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Associative Accounts of Recovery-from-Extinction Effects

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Abstract

Recovery-from-extinction effects (e.g., spontaneous recovery, renewal, reinstatement, and facilitated reacquisition) have become the focus of much research in recent years. However, despite a great deal of empirical data, there are few theoretical explanations for these effects. This paucity poses a severe limitation on our understanding of these behavioral effects, impedes advances in uncovering neural mechanisms of response recovery, and reduces our potential to prevent relapse after exposure therapy. Towards correcting this oversight, this review takes prominent models of associative learning that have been used in the past and continue to be used today to explain Pavlovian conditioning and extinction, and assesses how each model can be applied to account for recovery-from-extinction effects. The models include the Rescorla-Wagner (1972) model, Mackintosh's (1975) attentional model, Pearce and Hall's (1980) attentional model, Wagner's (1981) SOP model, Pearce's (1987) configural model, McLaren and Mackintosh's (2002) elemental model, and Stout and Miller's (2007) SOCR (comparator hypothesis) model. Each model is assessed for how well it explains or does not explain the various recovery-from-extinction phenomena. We offer some suggestions for how the models might be modified to account for these effects in those instances in which they initially fail.

Keywords

Extinction; Recovery from extinction; Spontaneous recovery; Renewal; Reinstatement; Associative theories

Extinction and Recovery from Extinction

Pavlovian conditioning describes the process of systematically pairing a neutral stimulus (e.g., tone) with a biologically significant stimulus such as a footshock (i.e., the unconditioned stimulus; US). Typically, this results in the neutral stimulus becoming a conditioned stimulus (CS) that asserts behavioral control, such that presentation of this stimulus evokes a conditioned response (CR) which is mimetic or compensatory of the reaction produced by the US. Following sufficient CS-US pairings, the probability of a subject's responding upon presentation of the CS is high and the strength of the CR is strong. But both CR probability and magnitude can be weakened by subsequently repeatedly presenting the CS without reinforcement (e.g., tone-footshock pairings followed by tone-no

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footshock). The CS-alone presentations constitute operational extinction (i.e., extinction treatment), and the response decrement is called behavioral extinction (Pavlov, 1927). If a sufficient number of extinction trials are given, the CR may completely disappear.

Over the years, many theories have been developed to try to explain Pavlovian conditioning and extinction. Most major theories of learning agree that conditioned responding is supported by an association(s) between mental representations of the CS and the US. With regards to extinction, theories are split into two general categories, those that assume the excitatory association is degraded (e.g., Mackintosh, 1975; Rescorla & Wagner, 1972), and those that assume that a second association is established between the CS and the US representations that is inhibitory in nature (e.g., Wagner, 1981); some theories alternatively frame the second association as an excitatory one between the CS and a no-US representation (e.g., Pearce & Hall, 1980). The general inhibitory consequences of these two variations concerning the second association are the same, at least for the purposes of this review. The inhibitory association is assumed to summate with the excitatory association resulting in a net reduction in responding. The view that extinction results in new inhibitory learning is supported by studies showing brain areas believed to underlie inhibition being activated during extinction and behavioral observations of recovery from extinction (e.g., Quirk, Garcia, & González-Lima, 2006).

Recovery from extinction refers to the re-emergence of excitatory responding to the CS despite a reduction or even elimination of the CR by the end of the extinction procedure. Recovery-from-extinction effects include spontaneous recovery, renewal, reinstatement, and facilitated reacquisition (among others). In each of these effects, the subject reverts to expressing the initial acquisition information after extinction treatment given appropriate circumstances at test (e.g., Quirk, 2002). Briefly, spontaneous recovery refers to an increase in responding to an extinguished CS following a long retention interval after extinction during which the CS received no additional training (Pavlov, 1927). Renewal refers to an increase in responding to an extinguished CS as a result of testing taking place outside of the context in which extinction treatment occurred. There are three basic types of renewal designs, ABA, ABC, and AAC (a.k.a. AAB), which are designated by letters representing the contexts of acquisition, extinction, and testing, respectively (e.g., Bouton & Bolles, 1979a). Reinstatement refers to an increase in responding to an extinguished CS as a result of presentation of the US alone (Rescorla & Heth, 1975), and facilitated reacquisition refers to rapid reacquisition of behavioral control when an extinguished CS is again reinforced (Napier, Macrae, & Kehoe, 1992). These effects are described in more detail below. Collectively, they support the view that information acquired during initial acquisition is not destroyed or unlearned as a result of extinction treatment. However, recovery from extinction is generally not complete, which suggests that some limited attenuation of the initial excitatory CS-US association may have occurred (e.g., Thomas & Ayres, 2004).

Notably, recovery-from-extinction effects are only observed following a reduction in behavioral control, for example, extinction and the overexpectation effect (i.e., the decrement in responding to a conditioned excitor after reinforcing it in compound with another conditioned excitor; Rescorla, 2006b), relative to otherwise equivalent simple reinforcement. That is, only stimuli that have suffered an impairment in behavioral control will show an increment in behavioral control with the passage of time (i.e., spontaneous recovery; e.g., Rescorla, 2007 [magazine approach preparation with rats]), a change in context (i.e., renewal; e.g., Rosas & Callejas-Aguilera, 2007 [conditioned taste aversion preparation with rats]), or the presentation of the US in the test context (i.e., reinstatement; e.g., Bouton, 1984; Bouton & Bolles, 1979b; Rescorla & Heth, 1975 [all using conditioned suppression preparation with rats]). Stimuli that have not experienced attenuation in

behavioral control relative to equivalent simple acquisition will not show enhanced responding following any of these manipulations.

Theories of associative learning have long guided the study of extinction. This research is particularly important because experimental extinction is commonly used as a model for exposure therapy for treating anxiety disorders, and just as there are observations of recovery from extinction in the laboratory, so too are there observations of relapse after exposure therapy (e.g., Hofmann, 2008; Laborda, McConnell, & Miller, 2011). Our understanding of the theoretical mechanisms of extinction itself has become extensive and complex, and these theories are now being tested with neuroscientific data. However, there is considerably less known about the theoretical and neurological mechanisms responsible for recovery-from-extinction effects. It is clear that return of fear occurs readily in both laboratory and clinical settings, but why and how these effects occur is less apparent.

Schmajuk, Lam, and Gray's Neural Network Model

To date, only a couple theories have attempted to provide a comprehensive account of extinction and recovery-from-extinction effects. We discuss these two theories separately from the other models, which have not been systematically applied to account for recovery effects, and hence their accounts and shortcomings will be addressed as the various recovery phenomena are introduced. Larrauri and Schmajuk (2008) offered an explanation for recovery effects based on the Schmajuk, Lam, and Gray (SLG; 1996) model. In brief, the SLG model incorporates multiple processes that operate in parallel to form an internal representation of the stimulus that is modulated by attention to novelty. CSs that are attended to are assumed to activate their internal representations upon presentation and are used to predict the occurrence of other events. Selective attention to stimuli modulates not only the retrieval of associations, but their later retrieval. Extinction is the result of both a decrease in the CS-US association and strengthening of an inhibitory context-US association. If the context is sufficiently salient, then the inhibitory context-US association will prevent the CS-US association from decreasing completely to zero strength. During extinction and a retention interval, attention to the CS, the context, and the context-CS association is posited to decrease, thereby resulting in increased novelty when the CS is presented again. This novelty should trigger an increase in attention to the CS, which should lead to spontaneous recovery. CS-extinction context associations that are inhibitory in nature that are formed during extinction training are assumed to be responsible for renewal, such that testing outside of the extinction context should result in a recovery of responding. The SLG model assumes that contextual conditioning is also responsible for reinstatement, such that presenting the US in the reinstatement context should produce an excitatory context-US association, which drives responding, an account that is consistent with reinstatement being context specific (e.g., Bouton, 1984). Facilitated reacquisition reflects residual excitatory strength between the CS and US; however, if attention to the CS is reduced too much during extinction, then reacquisition will be retarded.

Although the SLG (Schmajuk et al., 1996) model provides a fairly comprehensive explanation of extinction and recovery from extinction, the complexity of the model (e.g., three simultaneously operating error correction processes), the need to add special assumptions to the model to explain many different phenomena, and the exceptionally large number of free parameters that the model contains (11 plus 1 more for each stimulus) seems to have discouraged researchers from widely using it to account for extinction or recovery-from-extinction effects.

