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CHAPTER 11

EXTINCTION: A REVIEW OF THEORY AND THE EVIDENCE SUGGESTING THAT MEMORIES ARE NOT ERASED WITH NONREINFORCEMENT

William A. Falls

Pavlov (1927) observed that if a well-trained conditioned stimulus (CS) was presented several times in the absence of the unconditioned stimulus (US) with which it was originally paired, the CS would lose its ability to elicit the conditioned response (CR). He used the term *extinction* to describe the loss of the CR that occurred as a consequence of nonreinforcement. Presently, the term *extinction* (and its derivatives) is used interchangeably to describe both a procedure, "testing occurred in extinction," and the consequence of the procedure, "nonreinforcement of the CS led to extinction of the CR" (Mackintosh, 1974). Sometimes the term *extinction* is also used to describe the *mechanism* or *process* that is responsible for the loss of the CR, as in "the loss of the CR was due to extinction." This last use of the term can be misleading because it implies that the process that underlies the loss of the CR is understood when it is not.

The basic characteristics of extinction are that (1) extinction occurs only with the omission of reinforcement and does not occur if CS-US training continues and (2) extinction is an *active learning process* requiring the presentation of the CS. Therefore it differs from "forgetting," which refers to a loss of responding that results merely from the passage of time. Thus, through some learning process, presentation of the CS without the US causes a reduction in the ability of the CS to elicit a CR. What might this process be? Although there are many theories of extinction, the theories can be forced into two general classes (Mackintosh, 1974). Theories in the first class argue that the CR-producing associations (one may wish to read "memory") are *erased* as a consequence of nonreinforcement. Theories in the second class argue that the CR-producing associations are intact but are influenced (reduced or inhibited) by competing associations

that are acquired as a consequence of nonreinforcement. Experiments have been unable to definitively support one or the other of these alternatives. However, what experiments have shown is that nonreinforcement may not erase all of the CR-producing associations. For example, the phenomena of spontaneous recovery, rapid reconditioning, and reinstatement all show that under certain experimental conditions the CR can return to preextinction levels. This suggests that some portion of CR-producing associations survives extinction.

This chapter begins with an overview of these extinction phenomena and the theories of extinction that have been proposed in their wake. It next discusses the paradox of avoidance behavior relevant to extinction followed by a brief overview of the salient issues in extinction of instrumental responses. It then reviews clinical implications of data related to extinction and avoidance behavior. Finally, it discusses why some traumatic memories may not be easily extinguished and how the extinction procedure may be augmented to facilitate extinction in these cases.

PHENOMENA RELATED TO THE RECOVERY OF THE CR AFTER EXTINCTION

Spontaneous Recovery

Pavlov (1927) observed that an extinguished CR would recover to a preextinction baseline with relatively minor disturbances in the experimental protocol. If left undisturbed for some period of time, an extinguished salivary CR would spontaneously recover. For example, in one experiment, three nonreinforced presentations of a well-trained CS at a 10-minute intertrial interval resulted in complete extinction of a salivation CR (from 8 drops of saliva to 0). After an interval of 20 minutes, CS was again presented, and a recovery of the CR was observed (7 drops). Spontaneous recovery is often observed in the first few CS-alone presentations that follow after a session of nonreinforced CS presentations (Wagner, Siegel, Thomas, & Ellison, 1964).

Pavlov's Theory of Internal Inhibition

For Pavlov, spontaneous recovery indicated that extinction could not be regarded as an "irreversible destruction of the nervous associations" that first allowed the bell to elicit the salivary CR (Pavlov, 1927, p. 60). Instead, Pavlov believed that under conditions of nonreinforcement a well-trained CS became inhibited. He further assumed that the inhibition was fragile and that with the passage of time the inhibition would decay causing a restoration of the CR. Consistent with a time-dependent decay of inhibition was the observation that the restoration of the CR is more complete the greater the interval between extinction and testing.

Pavlov (1927) conceived of learning as taking place in the cortex of the animal. As a consequence of nonreinforcement, the CS produced an "inhibitory effect" in the cerebral hemispheres. Internal inhibition, as it was called, was due to a spreading wave of inhibition initiated at the cortical center that corresponded to the cerebral location of the CS. Once inhibition was initiated at the cortical center, it would irradiate over the entire cerebral cortex in a spreading wave of inhibition that would compete with the CR-eliciting properties of the CS.

In his theorizing, Pavlov (1927) attempted to describe how the nervous system functioned to produce behavior. His concept of *irradiation* of inhibition was borne out of his understanding of nervous system function. However, within a short time, Pavlov's understanding of the nervous system was no longer accurate. Konorski (1948) recognized that Pavlov's concept of irradiation of inhibition could not be reconciled with the current understanding of the nervous system. But rather than aband Pavlov's concepts, Konorski (1948) reformulated Pavlov's (1927) notions into terms that were more consistent with modern principles of neurophysiology. In doing so, Konorski provided a more detailed explanation of the mechanism of extinction.

Konorski's Theory of Extinction Via Inhibitory Association

Konorski (1948) also argued that spontaneous recovery was strong evidence that the original associations were not erased as a consequence of nonreinforcement. Like Pavlov, Konorski (1948) believed that extinction was the result of active inhibition. He conceived of *neural centers* in the brain that are activated by specific stimuli and that over the course of training developed progressively stronger associations with other neural centers. He argued that during acquisition, positive or excitatory associations were strengthened between neural centers corresponding to the CS and the US. After training, activation of the CS center alone was capable of initiating activity in the US center, thus producing a CR. If the US was subsequently omitted, negative or inhibitory associations between neural centers corresponding to the CS and US were strengthened. As nonreinforcement continued, inhibition would gradually win out over excitation, and the CR would be abolished. Importantly, despite the absence of a CR, the excitatory CS-US associations remained intact.

Konorski believed that spontaneous recovery was caused by a decay in the inhibitory associations. However, this decay process was not unique to inhibition. Konorski argued that all weak and recent associations, regardless of sign, would decay over time. Hence, newly established excitatory associations would decay in much the same way as a newly established inhibitory association. Spontaneous recovery occurred because the CR-producing associations were firmly established while inhibitory associations were more recent and more weak and therefore subject to decay. In contrast to Pavlov, Konorski did not believe that inhibition was inherently fragile. In fact, Konorski believed that with extended nonreinforcement it was possible to firmly establish inhibitory associations that would be impervious to spontaneous recovery.

Perhaps the greatest contribution of this theory came in the statement of how these associative connections were formed. Excitatory CS-US associations were strengthened when the activation of the CS center coincided with an increase in activation of the US center caused by presentation of the US. Inhibitory associations were strengthened when the activation of the CS center coincided with a *decrease* in activation of the US center. The latter occurred when the well-trained CS was presented in the absence of the US. Konorski's theory is important not only because it makes explicit the notion that extinction is the result of an accrual of competing inhibition but also because it makes specific predictions concerning the formation and detection of the competing inhibition, for example:

1. Because the strength of the CR is proportional to the number of excitatory associations, extinction of the CR will require an equal number of associations and so will also be proportional to the strength of the CR.
2. Presentation of the CS is required for extinction.
3. Extinction will not occur unless there is a fall in activation of the US center. Hence, any event that prevents the fall in activation (e.g., a US, an over-conditioned CS, or perhaps a traumatic memory) will prevent extinction.
4. If the inhibitory associations formed during extinction could be temporarily removed, the excitatory activation of the US center, and therefore the CR, would be restored.

Extinction as Unlearning

Because the acquisition of a CR is commonly thought to occur as a result of the strengthening of excitatory associations, extinction may be a symmetrical process: an unlearning or rewriting of excitatory associations. Both Skinner (1950) and Estes (1955) proposed that extinction was the result of the acquisition of a new response that was more appropriate to nonreinforcement. Importantly, the associations that were formed during nonreinforcement overwrote and therefore erased the associations that produced the original CR. Rescorla and Wagner (1972) also

conceived of extinction as a *weakening* of the previously established excitatory associations (see Pearce & Hall [1980] for a review of the Rescorla-Wagner model and extinction).

If the original associations are erased, what is the cause of spontaneous recovery? Perhaps extinction does not erase all of the excitatory associations but erases enough so that they are subthreshold for performance of the CR. With the passage of time, the subthreshold associations somehow become suprathreshold, causing the CR to spontaneously recover. Estes (1955) argued that the cues present during nonreinforced CS presentations are likely to be a subset of the cues present during original training. Hence, extinction would erase only a subset of the original associations, and the CR would be abolished in the presence of this subset of cues. However, testing sometimes would later involve a different subset of the training cues whose associations have not been erased. The intact associations would produce spontaneous recovery.

