Spontaneous recovery and ABC renewal from retroactive cue interference

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Abstract Two conditioned suppression experiments with rats were conducted to determine whether the spontaneous recovery and renewal that are commonly observed in retroactive outcome interference (e.g., extinction) also occur in retroactive cue interference. Experiment 1 showed that a long delay between Phase 2 (the interfering phase) and testing produces a recovery from the cue interference (i.e., the delay enhanced responding to the target cue trained in Phase 1), which is analogous to the spontaneous recovery effect observed in extinction and other retroactive outcome interference procedures. Experiment 2 showed that, when target and interfering cues are trained in separate contexts and testing occurs in a different but familiar context, a recovery from the cue interference is also observed (i.e., the context shift enhanced responding to the target), which is analogous to ABC renewal from extinction. The results are discussed in terms of the possibility that similar associative mechanisms underlie cue and outcome interference.

Keywords Retroactive interference · Cue interference · Spontaneous recovery · Renewal · Pavlovian conditioning · Associative learning · Classical conditioning

Interactions between cues or between outcomes that attenuate acquired behavior can be classified as either stimulus competition (e.g., overshadowing and blocking) or stimulus interference (i.e., retroactive and proactive interference), depending on whether the stimuli are presented together

(i.e., competition) or in different sequential phases (i.e., interference). From this conceptualization, one can create a 2×2 matrix (see Fig. 1) that incorporates whether the interacting stimuli occurred together or apart (i.e., competition or interference, respectively) and whether the interacting stimuli were cues or outcomes (i.e., antecedent or subsequent events, respectively). Within the Pavlovian conditioning literature, stimulus competition (particularly cue competition) has received the greatest amount of attention, followed by attention to retroactive outcome interference phenomena (particularly extinction and counterconditioning). In a counterconditioning design, a target cue (X) is followed by an outcome (A) (e.g., X–A) in phase 1, and then the same cue is followed with an outcome of the opposite valence (e.g., X–B) in phase 2. On a test of the X–A association (i.e., X is presented), this arrangement produces a deficit in responding as result of phase 2 treatment, relative to a control group that lacks the X–B training in phase 2. In an extinction design, X–A pairings in phase 1 are followed by X–no-A trials in phase 2, and a deficit in responding based on X–A training is observed as result of phase 2 treatment.

Traditional models of associative learning (e.g., Mackintosh, 1975; Miller & Matzel, 1988; Pearce, 1987; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981) all do well in accounting for competition between cues trained together in at least one phase (Cell 1 of Fig. 1; e.g., overshadowing [Pavlov, 1927], blocking [Kamin, 1968], relative stimulus validity [Wagner, Logan, Haberlandt, & Price, 1968], and overexpectation [Rescorla, 1970]). However, only the Miller and Matzel (1988) model can currently explain competition between outcomes (Cell 2 of Fig. 1), as well as competition between cues (see Miller & Escobar, 2002). This relative neglect of competition between outcomes, as compared with competition between cues, might have occurred because evidence of outcome

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Fig. 1 A 2×2 matrix of different types of associative interference and competition, all of which produce a decrease on control of behavior of the target stimulus. In each cell some representative examples are listed (after Miller & Escobar, 2002). O is an outcome; O1 and O2 are outcomes of opposite valence; X and Z are cues. See the text for a discussion

	Interacting Events	
	Antecedent Events	Subsequent Events
Trained Together (Competition)	Cue competition (overshadowing, relative validity, overexpectation) Cell 1	Outcome competition (Rescorla, 1980; Esmoris-Arranz, Matute, & Miller, 1997; Miller & Matute, 1998) Cell 2
Trained Apart (Interference)	Retroactive cue interference, proactive cue interference (e.g., Escobar, Matute, & Miller, 2001; Admundson, Escobar, & Miller, 2003) Cell 3	Retroactive outcome interference, proactive outcome interference (extinction, latent inhibition, counterconditioning) Cell 4

competition is much scarcer (e.g., Esmoris-Arranz, Miller, & Matute, 1997; Miller & Matute, 1998; Rescorla, 1980).