Bouton's Retrieval Theory

Bouton's (1993; also see 1994; 2006) theory of retrieval is the most widely cited account of recovery from extinction. However, it is important to note that this theory does not speak to how associations are learned or most other traditional Pavlovian conditioning phenomena such as cue competition (e.g., blocking, overshadowing, etc.); thus, it is not a comprehensive theory of learning nor did Bouton ever mean it to be one. It was designed specifically to account for associative outcome interference phenomena (i.e., situations in which a cue has been paired with two different outcomes in separate phases of treatment), such as extinction, latent inhibition, and counterconditioning.

According to Bouton's (1993) theory, when a CS is ambiguous (e.g., it has a history of reinforcement followed by extinction), subjects use the context in which the ambiguity arose (i.e., the extinction context) as a signal for when the extinction contingency is in force (Bouton, 1994). This results in the context of extinction functioning as an occasion setter (Holland, 1983; Miller & Oberling, 1998), which modulates the expression of an association between the target CS and nonreinforcement (Bouton & Swartzentruber, 1986). Whether the association to reinforcement or nonreinforcement is expressed on a later test depends on the relative similarity between the test context and the extinction context. The more similar the test context is to the extinction context, the greater the likelihood that the inhibitory-like CS-nonreinforcement association will be expressed (i.e., weak responding to the CS). When the test context differs appreciably from the extinction context, the excitatory CS-US association will be expressed. Thus, in this theory, high similarity between the extinction and test contexts is critical for retrieving the memory of extinction.

Bouton's definition of a context is broad, and it has been expanded to include not only conventional contextual stimuli (that is, stimuli diffusely distributed in space; Bouton & Bolles, 1979a), but also time of occurrence (Bouton & Brooks, 1993), characteristics of a particular situation such as reinforcement schedules (Winterbauer & Bouton, 2010) and intertrial intervals (Bouton & García-Guiérrez, 2006), and internal drug-induced states (Bouton, Kenney, & Rosengard, 1990). As such, Bouton's (1993) theory is able to account for a wide range of recovery-from-extinction effects, including spontaneous recovery, renewal, reinstatement, and facilitated reacquisition, using the same contextual-modulation mechanism. The view that the various recovery-from-extinction effects rely on a common process is supported by many studies showing similarities between these effects. For example, across all of these phenomena more extinction trials appear to reduce recovery from extinction (e.g., Denniston et al., 2003 [reduced renewal in conditioned suppression preparation with rats]; García-Gutiérrez et al., 2005 [reduced reinstatement in predictive learning preparation with humans]; Leung et al., 2007 [reduced facilitated reacquisition in freezing preparation in rats]; Rosas & Bouton, 1996 [reduced spontaneous recovery in conditioned taste aversion preparation with rats]).

Although Bouton's (1993) theory does provide a data-supported, parsimonious, and simple foundation for understanding most recovery effects, there are reasons to believe that it is not sufficient to tell the whole story. For example, this theory predicts that so long as testing occurs outside of the extinction context, recovery of responding should be equal across all three forms of renewal; minimally, ABC and AAC renewal should be equal because, in both designs, the subject is tested in a neutral environment. That is, this theory assumes that the degree of similarity between the extinction and test contexts, which should be matched between AAC and ABC paradigms, modulates renewal. However, this is not empirically supported. AAC renewal is much more difficult to obtain, and, when it is observed, the size of the recovery is generally far smaller than what is observed in ABC renewal (e.g., Laborda et al., 2011; Thomas, Larsen, & Ayres, 2003; Üngör, & Lachnit, 2008). Moreover, as we previously noted, Bouton's model is not a complete theory of learning, and additionally it

has not been mathematically formalized thereby limiting it to only being able to make qualitative predictions. In addition, some questions have arisen challenging the conceptual validity of Bouton's theory regarding time as a modulating context (e.g., Riccio, Richardson, & Ebner, 1984, 1999; but see replies by Bouton, Nelson, & Rosas, 1999a, 199b), and there is some doubt as to whether the same process underlies all recovery effects (e.g., García-Gutiérrez & Rosas, 2003b). Thus, while acknowledging the considerable success of Bouton's account, we suggest that there are other mechanisms at work, either in place of or more likely in addition to Bouton's occasion setting mechanism.

Unfortunately, there is a dearth of comprehensive theoretical explanations for recovery-from-extinction effects. As stated above, Bouton's (1993) theory provides a framework for understanding many recovery effects, but it does not (and was never intended to) provide an account of how learning occurs, and surely some new learning happens during extinction treatment. To fully understand recovery-from-extinction effects, we need to understand the extent and the processes by which inhibitory learning concerning the extinction context and unlearning influence recovery in addition to the influence of occasion setting by the extinction context. This explanatory deficiency stands in contrast with the large number of theoretical explanations for learning and basic extinction. Indeed, there are many models of associative learning that are frequently used to interpret data about conditioning and extinction, but these models are rarely considered when trying to explain recovery-from-extinction effects. We believe that this presents a serious gap in our knowledge of how and why recovery from extinction occurs, and it poses a general limitation of our understanding about behavior plasticity as a whole. Thus, it is the purpose of this review to apply some of what we consider to be the most prominent models of associative learning to explaining recovery-from-extinction effects. Moreover, it is important for the sake of theoretical development to assess how well various learning theories can account for recovery effects because an adequate theory should be able to be applied to new situations. Theories that are restricted only to the phenomena that they were originally intended to explain have limited heuristic value, and we think it worthwhile to determine how well traditional learning theories can adequately explain some, if not all, recovery effects. Lastly, as noted by a reviewer of a prior version of this manuscript, Bouton's theory was developed “precisely because response restoration phenomena were not explained by existing comprehensive theories.” While this failure of traditional associative learning theories may have seemed true at the time, we believe that with a few further assumptions, several of these learning theories can account for recovery effects without using an occasion setting mechanism. Therefore, in the remainder of this review, we assess how well these learning models can account for recovery-from-extinction effects, which lay outside of their original explanatory domain. We emphasize and repeat that it is not our intention to displace Bouton's theory. Rather, we are investigating other theory-driven mechanisms that heretofore have not been duly considered due to a heavy reliance on Bouton's theory, but that might also influence the observation of recovery from extinction. In doing so, we hope to spur more research into these learning-based mechanisms that will further our understanding of why recovery from extinction occurs.

Explaining Recovery-from-Extinction Effects with Associative Learning Models

We have chosen to focus on seven models that we believe are representative and frequently cited models of basic associative learning. These include the Rescorla-Wagner (1972) model, Mackintosh's (1975) attentional model, Pearce and Hall's (1980) attentional model, Wagner's (1981) SOP model, Pearce's (1987) configural model, McLaren and Mackintosh's (2002) elemental model (see also McLaren, Kaye, & Mackintosh, 1989; McLaren & Mackintosh, 2000), and Stout and Miller's (2007) SOCR model (see also Denniston,

Savastano, & Miller, 2001; Miller & Matzel, 1988). Clearly, there are many more models of associative learning that could have been included in this review, but in the interest of brevity, we chose to focus only on these seven, which have a long history of guiding research, a solid basis of empirical support, and have made significant contributions in the past and present to understanding learning and behavior. The approach that each of these models uses to explain learning and performance in general has already been described in great detail elsewhere. For comprehensive introductions to each model, readers should refer to the original papers. Although these models have been used extensively over the years to guide research and interpret data, it is noteworthy that these models have rarely been used to account for recovery-from-extinction phenomena. Here we try to fill this gap. We tried to apply the models as best as possible in their originally conceived forms. In instances in which the models proved insufficient, we offer some suggestions that could be incorporated into the models to aid them in accounting for the effect in question. We tried to keep our suggestions as simple and minimal as possible so as to not deviate from the basic principles of the model. The reader should keep in mind that these suggestions have not been systematically tested and certainly more thought is needed regarding their viability if one is to seriously incorporate them into the model. However, we believe that at first glance our suggestions provide a reasonable potential solution to the various problems the models encounter in trying to account for the recovery-from-extinction effects.