Because the CR could be shown to survive extinction, Pavlov (1927) and Konorski (1948) argued that the CR-producing associations were completely intact. Extinction had to be the result of competing inhibition. However, with a few simple assumptions, an erasure hypothesis of extinction can explain why the CR returns with the passage of time. In fact, spontaneous recovery is just one example of a class of phenomena that demonstrate that the CR survives extinction. Other phenomena of this type include disinhibition, rapid reacquisition of an extinguished CR, and reinstatement of the CR by presentation of an unconditioned stimulus.

Rapid Reacquisition

In a series of experiments employing salivary conditioning in dogs, Konorski and Szwejkowska (1950) showed that while extinction of the CR generally proceeded slowly, occurring in 20 to 40 trials, reacquisition to a preextinction baseline occurred in just two to three trials (see also Konorski, 1967; Szwejkowska & Konorski, 1952). Rapid reacquisition has been demonstrated by others as well (Frey & Butler, 1977; Smith & Gormezano, 1965).

Recall that Konorski (1949) viewed acquisition and extinction as symmetrical processes: A complete loss of the CR is equivalent to the algebraic summation of excitatory and inhibitory associations. If the inhibitory associations are equal and opposite to the excitatory associations, reacquisition should occur at the same rate as, not more rapidly than, the original conditioning. Konorski (1948) was forced to make special assumptions about the nature of the competing inhibition to explain rapid reacquisition. Because the inhibitory associations were the most recent, they would be disrupted by reacquisition trials, resulting in net excitation and the *appearance* of rapid reacquisition. However, it may not be necessary to assume that recent associations are more fragile. Alternatively, the competing inhibitory associations may not be equal to the excitatory associations but may merely be sufficient to keep activation of the US center below threshold for performance. Because performance is near threshold, retraining would cause reacquisition that is more rapid than the original acquisition. Notice, however, that this subthreshold idea can also explain rapid reacquisition if extinction is considered to be the result of an erasure of the original CR-producing associations. Again, a phenomenon thought to support an inhibition hypothesis of extinction only indicates that some portion of the CR survives extinction.

Rapid reacquisition may not occur in all situations. Using a conditioned emotional response (CER) procedure in which conditioned fear is indexed by a suppression in operant bar pressing, Bouton (1986) compared the rates of reacquisition of three groups with different histories of acquisition and extinction. Two groups of rats received tone (CS)-foot shock (US) pairings followed by either 24 or 72 nonreinforced tone presentations (groups 24E and 72E, respectively). A control group was given equal context exposure but never received acquisition or extinction. Following these treatments, all three groups were given tone-foot shock pairings (i.e., reacquisition for groups 24E and 72E). In contrast to the

data on rapid reacquisition, Bouton found that group 24E reacquired the fear to the tone CS at the *same rate* that the naive control group acquired fear to the novel tone (Bouton, 1986). Even more interesting, while both experimental groups showed equal and complete extinction of fear, group 72E showed significantly *slower reacquisition* than group 24E. More recently, Hart, Bourne, and Schatman (1995) have extended this finding showing similar slow reacquisition in conditioned taste aversion paradigm. Slow reacquisition is consistent with the idea that extinction is caused by the accrual of competing inhibitory associations. Recall that extinction to a zero level of CR performance would occur if the inhibitory associations are equal and opposite to the excitatory associations. From this point, reacquisition would proceed at the same rate as original acquisition because neither excitatory nor inhibitory influences win out. In the Bouton (1986) experiment, group 24E and the naive control group showed comparable rates of reacquisition. Perhaps with those experimental parameters, 24 nonreinforced CS trials achieved a balance of excitatory and inhibitory associations. Carrying this one step further, perhaps if many more nonreinforced CS trials are given, extinction will be carried beyond zero (cf. Konorski, 1967; Pavlov, 1927; Rescorla & Wagner, 1972; Wagner & Rescorla, 1972). In this case, reacquisition of excitatory associations would have to first recover the net inhibition to produce a CR. Therefore, reacquisition would be much slower than acquisition to a novel stimulus. Perhaps 72 nonreinforced CS trials in the Bouton (1986) experiment succeeded in carrying extinction beyond zero. A theory of extinction based solely upon erasure of excitatory associations cannot explain slow reacquisition. In the limit, if all of the excitatory associations are erased, reacquisition should proceed at a rate similar to initial acquisition but never more slowly.

Reinstatement

A single presentation of the US can be sufficient to fully reinstate an extinguished CR. The basic finding is best exemplified by a study conducted by Rescorla and Heth (1975). In this experiment, rats were first given tone-foot shock training followed by tone-alone extinction trials. One half of the total number of rats were given a single "reminder" foot shock that was identical to the foot shock used in the original training. The remaining rats were given no treatment. Twenty-four hours later, all of the rats were tested for conditioned fear to the tone. Following nonreinforced tone presentations, the rats given no treatment after extinction showed a lack of conditioned suppression to the tone, that is, fear was extinguished. However, at testing, the group that received the reminder shock 24 hours earlier showed renewed suppression to the tone. The reminder shocks appeared to reinstate fear. A control group that was initially given unpaired tone and shock training followed by tone alone extinction trials did not show fear to the tone following a reminder shock. This suggests that the renewed fear was not the result of sensitization produced by the reminder shock. Instead, the reminder shock reinstated extinguished fear.

Reinstatement not only occurs following a single reminder shock but also occurs following systemic administration of drugs that are related to an organism's physiological response to stress. In one experiment, Richardson, Riccio, and Devine (1984) trained rats to avoid shock in a shuttle box. After they had obtained a stable avoidance baseline, the rats were given a session of avoidance trials in which shock was omitted, that is, extinction. Twenty-four hours later, the rats were tested for retention of the avoidance response. Just prior to this test, one half of the total number of rats were injected with the peptide aderenocorticotropin releasing factor (ACTH). The remaining rats were injected with water. The rats that were injected with water made few avoidance responses, indicating that the response had extinguished. In contrast, the rats injected with ACTH 24-hours earlier made significantly more avoidance responses. Like a reminder shock, systemic injection of ACTH reinstated the extinguished avoidance response.

Revaluation of the US

Reinstatement has been explained in several ways. Rescorla and Heth (1975) assume that extinction not only affects CS-US associations but also decreases how the US is represented. Nonreinforcement causes a deterioration in the representation of the US, rendering it less excitable. A reminder shock revalues the US representation of shock, therefore allowing it to be fully activated by the remaining net excitatory CS-US associations.

Rescorla and Heth's argument rests on the assumption that following extinction at least some of the associative CS-US associations remain intact. All of the evidence discussed thus far supports this assumption. Interestingly, within their model, it is possible to produce extinction nonassociatively, that is, without affecting CS-US associations at all. For example, if the US could be completely devalued, it would not allow for performance of the CR regardless of the strength of the CS-US associations (Rescorla & Heth, 1975). Therefore, according to their theory, if one could devise an extinction procedure that favors US devaluation over a reduction in the strength of CS-US associations, a reminder shock may lead to a greater magnitude of reinstatement since a reminder shock reinstates only the US representation. One such procedure involves extinction with continued US presentation.

Several reports have indicated that extinction will occur, albeit more slowly, when the US is presented in an unpaired fashion (Ayres & Decosta, 1971; Frey & Butler, 1977; Rescorla & Skucy, 1969). Rescorla and Heth (1975) argue that in comparison to a CS-alone procedure, continued reinforcement with a *gradual reduction* in US intensity should lead to extinction, but resulting more in part from a decrement in the US representation than from a change in CS-US connection. In a fourth experiment, Rescorla and Heth (1975) gave three groups of rats tone-foot shock pairings using a moderately intense US. Following acquisition, each group was subject to a different extinction procedure. Group Normal received CS-alone presentations, group Abrupt continued to receive CS-US pairings but with a much less intense US (one that normally does not support conditioning), and group Gradual also continued to receive CS-US pairings, but the intensity of the US was gradually reduced. Extinction was continued for six sessions. All of the groups attained a criterion level of extinction within the six extinction sessions, but they extinguished at different rates. Group Normal extinguished in three days, group Abrupt in four days, and group Gradual in six days. All groups were given four reminder shocks at the original training intensity. As expected, the groups that continued to receive CS-US pairings (Gradual and Abrupt) showed a greater magnitude of reinstatement than the group that received nonreinforced CS presentations (Normal). The extinction produced with continued presentation of a less intense US resulted in an extinguished response that was more susceptible to reinstatement.

