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Spontaneous recovery of excitation and inhibition

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Abstract

In two conditioned suppression experiments with rats as subjects, we examined two classes of accounts of spontaneous recovery. One view suggests that spontaneous recovery occurs due to greater temporal instability of inhibitory associations, whereas the other posits that spontaneous recovery occurs due to greater temporal instability of second-learned associations. These accounts diverge in predictions concerning spontaneous recovery when the first-learned association is inhibitory and the second-learned association is excitatory. Using different designs, Experiments 1 and 2 found spontaneous recovery of both excitation and inhibition. The results support the view that spontaneous recovery occurs due to faster waning of second-learned associations.

Keywords

spontaneous recovery; extinction; conditioned inhibition

Spontaneous recovery is typically defined as the reemergence of conditioned responding to an extinguished conditioned stimulus (CS) with the passage of time since extinction. Because acquisition followed by extinction constitutes a two-phase training regimen, a broader definition of spontaneous recovery is the reemergence of a first-learned association to a CS that subsequently has received contradictory training, with the passage of time since the contradictory training. This latter definition better engages the present discussion; thus, it will be our working definition of spontaneous recovery. Consistent with this definition, spontaneous recovery can also be regarded as an increase in primacy with delay of testing, which has been frequently observed (Konorski & Szwejkowska, 1952; Postman, Stark, & Fraser, 1968; Wheeler, Stout, & Miller, 2004).

In his seminal paper, Bouton (1993) proposed two types of explanations of spontaneous recovery from extinction. One account emphasizes the nature of the learning that happens during extinction, specifically the formation of an inhibitory CS-no unconditioned stimulus (US) association that is superimposed at test onto the previous CS-US excitatory learning (Bouton, 1993; Pavlov, 1927). This account explains spontaneous recovery from extinction of the conditioned response by assuming that the retrievability of the inhibitory memory fades faster over time than that for excitation (which presumably fades very slowly). The other type of explanation suggests that spontaneous recovery is a consequence of sequentially learning two contradictory associations (Bouton, 1993; Spear, 1971). According to this account, the impact of extinction fades with time *because* extinction is the second-learned association and consequently the more ambiguous of the two experiences (the first being acquisition). Toward differentiating between these two accounts, we noted that they differ in their expectations for spontaneous recovery in situations involving two contradictory learning experiences when the second experience includes reinforcement. If conditioned inhibition fades faster than

excitation, there is no reason to expect spontaneous recovery of conditioned inhibition. The other account, which assumes rapid fading of the second-learned association, would expect spontaneous recovery of the first-learned association regardless of whether the second-learned association was inhibitory or excitatory. Of note, in a subsequent chapter Bouton (1997) favored this second explanation.

An attentional account of spontaneous recovery from extinction proposed by Robbins (1990) suggests that extinction is a result of decreased attention to a CS and that spontaneous recovery is due to a natural restoration of attention over time. This theory is viable with respect to extinction, a situation in which a cue loses control of behavior, but does not appear to be readily applicable to second-learned inhibition or excitation training as both involve a change in control of behavior rather than a loss of control of behavior. Consequently, we will not here pursue this account.

The study of super-latent inhibition (De la Casa & Lubow, 2000, 2002; also see Wheeler, Stout, & Miller, 2004) begins to address this issue. Latent inhibition is the retarded emergence of behavioral control by a CS over repeated pairings of the CS and a US as a result of the CS having been previously presented without reinforcement (i.e., nonreinforcement followed by reinforcement; Lubow & Moore, 1959). Super-latent inhibition is the observation of greater retardation of behavioral control (i.e., greater latent inhibition) with a long interval between the acquisition of the excitatory association and test relative to a short interval. De la Casa and Lubow documented this effect using a conditioned taste aversion preparation with rats. First, their experimental subjects received four days of nonreinforced preexposure to a flavor (saccharin solution) while their control subjects received exposure to water. After the preexposure phase, all subjects received access to the saccharin solution followed by an injection of lithium chloride (i.e., reinforcement). Testing consisted of the subjects being allowed to drink the saccharin solution without lithium chloride injections. A test soon after conditioning showed that animals that were preexposed to the saccharin solution drank more than animals that had not received saccharin preexposure (i.e., latent inhibition). This difference in conditioned rejection of saccharin was enhanced by a 21-day delay in testing (i.e., super-latent inhibition). There was no significant effect of delay in the no preexposure condition. This experiment is an example of spontaneous recovery (given our broader definition of spontaneous recovery) of a CS-noUS association as well as a demonstration of the super-latent inhibition effect, which suggests that super-latent inhibition could be caused by the same mechanism as spontaneous recovery from extinction. Of note some laboratories have failed to document the super-latent inhibition effect (Aguado, Symonds, & Hall, 1994; Kraemer & Roberts, 1984) That is, they observed decreased latent inhibition as the retention interval increased, but that was likely because the conditioning context was extinguished due to all stages of the experiments being conducted in one context. Super-latent inhibition appears to occur only when the retention interval is spent outside the training context.