Spontaneous recovery

Spontaneous recovery from extinction was first documented by Pavlov (1927). He observed that responding recovered to almost full strength after a relatively long period of time following extinction during which no additional training occurred. This was observed even when responding to a CS had completely ceased at the end of extinction treatment. Bouton's (1993) theory has proven capable of accounting for many of the findings related to spontaneous recovery. The theory assumes both physical and temporal aspects of the situation can define the context. Therefore, imposing a long retention interval between extinction and testing results in the subject at test being in a new temporal context different from the temporal context of extinction, which should interfere with retrieval of the CS-nonreinforcment memory that is acquired during extinction treatment. Consequently, recovery of responding should be observed. However, Riccio et al. (1984) have argued against this interpretation of spontaneous recovery based on findings that, after a retention interval, conditioned responding is less stimulus specific. They reasoned that subtle changes in the contextual stimuli induced by the passage of time should be insufficient to produce retrieval failure given that much more salient changes in the stimuli fail to produce such an effect. The debate between Riccio et al. (see also Riccio et al., 1999) and Bouton and his colleagues (Bouton et al., 1999a, 199b) is still unresolved, but we believe that the questions raised are sufficient to justify consideration of alternative explanations of spontaneous recovery.

In their present forms, none of the traditional learning models can account for spontaneous recovery. However, with relatively minor modifications, models of extinction that assume an inhibitory CS-US association is formed during extinction, such as SOP (Wagner, 1981), Pearce and Hall's (1980) model, Pearce's (1987) model, and McLaren and Mackintosh's (2002) model, can do so. These models assume that weak responding at the end of extinction is the result of learning during extinction an inhibitory CS-US association that negatively summates with, but does not degrade, the previously acquired excitatory CS-US association. Therefore, if one assumes that inhibitory associations are more vulnerable to decay over time than are excitatory associations, spontaneous recovery from extinction would be expected. One possibility is that inhibitory associations, but not excitatory associations, decay over long retention intervals, or at least that inhibitory associations decay faster than

excitatory ones. The McLaren and Mackintosh model offers a framework in which this might occur. In this model, associations are formed between the microelements of a stimulus to form a unitized (i.e., integrated) representation of the CS and then this unitized representation becomes associated with the outcome. When a CS is presented, connections formed between concurrently activated elements are strengthened. However, a decay function is also implemented such that the connections are simultaneously decaying as they are being incremented until the connections have decayed to some fixed proportion of their original strength. In other words, during reinforced training, associations between stimuli are strengthened by reinforcement, but at the same time they are being degraded due to associative decay until a set point in which decay stops. At that point, the association is said to be stabilized and contributes to the long-term response potential and associatively integrated representation of the CS. We suggest that a decay function can also operate on the associations between the unitized representation of the CS and the US such that inhibitory associations have a lower fixed point of decay than excitatory associations. Soon after extinction, the association between the CS and US may be composed of inhibitory connections that have just been strengthened and excitatory connections that have partially decayed. However, after a long retention interval, all of the associations should have decayed to their fixed point, at which time the excitatory connections will be stronger than the inhibitory connections. Thus, excitatory responding should be observed.

Differential decay rates are consistent with a proposal by Pavlov (1927) that inhibitory associations are more labile than excitatory associations (for supportive data, see Rescorla, 2005; but also see Sissons & Miller, 2009). Assuming that the rate of decay decreases as the retention interval increases, this view would also explain why, all other things being equal, spontaneous recovery increases as the interval between extinction and testing increases (e.g., Kraemer & Spear, 1992). Moreover, on a simply qualitative basis, this process would explain some of the data from Johnson, Escobar, and Kimble (2010) who compared the effects of a long or short delay between acquisition and extinction and between extinction and testing against a no-extinction control. They observed strong spontaneous recovery when acquisition, extinction, and testing all proceeded relatively soon after the other. However, behavior consistent with extinction treatment (i.e., less spontaneous recovery) was seen when extinction training was delayed, at least when there was a relatively long extinction-test interval. According to a differential decay interpretation, this occurs because a long delay between acquisition and testing allows the excitatory association to at least partially decay, whereas a shorter interval between extinction and testing can be insufficient for the inhibitory association to weaken to a similar degree. However, if both intervals are short, the differential rates of decay should result in the excitatory association being stronger than the inhibitory association, at least under certain parameters. A differential rates of decay approach is a challenged, though, to explain why delayed extinction results in more spontaneous recovery than immediate extinction (both compared to a no-extinction control) when a long retention interval is imposed between extinction and testing. Presumably, the long retention interval should allow both the excitatory and inhibitory associations each to decay toward their asymptotic values, with the inhibitory association decaying more than the excitatory association. All other things being equal, immediate or delayed extinction should not greatly affect the strength of the excitatory association, and if anything, immediate extinction should result in stronger recovery because the excitatory association has less time to decay. However, Bouton's (1993) occasion setting theory also has trouble explaining all of Johnson et al.'s results. In the framework of this theory, conducting acquisition, extinction, and testing all in close temporal proximity to each other should produce an AAA-like design relative to when extinction is delayed, which should create more of an ABB-like design. Johnson et al. observed stronger spontaneous recovery in the AAA-like group than the ABB-like group, which is not consistent with Bouton's model of recovery. One could theorize that their 48-hour retention interval between extinction and testing actually did

induce a temporal context shift such that the immediate extinction and immediate testing group was more representative of an AAC form of renewal rather than an AAA design, but this would necessitate regarding the ABB-like group as an ABC-like renewal group, which should result in strong recovery, and this was not observed. Bouton's theory is also challenged to explain why immediate extinction and delayed testing (i.e., a 7-day retention interval) did not produce strong recovery because this design is akin to an AAC renewal procedure. But, the theory does correctly predict strong spontaneous recovery in the group that received delayed extinction and delayed testing (i.e., ABC-like renewal). Thus, Johnson et al.'s data seem to pose challenges to both a differential decay explanation and an occasion setting explanation of spontaneous recovery. Such difficulties support our assertion that there are multiple processes that underlie recovery, and that we should not contain ourselves to focus on only one mechanism.

Because those models that assume extinction establishes CS-nonreinforcment or inhibitory CS-US associations (e.g., McLaren & Mackintosh, 2002; Pearce, 1987; Pearce & Hall, 1980; Wagner, 1981) do postulate that excitatory associations are not erased during extinction, they are [relatively] readily compatible with various recovery effects, such as a long retention interval unmasking the excitatory association, which is preserved throughout extinction treatment. It is important to note that the assumption that inhibition decays faster or more than excitation is currently not part of any of these models and its addition is post-hoc to describe the observed behavior, but we believe that this idea is worth investigating. Perhaps one way to do this would be to compare responding after a long retention interval (i.e., spontaneous recovery) between a group that receives 50% reinforcement of a CS's presentations versus a group that receives only simple acquisition (same number of trials as the partial reinforcement group). If inhibition decays faster than excitation, then the difference in responding between these two groups should grow smaller as the retention interval is increased. Of course, additional control groups and experiments would likely be necessary before any firm conclusions could be made.

If the retention interval is spent in the experimental context, then models capable of accounting for retrospective revaluation (Dickinson & Burke, 1996; Stout & Miller, 2007; and Van Hamme & Wasserman, 1994) can explain spontaneous recovery as a consequence of retrospective revaluation mediated by the context. Retrospective revaluation refers to situations in which responding to a target cue changes as a function of posttraining associative manipulations of an associate of the target cue (e.g., responding to an overshadowed cue increases if the overshadowing cue is subsequently extinguished [e.g., Kaufman & Bolles, 1981; Matzel, Schachtman, & Miller, 1985; Shanks, 1985]). In the framework of Van Hamme and Wasserman's (1994) and Dickinson and Burke's (1996) retrospective revaluation models, stronger responding to the target CS could occur because the CS-US association is strengthened during the retention interval by extensive exposure to contextual cues that have associations to both the CS and US. In the framework of the comparator hypothesis (Stout & Miller, 2007), responding to the target cue soon after extinction was weak because of a strong indirectly activated representation of the US mediated by a CS-context association (Link 2; see Figure 1) which was strengthened during extinction of the CS and only a limited decrement of the CS-US association (Link 1). Extensive context exposure over a long post-extinction retention interval should weaken the comparator process due to attenuation of the effective context-US association (Link 3), resulting in recovery of responding to the target cue. Problematic for a retrospective revaluation account is that nearly all demonstrations of spontaneous recovery remove the subject from the context during the retention interval. A retrospective revaluation account could still be applied if one assumes that the experimental context and home cage where the retention interval is spent share appreciable common elements that have associations with the US and the CS (e.g., Killcross, Kiernan, Dwyer, & Westbrook, 1998). However, such

associations are likely to be weak if they exist at all, and retrospective revaluation generally depends on strong within-compound associations (Witnauer & Miller, 2010, but see Vadillo, Castro, Matute, & Wasserman, 2008). But these models may still be able to account for spontaneous recovery if the CS-US association is compared to a more general and global context, which includes the time spent outside of the experimental context.