Few models have been developed in a Pavlovian framework to account for associative interference. One example of such models is Bouton's (1993) model of retrieval interference that was developed to account for interference between outcomes trained in different phases (Cell 4 of Fig. 1; e.g., latent inhibition [Lubow & Moore, 1959], extinction, and counterconditioning [Pavlov, 1927]). Less attention has been paid to developing accounts of interference between cues (e.g., Cell 3 of Fig. 1), as compared with interference between outcomes. Bouton's (1993) account of outcome interference does not lend itself to explaining cue interference without some modification. Miller and Escobar (2002) proposed a modification of Bouton's (1993) model that anticipates parallel effects between outcome and cue interference. Basically, they posited that activation of a binary sequential association inhibits activation of other associations containing either element in the same sequential order as the association already activated. Many reports have documented that retroactive outcome interference is subject to recovery, which is exemplified by spontaneous recovery (e.g., Brooks & Bouton, 1993; Pavlov, 1927) and renewal (e.g., Bouton & Bolles, 1979). The question addressed in this report is whether retroactive cue interference is also subject to spontaneous recovery and renewal.

In the case of extinction, reinforced presentations of a conditioned stimulus (CS) in one phase are followed by nonreinforced presentations of that CS in a subsequent

phase. This procedure causes reduced responding to the CS at test, relative to a group that has not received non-reinforced exposure to the target cue. The common element in both phases is the CS, which was followed by two different outcomes: the presence of the unconditioned stimulus (US) in phase 1 and the absence of the US in phase 2. In a broad sense, the response reduction at test is produced because the inhibitory-like learning which occurred during phase 2 interferes with the expression of the excitatory learning that occurred in phase 1 (e.g., Pavlov, 1927). Although other accounts for experimental extinction have been purposed based on erasure of the learning that occurred in phase 1 induced by the nonreinforced presentation of the CS in phase 2 (e.g., Rescorla & Wagner, 1972), these accounts are incompatible with considerable data showing recovery from extinction, which is readily explained by regarding extinction as an instance of retroactive outcome interference.

Although attenuated excitatory responding to the CS is observed soon after extinction treatment, a signature phenomenon of extinction (and outcome interference in general; see Westbrook & Bouton, 2010) is spontaneous recovery. This refers to a long delay between extinction treatment in phase 2 and testing, which results in a partial recovery of the extinguished response (e.g., Brooks & Bouton, 1993; Pavlov, 1927). Sissons and Miller (2009; see also Nelson, 2002), demonstrated that spontaneous recovery of phase 1 training can occur regardless of whether the phase 1 association is inhibitory-like (i.e., nonreinforcement) and the phase 2 association is excitatory, or when the relationship

is reversed (i.e., phase 1 being excitatory and phase 2 being Inhibitory). This suggests that spontaneous recovery depends less on the status of the interfering outcome (i.e., presence or absence) and, instead, depends largely on the phase 2 association's losing its effectiveness with the passage of time (e.g., Bouton, 1993, 1997; Spear, 1976). Spontaneous recovery has also been reported in counterconditioning preparations. In this situation, a CS is paired with one outcome (e.g., appetitive) during phase 1 of training and then is paired with an outcome that provokes an opposing response (e.g., aversive) during Phase 2. Bouton and Peck (1992) found that when testing occurred immediately after the second learning experience (i.e., CS–US2), behavior was controlled by the most recent US; however, with delayed testing, the first-learned association (i.e., CS–US1) came to control responding to the CS.