Bouton's (1997) explanation of spontaneous recovery in terms of the ambiguity of the second-learned associations accounts for super-latent inhibition by assuming that during CS preexposure animals learn that drinking saccharin has no negative consequences (first-learned association). Then during conditioning, a new saccharin-outcome (illness) association is learned, which is ambiguous because it is a second-learned association for the CS. With an immediate test, animals show a greater tendency toward expressing the most recently learned association compared to a delayed test in which animals more strongly express the first-learned association (i.e., a shift from recency to primacy). However, there is a reason to doubt that CS-preexposure treatment makes a CS into a conditioned inhibitor (e.g., Reiss & Wagner, 1972). Thus, the observation of super-latent inhibition, although suggestive, does not clearly differentiate between the two accounts of spontaneous recovery. An important point here is that the loss of excitatory responding observed following the retention interval does not

necessarily demonstrate that latent inhibition was recovered, only that excitatory responding was lost.

Counterconditioning is another phenomenon that speaks to the two accounts of spontaneous recovery. Counterconditioning is the procedure of associating one outcome with a CS during the first phase of training, and then associating an opposing outcome with the same CS during the second phase of training. For example, a CS may first be paired with an appetitive US followed by an aversive US; or vice versa. Bouton and Peck (1992) presented evidence of spontaneous recovery from counterconditioning using this procedure with rats. That is, immediately after the second learning experience behavior was controlled by the most recent US. But with delayed testing the first-learned US came to control responding to the CS. Bouton's (1997) view accounts for recovery from counterconditioning in much the same way as superlatent inhibition. The second-learned association, regardless of the motivational system it engages, wanes more rapidly with the passage of time. However, as the Pavlovian account of spontaneous recovery from extinction depends on an inhibitory association being superimposed upon the original association, counterconditioning does not clearly differentiate the two accounts either.

Nelson (2002) reported training a cue as a conditioned inhibitor during Phase 1, followed by training of the same cue as a conditioned excitor in Phase 2, or alternatively, he switched the order of the inhibition and excitation training. In both cases, when he switched the context at test, responding to the second-learned association was reduced while responding to the first-learned association was unimpaired. These results suggest that the second-learned association is more susceptible to context change than the first-learned association, whether that association is inhibitory or excitatory. These results complement the experiments reported here in that Nelson manipulated the physical context whereas we manipulated the temporal context by imposing a retention interval. As will be evident, we found spontaneous recovery of the first-learned association regardless of whether the association was excitatory or inhibitory, which is analogous to Nelson's observation of renewal in his experiments.