A more specific modification we suggest that would allow the SOCR model (Stout & Miller, 2007) to account for spontaneous recovery is to add a uniform decay rate for all links. Note that the most recent version of the comparator model (Stout & Miller) does allow for decreases of the CS-outcome association during extinction treatment as one mechanism that contributes to empirical extinction. This would make the indirectly activated US representation less durable than the directly activated US representation because the indirectly activated US representation depends on the product of two independent associations (Links 2 and 3), whereas the directly activated US representation depends on only one (i.e., Link 1). Thus, if Links 1, 2, and 3 decayed at the same rate, then the strength of the indirectly activated US representation should wane more rapidly over time than the directly activated US representation. This suggested addition would allow the comparator model to account for spontaneous recovery in much the same way as the models that assume extinction establishes an inhibitory CS-US association, but another parameter would have to be added to reflect the rate of decay, just as do the inhibitory models.

The Rescorla-Wagner (1972) model and Mackintosh's (1975) model are greatly challenged in trying to explain recovery from extinction. This is because they assume that weak responding at the end of extinction is the result of the actual CS-US association being degraded (i.e., unlearned), whereas recovery effects, particularly spontaneous recovery, demonstrate that the original excitatory association was not destroyed, at least not completely. Even if one were to assume that the CS retained residual excitatory associative strength at the end of extinction, these models are still unable to explain why it should increase after a period of time during which no training occurred. Mackintosh's model assumes that attention (akin to associability in this model) to a CS modulates learning, such that attention increases to CSs that are good predictors of the outcome. As such, it separates associative strength and attention. Formally, the model assumes that responding is determined solely by the sum of the associative strengths of all stimuli present on a test trial. However, Mackintosh entertained the possibility that a stimulus may retain high associative strength with a US, but if it suffers a decline in attention, then expression of that associative strength would decrease. This speculation was not incorporated into the formal model, but Mackintosh clearly left open the possibility that attention may influence behavior at test as well as new learning during training. We could further speculate that during extinction attention to the CS decreases rapidly, but the associative value of the CS does not diminish greatly. Weak or no responding at the end of extinction treatment could principally reflect reduced attention to the CS. To account for spontaneous recovery, one would have to make the further assumption that attention increases due to the passage of time, allowing responding to recover and reflect the still-present associative strength. One can speculate that attention increases because the passage of time causes the stimulus to be perceived as more novel again.

Renewal

Renewal refers to an increase in responding to an extinguished CS when it is tested outside of the extinction context relative to when it is tested in the extinction context. As previously stated, there are three different forms of renewal based on the contexts of acquisition, extinction, and testing. ABA renewal (Bouton & Bolles, 1979a) refers to when the subject is trained in one context, extinguished in another, and then tested back in the acquisition context. ABC renewal (Bouton & Bolles, 1979a) refers to when acquisition, extinction, and

testing all occur in different contexts. Lastly, AAC (a.k.a. AAB) renewal (Bouton & Ricker, 1994) refers to when both acquisition and extinction are conducted in one context and testing occurs in a second. Although Bouton's (1993) model has a great deal of explanatory power, it erroneously anticipates equal degrees of renewal provided testing occurs outside the extinction context. At the very least, it predicts equal recovery in ABC and AAC designs because both involve testing in a completely neutral context. However, the observed differences in recovery across the renewal designs, particularly the data showing that ABA and ABC renewal are far more robust than AAC renewal (e.g., Laborda, Witnauer, & Miller, 2011 [conditioned suppression preparation with rats]; Thomas et al., 2003 [conditioned suppression preparation with rats]; Üngör & Lachnit, 2008 [predictive learning preparation with humans]), and the often observed greater recovery observed in ABA renewal than ABC renewal (e.g., Effting & Kindt, 2007 [fear conditioning preparation with humans]; Thomas et al., 2003 [conditioned suppression preparation with rats]), are evidence that there are other mechanisms at work to produce renewal instead of or in addition to occasion setting by the test context.

One such possible mechanism is that of contextual conditioning (i.e., inhibitory and excitatory context-US associations) and associative summation of these associations with those of the CS at test. This approach would be consistent with the frameworks of Mackintosh's (1975) model, Pearce and Hall's (1980) model, the Rescorla-Wagner (1972) model, and Wagner's (1981) SOP model. In general, the contextual conditioning approach to renewal assumes that the acquisition context acquires some excitatory associative strength, which summates with the excitatory associative strength of the CS on subsequent test trials in the acquisition context (ABA renewal). The extinction context (assuming it was associatively neutral prior to extinction) acquires inhibitory associative strength, which negatively summates with the excitatory strength of the CS on subsequent test trials in the extinction context (i.e., the ABB control condition for ABA and ABC renewal). Not only should the extinction context become inhibitory in simple extinction situations thereby reducing subsequent responding to the CS in that context, but during extinction, the extinction context's inhibitory status should offer some protection to the CS against it losing excitatory associative strength (e.g., McConnell & Miller, 2010; Rescorla, 2003; Soltysik, Wolfe, Nicholas, Wilson, & Garcia-Sanchez, 1983). Therefore, the CS's associative strength from initial acquisition should be partially preserved during extinction thereby increasing responding to the CS outside of the extinction context. Importantly, the Mackintosh model does not predict that the extinction context will become inhibitory, and hence does not predict that the extinction context will protect the CS from unlearning because it uses a local error correction term (i.e., it only considers the associative strength of the stimulus in question, not the aggregate associative strength of all cues present on a given trial) in its rule for new learning (inhibitory learning in this case). However, this model can account for the CS's preserved excitation because it assumes that changes in associative strength are equal to attention (i.e., associability) times the local error correction term. If attention to the CS is low because it is a poor predictor of nonreinforcement early in extinction treatment, then even a large error term may have minimal impact on the overall change in associative strength of the CS. However, this account fails to address the context specificity of extinction (e.g., ABC renewal).

According to the contextual conditioning approach, when the CS is tested in the acquisition context (ABA renewal) or in a neutral context (ABC renewal), it is no longer in the presence of the conditioned inhibitor (i.e., the extinction context), which should allow the combined excitatory associative strength of the context and the residual strength of the CS to be expressed in ABA renewal, or just the residual excitatory associative strength of the CS to be expressed in ABC renewal. Thus, the contextual conditioning account can explain why ABC renewal is commonly observed to be weaker than ABA renewal, which is a

shortcoming of the Bouton (1993) model of renewal. AAC renewal is explained as the result of deepened extinction (Rescorla, 2006a) to both the target CS and Context A. That is, both the target CS and context should have some excitatory value due to both having been present during acquisition. But, the context should be less excitatory than the target due to its lower salience, and therefore its presence should not greatly deepen extinction. In the framework of Pearce and Hall (1980), Rescorla and Wagner (1972), and Wagner (1981), at some point, the A context in an AAC procedure may become somewhat inhibitory because it is co-present with the still excitatory CS during extinction trials on which the US is not present. Testing outside of this context should produce recovery (relative to an AAA control condition) because the inhibitory Context A is not present at test, but recovery should only be slight due to Context A's weak inhibitory status. Thus, the context conditioning approach also can explain why AAC renewal is generally weaker than ABA or ABC renewal, which is not accounted for by Bouton's model. Importantly, this explanation assumes that the context was of low salience during acquisition and its saliency increased during extinction, which is how it acquired inhibition. One could speculate that this is due to the new reinforcement contingency, which causes the animal to look to other causes of the new outcome than of the initial reinforcement.