A second signature phenomenon of extinction (and outcome interference in general; see Westbrook & Bouton, 2010) is renewal. In this situation, partial recovery of an extinguished response occurs when testing takes place outside the extinction context (e.g., Bouton & Bolles, 1979; Bouton & King, 1983; Bouton & Ricker, 1994). There are three types of renewal. ABA renewal is the expression of the phase 1 association when testing occurs back in the phase 1 context after the interfering association was trained in a different context, relative to when phase 2 and testing occur in the same context (e.g., Bouton, & King, 1983). ABC renewal is the expression of the phase 1 association when phase 1 training, phase 2 training, and testing all take place in different contexts (e.g., Bouton & Bolles, 1979). AAC renewal, the weakest of the three renewal effects (e.g., Laborda, Witnauer, & Miller, 2011), is the expression of the phase 1 association after phase 1 and phase 2 take place in the same context and testing occurs in a different context (e.g., Bouton & Ricker, 1994). Renewal after retroactive outcome interference is typically reported when the first outcome enters into an excitatory association (e.g., Bouton & Bolles, 1979; Bouton & King, 1983; Bouton & Ricker, 1994). However, Nelson (2002) reported attenuated responding to the target cue in an AAC condition when phase 1 training was inhibitory-like (i.e., nonreinforcement) and the phase 2 association was excitatory, relative to an AAA condition. This can be viewed as an instance of recovery from retroactive outcome interference, with the first-learned association being inhibitory-like. ABA and AAC renewal of retroactive cue interference have also been reported, relative to an ABB control group in human causal (and predictive) learning by Matute and Pineño (1998a, Experiment 3). ABA renewal of retroactive cue interference has additionally been reported in sensory preconditioning in a conditioned suppression task with rats by Escobar, Matute, and Miller (2001, Experiment 3). These prior findings suggest the generality of recovery

effects following both retroactive outcome interference and retroactive cue interference.

Early reports of spontaneous recovery-like effects in cue retroactive interference come from the verbal-learning literature (e.g., Chandler, 1993; Underwood, 1948); however, verbal-learning tasks possibly make use of cognitive faculties that are not engaged during basic learning tasks like Pavlovian conditioning (see Miller & Escobar, 2002). Similarly, spontaneous recovery-like effects have been demonstrated using various species in serial list learning, in which the relative position of the last items of a list are better recalled if testing occurs with a short delay after the study phase but the position of the initial items are better recalled with long retention intervals (e.g., Wright, Santiago, Sands, Kendrick, & Cook, 1985). More recently, Pineño, Ortega, and Matute (2000) and Luque, Moris, and Cobos (2010) have reported spontaneous recovery of retroactive cue interference in human causal learning, which in some ways is analogous to Pavlovian conditioning. But until now, no evidence of spontaneous recovery of cue interference has been reported in a Pavlovian preparation or in a nonhuman species.

Experiment 1 was designed to illuminate the nature of retroactive cue interference by studying potential spontaneous recovery from retroactive cue interference in a Pavlovian conditioned suppression preparation with rats. Specifically, we asked whether a delay between Phase 2 and testing produces stronger responding to the cue trained in Phase 1 (i.e., spontaneous recovery), relative to groups that did not receive a delay between Phase 2 and testing.. Experiment 2 assessed recovery from retroactive cue interference in an ABC renewal design analogous to those typically studied in retroactive outcome interference (e.g., extinction). To date, no report has been published of spontaneous recovery or ABC renewal following retroactive cue interference in a Pavlovian conditioning preparation.

Experiment 1: Spontaneous recovery

Experiment 1 consisted of four groups with treatment embedded in a sensory preconditioning paradigm (see Table 1). We used a sensory preconditioning procedure because retroactive cue interference is severely attenuated if the target cue acquires high biological significance prior to the interference treatment (Escobar et al., 2001), due to difficulty in attenuating, through indirect means, the response to a cue that has already acquired the potential to elicit a vigorous response (e.g., Denniston, Miller, & Matute, 1996; Escobar et al., 2001; Miller & Matute, 1996; Oberling, Bristol, Matute, & Miller, 2000). In our sensory preconditioning design, we controlled for the time between