Rescorla (2005) sought to compare the inhibitory and second-learned accounts of spontaneous recovery. He wanted to see if spontaneous recovery of inhibition after reversal treatment (i.e., reinforced training) was equivalent to spontaneous recovery of excitation following inhibition training. To answer this question, he used a number of different designs and methods. In Experiments 1-3, he only sought spontaneous recovery of conditioned inhibition without including control groups to demonstrate spontaneous recovery of conditioned excitation under comparable conditions. Thus, despite Rescorla's failure to observe spontaneous recovery of inhibition (i.e., null results), it is unclear whether conditioned inhibition is not subject to spontaneous recovery or simply that his procedure was insensitive to spontaneous recovery. However, in Experiments 4 and 5 Rescorla reported evidence of spontaneous recovery from conditioned excitation but not from conditioned inhibition. In those two experiments he administered Pavlovian inhibition training in which one cue, an excitor (A), was consistently reinforced elementally and another cue, the inhibitor (X), was consistently nonreinforced in compound with A in Phase 1 (A+/AX-, where + indicates reinforcement and - indicates nonreinforcement). Then, in Phase 2, he reversed the roles of the CSs by reinforcing the inhibitor and not reinforcing (i.e., extinguishing) the excitor (A-/X+). Thus, A received excitation training followed by differential inhibition training, whereas X received Pavlovian inhibition training followed by excitation training. Rescorla found that the behavioral control of stimuli, trained either as excitors or inhibitors and then given a second phase of training with reversed reinforcement contingencies, changed differently over time depending on whether the second learning experience was reinforced or nonreinforced. Specifically, Rescorla observed spontaneous recovery of the first-learned reinforced (excitatory) experience but not the first-learned nonreinforced (inhibitory) experience. He took this as support for the view

that there is greater temporal instability for the learning that occurs during inhibition training. But this conclusion was predicated on the assumption that the inhibitory training in the first and second phase was equivalent other than the phase in which it occurred. Notably, the following criticisms of Experiments 4 and 5 do not apply to Rescorla's Experiment 1. However, as there was no comparable control for spontaneous recovery of conditioned excitation, it does not tell the full story.

Due to the interrelationship of intervals between training and extinction, training and test, and extinction and test, there are always confounds within spontaneous recovery designs (Rescorla, 2004). Rescorla (2005) addressed this problem by using two different experimental designs, each of which confounded different time intervals. However, there are other difficulties in his designs that, if corrected, might have produced different results. A key limitation with the reversal treatment employed in these studies lies in work pioneered by Lysle and Fowler (1985; also see Hallam, Matzel, Sloat, & Miller, 1990). They found that extinction of the excitor used in Pavlovian inhibition training (A) partially deactivates the potential of the inhibitor to reduce responding. Thus, in Phase 2 of Rescorla's design, extinction of A, which was the excitor used to make X inhibitory in Phase 1, may have weakened the inhibitory potential of X, thereby creating a bias against his observing spontaneous recovery of conditioned inhibition. A further potential problem with Rescorla's studies is that the type of inhibition given in the two phases was different. For inhibition training of X before excitation training, he used a conventional Pavlovian inhibition procedure (i.e., A+/AX-), whereas for excitation training before inhibition training, the inhibition training took the form of differential inhibition (i.e., X+/A-). In prior research (Urcelay & Miller, 2006), we found that Pavlovian and differential inhibition are best observed under widely different conditions. Pavlovian inhibition is greater with spaced trials and differential inhibition is greater with massed trials, all other factors being equal. But even without concern for which parameters favor one type of inhibition over the other, the fact that the type of inhibition treatment and phase were confounded is reason to question the conclusions that Rescorla reached. Possibly, his observation of less spontaneous recovery from reinforced training in Phase 2 than from nonreinforced training in Phase 2 could have simply been the result of the nature of inhibitory training differing between the reinforced-nonreinforced and the nonreinforced-reinforced sequences.

The aim of the present research was to reassess the possibility of observing spontaneous recovery of excitation and inhibition while addressing the aforementioned limitations of Rescorla's (2005) prior studies. We equated the number of trials administered in each phase to create symmetry between phases and used Pavlovian conditioned inhibition treatment in each phase to equate inhibitory treatment between phases. Furthermore, we used a nontarget excitor for inhibition training to minimize the possibility of indirectly decreasing the inhibitory value of the stimulus trained as an inhibitor in Phase 1 when we nonreinforced the target excitor in Phase 2.