Within the framework of SOCR (Stout & Miller, 2007), if one makes the added assumption that the comparator processes for the target cue and the context summate to determine responding (thus allowing the model to make predictions for testing stimuli in compound), then SOCR does anticipate the basic renewal effects based on contextual conditioning (i.e., excitatory acquisition context and inhibitory-like extinction context), as well as the different levels of recovery observed between ABA and ABC renewal. Specifically, according to SOCR, the target CS acquires behavioral control during acquisition due to a strong CS-US association (i.e., Link 1). The acquisition context, a comparator stimulus for the CS, should acquire a weaker association with the US during training (i.e., the target CS's Link 3) due to its lower salience. If extinction occurs in a different context, then the association between the target CS and the acquisition context should not be further strengthened, thereby limiting the acquisition context's potential to down-modulate responding to the target CS; but the association between the target CS and the extinction context (i.e., Link 2) should gain strength, which should cause the extinction context to exert an inhibitory-like influence on responding within that context. Thus, SOCR is able to provide an account of increased responding in ABA and ABC renewal relative to an ABB control, and if one accepts the assumption of associative summation, then it also predicts the difference in observed ABA and ABC renewal. If extinction is conducted in the same context as acquisition, then the acquisition/extinction context should exhibit a weak inhibitory-like influence on responding to the CS due to the within-compound association between the target cue and the acquisition context (i.e., Link 2) being strengthened during extinction treatment. This effect should be observed regardless of whether testing occurs in (AAA) or outside (AAC) the training context because comparator stimuli are presumably established during training, not testing. Thus, SOCR fails to account for AAC renewal (the weakest form of renewal) without a further assumption. One such assumption is summation between the CS's residual excitatory value and the [weak] inhibitory value of the A context. For an alternative assumption to account for AAC renewal in a comparator framework, see Witnauer, Wojick, Polack, and Miller (2012), but describing this would be too great a digression from the goal of this paper.

In actuality, there are likely many mechanisms, which are not necessarily mutually exclusive, that contribute to renewal. Which mechanism plays the primary role in producing a specific instance of renewal may depend on the parameters and variables of the specific situation. A contextual conditioning account of renewal is supported by studies showing that contextual associations do contribute to renewal under some circumstances (e.g., Laborda et

al., 2011; Polack, Laborda, & Miller, 2012 [both used a conditioned suppression preparation with rats]), but it is challenged by other findings of renewal in the absence of such associations (e.g., Bouton & King, 1983 [conditioned suppression preparation with rats]; Nelson, Sanjuan, Vadillo-Ruiz, Pérez, & León, 2011 [conditioned suppression preparation with humans]). The SLG (Larrauri & Schmajuk, 2008; Schmajuk et al., 1996) model explains the apparent lack of observable inhibition as a result of reduced attention to the context, which suggests that inhibitory context-US associations should be difficult to detect even if they are present. It is also problematic for an account of renewal based purely on context conditioning that renewal is sometimes still observed when the associative histories of the acquisition, extinction, and test contexts are equated (e.g., Harris, Jones, Bailey, & Westbrook, 2000 [conditioned suppression preparation with rats]; Rescorla, 2008 [sign-tracking preparation with pigeons]; but see Lovibond, Preston, & Mackintosh, 1984 [conditioned suppression preparation with rats]). Moreover, there is some evidence that CS and contextual associations do not always summate (e.g., Bouton & King, 1986 [conditioned suppression preparation with rats]). These findings suggest that, although contextual stimuli can modulate a conditioned response, this explanation is not sufficient to account for all instances of renewal, just as simple occasion setting is not sufficient to account for all instances of renewal. This lends support to our central point that researchers should consider multiple mechanisms when trying to explain recovery-from-extinction phenomena.

As an alternative to direct contextual conditioning, Wagner's (1981) SOP model offers another explanation of renewal that draws on the same mechanism that it uses to account for latent inhibition (Lubow & Weiner, 2010). According to this account, during extinction in Context B, the Context B-CS association should be strengthened. If the subject is returned to this same context for test, then the context should prime some CS nodes into A2. This should leave fewer CS nodes available to enter A1 than if the subject is tested outside of Context B. Consequently, the CS should have less behavioral control when the subject is tested in the extinction context than if the subject is outside of this context. However, this account fails to explain the observed differences in robustness of the three renewal designs.

In Pearce's (1987) theory, associations are formed between stimulus patterns that represent entire perceptual fields at a given moment (i.e., so-called configured stimuli consisting of presented CSs and the context). Responding to a configured stimulus reflects the amount of excitatory and inhibitory associative strength that is conditioned to the stimulus directly and associative strength that is generalized to it from other configured CSs. Generalization is assumed to occur as a function of similarity, such that more generalization occurs between stimuli that share more common stimulus elements and when the common elements are more salient than the unique elements. ABA renewal is explained because the subject is expected to form two separate configural units during treatment, XA during acquisition, which forms an excitatory association with the US, and XB during extinction, which forms an inhibitory association with the US (where A and B represent distinct contexts, and X is the target CS). Testing X in Context A (ABA renewal) should most strongly activate the XA acquisition configural unit, despite some generalization of inhibition from XB, which in turn should activate the US representation. The acquisition configural unit is more strongly activated because it is identical to what is presented during testing. Conversely, testing in Context B should more strongly activate the extinction configural unit, which in turn should activate the inhibitory association, despite some generalization of excitation from XA, and consequently support little responding relative to XA. Responding to X in Context C (i.e., ABC renewal) should be strong relative to X being tested in Context B (i.e., ABB control) because generalization of inhibition from XB to XC should suffer generalization decrement, thus reducing the amount of inhibition expressed in Context C. This account predicts weaker ABC renewal relative to ABA renewal because the excitation from XA should also suffer generalization decrement when generalizing to XC. This is consistent with reports regarding

the strengths of the different types of renewal (e.g., Effting & Kindt, 2007), although this relationship is not always true; there are demonstrations of comparable ABA and ABC renewal (e.g., Thomas et al., 2003). Pearce's (1987) model is unable to account for AAC renewal. This is because at the end of acquisition, it assumes that XA is excitatory, and XA acquires inhibitory associative strength during extinction. When the subject is moved to a new context, there should be equal generalization of excitation and inhibition to XC, which would result in responding that is comparable to an AAA control.

McLaren and Mackintosh's (2002) model distinguishes between core and peripheral units of stimulus representation. Core units are those that are activated by the stimulus and are not dependent on other stimuli for activation. In contrast, peripheral units are activated by combinations of elements from multiple stimuli; their activation is largely dependent on the presence of other stimuli as well as the target (see Wagner & Brandon, 2001, and Wagner & Rescorla, 1972, for a similar idea). Peripheral stimuli are sensitive to changes in the context and accompanying stimuli. One may think of these peripheral stimuli as being analogous to unique units, which are only activated when certain combinations of stimuli are present. When two stimuli are combined, the compound consists of some proportion of the core elements from each stimulus and some proportion of peripheral elements that make up a unique element for the compound. Within this framework, we suggest that renewal can be explained by assuming that inhibition is conditioned primarily to the unique cue generated by a compound made up of the CS and the extinction context. Because these peripheral elements are context-sensitive, moving the subject to a new context for testing should disrupt the inhibition provided by these unique elements, thereby allowing excitatory responding to be expressed. Our application of the McLaren and Mackintosh model assumes that excitation is conditioned primarily to the core elements and that the inhibition that is conditioned to the unique element is post hoc; but it is consistent with Bouton's (1993) suggestion and data from Rescorla (2005; see also Swartzentruber & Bouton, 1992; but see Sissons & Miller, 2009) showing that inhibition is more context-dependent than excitation. This account can explain all forms of renewal. Moreover, it explains why Bouton and King (1983) did not find inhibition conditioned to the extinction context. If the peripheral units acquired most of the conditioned inhibition, then it would not be expected that the core units of the extinction context alone would display inhibitory properties when tested with transfer excitors rather than the target CS.

Reinstatement

Reinstatement refers to the recovery of responding to an extinguished CS that is often observed following extinction treatment, when the US is presented alone prior to testing (Rescorla & Heth, 1975). It is often assessed in a situation in which acquisition, extinction, reinstatement, and testing all occur in the same context. Bouton's (1993) model explains the reinstatement effect as being due to retrieval of the memory of extinction being impaired at test context after USs have been delivered in context as a result of these USs making the test context dissimilar to the extinction context. In this framework, the extinction context is defined in part by the absence of US presentations (and the contextual and internal excitatory properties that accompany such experiences). Alternatively stated, in Bouton's account of reinstatement, making the test context different from the extinction context is the critical consequence of the reinstating USs. Bouton does not subscribe to summation of the increased associative strength between the reinstatement context and the US summating with the associative strength of the CS being a factor. Thus, according to Bouton's theory, reinstatement should be context specific, meaning that it should only be observed in the context in which the reinstating US was administered. This is generally consistent with empirical data.