Rescorla and Heth (1975) argue that the greater susceptibility to reinstatement reflects the disproportionate contribution of US devaluation to extinction performance when the extinction procedure is carried out with continued reinforcement. However, this interpretation of extinction is derived from the assumption that reinstatement of the CR occurs by reinstating the US representation. Little direct evidence exists that extinction weakens the US representation. For example, nonreinforcement of one CS does not affect performance to another CS trained with the same US (Bouton & King, 1983). In addition, under certain conditions, presentation of the US alone may not be sufficient to produce reinstatement (Bouton & Bolles, 1979b; Callen, McAllister, & McAllister, 1984).

Facilitated Memory Retrieval

Ahlers and Richardson (1985) have offered another nonassociative account of reinstatement that is based on their findings with pretest administration of ACTH. They argue that ACTH is released during training (perhaps as a consequence of shock) and as a result becomes an element of the training memory. Subsequent pre-

test exogenous ACTH increases the similarity of training and testing and leads to facilitated retrieval of the training memory. To test this hypothesis, Ahlers and Richardson (1985) sought to reduce the contribution of ACTH to the training memory. They administered dexamethasone (DEX), a synthetic glucocorticoid that inhibits the endogenous release of ACTH, just prior to avoidance training. The avoidance response was then extinguished in the absence of DEX. Pretest administration of ACTH reinstated extinguished avoidance responding in rats given water during training. However, ACTH did not reinstate avoidance responding in rats given DEX during training. Thus, reinstatement by exogenous ACTH seems to depend on the participation of endogenous ACTH during training. It is possible that any postextinction event that reintroduces elements of the training memory can facilitate retrieval of the entire training memory. For example, shocks or other stressors can cause the release of ACTH, which may reinstate the CR (Ahlers & Richardson, 1985).

Reconditioning of CS-US Associations

There are, however, aspects of the effect of ACTH that cannot be easily explained by the facilitated memory retrieval hypothesis. In a subsequent experiment, Ahlers and Richardson (1989) showed that a single injection of ACTH 24 hours after extinction reinstated avoidance responding seven days later (Ahlers, Richardson, West, & Riccio, 1989). Facilitated retrieval would not persist over this long interval. Instead, effects that occur over very long intervals are oftentimes indicative of some form of learning. Alternatively, ACTH may act to strengthen the associative CS-US associations formed during training (Bohus, Nyakas, & Endroczi, 1967; Izquierdo & Pereira, 1989). This associative effect of ACTH would be expected to persist over very long injection-to-test intervals. Consistent with this, pretraining administration of ACTH or another peptide, vasopressin, has been shown to facilitate *acquisition* of active avoidance (Bohus & Endroczi, 1965), and posttraining administration of ACTH or vasopressin has been shown to facilitate the retention of active avoidance (Izquierdo & Pereira, 1989). Interestingly, Bohus, Nyakas, and Endroczi (1967) found that the facilitatory effect of ACTH did not occur until after a minimum amount of conditioning was demonstrated. Together these results suggest that ACTH may act to strengthen CS-US associations.

Similarly, Callen, McAllister, and McAllister (1984) argued that reinstatement was the result of reconditioning. In their experiment, fear was conditioned to apparatus cues in one side of a hurdle-jumping apparatus and then extinguished. The rats were then shocked either in the presence of these same apparatus cues or in a distinctively different apparatus. Despite the fact that both groups were shocked, reinstatement of conditioned fear occurred only in the rats that were shocked in the presence of the original apparatus cues. According to the nonassociative accounts of reinstatement (Ahlers & Richardson, 1985; Rescorla & Heth, 1975), reinstatement should occur regardless of where the shocks are given. Instead, these data show that shock must be given in the presence of the stimuli associated with original training, suggesting the possibility that reinstatement is the result of reconditioning or strengthening of residual CS-US associations (Callen et al., 1984).

Summation With an Excitatory Context

It is well established that contextual cues can acquire associative strength and can have a profound influence on acquisition and performance of CRs (Bouton, 1984; Bouton & Bolles, 1979a; Bouton & Bolles, 1979b; Dweck & Wagner, 1970; Odling-smee, 1978). One way to assess conditioning to the context is to place a weakly excitatory CS in the putative excitatory context and assess the CS's ability to elicit a CR. The weak CS does not produce a CR on its own, but when placed in compound with an excitatory context, the excitatory tendencies of both the CS and the context summate, and the CS elicits a CR. Perhaps reinstatement results from the summation of the weak extinguished CS with a context that was made excitatory by the "reminder"

US. Neither the context nor the weak CS would produce a CR on its own, but together, the combined associative strengths may result in a renewed CR. If so, reinstatement would occur only in the context where the reminder shock was given.

Rescorla and Heth (1975, Experiment 2) attempted to test this associative mechanism of reinstatement by giving the reminder shock in a context that was different from the one that was going to be used in testing. In support of their nonassociative hypothesis, they found reinstatement even though the rats were tested outside of the shocked context. However, as Bouton and Bolles (1979b) point out, the contexts used by Rescorla and Heth (1975) may not have been different enough to allow the rat to discriminate between them, as would be required to test the context dependency of reinstatement. In a direct test of context-specific reinstatement, Bouton and Bolles (1979b) gave reminder shocks to rats either in a context in which they were to be tested or in a context discriminatively different from the test context. Using the CER procedure, two groups of rats received tone-foot shock pairings followed sometime later by tone-alone extinction trials. One group (group CC) was given reminder shocks in the conditioning context, and the other group (group TC) was given the shocks in the test context. Both groups were tested for reinstatement in the test context. The results were quite clear. Reinstatement occurred only when the rats were tested in the shocked context. In addition, extinction of the shocked context prevented or erased reinstatement (Bouton & Bolles, 1979b, Experiment 2; cf. Rescorla & Cunningham, 1977). Similar results have been obtained more recently by Callen, McAllister, and McAllister (1984) using a passive avoidance procedure. It would thus appear that neither a US revaluation, facilitated retrieval, nor a strengthening of CS-US associations accounts of reinstatement are necessary. Summation of an excitatory context with residual excitation of the extinguished CS may account for the reinstatement of CRs seen following reminder shocks.

A context-US association account of reinstatement rests on the assumptions that the extinguished CS retains some level of excitation following extinction. This still leaves open the question of whether extinction is due to a build-up of competing associations or a to reduction in the number or strength of excitatory CS-US associations. Investigations of reinstatement have not provided clear evidence supporting either theory. However, similar work on the role of context in extinction suggests that little or none of the excitatory CS-US associations are erased with extinction. And more importantly, these results strongly suggest that inhibitory context-US association is formed during extinction.

Another Look at the Role of the Context

Bouton (1991) has noted that the strength of context conditioning is directly related to the amount of reinstatement observed in that context. But this context conditioning is often weak, and sensitive procedures have to be used to detect it (Bouton, 1991). So, contextual associations are weak, residual CS associations may be considered weak, yet summation of these two effects is very robust, often returning the CR to its preextinction level. Perhaps residual associations of an extinguished CS are especially sensitive to summation with weak contextual associations.

If reinstatement is due to summation of these weak associations, both an extinguished CS and a weak nonextinguished CS should produce comparable levels of conditioned responding following US exposure. To test this, Bouton (1984) arranged conditions so that an extinguished CS and a nonextinguished CS both produced comparable levels of pretest conditioned responding. This was accomplished in the following way. In one group of rats, a CS was paired with a strong shock (3.0 mA) and then followed by extinction; in the other group, the CS was paired with a weak (0.3 mA) shock and not followed by extinction. Both groups were given the intense shock alone. Despite equivalent amounts of context conditioning in both groups, only the group that had been extinguished showed enhanced fear to the CS. The context did not enhance fear to the weak CS. Hence, the contextual associa-

tions preferentially affected the extinguished CS. This result would not be expected if reinstatement was simply the result of summation of residual CS and weak contextual associations.

Bouton has argued that unlike a weak, nonextinguished CS, an extinguished CS is "ambiguous" because it has been associated with the US in some circumstances and the absence of the US in other circumstances. This ambiguity makes it susceptible to reinstatement by an excitatory context. More specifically, extinction is thought to result from a discrimination between the circumstances in which the CS is and is not followed by the US. In other words, CS-US associations are intact following extinction but are accompanied by associations that are specific to nonreinforcement. Whether or not the CR occurs in any situation will depend on which set of associations is active. Excitatory context-US associations may reinstate the CR either by retrieving the CS-US associations or by inhibiting the competing associations.