Experiment 1

Experiment 1 was designed to assess the possibility of observing spontaneous recovery of excitation and inhibition when the first-learned association and contrary second-learned association were equated in terms of treatment. Our parameters were similar to those used by Urcelay and Miller (2006), who successfully demonstrated conditioned inhibition with summation and retardation tests. Therefore, we felt it unnecessary to conduct these standard tests for conditioned inhibition. In this experiment, all animals received Phase 1 and Phase 2 at the same times, but the NoDelay Groups were tested 4 days after the last day of Phase 2 training and the Delay Groups were tested 27 days after the last day of Phase 2 training. Subjects in each of these two conditions were assigned to be tested on either the initially excitatory or initially inhibitory stimulus. See Table 1 for the design of Experiment 1.

Method

Subjects

The subjects were 24 male and 24 female Sprague-Dawley, experimentally naïve, young adult rats ($N = 48$), bred in our colony. Body weight range for females was 171–219 g and for males was 245–306 g. Subjects were individually housed and maintained on a 16-hr light / 8-hr dark cycle with experimental sessions occurring roughly midway through the light portion of the cycle. All subjects were handled for 30 s three times per week from weaning until the initiation of the study. Subjects had free access to food in the home cage. One week prior to initiation of the experiment, water availability was progressively reduced to 30 min per day, provided shortly after any scheduled treatment.

Apparatus

The apparatus consisted of 12 operant chambers each measuring $30.5 \times 27.5 \times 27.3$ cm ($l \times w \times h$). All chambers had clear Plexiglas ceilings and side walls, and metal front and back walls. On one metal wall of each chamber, there was an operant lever and adjacent to it a niche ($4.5 \times 4.0 \times 4.5$ cm) centered 3.3 cm above the floor where a drop (0.04 cc) of distilled water could be presented by a solenoid valve. Chamber floors were 4-mm diameter grids spaced 1.7 cm apart center-to-center, connected with NE-2 neon bulbs, which allowed constant-current footshock to be delivered by means of a high voltage AC circuit in series with a 1.0-M Ω resistor. All chambers were housed in sound- and light-attenuating cubicles. Each chamber was dimly illuminated by a #1820 houselight. Additionally there was a 60-W (nominal at 120 VAC, driven at 60 VAC) incandescent bulb that was illuminated for 0.5 s simultaneously with each water reinforcement. Chamber assignments within each of the four experimental groups were counterbalanced. Three 45- Ω speakers mounted on different interior walls of each environmental chest could deliver a high-frequency complex tone (3000 and 3200 Hz) 8 dB (C-scale) above the ambient background, a 6-Hz click train 8 dB above background, or a white noise stimulus 8 dB (C) above background. The ambient background sound of 78 dB was produced primarily by a ventilation fan. The white noise was used as the training excitator in inhibition training (CS A), and the clicks and tone were used as CSs B and C, counterbalanced within groups. A 0.7-mA, 0.5-s footshock, which served as the US, could be delivered through the chamber floors.

Procedure

Subjects were randomly assigned to one of four groups, counterbalanced for sex ($n_s = 12$). All phases of the study were conducted in the same chamber; the levers were deactivated during Phases 1 and 2.

Shaping—Prior to Phase 1 of training, 5 days of acclimation to the apparatus and shaping of lever press behavior were conducted in 60-min sessions. Subjects were shaped to lever press for a drop of water on a variable-interval 20-s (VI 20-s) schedule in the following manner. On Days 1 and 2, a fixed-time 120-s schedule of noncontingent water delivery was in force concurrently with a continuous reinforcement schedule. On Day 3, noncontingent reinforcers were discontinued and subjects were trained on the continuous reinforcement schedule alone. Subjects that emitted less than 50 responses on this day experienced a hand-shaping session later in the same day. On Days 4 and 5, a VI 20-s schedule was imposed. This schedule of reinforcement prevailed throughout the remainder of the experiment except during Phases 1 and 2 during which no water was available. Other than the water and light signal for water, no nominal stimulus was presented during shaping.

Phase 1—On Days 6–11, during daily 75-min sessions, all subjects received four A+, eight AB-, and one C+ trials per day, with the intent of making C excitatory and B inhibitory. CS

duration for all cues was 10 s. The mean ITI (CS termination to CS onset) was 5.75 min (values = 4.75, 5.75, and 6.75 min, each used 4 times/session). On reinforced trials, the US was presented in the last 0.5 s of the CS. Trial order was pseudo-randomized so that the sessions always began and ended with an A+ trial. There were never more than two reinforced trials in a row or more than 3 inhibitory compound trials in a row.