In contrast to Bouton (1993), most other models (e.g., Mackintosh, 1975; McLaren & Mackintosh, 2002; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981) also assume that the reinstating USs make the reinstatement context excitatory, but they emphasize context conditioning during the reinstating USs and subsequent summation of context excitation with the residual associative strength of the CS, whereas associative summation plays no role in Bouton's account. According to these models, responding is not expected when testing occurs in a different context from reinstatement treatment because the test context is not excitatory, and whatever excitatory strength remains to the CS is below a threshold for producing a robust response on its own (e.g., Reberg, 1972). Similarly, the context of reinstatement without the CS is below threshold for eliciting a response. Notably, these models do not include Bouton's notion of failure to retrieve the memory of extinction due to the ‘absence’ of the extinction context created by making the test context excitatory.

Pearce's (1987) model also assumes contextual conditioning underlies reinstatement, but this explanation, of course, is expressed in terms of generalization between configural units. In the framework of this model, acquisition and extinction (in the same context) should result in the test configural unit (composed of the discrete CS and the context) having both excitatory and inhibitory associations with the US. Presenting the CS at test in this context should normally not produce any responding because the opposing associations should cancel out each other. However, reinforcing the test context following extinction should create another context configural unit, which shares some common features with the CS-context configural unit (i.e., the common contextual cues). Therefore, at test, the target configural unit (CS + context) should activate excitatory and inhibitory associations based on its own training, and it should receive additional excitatory associative strength that is generalized from the context configural unit, which should be greatest if testing occurs in the context of reinstatement treatment. The addition of the generalized excitatory strength from the context should result in a conditioned response that was otherwise nullified by the inhibitory connections between the test configural unit and the US representation.

Contextual change accounts (Bouton, 1993) and contextual conditioning accounts of reinstatement are both congruent with many studies showing context-specificity of reinstatement (e.g., Bouton, 1984 [conditioned suppression preparation with rats]; Bouton & Bolles, 1979b [conditioned suppression preparation with rats]; Bouton & King, 1983 [conditioned suppression preparation with rats]; Bouton & Peck, 1989 [appetitive conditioning preparation with rats]; Brooks, Hale, Nelson, & Bouton, 1995 [counterconditioning preparation with rats]; García-Gutiérrez & Rosas, 2003a [casual learning preparation with humans]; LaBar & Phelps, 2005 [fear conditioning preparation with humans]) and studies showing that this effect is attenuated when the context is extinguished between reinstatement treatment and testing (e.g., Bouton & Bolles, 1979b; Westbrook, Iordanova, McNally, Richardson, & Harris, 2002 [freezing preparation with rats]).

One potential challenge to the contextual conditioning account of reinstatement comes from García-Gutiérrez and Rosas (2003a). Using only one context, they trained humans on a retroactive outcome interference task (A-B, A-C), where A was a particular food and B and C were diarrhea and constipation, counterbalanced. They showed reinstatement of responding based on the Phase 1 association (A-B) even though the reinstatement treatment consisted of presentations of the Phase 2 outcome (C). This problem can be resolved if one were to think of the outcome presentation as increasing the overall excitatory status of the context, irrespective of which US is presented, which would be in line with the idea of context reinstatement arising from a change from the context of extinction as suggested by Bouton (1993). Future research should determine whether reinstatement by a different US depends on the USs sharing the same motivational state or similar physical characteristics.

Also problematic for accounts of reactivation that assume simple contextual conditioning and associative summation are responsible for reinstatement are demonstrations of reinstatement after the reinstating US presentations were given outside of the test context (e.g., (e.g., Neumann, 2008 [conditioned suppression preparation with humans], Schachtman, Brown, & Miller, 1985 [conditioned taste aversion preparation with rats]; Westbrook et al., 2002 [freezing preparation with rats]). This is also problematic for the explanation given by Bouton's theory. Thus, like renewal, it appears that there are multiple mechanisms that contribute to reinstatement, including but not limited to contextual conditioning and occasion setting by the context, and that one mechanism alone is not likely to be sufficient to account for all instances of recovery.

The SOCR model (Stout & Miller, 2007) is challenged to account for reinstatement because, as initially proposed, it lacks a summation rule and predicts less responding to the CS due to increased competition at test from the reinstatement/test context, provided the CS has an association with that context, as would be the case if all treatment occurred in a single physical context. In the comparator framework, this increased competition occurs because presenting the US alone in that context should strengthen the target's Link 3 (the reinstatement context-US association), making the indirectly activated US representation stronger, provided there was a previously established CS-reinstatement context association (i.e., if the reinstatement/test context was also the acquisition or extinction context, which should establish an effective Link 2 for the target CS). Thus, this model predicts weak responding to the target CS following reinstatement treatment when the same context is used for reinstatement and testing as was used for acquisition and/or extinction, and no effect if different contexts are used (no Link 2). However, modification of the comparator hypothesis to include associative summation between the target CS and test context provides a potential solution, and with such a modification, the model can address reinstatement in the same manner as the previously discussed models that use simple associative summation to account for reactivation.

Facilitated (and retarded) reacquisition

It seems clear that the excitatory associative strength of a CS is not completely destroyed (i.e., unlearned) during nonreinforced presentations of the CS. For example, the phenomenon of spontaneous recovery demonstrates that the association between the CS and US representations is at least partially intact following complete behavioral extinction because responding to the CS becomes strong again without any further training. Facilitated reacquisition is also consistent with the view that extinction does not fully erase the CS-US association. This effect refers to an extinguished CS showing rapid, or facilitated, reacquisition of behavioral control when it is reinforced again relative to a novel cue gaining behavioral control for the first time with the same number of reinforced pairings (e.g., Napier et al., 1992). By itself, it is not conclusive proof because it is possible that extinction treatment might have fully erased the CS-US association but left the subject a better learner on the reacquisition trials. But, in conjunction with studies showing latent CS-US associations after extinction (e.g., Reberg, 1972), facilitated reacquisition indicates that an extinguished CS has latently preserved some part of its previous excitatory associative strength, which results in it taking fewer trials for the extinguished CS to gain behavioral control relative to a novel stimulus (e.g., Kehoe, 2006; Macrae & Kehoe, 1999; Napier et al., 1992; Weidemann & Kehoe, 2003, all using nictitating membrane response preparation with rabbits). Reacquisition is sensitive to the number of initial acquisition and extinction trials the subject experienced. Reacquisition is strengthened when many initial acquisition trials are given (e.g., Fishbein, 1967 [eyelid conditioning preparation with humans]) and, correspondingly, reacquisition is attenuated when few initial acquisition trials are given (e.g., Ricker & Bouton, 1996 [appetitive conditioning preparation with rats]).

Somewhat puzzling are reports of the opposite effect, retarded reacquisition, during retraining of an extinguished CS (e.g., Bouton, 1986; Bouton & Swartzentruber, 1989 [both conditioned suppression preparation with rats]; Bouton, Woods, & Pineño, 2004 [appetitive conditioning preparation with rats]; Danguir & Nicolaidis, 1977 [conditioned taste aversion preparation with rats]; Denniston & Miller, 2003 [conditioned suppression preparation with rats]; Hart, Bourne, & Schachtman, 1995 [conditioned taste aversion preparation with rats]). One complication in resolving these discrepant results is that most studies showing facilitated reacquisition use rabbits as subjects in a nictitating membrane response preparation, whereas most studies showing retarded reacquisition typically use rats in either conditioned taste aversion or conditioned suppression preparations. The different preparations make it difficult to draw comparisons between experiments concerning parameters such as number of extinction trials, intertrial intervals, and stimulus duration. However, these opposing effects cannot be attributed solely to the different preparations in that Ricker and Bouton (1996) observed both facilitated and retarded reacquisition relative to a rest control (i.e., a group that did not experience conditioning before the reacquisition phase) in an appetitive conditioning preparation with rats. They suggest that the rate of reacquisition is determined by prior experiences with reinforcement and nonreinforcement as well as interference between the two resultant memories. The influence of previous learning depends on the amounts of acquisition and extinction training, such that extensive acquisition training appears to allow subjects to learn that a reinforced trial signals that the CS will be reinforced again on the next trial. Likewise, extensive extinction treatment appears to result in nonreinforced trials becoming a cue for more nonreinforced trials. During reacquisition consisting of partial reinforcement, subjects that were previously given sufficient training to learn associations within trial types showed higher responding (i.e., facilitated reacquisition) following reinforced trials and lower responding (i.e., retarded reacquisition) following nonreinforced trials. Moreover, slow reacquisition was observed when insufficient acquisition trials (and many extinction trials) were used that prevented subjects from learning the association between reinforced trials, allowing only interference from extinction treatment to be observed. Ricker and Bouton suggested that this type of sequential learning (e.g., Capaldi, 1966) is not necessarily cue-specific since learning is focused on the outcome as a signal for the next trial. Consequently, the same facilitative reacquisition effect should be observed in a group that was trained and extinguished with a different cue; however, this prediction has not yet been tested.