Context Specific Extinction

Context may also play a more direct role in the extinction process. In an impressive series of experiments Bouton and colleagues have shown that extinction is specific to the context in which nonreinforcement occurs (Bouton, 1991; Bouton & Bolles, 1979a; Bouton & Bolles, 1985; Bouton & King, 1983; Bouton & King, 1986). In one experiment, rats were first given tone-shock pairings in a training chamber. After conditioning, the rats were given tone-alone extinction trials in one of two different contexts. To evaluate whether extinction would be specific to the context where the tone was nonreinforced, the rats were tested either in the extinction context or in the alternate context. Rats tested in the extinction context showed extinction of conditioned fear, whereas rats tested in the alternate context showed renewed fear to the tone. The data clearly show that under circumstances in which the rat can discriminate between different contexts, extinction is specific to the context where nonreinforcement occurred.

An erasure hypothesis of extinction cannot easily explain context-specific extinction. Erasure would reduce the likelihood of the CR regardless of where it was tested. On the other hand, a competing association hypothesis can explain context specificity. Stimuli present during nonreinforcement acquire inhibitory CS-US associations that compete with existing excitatory CS-US associations. Perhaps Bouton and Bolles (1979a) succeeded in allowing the context to acquire a great deal of the inhibition. Outside that context there would be no inhibition, and the CR would be renewed. Note that this is similar to the conventional conditioned inhibition procedure in which a neutral stimulus is nonreinforced in the presence of an excitatory CS (see next subsection).

To test whether the extinction context was inhibitory, Bouton and King (1983) used a summation test in which fear to a nonextinguished light was tested in a context in which another CS had been extinguished. If the extinction context was inhibitory, fear to the nonextinguished light should be reduced because of the competing associations. Surprisingly, fear to the light was not reduced. It appears as though the extinction context was not inhibitory (Bouton & King, 1983).

Bouton has argued that as a result of its history of reinforcement and nonreinforcement, an extinguished CS may be considered "ambiguous" (Bouton, 1984; Bouton, 1991; Bouton & King, 1986). The extinction context is not inhibitory but may act to remove the ambiguous meaning of the CS. For example, if a CS is paired with shock in one context and extinguished in another context, the former context may signal that the CS will be followed by shock, while the later context may signal that the CS will not be followed by shock. Hence, CS-US associations may be formed in both training and extinction, and the context determines which associations are selected for expression. Just how the context selects the associations is unclear. One possibility is that the extinction context plays a permissive role gating the *inhibitory CS-US associations* that are formed during nonreinforcement. (Of course, this assumes that inhibitory CS-US asso-

ciations are formed during extinction. Up to this point, there has been no evidence to suggest that the CS acquires inhibitory CS-US associations.) Alternatively, the extinction context may be directly responsible for extinction, inhibiting the expression of excitatory CS-US associations. In both cases, excitatory associations are intact, and extinction is the result of the accrual of new associations. One way to begin to evaluate these alternatives is to ask whether evidence exists that an extinguished CS is inhibitory.

The Relationship of Extinction to Feature Negative Discriminations

It is well established that a neutral stimulus can acquire the ability to reduce a CR following training in which it is placed *in compound with an excitatory CS and not reinforced* (Konorski, 1948, 1967; Pavlov, 1927; Rescorla, 1969; Wagner & Rescorla, 1972). The result of this so-called feature negative discrimination procedure leaves little doubt that the neutral stimulus, referred to as a feature, has acquired the ability to somehow inhibit the production of the CR. The classic interpretation of a feature negative discrimination is that the feature inhibits the representation of the US, so when it is placed in compound with the target CS, the CR is reduced. On the face of it, the feature negative discrimination procedure does not differ substantially from the normal extinction procedure. Both involve *nonreinforcement* in the presence of an otherwise *excitatory CS,* and both result in a decrement in the CR. Therefore, one can ask whether it is necessary to assume a separate mechanism for extinction and a feature negative discrimination.

Despite all the evidence in support of the idea that extinction is caused by some form of competing association, researchers have been reluctant to entertain the notion that the CS acquires inhibitory CS-US associations. This reluctance is based on the failure to detect inhibition by an extinguished CS (Reberg, 1972; Rescorla, 1969, 1979). However, one would have no reason to suspect that an extinguished CS and a conditioned inhibitor would yield the same results on a summation or retardation test. As Rescorla (1969) points out, an extinguished CS would have both excitatory and inhibitory CS-US associations (cf. Konorski, 1948; 1967). Inhibition by an extinguished CS can be detected only with the traditional procedures if the net CS-US inhibition is greater than the net CS-US excitation. A feature, on the other hand, is a neutral CS at the outset of conditioning. It only acquires inhibition and has no competing excitatory CS-US associations. Therefore, inhibition is easily detected.

The task would be to demonstrate that extinction and conditioned inhibition are functionally equivalent. One approach is to demonstrate that the inhibition and extinction procedures are interchangeable. Along these lines, Devito and Fowler (1987) conducted an experiment in which they demonstrated that conditioned inhibition can be enhanced by extinction of the conditioned inhibitor. Using a lick suppression procedure, two groups of rats given feature negative discrimination training in which a clicker was paired with shock and a tone+clicker compound was presented without shock. Group Extensive was given a total of 144 tone+clicker trials, while the other, group Moderate, was given 42 tone+clicker trials. In a third phase, both groups were given tone-alone extinction trials. A third group, group Control, was given identical training to group Moderate, but the tone-alone phase was omitted. Prior to tone-alone trials, group Extensive acquired greater inhibition to the tone than groups Moderate and Control. Following nonreinforced tone presentations, group Moderate showed the same level of inhibition as group Extensive, and they both showed reliably more inhibition than group Control. Therefore, for group Moderate, the intervening tone-alone extinction enhanced an already moderate level of inhibition. Although these data are by no means conclusive, they are consistent with the notion that feature negative discriminations and extinction operate through a similar mechanism (Devito & Fowler, 1987). This mechanism may be the accrual of inhibitory CS-US associations.

Evidence is accumulating, however, suggesting that feature negative discriminations may not always result in the feature acquiring inhibitory feature-US associations. Under some experimental conditions, the feature acquires the ability to modulate the expression of the excitatory CS-US associations (Holland, 1985, 1986, 1989, 1990; Holland & Morell, 1993). These features "set the occasion for nonreinforcement" of the CS essentially by inhibiting the ability of the training target to activate the US representation (Holland, 1985, 1990; Rescorla, 1985). Once again, the feature negative discrimination is procedurally similar to extinction, which may involve a similar occasion setting mechanism. Some stimulus present during nonreinforcement may acquire the capacity to inhibit the ability of the CS to activate the US representation. Recall that Bouton has proposed that the extinction context disambiguates the meaning of the CS. The extinction context could act like this feature and inhibit the ability of the CS to activate the US representation. In this context, the CR would be extinguished; outside this context, the modulation does not occur and the CR is renewed (Bouton, 1991). Because the CS does not inhibit the US representation and because the context does not inhibit the US representation, this hypothesis explains why there have been repeated failures to detect inhibition to the CS and the context after extinction.

Summary

Taken together, the data reviewed thus far clearly indicate that extinction does not result in a complete erasure of the original CS-US association. Despite the appearance of no further memory toward the end of an extinction session where no conditioned response whatsoever may occur, evidence of some remaining amount of CS-US association can be seen following a period of time (spontaneous recovery), presentation of a US alone (reinstatement), or testing in a different context (renewal). However, these paradigms do not directly implicate active inhibition as the mechanism of extinction. On the other hand, there is wide agreement that a closely related phenomenon known as feature negative discrimination does involve some form of inhibition. In fact, it has been argued that extinction is a special case of feature negative discrimination. Whether extinction is more like traditional feature negative discrimination or like occasion setting must still be determined. Complicating this picture is the possibility that extinction can involve either process or both processes and that the contribution of one over the other is dependent on procedural differences that we do not yet understand (Falls & Davis, submitted; Holland, 1989b). Interestingly, Bouton (1991) reports that the context specificity of extinction can be reduced if extinction is also carried out in the training context. Perhaps this procedure reduces the contribution of negative occasion setting (Falls & Davis, 1993). Clearly more work is needed not only to explore the relationship between extinction and feature negative discriminations but also to determine the conditions under which feature negative discriminations result in traditional feature-US inhibition or occasion setting.