Phase 2—On Days 12-17, during daily 75-min sessions, all subjects received four A+, eight AC-, and one B+ trials per day. Except for the switching of B and C, training was identical to Phase 1.

Reshaping—On Day 18, due to an experimenter error, all squads received a 1-h session of VI 20-s reshaping.

Reshaping 1—On Days 19 and 20, subjects in Groups E No-delay and I No-delay experienced 1-h sessions to restabilize lever pressing on the VI 20-s schedule. Groups E Delay and I Delay remained in their home cages. Baseline responding was assessed by recording the number of responses made in the two minutes before the first test trial that would be given in the Test phase.

Test 1—On Day 21, suppression of baseline responding during presentation of the CS was assessed in Groups E No-Delay and I No-Delay. Each subject received four nonreinforced 60-s presentations of their designated CS (i.e., C for Group Test E No-Delay and B for Group Test I No-Delay) during a 25-min session with the onset of the CS occurring at 4, 10, 16, and 22 min during the session. The response rate (number of lever-presses/min) during the 60-s period preceding each CS exposure (pre-CS score) and that during the 60-s CS exposure (CS score) was recorded.

Retention Interval—On Days 19 through 39, subjects in Groups E Delay and I Delay remained in their home cages, but were handled for 30 s three times a week. The deprivation schedule was maintained throughout the retention interval.

Reshaping 2—On Days 40-43, subjects in Groups E Delay and I Delay experienced 1-h daily sessions to restabilize lever pressing on the VI 20-s schedule. Baseline responding was assessed by recording the number of responses made in each of the two minutes before the first test trial would be given in the Test phase. Due to initial baseline differences in responding, all animals received a total of four days of reshaping.

Test 2—On Day 44, each subject in Groups E Delay and I Delay was tested exactly like the No-Delay groups on Day 21. Group E Delay was tested on CS C and Group Test I Delay was tested on CS B.

Data Analysis

A suppression ratio (Annau & Kamin, 1961) for each subject was calculated by the formula $CS/(CS+pre-CS)$, where CS is the mean rate of lever pressing across all four of the 60-s CS presentations and pre-CS is the mean rate of lever pressing during the first 120-s pre-CS period. We used this as the basis of our pre-CS score the rate of lever pressing before the first CS presentation because we observed some carryover of fear from earlier CS test presentations on pre-CS performance of test trials following the first CS test presentation (also see Bolles, Collier, Bouton, & Marlin, 1978). Suppression on later test CS presentations was likely the result of fear induced both on that trial and fear held over from earlier trials. Thus, the only pure measure of baseline drinking was the time prior to the first presentation of the test CS. This ratio was used as an index of the subject's fear to the presentation of the target CS. The

pre-CS rates and suppression ratios were compared using a 2 (Test stimulus condition: Excitor vs. Inhibitor) $\times$ 2 (Delay condition: Delay vs. No-Delay) analysis of variance (ANOVA). Critical planned between-subject comparisons between Groups E No-Delay and E Delay and between Groups I No-Delay and I Delay used the error term from the omnibus ANOVA. This was intended to determine the effect of the retention interval on spontaneous recovery of an excitor versus an inhibitor. Alpha was set at .05 for all inferential tests. Effect sizes were calculated using Cohen's f (Myers & Well, 2003, p. 210).

Results and Discussion

Figure 1 illustrates mean suppression ratios to the target CS observed in Experiment 1. Greater suppression was observed in Group E Delay compared to Group E No-Delay, reflecting spontaneous recovery of excitation as a result of a long delay imposed between Phase 2 and Test. In contrast, less suppression was observed in Group I Delay compared to Group I No-Delay, suggesting that a long delay imposed between Phase 2 and Test resulted in spontaneous recovery of inhibition. These observations were supported by the following analyses.