Capaldi's (1966) sequential learning can be reframed in Bouton's (1993) retrieval theory as learning about a general context of reinforcement. When the CS is reinforced during reacquisition treatment with partial reinforcement, the subject is switched out of the context of nonreinforcement, which was present during extinction training, and into the context of reinforcement (regardless of the CS), thus resulting in greater responding on the subsequent reacquisition trial. Likewise, nonreinforced trials intermixed during reacquisition should retrieve the context of extinction and result in lower responding on the next trial. This pattern of results was observed in Ricker and Bouton's (1996) group that was trained, extinguished, and reconditioned with the same cue, whereas a group that was trained and extinguished with a different cue than the one used during reacquisition showed general elevated responding but no differentiation between reinforced and nonreinforced trials during reacquisition. The behavior by the latter group does not support the explanation of facilitated and retarded reacquisition by retrieval of contexts of reinforcement and nonreinforcement, respectively, unless one makes the additional assumption that only reinforced trials will create a general context of reinforcement (i.e., across different CSs), whereas nonreinforced trials will result in only a CS-specific context of nonreinforcement. The authors themselves admit that this differentiation in the degree of generalization between reinforced and nonreinforced trials is speculative; however, they add that context-specificity of nonreinforcement only is a relative matter in light of comparisons of observed

generalization of acquisition versus extinction from the context of treatment to alternative test contexts.

Most likely, the relative amounts of acquisition and extinction training play a critical role in determining whether facilitated or retarded reacquisition is observed, but it is possible that this factor influences responding through a process other than the one proposed by Bouton's (1993) retrieval theory. In the frameworks of the Rescorla-Wagner (1972) model and Mackintosh's (1975) model, facilitated reacquisition can be explained if extensive acquisition and minimal extinction training is given by assuming that the CS retained residual excitatory strength at the end of extinction training. Because the associative value of the extinguished CS is greater than zero, this effectively reduces the amount of re-training required to reach the threshold for responding relative to a novel stimulus. Importantly, if many extinction trials are used, then Mackintosh's model predicts that the CS's associability should be low because of its reduced validity as a signal for reinforcement. The low associability should prevent the CS from being attended to on the first few reacquisition trials, which should retard the rate of reacquisition. The Rescorla-Wagner model fails to predict retarded reacquisition because it assumes that extinction, even massive extinction, cannot give the CS inhibitory value unless it occurs in a highly excitatory context.

The Pearce and Hall (1980) model assumes that attention, and thus associability, to a CS increases if it is not a good predictor of the outcome. A consequence of a CS having high associability is that learning about it will proceed more rapidly. Thus, reacquisition should be facilitated if a CS's associability, which is initially elevated at the start of extinction, is not greatly reduced at the end of extinction (i.e., if only a few extinction trials have been administered). Retarded reacquisition is expected after many extinction trials because this should reduce the CS's associability. Somewhat problematic for the Pearce and Hall account of retarded reacquisition are reports that presenting surprising outcomes during extinction slows reacquisition (e.g., Bouton et al., 2004 [appetitive conditioning preparation with rats]) or does not have an effect on reacquisition (Dopheide, Smith, Bills, Kichnet, & Schachtman, 2005 [conditioned taste aversion preparation with rats]). According to the Pearce and Hall model, a surprising US (Bouton et al., 2004) or a surprising stimulus (Dopheide et al., 2005) administered occasionally immediately following the CS during extinction treatment should have increased the CS's associability or at least prevented it from decreasing, which should have facilitated subsequent conditioning. However, these studies provided evidence to the contrary. We suggest that these results can be reconciled with the Pearce and Hall framework by assuming that the surprising outcomes did in fact increase associability during extinction and consequently resulted in enhanced inhibitory learning on later extinction trials.

Wagner (1981) and Pearce (1987) can account for facilitated reacquisition as the result of net excitatory associative strength, typically occurring when acquisition training is massive and extinction treatment is kept to a minimum. A potential challenge to accounts of facilitated reacquisition based on the net value of concurrent inhibition and excitation is the fact that most reacquisition studies show little or no responding at the end of extinction treatment, suggesting the presence of an inhibitory association strong enough to fully counter the excitatory association that was established during initial acquisition. It is unclear why these inhibitory associations should no longer be effective in suppressing activation of the US representation early in reacquisition. Notably, later versions of Pearce's model (Pearce, 1994, 2002) assume that extinction degrades associations rather than forms separate inhibitory associations (Pearce, 1987). Thus, the 1994 version would explain facilitated reacquisition in the same manner as the Rescorla-Wagner (1972) model, but it would be similarly challenged to account for retarded reacquisition.

McLaren and Mackintosh's (2002) model can account for facilitated reacquisition because it assumes that the stimulus representation for the extinguished CS is still unitized at the start of reacquisition. The excitatory connections between the microelements that comprise the CS should still be excitatory because the CS was presented during extinction, which, if anything, should serve to further unitize the stimulus representation in long-term memory. The only effect of extinction should be to create inhibitory connections between the CS configural unit and the US. When the CS is retrained, presentation of the CS externally activates some proportion of the nodes that represent that stimulus, and those nodes will immediately internally activate the other CS nodes. The whole CS representation will then further increase its excitatory association with the US. In contrast, a novel stimulus must be presented multiple times before the CS representation will be unitized and a strong excitatory association can be established between the unitized CS representation and the US. Thus, the McLaren and Mackintosh framework elegantly explains rapid reacquisition by assuming that training resulted in some degree of perceptual learning about the target CS's representation, which enhances the rate with which microelements of the CS become excitatory during reacquisition. Retarded reacquisition may be expected after extensive extinction, which reduces the associability of the stimulus, thus resulting in a latent inhibition effect. The balance between the beneficial effect of unitization reducing stimulus sampling variability and the retarding effect of exposure reducing stimulus associability will depend on specific parameters of the situation.

The SOCR model (Stout & Miller, 2007) predicts facilitated and retarded reacquisition as a function of the strength of the within-compound association between the CS and the acquisition context (Link 2) and the strength of the CS-US association (Link 1). If the CS is trained and extinguished in the same context, then there should be a strong comparator process (due to a strong CS-context association) down-modulating the target CS's behavioral control. When the target is retrained, it should be retarded in expressing behavioral control relative to a novel stimulus that is not being down-modulated by a strong CS-context association. But, if extinction occurs outside of the acquisition context, then the association between the CS and the acquisition context (i.e., Link 2) should only be of moderate strength, and the CS should be facilitated in re-expressing behavioral control relative to a novel stimulus that does not already have an association with the US (Link 1). Supporting this account is the observation that when all training occurred in the same context and extensive extinction training was administered (which, in the comparator framework, strengthened Link 2), slower reacquisition was observed relative to a novel cue (Bouton, 1986). But moderate extinction training (which would not yield as strong a Link 2) resulted in a reacquisition rate that was indistinguishable from a novel cue (Bouton, 1986). Furthermore, facilitated reacquisition was observed when reacquisition training occurred in a neutral context (Bouton & Swartzentruber, 1989). These results are consistent with SOCR's prediction that the rate of reacquisition depends largely on the strength of the CS-context (i.e., Link 2) association. Of course, this explanation is challenged by demonstrations of facilitated reacquisition when all three phases occur in the same context (e.g., Napier et al., 1992; Ricker & Bouton, 1996).

