a group that had received no treatment during Phase 2. Results were interpreted as suggesting that treatments that allow habituation of the US might be especially effective at preventing relapse effects. Although postconditioning habituation of a US is known to decrease fear of a CS that has been associated with it (Rescorla 1973), the ultimate implications, and how they should be translated into practice, are not clear at the present time.

A final implication is also worth mentioning. As I have noted elsewhere (e.g., Bouton 2000; Bouton and Nelson 1998), the possibility that the first learning is not destroyed suggests that, in the interest of reducing psychopathology, we should encourage greater efforts in prevention. As described earlier, the first-learned things seem relatively stable and immune to the effects of changing the physical and temporal context. We should thus do our best to ensure that the first things learned are positive things. The value of prevention receives support from laboratory research on basic learning and unlearning processes.

Conclusion

A large body of research suggests that extinction and retroactive interference do not cause unlearning. It may be tempting to conclude that therapeutic treatments are therefore doomed, but this is not the case. Although it is probably best to assume that the original information is always available, recognition of that fact will promote better, more realistic therapies. The ambiguity framework suggests that the trick will be to develop methods that promote ubiquitous retrieval of the second meaning. Although we have reached a reasonably good understanding of the various context effects that can help generate relapse, further basic research is needed to understand how they can be reduced and controlled.

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