Mean baseline rate responding was 27.2 for Group E Delay, 21.1 for Group E No-Delay, 33.1 for Group I Delay, and 32.5 for Group I No-Delay. An ANOVA with test stimulus initial training (Excitor vs. Inhibitor) and retention interval (Delay vs. No-Delay) as main factors conducted on baseline lever pressing during the 120 s before the first test trial detected no significant main effect nor interaction ($ps > .36$). This indicates that there were no appreciable differences among groups in fear of the test context. A similar ANOVA was conducted on the suppression ratios. This analysis revealed no main effects, but critically there was an interaction between the two factors, $F(1, 44) = 22.21$, $MSE = .06$, Cohen's $f = .66$.

Planned comparisons using the overall error term from the latter ANOVA were conducted to assess the source of the interaction. Comparisons of responding to each test stimulus with and without the delay showed significant changes as a function of the retention interval. Suppression to C in Group E Delay was greater than Group E No-Delay, indicating spontaneous recovery of excitation, $F(1, 44) = 10.07$. Suppression to B in Group I Delay was weaker than Group I No-Delay, indicating spontaneous recovery of inhibition, $F(1, 44) = 12.19$. An alternative account to spontaneous recovery from conditioned inhibition is that the conditioned excitation trained in Phase 2 was simply forgotten over the long retention interval. However, this is implausible as previous research from our laboratory has documented that simple excitation does not wane using parameters and retention intervals similar to those used in this experiment (Pineno, Urushihara, & Miller, 2005; Wheeler, Stout, & Miller, 2004).

Experiment 1 demonstrated spontaneous recovery of both excitatory and inhibitory behavioral control. This finding is more in accord with the view that spontaneous recovery occurs because the second-learned association wanes faster than the first-learned association than with the view that inhibition fades faster than excitation.

Experiment 2

Experiment 2 was conducted to assess the possibility that we observed differential responding in Experiment 1 simply because of testing at two different points in time relative to Phase 1 rather than the interval between Phase 2 and testing. This concern was raised by Rescorla (1997, 2004). He criticized testing for spontaneous recovery at two different times because the state of the animals and (temporal) context of testing could be quite different across groups. Therefore, in Experiment 2, all groups received Phase 1 treatment at the same time. But Phase 2 was split so that the Delay groups received Phase 2 training immediately after Phase 1 followed by a 28-day retention interval and the No-Delay groups received Phase 2 training immediately prior to testing. Thus, all animals received Phase 1 training at the same time and

were tested at the same time (see Table 2). Such a design necessitated confounding the retention interval with the time between Phase 1 and 2, but Experiment 1 avoided this confound.

Method

Subjects and Apparatus

Subjects were 24 female (181–240 g) and 24 male (254–324 g) Sprague-Dawley, experimentally naïve, young adult rats ($N = 48$), bred in our colony. The animals were housed and maintained as in Experiment 1. The apparatus and stimuli were identical to those used in Experiment 1.

Procedure

Subjects were randomly assigned to one of four groups, counterbalanced for sex ($ns = 12$). All phases of the study were conducted in one context; levers were deactivated during Phases 1 and 2. Shaping, Phase 1, Reshaping and Test were conducted in the same manner as Experiment 1 with the exception that all animals received a total of four days of reshaping.

No-Delay Condition—On Days 12 through 32, subjects in Groups E No-Delay and I No-Delay remained in their home cages, but were handled for 30 s three times a week. The deprivation schedule was maintained throughout the retention interval.

Phase 2a—On Days 12–17, subjects in Groups E Delay and I Delay received Phase 2 treatment.

Delay Condition—On Days 18 through 38, subjects in Groups E Delay and I Delay remained in their home cages, but were handled for 30 s three times a week. The deprivation schedule was maintained throughout the retention interval.

Phase 2b—On Days 33–38, during daily 75-min sessions, subjects in Groups E No-Delay and I No-Delay received the same treatment as Groups E Delay and I Delay in Phase 2a.

Results and Discussion

Figure 2 illustrates mean suppression ratios to the target CS observed in Experiment 2. The data were very similar to those of Experiment 1. Greater suppression was observed in Group E Delay compared to Group E No-Delay, once again reflecting spontaneous recovery of excitation as a result of a long delay imposed between Phase 2 and Test. Also in accord with Experiment 1, less suppression was observed in Group I Delay compared to Group I No-Delay, suggesting that a long delay imposed between Phase 2 and Test caused spontaneous recovery of inhibition. These observations were supported by the following analyses.