AVOIDANCE OF THE CS: IMPLICATIONS FOR EXTINCTION

Most of the experiments discussed thus far have employed Pavlovian fear-conditioning procedures to evaluate the extinction process. The advantage of these procedures is that the exact presentation and timing of stimuli can be controlled. Hence, experimenters can be reasonably assured that each stimulus is fully encountered by the animal. However, this assurance is rare outside of the laboratory and outside of these procedures. More typically, animals, if allowed, will escape from a stimulus that elicits fear. This can have a profound impact on the extinction process.

The Paradox of Active Avoidance

Consider the following experiment. A dog is placed into one side of a two-compartment box. The compartments are separated by a low barrier. After a short period of time, a tone is presented

to the animal, and 5 seconds later, foot shock is presented through the floor of the box. The foot shock elicits vigorous activity, a consequence of which is that the dog jumps over the barrier and escapes the shock. A few minutes later, the same sequence is repeated. In a few trials, the dog will jump the barrier shortly after the tone is presented, thereby avoiding the shock. Over the next several trials, the time it takes for the dog to avoid the shock is reduced. Now the dog may jump the barrier immediately after the tone comes on. Depending on various parameters, the dog may make hundreds of successful avoidance responses, never getting shocked.

The explanation for this avoidance behavior seems straightforward. The tone signals that shock will occur and the dog learns to jump over the barrier to avoid getting the shock. But as Mowrer (1947, as cited by McAllister & McAllister, 1995) pointed out, how can a shock that is avoided and therefore not experienced serve to reinforce a barrier-jumping response? In addition, even if the dog is avoiding the shock when the tone comes on, why doesn't the avoidance behavior extinguish after the shock is not experienced?

Two-Factor Theory of Avoidance

To resolve this paradox, Mowrer (1947) proposed that active avoidance behavior involved a few distinct processes. Initially, the dog learns to be fearful of the CS by virtue of its being paired with shock. Fear of the CS activates the dog and eventually leads the dog to escape this aversive CS by jumping the barrier. This escape response results in a reduction of fear that reinforces the barrier-jumping response. So, when the dog is placed in the shock compartment and presented with the CS, fear elicited by the CS motivates the escape response and results in a reduction of fear, which reinforces the escape response.

Mowrer's (1947) theory resolved the avoidance paradox, although it has been criticized on two grounds. For one thing, animals well trained in active avoidance paradigms did not seem fearful, but merely jumped the barrier as soon as the CS came on (Seligman & Johnston, 1973). In addition, even if fear motivated the barrier-jumping response, this response often took much longer to extinguish than fear measured in other Pavlovian conditioning paradigms.

The criticism that fear is no longer present in a well-trained dog may, however, be without empirical basis (McAllister & McAllister, 1991). Several experiments have indicated that when avoidance responding is well learned, a substantial amount of fear to the CS remains (Levis & Boyd, 1979a; Mineka & Gino, 1980; Starr & Mineka, 1977). For example, Mineka and Gino (1980) showed that rats trained to a criterion of 27 avoidance responses still showed substantial fear to the CS in a CER procedure.

To explain the very slow rates of extinction, Solomon and Wynne (1954) introduced their "conservation of anxiety hypothesis," based on a few observations. First, they noticed that over the course of training, the avoidance latencies got progressively shorter, while the signs of fear seen early in training were no longer evident. However, if the animal happened not to make a long latency response, the continued presence of the CS elicited signs of fear.

Given this behavior, the animal arranged conditions in which the CS was on for only a very short period of time. Solomon and Wynne (1954) suggested that this protected them from extinction, a conclusion consistent with later work showing that rate of extinction is directly related to the total duration of CS exposure (Malloy, 1981; Shipley, 1974). In fact, in an avoidance study in humans, subjects who failed to extinguish had shorter avoidance latencies than subjects who did extinguish (Williams & Levis, 1991).

This now, however, exposes the animal to a part of the CS that has not become extinguished and leads to a high level of fear, which then serves as a reconditioning trial, making the first part of the CS fearful once again (i.e., reinstatement). This then leads to a very short latency avoidance response and once again protects the

rest of the CS from being exposed and hence protects it from extinction. In fact, Levis and Boyd (1979b) have data on rats that show a striking oscillation of avoidance latencies over trials where a long latency response tends to be followed by a short latency response, and vice versa. With extended training, the animal begins to make the avoidance response at a longer latency, thus extending the duration of CS exposure and hastening extinction.

These ideas account for a good deal of data within the literature on active avoidance, although they have not gone unchallenged. For example, Seligman and Johnston (1973) assert that although fear is necessary for the acquisition of active avoidance behavior, it does not motivate sustained asymptotic avoidance behavior. They pointed out that Solomon's dogs sometimes responded for hundreds of trials, whereas fear measured in a conditioned emotional response paradigm extinguished after only 40 trials (Annau & Kamin, 1961). Because of this, they concluded that fear would have extinguished long before avoidance behavior, and hence fear could not continue to produce the avoidance response. Instead, they proposed a more cognitive account where the avoidance response continued because the animal expected that the absence of a response will lead to a shock.

This cognitive account may not be necessary, however (Levis, 1989; McAllister & McAllister, 1991, 1995). Levis (1989) pointed out that the total duration of CS exposure in a typical Solomon avoidance study where a dog made 500 avoidance responses without receiving a shock would be about 1,000 seconds, assuming an average response latency of 2 seconds. In contrast, total CS exposure for complete extinction in the Annau and Kamin study quoted by Seligman and Johnston (Seligman & Johnston, 1973) was 2,400 seconds. Furthermore, even when animals are making short latency avoidance responses, they still appear to be fearful of the CS, provided sensitive enough tests are used. For example, Levis and Boyd (1979b) trained rats to an asymptotic level of active avoidance and showed that the onset of the CS still caused substantial suppression of bar pressing in a conditioned emotional response paradigm. Hence, lack of exposure to the CS can account for the persistence of the avoidance response.

Eventually the avoidance response will extinguish. Although the preceding arguments describe why the escape response is slow to extinguish, they do not explain how extinction occurs. McAllister and McAllister have described how the extinction process might occur. Following Mowrer (1947), they assume that escape from the CS results in the reduction of fear and reinforcement of the escape response. Fear reduction can occur in one of two ways: either with the removal of fear-eliciting stimuli or through generalization decrement, as when a neutral stimulus or so-called feedback stimulus is presented (McAllister & McAllister, 1992). The reduction of fear results in a response of relaxation that can be conditioned to the feedback stimulus (Denny, 1991). Since relaxation is antagonistic to fear, the feedback stimuli acquire the ability to reduce fear. The instrumental avoidance response is reinforced by relaxation elicited by the feedback stimuli. Importantly, the feedback stimuli do not act as positive reinforcers of the escape response. If they did, the avoidance response would be maintained indefinitely. Instead, the avoidance response is maintained by fear reduction (i.e., relaxation). Hence, as long as fear persists, feedback stimuli will be effective as fear reducers, and the avoidance response will be maintained. Once fear is extinguished, the avoidance response is no longer reinforced and will cease to occur.

Extinction of fear occurs as a result of a competing response of relaxation. Initially, feedback stimuli and traces of the CS acquire relaxation. But as nonreinforcement continues, the CS itself can acquire the ability to elicit relaxation. Therefore, through counterconditioning of fear, fear to the CS will extinguish. As the animal exposes itself to more and more of the CS, more of the CS acquires relaxation, and the escape response stops.

EXTINCTION OF INSTRUMENTAL RESPONSES

The preceding discussion has focused on extinction of Pavlovian conditioned responses. However, biologically significant stimuli have the ability to reinforce instrumental responses, and the omission of the reinforcer after the instrumental response is acquired leads to the reduction of the response. For example, rats will learn to press a bar to receive a food reinforcer. The bar pressing will be maintained as long as food is delivered on some schedule. Once the food no longer follows a bar press, the frequency of the bar-press response will gradually decline.

Inhibitory S-R Associations

Like Pavlovian extinction, the process that underlies extinction of instrumental responses is not yet understood. Like extinction of Pavlovian CRs, extinction of instrumental responses begs the same questions: Have the associations that lead to the instrumental response been somehow masked, or have they been erased or otherwise overwritten? But answering these questions for extinction of instrumental responses may be more difficult because the nature of the associations that are formed in instrumental learning is more complex. As Rescorla (1993) points out, recent analysis of instrumental learning suggests that a variety of associations are formed as a consequence of instrumental learning procedures. For example, exists evidence to suggest that associations are formed between the instrumental response (R) and the reinforcer (more accurately referred to as the earned outcome (O), between the discriminative stimulus (S) and the reinforcer, and between the discriminative stimulus and the response. Evidence also points to higher order associations between S and the R-O association (Rescorla, 1993). It is possible that any one or a combination of these associations is affected by nonreinforcement.