One important failure of all of the associative learning accounts of facilitated and retarded reacquisition is that they predict these effects should be CS-specific (i.e., they do not explain the learning-to-learn effect). However, Bouton's (1993) retrieval theory is also challenged by the learning-to-learn effect, unless one accepts Ricker and Bouton's (1996) suggestion of differential generalization of outcome-based contexts (i.e., contexts of prior reinforcement better predict subsequent reinforcement better than contexts of nonreinforcement predicting subsequent nonreinforcement). Interestingly, the idea that sequential learning creates a context of reinforcement that is CS-general is homologous to the suggestion that reinstatement is the result of the US reinstatement treatment creating a US-defined context

similar to that of acquisition . Drawing on this, it might be interesting to investigate whether facilitated reacquisition will be observed if different USs of the same (and different) valence are used between training and reacquisition.

Conclusions

There is a long history of experimental research investigating the phenomenon of extinction, and consequently, there are many theories to account for basic extinction effects. Recovery-from-extinction effects have a much shorter experimental history, and there are far fewer theoretical explanations to account for their occurrence. Somewhat oddly, theories that have proven successful over the years in predicting and explaining basic extinction effects have not been extensively applied to account for these newer recovery-from-extinction phenomena. We believe that this oversight has resulted in a tunnel-vision approach to understanding recovery-from-extinction effects. Even in the clinical literature, theories about return of fear are based almost exclusively on Bouton's (1993) occasion setting-based theory of retrieval. This theory has held up well against much empirical data, and it explains recovery effects more parsimoniously than any other approach. However, there are appreciable data that are not in accord with this theory's predictions, which suggests that there are other processes at work producing recovery effects in place of or in addition to the extinction context serving as an occasion setter.

We think that it is important that researchers examine all potential mechanisms to better understand recovery from extinction, particularly because of the clinical implications for reducing or even eliminating relapse following exposure therapy. More generally, we believe that the lack of adequate psychological explanations for recovery-from-extinction effects has hindered advances in uncovering the neural mechanisms responsible for these effects. Without a behaviorally-grounded psychological framework for understanding recovery-from-extinction effects, it is difficult for researchers to appreciate the roles of different brain regions and neural systems in recovery from extinction. Neuroscientists have made a lot of progress towards understanding how learning and extinction occur, and much of this research, particularly with respect to the dopamine system, was guided by the Rescorla-Wagner (1972) model (e.g., Niv, 2009). However, its supposition that extinction causes unlearning has been poorly supported at the behavioral level, making it an inappropriate model to use when investigating recovery-from-extinction effects. As previously stated, Bouton's (1993) theory focuses exclusively on retrieval, and does not speak to the learning, which necessarily underlies retrieval; therefore, it too is deficient in providing a comprehensive understanding of recovery effects. Hence, there is a clear need for theories that account for inhibitory or inhibitory-like learning (e.g., McLaren & Mackintosh, 2002; Pearce, 1987; Pearce & Hall, 1980; Stout & Miller, 2007; Wagner,1981) to be guide behavioral and neurological research concerning extinction and recovery from extinction.

Taken as a whole, we are beginning to understand what roles, in addition to a modulatory one, the context plays in facilitating recovery from extinction. Most associative theories predict that the acquisition and extinction contexts acquire associative values, which drive extinction behavior and recovery from extinction during renewal and reinstatement tests. Most of these theories also predict that the amount of acquisition versus extinction training that is administered will directly influence the observation of facilitated or retarded reacquisition. None of the theories that we considered is presently able to account for spontaneous recovery, but we suggested that adding to the models that assume extinction results in new inhibitory learning a decay function which differentially influences inhibitory and excitatory associations could address this failing. This suggestion is post-hoc with respect to recovery from extinction, but it is consistent with previous suggestions about

associative learning in general (e.g., Pavlov, 1927) and would make these models more compatible with behavioral observations. Without further research and simulations, it is difficult to know what kind of implications such a modification would have on the theories' broader explanatory power. But, we believe that adding associative decay is a potential constructive solution.

Lastly, we would like to state that as the field of associative learning moves away from basic behavioral research towards a more neuroscientific approach, there is still a need for a theoretical framework to understand how certain behaviors occur at the psychological level, not just at the neurological level. Additionally, researchers are starting to realize how learning contributes to the development and maintenance of psychopathology, giving significance to research that models treatment of such disorders. Therefore, it is important to develop theoretical frameworks that we can use to interpret these data and lead us to new techniques for reducing relapse from therapy. In conclusion, we believe that the more general theories of learning that have long guided behavioral research, including some of those that were not examined in this review, are still useful in understanding recovery from extinction and should be duly considered.

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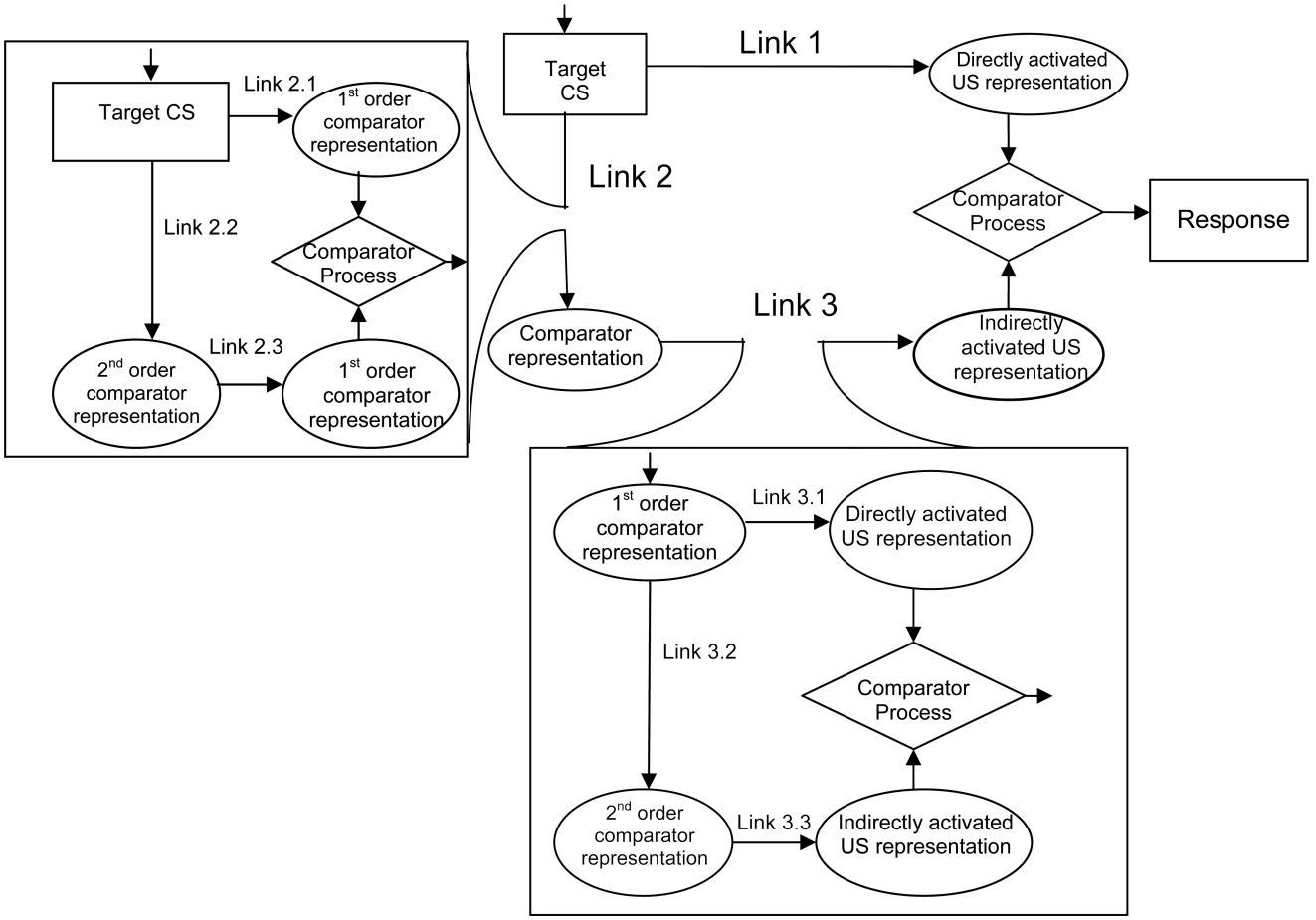

Figure 1.

Denniston, Savastano, and Miller's (2001) extended comparator hypothesis. Ovals depict stimulus representations; rectangles depict physical events; diamonds represent the comparator process; arrows represent associations.