Mean baseline rate responding was 23.2 for Group E Delay, 27.2 for Group E No-Delay, 29.1 for Group I Delay, and 30.8 for Group I No-Delay. An ANOVA with Test stimulus condition (Excitor vs. Inhibitor) and Delay condition (Delay vs. No-Delay) as main factors conducted on baseline lever pressing during the 120 s before the first test trial detected no significant main effect nor interaction ($ps > .28$). This indicates that there were no appreciable differences among groups in fear of the test context. A similar ANOVA was conducted on the suppression ratios. This analysis revealed no main effects, but found an interaction between the two factors, $F(1, 44) = 20.85$, $MSE = .07$, Cohen's $f = .64$.

Planned comparisons using the overall error term from the latter ANOVA were conducted to assess the source of the interaction. Comparisons of responding to each test stimulus with and

without the delay showed significant changes as a function of the retention interval between Phase 2 and Test. Suppression to C in Group E Delay was greater than Group E No-Delay, indicating spontaneous recovery of excitation, $F(1, 44) = 9.02$. Suppression to B in Group I Delay was weaker than Group I No-Delay, indicating spontaneous recovery of inhibition, $F(1, 44) = 11.93$.

Experiment 2 replicated the findings of Experiment 1 by demonstrating spontaneous recovery of Phase 1 training with both the excitatory stimulus and the inhibitory stimulus. Additionally, Experiment 2 shows that spontaneous recovery of inhibition can be found when the Phase 1-Test interval is held constant and therefore is not merely a product of testing at different times relative to Phase 1. Once again, this finding is more in accord with the view that spontaneous recovery occurs due to the second-learned association waning faster than the first, than with the view that inhibition fades faster than excitation.

General Discussion

In two experiments we assessed the possibility of observing spontaneous recovery of excitation and inhibition. Experiment 1 used a standard spontaneous recovery design in which training for all groups was conducted simultaneously and testing was conducted 4 days or 27 days later, which confounded the time between Phase 1 and Test. Experiment 2 controlled for the interval between Phase 1 and test, but it confounded the interval between Phase 1 and Phase 2. Though the two designs confounded different time intervals, each demonstrated spontaneous recovery of both excitation and inhibition. This is contrary to Rescorla's (2005) conclusion that spontaneous recovery occurs for excitation but not for inhibition. The present results favor the view that spontaneous recovery occurs due to the more rapid fading of second-learned associations (Bouton, 1993, 1997; Spear, 1971) over the view that spontaneous recovery occurs due to inhibition fading faster than excitation (Bouton, 1993; Pavlov, 1927).

The most likely reason that spontaneous recovery of inhibition was observed in the present experiments is because inhibitory training was equated between phases and the excitor from inhibition training in Phase 1 was not extinguished in Phase 2. Regardless of whether inhibition training occurred in Phase 1 or Phase 2, the same type and number of trials were administered. The same is true of excitatory training. This presumably more closely equated the opportunity across phases to learn about each relationship, which provides a more impartial baseline from which to observe spontaneous recovery. Obviously a balance of the two competing memories is important to observe spontaneous recovery. With too little Phase 2 training, there will be little interference from which recovery could occur; with too much Phase 2 training, there will likely be so much interference that little recovery will be observed.

The present experiments also diverge from Rescorla's (2005) experiments with respect to the means used to produce excitators and inhibitors. Excitatory training was the same in Phase 1 and Phase 2, and inhibitory training was the same in Phase 1 and Phase 2. Moreover, we used a separate excitor (A) for inhibition training that was never altered in terms of its elemental reinforcement history. Instead, it was used in each phase to train the inhibitor of that phase. This allowed us to circumvent the possibility of altering the first-learned inhibitory association by extinguishing the excitor used to train that inhibitor, a procedure known to sometimes degrade the inhibitory strength of a CS even in the absence of reinforced presentations of the inhibitor (e.g., Lysle & Fowler, 1985).