the presentation of the biological significance event and testing to equate nonassociative factors evoked by biologically significant events. On the basis of analogous situations with retroactive outcome interference, we expected to observe weak behavioral control in Groups Interfere-Short and Interfere-Long. In those groups, Phase 2 Z–O training occurred close in time (i.e., in the same temporal context; Bouton, 1993) to testing, which should have allowed the latter treatment to interfere with the retrieval of the X–O association at test. We expected to observe strong responding to CS X in Group Spon-Rec because second-learned information concerning O (i.e., Z–O) is prone to disruption during a long retention interval (e.g., Bouton, 1997; Sissons & Miller, 2009), presumably because of the change in temporal contexts that occurs during the retention interval. This should result in a mismatch between the temporal contexts of the interference treatment with that of testing, thereby reducing interference. Therefore, Phase 2 learning should not appreciably interfere with expression of Phase 1 training at test. Group Unpaired was included to control for nonassociative interference from Z and O presentations in Phase 2 by providing unpaired presentations of Z and O, rather than Z–O pairings, during that phase. A second Unpaired group with a long delay between Phase 1 and Phase 2 was omitted because similar preparations have shown no differences in performance from the present Unpaired group (e.g., Miguez, Laborda, & Miller, 2011). All subjects started the experiment and were tested at the same time to control for the duration of the experiment. This was done by inserting a 21-day retention interval at different locations for all the groups

Method

Subjects

The subjects were 24 male and 24 female experimentally naive, Sprague-Dawley-descended rats obtained from our own breeding colony. Body weight ranges were 213–303 g

for males and 160–217 g for females. Subjects were randomly assigned to one of four groups ($n_s = 12$; Spon-Rec, Interfere-Short, Interfere-Long, Unpaired), counterbalanced within groups for sex. The animals were individually housed in standard hanging stainless steel wire mesh cages in a vivarium maintained on a 16:8-h light:dark cycle. Experimental manipulations occurred near the middle portion of the light phase. The animals received free access to Purina Lab Chow, whereas water availability was limited to 20 min per day following a progressive deprivation schedule initiated 1 week prior to the start of the study. Rats were water deprived throughout the experiment. From the time of weaning until the start of the study, all animals were handled for 30 s, 3 times per week.

Apparatus

Six identical copies of each Chamber V and Chamber R were used. The use of two contexts was due to the equipment available. Any given subject experienced only one context. The two contexts were perfectly counterbalanced within each group. Chamber V was a 27-cm-long box in a truncated-V shape (29.5 cm high, 21.5 cm wide at the top, and 5.5 cm wide at the bottom). The floor consisted of two 27-cm-long, 2-cm-wide stainless steel plates with a 1.5-cm gap between the two plates. A 0.7-mA, 0.5-s constant-current footshock, produced by a high voltage AC circuit in series with a 1.0-M Ω resistor, could be delivered through the metal walls and floor of the chamber. The ceiling was clear Plexiglas, the front and back walls were black Plexiglas, and the sidewalls were stainless steel. Each of six copies of Chamber V was housed in a separate sound- and light-attenuating environmental isolation chest. The chamber was illuminated by a 7-W (nominal at 120 VAC, but driven at 50 VAC) incandescent light bulb, which was mounted on the inside wall of the environmental enclosure, approximately 30 cm from the center of the experimental chamber. The light entered the chamber primarily by reflection from the ceiling of the environmental chest.