Rescorla has conducted several experiments with rats that suggest that the S-O and R-O associations are intact following nonreinforcement (Rescorla, 1992, 1993). In a typical procedure, an auditory cue (S) signals that a lever press (R) will lead to a food pellet (O). After the rat has acquired this instrumental behavior, reinforcement is removed, and the lever press response extinguishes. To evaluate the integrity of S-O association, Rescorla presented the original auditory stimulus in the context of a unique response that was previously associated with the same food pellet outcome. Despite previous nonreinforcement, the original auditory cue retained its ability to augment responding. This suggests that the original S-O association was intact.

Similarly, in a test of the integrity of the R-O association, rats were presented with a unique stimulus that had previously signaled a different response that led to the same food pellet outcome. Once again, despite extinction, the bar-press response returned, suggesting that the R-O association was also intact. If both the S-O and R-O associations are intact following nonreinforcement, why does the response extinguish with nonreinforcement? Rescorla (1993) suggests that nonreinforcement results in the acquisition of an inhibitory S-R association. In a test of whether an S present at the time of nonreinforcement acquires an inhibitory association with, Rescorla (1993, Experiment 4) trained rats to press a lever for food pellets. This response was extinguished in the presence of a visual stimulus. The bar-press response was then retrained. In a subsequent test, the visual stimulus suppressed the bar-pressing response but did not affect a chain-pull response that had also been reinforced with food pellets. This suggests that nonreinforcement in the presence of the visual stimulus causes the visual stimulus to acquire the ability to inhibit the bar-press response; that is, extinction resulted in an inhibitory S-R association. Hence, like Pavlovian extinction, instrumental extinction does not appear to involve the erasure of the associations that led to the original response but rather appears to involve buildup of competing associations that mask the expression of the associations that led to the instrumental response. Moreover, the inhibition appears to be under the control of stimuli that are associated with nonreinforcement.

The Role of Competing Responses in Instrumental Extinction

In his review of the major theories of extinction, Mackintosh (1974) distinguished between the inhibition theories of extinction (Rescorla's inhibitory S-R association would come under this class) and interference theories of extinction. We have discussed the inhibition theories in some detail. The interference theories, on the other hand, believe that the omission of the reinforcer establishes a set of new responses that are incompatible with the original instrumental response. Therefore, as the strength of the new response builds, the original instrumental response is overcome. As Mackintosh (1972) points out, extinction is often accompanied by an increase in overt behaviors that appear to be incompatible with the instrumental response. However, an increase in overt behavior does not mean that these behaviors themselves are the cause of extinction. According to Mackintosh (1972), what is required is a conniving demonstration that independent manipulation of the overt behaviors systematically alters the course of extinction. For example, will prevention of these behaviors during nonreinforcement block extinction? Or will prevention of these behaviors result in reinstatement of the extinguished instrumental response? Little evidence exists that the overt behaviors themselves cause instrumental extinction.

Conditioned Frustration

These overt responses do occur, however. Perhaps they are indicative of the process that leads to instrumental extinction. That is, these overt behaviors may occur as a consequence of the process that leads to extinction. Skinner (1950) observed that when key pecking was no longer reinforced, a pigeon would begin to coo, move rapidly about the cage, defecate, or flap its wings. These behaviors gave the appearance of an emotional reaction to nonreinforcement. Several authors have suggested that the omission of reinforcement leads to the emotional response of frustration, which in turn contributes to extinction of the instrumental response (Amsel, 1958, 1962; Skinner, 1950; Spence, 1960). Azrin, Hutchinson, and Hake (1966) provided striking evidence for frustration. In their experiment, two birds were placed in an operant box. One bird was reinforced for pecking a key, while the other bird was lightly restrained in the corner of the box. When reinforcement was discontinued, there was a high probability that the formerly reinforced pigeon would attack the restrained pigeon (Azrin, Hutchinson, & Hake, 1966). It's been argued that this aggression is due to the frustration caused by the withdrawal of reinforcement (Terrace, 1971). Although nonreinforcement may cause frustration and an overt aggressive display, little evidence exists to suggest that these behaviors cause extinction. In fact, Azrin, Hutchinson, and Hake (1966) found that both the probability of aggressive displays and the instrumental response decrease over the course of nonreinforcement. If the aggressive display were causing the decrease in instrumental performance, the aggressive displays should increase as instrumental performance decreases.

Amsel (1958, 1962) suggested that frustration could be conditioned to stimuli that preceded nonreinforcement. Importantly, because conditioned frustration was thought to be aversive and because aversive states are incompatible with instrumental performance, the presence of conditioned frustration would reduce instrumental performance. Thus, instrumental extinction can be explained through Pavlovian conditioning of frustration. To take a specific example, frustration is elicited in a rat when food reinforcement is omitted from the goal box of a maze. As a result, the goal box acquires the ability to elicit conditioned frustration, which is aversive and which the rat will avoid. The conditioned frustration grows with continued nonreinforcement, and over trials, the portions of the maze preceding the goal box also acquire the ability to elicit conditioned frustration. The tendency to avoid the cues that signal frustration competes with the tendency to approach cues that signal instrumental reinforcement, and soon the instrumental response ceases to occur.

Is frustration aversive, and can it be condi-

tioned to stimuli associated with nonreinforcement? A variety of studies suggest that nonreinforcement is aversive (cf. Amsel, 1958, 1962). In one notable experiment, Wagner (1963) trained rats to run a U-shaped maze to a goal box where food reinforcement was provided. After this habit was established, rats were reinforced on only 50 percent of the trials. On trials in which nonreinforcement occurred, a CS of flashing lights and interrupted noise was provided. The intention was to use nonreinforcement to condition frustration to this CS. After this, the aversiveness of the CS was tested in a hurdle-jumping apparatus in which the rats could terminate the CS by jumping a hurdle. Rats exposed to the CS during nonreinforcement escaped from the CS more quickly than did control rats that had not experienced the CS. In addition, the amplitude of the acoustic startle response was facilitated during the CS in these rats as well. Facilitated startle is a reliable measure of aversive Pavlovian conditioning (Brown, Kalish, & Farber, 1951; Davis & Astrachan, 1978). Hence, these data suggest that stimuli present during nonreinforcement of an instrumental response can acquire aversive properties. To the extent that frustration is aversive and is incompatible with instrumental performance, conditioned frustration can explain extinction of instrumental responses. But as Mackintosh (1974) points out, this explanation of extinction seems to apply only to instrumental situations where food is the reinforcer. However, in situations where the instrumental response is reinforced by an aversive outcome, nonreinforcement may elicit a response of relaxation (Deny, 1991).

In summary, data suggest that nonreinforcement of an instrumental response elicits frustration, which has two primary consequences: (1) elicitation of unlearned, incompatible behavior that may interfere with the performance of the instrumental response and (2) acquisition of conditioned frustration to the stimuli preceding nonreinforcement. Conditioned frustration may result in an affective state whose consequences are incompatible with production of the instrumental response. As with extinction of Pavlovian CRs, direct competition between these associations leads to a reduction in responding. Also like Pavlovian extinction, the competing associations are conditioned to the stimuli that signal nonreinforcement. Therefore, any manipulation that removed these stimuli would result in a return of the instrumental response.

The Partial Reinforcement Extinction Effect

Any discussion of instrumental extinction would be incomplete without consideration of the partial reinforcement extinction effect (PREE). Simply stated, the PREE is increased resistance to extinction of an instrumental response that has been partially reinforced in training. In the most simple case, maze running that is followed by food on 75% of occasions (partial reinforcement) will extinguish less quickly than maze running that is reinforced on 100% of occasions (continuous reinforcement). Amsel (1958, 1962) argued that the PREE occurs because animals trained under partial reinforcement learn to respond under conditions of frustration. As we have seen, nonreinforcement elicits frustration that is conditioned to the stimuli preceding nonreinforcement. However, when reinforcement is partial, the rat is rewarded in the presence of cues that signal frustration. Because the rat is rewarded more than it is frustrated, the approach response prevails. When reinforcement is totally withdrawn (i.e., extinction), the rat will resist extinction and will continue to run the maze because it has learned to run in the presence of stimuli signaling frustration. On the other hand, a rat that is given continuous reinforcement does not learn to run in the presence of stimuli signaling frustration and will extinguish more quickly. As a final note, although the PREE has been demonstrated in a large number of studies and is one of the more reliable findings in instrumental conditioning, a number of studies have failed to find evidence for the PREE in Pavlovian conditioning (Berger, Yarczower, & Bitterman, 1965; Gonzalez, Mil-

stein, & Bitterman, 1962; Longo, Milstein, & Bitterman, 1962; Thomas & Wagner, 1964; Wagner, Siegel, & Fein, 1967). Therefore, Pavlovian CRs acquired with partial reinforcement may not be more difficult to extinguish.