The present data are congruent with Nelson's (2002) findings in that, when conditioned inhibition is first learned in a two-phase learning situation with differing outcomes, it, like conditioned excitation, appears to be subject to recovery as a result of either removal from the spatial context of Phase 2 training (i.e., renewal) or removal from the temporal context of Phase

2 (i.e., spontaneous recovery). These convergent findings are consistent with Bouton's (1993, 1997) view of spatial and temporal contexts having much in common, and departure from the spatiotemporal context of a second-learned relationship encouraging retrieval of the first-learned relationship.

It should be noted that the point of these experiments is not to suggest that conditioned inhibition does not fade over time. Conditioned inhibition (as well as conditioned excitation) has been observed to fade over time (e.g., Henderson, 1978; Thomas, 1979). However, those experiments did not train inhibition and excitation sequentially. The question here is whether conditioned excitation and conditioned inhibition appreciably differ in the rate at which they fade when they are second learned as opposed to first learned, all other factors being equal.

In summary, these experiments demonstrate that spontaneous recovery of both Pavlovian excitation and inhibition can be observed at least with select parameters. These demonstrations show that there is merit to the assumption that spontaneous recovery is a result of second-learned associations fading more rapidly than first-learned associations, regardless of the nature of that learning (Bouton, 1993, 1997; Spear, 1971).

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Figure 1.

Results of Experiment 1. Mean suppression ratios to the target cues C (first trained as an excitor) and B (first trained as an inhibitor) at test. Note that with suppression ratios the scale is inverted; lower values reflect greater conditioned suppression. Error bars represent the standard error of the means. See text and Table 1 for details.

Figure 2.

Results of Experiment 2. Mean suppression ratios to the target cues C (first trained as an excitator) and B (first trained as an inhibitor) at test. Note that with suppression ratios the scale is inverted; lower values reflect greater conditioned suppression. Error bars represent the standard error of the means. See text and Table 2 for details.

Table 1

Design summary of Experiment 1

Group	Phase 1	Phase 2	Test 1	Retention Interval	Test 2	2 nd learned	CI
E No-delay	24 A+/ 48 AB-/ 6 C+	24 A+/ 48 AC-/ 6 B+	C			cr	cr
E Delay	24 A+/ 48 AB-/ 6 C+	24 A+/ 48 AC-/ 6 B+		27 days	C	CR	CR
I No-delay	24 A+/ 48 AB-/ 6 C+	24 A+/ 48 AC-/ 6 B+	B			CR	CR
I Delay	24 A+/ 48 AB-/ 6 C+	24 A+/ 48 AC-/ 6 B+		27 days	B	cr	CR

E = target first trained as an excitator, I = target first trained as an inhibitor, numbers = number of trials, A = excitatory cue used for inhibition training, B and C = target cues, + = footshock, - = no footshock, 2nd learned = predictions based on seconded-learned experience being forgotten rapidly, CI = predictions based on inhibitory information being forgotten rapidly, cr = weak conditioned suppression expected, CR = strong conditioned suppression expected.

Table 2

Design summary of Experiment 2

Group	Phase 1	Pre-Phase 2 Retention Interval	Phase 2a	Post-Phase 2a Retention Interval	Phase 2b	Test	2 nd learned	CI
E No-Delay	24 A+/ 48AB-/ 6 C+	28 days			24 A+/ 48 AC-/ 6 B+	C	cr	cr
E Delay	24 A+/ 48AB-/ 6 C+		24 A+/ 48 AC-/ 6 B+	28 days		C	CR	CR
I No-Delay	24 A+/ 48AB-/ 6 C+	28 days			24 A+/ 48 AC-/ 6 B+	B	CR	CR
I Delay	24 A+/ 48AB-/ 6 C+		24 A+/ 48 AC-/ 6 B+	28 days		B	cr	CR

E = target first trained as an excitator, I = target first trained as an inhibitor, numbers = number of trials, A = excitatory cue used for inhibition training, B and C = target cues, + = footshock, - = no footshock, 2nd learned = predictions based on seconded-learned experience being forgotten rapidly, CI = predictions based on inhibitory information being forgotten rapidly, cr = weak conditioned suppression expected, CR = strong conditioned suppression expected.