CLINICAL IMPLICATIONS

Therapeutic techniques such as systematic desensitization, flooding, and implosion are based on the idea that exposure to fear-eliciting stimuli will result in extinction of fear. These therapies can be successful in eliminating phobias. However, simply because the fear or phobia is no longer observable does not mean that it has been permanently removed. As we have seen, basic research has shown that extinction does not erase the original memory. The memory seems to be indelible, and extinction appears to involve a process that somehow inhibits or modulates the expression of the memory. The downside to the indelibility of the memory is that the extinction process is easily disrupted and unstable (Bouton & Swartzentruber, 1991) Therefore, under certain conditions, the extinguished memory can return. Therapists should be aware of these conditions, anticipate them, and consider them in the course of therapy.

Reinstatement

Exposure to the US itself or to some component of the original training experience can result in the return of a previously extinguished behavior. Reinstatement has four characteristics that the clinician should be concerned with. First, a single exposure to the US can result in reinstatement. Second, the effect of a single exposure to the US can persist long after US exposure. The reinstated response may remain until further extinction is carried out. Third, reinstatement can occur despite extensive nonreinforcement (Bouton & Swartzentruber, 1991). Hence, extensive behavioral therapy may not inoculate a client against the consequences of reexperiencing the US. Fourth, and perhaps most important, reinstatement does not have to be produced by the original US. Reinstatement can occur following exposure to stimuli that are seemingly unrelated to the original US.

Recall that Bouton and colleagues have shown that reinstatement of conditioned fear will occur if a rat is placed in a context that was previously paired with shock. Hence, stimuli associated with the same US can be sufficient to reinstate extinguished memories. In addition, reinstatement may not require new learning. Reintroduction to components of the original learning may be sufficient to produce reinstatement. Ahlers and Richardson (1985) have shown that reinstatement of fear occurs if a rat is injected with the stress-related peptide ACTH. ACTH is probably an element of the original training memory. This suggests that stimuli and events seemingly unrelated to the original learning can produce reinstatement if they activate a subset of the elements of the original experience. For example, burning dinner (often mildly traumatic) could reinstate an extinguished traumatic memory if it happens to activate similar visceral responses that were activated by the traumatic experience. It is especially interesting in this regard that drugs like yohimbine, which activate the release of norepinephrine in the brain, often produce flashbacks (memory of prior trauma) in patients with post traumatic stress disorder (Southwick et al., in press). Because elevated levels of brain norepinephrine would have accompanied the traumatic event, yohimbine may reinstate a component of the traumatic memory. Presumably, any event that releases norepinephrine in sufficient quantity would also produce flashbacks.

There is evidence to suggest that extinction of stimuli capable of producing reinstatement may reduce their ability to subsequently produce reinstatement. For example, Bouton and Bolles (1979b) showed that extinction of the context in which reminder shocks occurred reduced the context's ability to produce reinstatement. Similarly, extinction carried out in the presence of exogenous ACTH reduced the ability to subse-

quently produce reinstatement. Although therapists cannot identify and extinguish all stimuli that could potentially produce reinstatement, they might consider identifying the stimuli that the client is likely to encounter that may be related to the original learning. Extinction of these stimuli may reduce the likelihood of reinstatement.

Context Specificity of Extinction

If extinction is carried out in a distinguishing context (e.g., the therapist's office), extinction may be evident only in that context. The response may be renewed outside of that context. The goal of the therapist should be to reduce the context specificity of extinction. Bouton (1991) reports preliminary evidence suggesting that context specificity can be reduced if extinction is carried out in the same context in which training took place. In this case, the context does not serve as a discriminative stimulus that removes the ambiguous meaning of CS. In other words, there is no salient cue to signal when or where the CS will or will not be reinforced. One way to minimize context specificity would be to conduct behavior therapy in the client's natural setting. This setting may be the context in which the stimuli to be extinguished are normally encountered. Note that this would also tend to extinguish other stimuli in the client's environment that could produce reinstatement (see reinstatement). Another way to minimize context specificity would be to vary the context in which behavior therapy is given. This would create several contexts associated with nonreinforcement and may promote generalization to yet unexperienced, novel contexts.

Another implication of context-dependent extinction is that renewed fear can occur if drugs are made part of the extinction context. If a drug is used as an adjunct to therapy, renewal could occur when the extinguished stimulus is encountered in the absence of the drug. In fact, animal experiments have shown that when benzodiazepines are given during extinction of conditioned fear, fear of the CS is renewed when testing occurs in the absence of the drug (Bouton, Kenney, & Rosengard, 1990).

Avoidance Conditioning

Mowrer (1947) believed that abnormal behaviors came about because they had for the individual the appearance of lessening or reducing anxiety. According to his theory, instrumental escape responses are motivated by fear and reinforced by the reduction of fear. When an individual performs a response to escape fear, the entire fear-eliciting stimulus is not experienced. Because extinction is a function of the amount of exposure to the CS (Shipley, 1974; Shipley, Mock, & Levis, 1971), fear will not extinguish and will continue to motivate the avoidance response (McAllister & McAllister, 1995). Flooding and implosive therapy are successful because these procedures control the exposure to the fear-eliciting stimuli, thereby ensuring extinction of fear and preventing the instrumental escape responses that would otherwise be reinforced by fear reduction (McAllister & McAllister, 1995).

Extinction of fear through flooding or implosive therapy may not extinguish the instrumental escape response but may extinguish only the fear that motivates it. If fear is reinstated or renewed, the instrumental escape response is likely to recur as well. In one experiment, McAllister, McAllister, Scoles, and Hampton (1986) gave rats CS+shock training in one side of a hurdle-jumping apparatus. Rats were then allowed to learn to escape fear by jumping the hurdle. No shock was given during escape trials. The escape response was readily learned and maintained and after 225 trials eventually ceased. Next, the rats were given a single CS+shock pairing (analogous to a reminder shock). This resulted in an immediate return of the escape response, suggesting that when fear is extinguished (as evidenced by a cessation in escape responding), the capacity to perform the escape response is maintained. Other experiments have also shown a similar dissociation of fear extinction and the extinction of the avoidance response (Miller, Mineka, & Cook, 1982; Mineka & Gino, 1979, 1980).

The implication of this is that even if fear is extinguished through flooding or implosive therapy, any event that causes fear to recur could result in a return of symptomatic escape behavior and continued maintenance of fear. Hence, therapy should not only involve the extinction of fear and consideration of the conditions in which fear might recur (see context specific extinction) but also consider the elimination of symptomatic escape behaviors, perhaps by replacing them with more appropriate behaviors (McAllister & McAllister, 1995). Therefore, if fear were to recur as a result of a change in context (i.e., renewal) and the symptomatic escape behavior not occur because it was extinguished or replaced with more appropriate behavior, the individual would experience a fuller exposure to the new context, thus providing a better opportunity for learning that this context was also safe. As Levis has stressed, central to behavior therapy is the need to repeatedly expose the patient not only to "CS patterns directly correlated with symptom onset but also to cues reactivated by the exposure procedure and those *hypothesized to be responses for symptom development*" [emphasis added] (1985, p. 67).

Conditions Under Which Extinction Might Not Occur

Levis (1985) assumes that extinction produced by behavior therapies will be directly related to the level of response that is generated during presentation of the CS pattern. This is consistent with animal work showing that the amount of extinction is a function of the initial level of conditioning. However, conditions in which an excessive response occurs to a CS may be detrimental to extinction. For example, animal research has shown that intermittent presentation of the US during nonreinforcement of the CS retards extinction (Ayres & Decosta, 1971; Frey & Butler, 1977; Rescorla & Skucy, 1969). This is understandable because US presentations can serve as additional conditioning trials or as "reminder" USs (see reinstatement). Recall that Konorski (1948) argued that extinction occurred when the CS was presented at the time of a fall in activation of the US center. The fall in activation occurred only when the CS was presented in the *absence* of the US. Presentation of the US precludes a fall in activation of the US center and prevents extinction.

It is important to realize that there is no true distinction between a stimulus that can serve as a CS and a stimulus that can serve as a US. The distinction is based solely on the response that is elicited by the stimulus. A conditioned stimulus is initially neutral and does not elicit the CR to be measured. A US, on the other hand, elicits this or a similar response. However, once conditioned, a potent CS can function as a US to produce conditioning to a new CS (Rescorla, 1973, 1980). This is referred to as second-order conditioning and is generally weaker than primary conditioning, owing to the fact that the first-order CS is a weaker reinforcer than the original US. However, if the first-order CS is very potent (e.g., fear conditioned to a stimulus coincident with a traumatic experience), this stimulus could act like a US. This potent CS would resist extinction. In terms of Konorski's (1948) theory, if a CS has become so well trained that it activates the US center like the original US, CS-alone extinction trials *would not* accompany a fall in activation of the US center, and extinction would not occur.

This idea may be more easily understood by considering extinction in terms of relaxation (McAllister & McAllister, 1995). According to this theory, extinction of fear occurs as a result of the accrual of a competing response of relaxation. Relaxation occurs when fear is reduced, typically, when the CS is presented and nonreinforced. If the US were to occur, fear would not be reduced and relaxation would not occur. Similarly, a potent CS might also not allow for relaxation and might not extinguish. But this also tells us that any event that reduces fear at or about the time the CS is presented can produce relaxation.

Fear reduction may be aided in several ways.

Anxiolytic Drugs

Anxiolytics would certainly reduce fear and permit relaxation. However, benzodiazepines

have been shown to produce state-dependent extinction (Bouton et al., 1990) in which learning in the presence of the drug does not transfer outside of the drug state (Overton, 1966). In addition, there is evidence to suggest that blockade of GABAergic mechanisms in the brain may facilitate extinction of fear (McGaugh, Castellano, & Brioni, 1990). Because benzodiazepines facilitate GABAergic systems, they may interfere with the physiological mechanisms of extinction. Therapists may want to avoid benzodiazepines as adjuncts to exposure therapy.

Safety Signals

Another way to reduce fear is by presenting a safety signal. Recall that a stimulus trained as part of a feature negative discrimination acquires the ability to inhibit fear. Presentation of a safety signal together with a potent CS should reduce fear to the CS and augment extinction. Hawk and Riccio (1977) evaluated this and found that presentation of a safety signal during nonreinforcement hastened extinction of an avoidance response. Hence, extinction to a potent CS may be aided by the presentation of a safety signal.

Neutral Stimuli

In the Hawk and Riccio (1977) study, extinction was also hastened by presentation of a novel stimulus during nonreinforcement. Pavlov (1927) observed that novel stimuli could consistently reduce the effectiveness of the CS in eliciting the CR (Pavlov referred to this phenomenon as "external inhibition"). This phenomenon has also been labeled as distraction and generalization decrement. Baum and colleagues have evaluated the effect of presenting novel stimuli during extinction of an avoidance response in rats (Baum, 1987; Baum & Gordon, 1970; Baum, Pereira, & Leclerc, 1985). In one experiment, rats were trained to escape shock by jumping from an electrified grid floor of a box to a ledge located above the floor. After attaining an escape criterion, the rats were given extinction to the apparatus by placing them onto the unelectrified grid floor with the ledge retracted. In an experimental group, a novel continuous background noise was presented during extinction and in a control group, the noise was absent. Testing occurred in the absence of the noise and involved placing the rat onto the unelectrified grid floor and recording the number of escape responses onto the ledge. Rats given extinction in the presence of the novel noise showed fewer escape responses than rats not given the noise. Hence, the novel noise hastened extinction to the apparatus cues. Similar results were also reported by Baum and Gordon (1970) and Hawk and Riccio (1977).

Despite the fact that a novel stimulus should reduce fear and allow relaxation, according to some influential theories of learning, a novel stimulus should not hasten extinction but in fact should retard extinction (Kamin, 1969; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1980; Wagner & Rescorla, 1972). And considering the influence of context on extinction, one might expect that a neutral stimulus would become part of the extinction context. If so, extinction would not transfer outside of the presence of the neutral stimulus and might be present only in the presence of that stimulus (Brooks & Bouton, 1994). This is akin to the phenomenon of negative occasion setting discussed earlier (Bouton, 1991; Holland, 1985). Whether facilitated extinction, protection from extinction, or negative occasion setting occurs may depend on experiment parameters. More work is needed to determine the conditions under which novel stimuli may or may not hasten extinction and whether this procedure would have to be qualified by the potential for contextual effects.

Distraction

In many respects, presentation of a novel stimulus is like distracting the subject away from the fear-eliciting stimulus. Because it is difficult to define *distraction*, the term is rarely used in the animal literature. However, there have been a few studies in humans that have evaluated the effect of distraction on desensitization therapy and are therefore worth discussing in this context. (A review of this literature is beyond the scope of the present discussion, but the reader may wish to consider a recent review by Rodriguez and

Craske [1993].) Distraction from the fear-eliciting stimulus should have similar effects as presenting a novel stimulus: Fear would be reduced, allowing relaxation. Studies have shown that high levels of fear may interfere with in vivo desensitization to phobic stimuli (Borkovec & Sides, 1979; Foa & Kozak, 1986). However, the evidence is mixed as to whether distraction during exposure to the phobic stimulus can reduce fear and whether this has any effect on the long-term success of exposure therapy (i.e., extinction). As reviewed by Rodriguez and Craske (1993), the mixed results may have to do with the level of distraction and how distraction is defined. In most of these experiments, distraction involved engaging in some task while undergoing exposure, such as playing a video game or listening for target words. These tasks might produce so much distraction that the CS itself is not attended to. Herein lies the difficulty in evaluating these experiments with respect to fear reduction and relaxation. In the limit, extinction will not occur if the CS is not presented (recall Konorski, 1948). So, if the individual is fully distracted, extinction will not occur. The goal should be to partially distract the subject, reducing fear but still allowing the CS to be processed. It is interesting to speculate that the apparent therapeutic advantage of eye movement desensitization and reprocessing, EMDR (Shapiro, Vogelmann-Sine, & Sine, 1994), may be that it involves a level of distraction sufficient to produce fear reduction but without causing the subject not to attend to the CS. (See Acierno et al., 1994, and Greenwald, 1994, for reviews of EMDR.) Nevertheless, to evaluate the effect of fear reduction and distraction on desensitization, it would seem better to follow the animal experiments of Baum and colleagues (Baum, 1987; Baum & Gordon, 1970; Baum et al., 1985) and present a novel "distracting background stimulus" during exposure rather than have the subject *actively* engaging in some distracting task (cf. Singh, 1976).

In summary, an excessive amount of fear to a CS can prevent or severely retard extinction. To the extent that the potent CS behaves like a US, extinction may occur only if the exaggerated fear elicited by the potent CS is somehow reduced. The animal literature suggests that presentation of a safety signal or a neutral stimulus can hasten extinction. However, the effectiveness of these procedures as therapeutic tools has yet to be evaluated.

SUMMARY

Pavlov (1927) believed that extinction was as important to the organism as the original learning itself. Extinction provided the means to correct learned behavior to meet the organism's current circumstance, a circumstance in which the CS is no longer followed by the US. The procedural definition of extinction—presentation of the CS in the absence of the US—suggests that extinction is a simple procedure that is easily carried out. However, executing the procedure does not guarantee extinction. For example, failure to present the entire CS can retard or prevent extinction and make the memory more susceptible to reinstatement. Similarly, even though the US may be physically absent, a potent CS may act like a US and prevent extinction. And although a CS may no longer elicit a conditioned response following extinction, it is clear that many, if not most, of the original CS-US associations still exist. These associations can return with the passage of time, following presentation of a US, or when testing takes place in a context different from the one used in extinction.

The mechanistic definition of extinction remains to be written. Konorski (1948) believed that extinction was the result of the accrual of inhibition that competed with the excitatory associations for activation of the US center. However, it has been very difficult to demonstrate experimentally that the CS acquires direct inhibitory associations with the US. A more contemporary view is that extinction results from a discrimination between occasions in which the CS is and is not followed by the US center. In this scheme, some aspect of the experimental situation, such as the experiment context, serves as an occasion setter or trigger to signal whether

the CS will or will not be followed by the US. The exact nature of this modulation is unclear.

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LEARNING AND BEHAVIOR THERAPY

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