Table 1 Design summary of Experiment 1

Group	Ret Int 1	Phase 1	Ret Int 2	Phase 2	Ret Int 3	Phase 3	Test
Spon-Rec		4 X–O		4 Z–O	21 days	4 O–US	X
Interfere-Short	21 days	4 X–O		4 Z–O		4 O–US	X
Interfere-Long		4 X–O	21 days	4 Z–O		4 O–US	X
Unpaired	21 days	4 X–O		4 Z / O		4 O–US	X

Note. “Spon-Rec” = spontaneous recovery. “X” was a click train. “Z” was a tone. The outcome (O) was a white noise. “US” denotes a brief footshock. Numbers preceding stimulus pairings indicate total number of presentations in that phase. Numbers in the Ret Int 1, Ret Int 2, and Ret Int 3 columns denote days of retention interval (Ret Int) between acclimation and Phase 1, Phase 1 and Phase 2, and Phase 2 and testing, respectively. Slashes denote interspersed presentation within a session. All training and testing occurred in the same context

Chamber R was rectangular, measuring $24.0 \times 9.0 \times 12.5$ cm ($l \times w \times h$). The walls and ceiling of Chamber R were clear Plexiglas, and the floor consisted of stainless steel rods measuring 0.5 cm in diameter, spaced 1.3 cm apart (center to center). The rods were connected by NE-2 bulbs, which allowed for the delivery of a 0.7-mA, 0.5-s constant-current footshock. Each of six copies of Chamber R was housed in separate light- and sound-attenuating environmental isolation chambers. Each chamber was dimly illuminated by a 2-W (nominal at 120 VAC, but driven at 50 VAC) incandescent houselight mounted on an inside wall of the environmental chest located approximately 30 cm from the animal enclosure. The light intensities inside the two illuminated chambers were approximately equal due to the difference in opaqueness of the walls of Chambers V and R.

Chambers V and R could be equipped with a water-filled lick tube that extended 1 cm into a cylindrical niche, which was 4.5 cm in diameter, left–right centered, with its bottom 1.75 cm above the floor of the apparatus and 5.0 cm deep. There was a photobeam detector 1 cm in front of the lick tube that was broken whenever the subject licked the tube. Three 45- Ω speakers on the inside walls of each isolation chest could deliver a click train (6 Hz) 6 dB above background, a complex tone (500 Hz + 520 Hz) 6 dB above background, and a white noise 6 dB above background. Ventilation fans in each enclosure provided a constant 76-dB background noise (all auditory cues were measured on the C-scale).

CSs X and Z were the click train and complex tone, respectively. The outcome (O) was always the white noise. During training, X, Z, and O were each 5 s in duration. The footshock served as the US. The type of training chamber (R or V) used throughout the experiment was consistent within any given animal but was counterbalanced within each group.

Procedure

Acclimation On Days 1 and 2, all subjects were acclimated to drinking in their experimental context during a daily 60-min session. During the acclimation phase, subjects had free access to the water-filled lick tube. There were no presentations of any nominal stimuli during this phase. At the end of acclimation, the water tubes were removed until reacclimation.

Retention interval 1 (Ret Int 1) On Day 3, the Interfere-Short and Unpaired groups were started on a 21-day retention interval. Subjects given this retention interval were handled for 30 s, 3 times per week.

Phase 1 On Day 3, Groups Spon-Rec and Interfere-Long and on Day 24 Groups Interfere-Short and Unpaired were presented with four X–O pairings (i.e., click–white noise),

starting at 12, 24, 36, and 48 min into a 60-min session. On each of these trials, the onset of O immediately followed the termination of X.

Retention interval 2 (Ret Int 2) On Days 4–24, Group Interfere-Long experienced a 21-day retention interval. These subjects were handled for 30 s, 3 times per week.

Phase 2 Group Spon-Rec received four Z–O (tone–white noise) pairings on Day 4, starting at 12, 24, 36, and 48 min into a 60-min session. On each of these trials, the onset of O immediately followed the termination of Z. Groups Interfere-Long and Interfere-Short received the same treatment on Day 25. Group Unpaired received unpaired presentations of Z and O on Day 25. Presentations of Z occurred at 7, 14, 34, and 47 min, whereas presentations of O occurred at 20, 27, 40, and 54 min into a 60-min session.

Retention interval 3 (Ret Int 3) On Days 5–25, subjects from Group Spon-Rec experienced a 21-day retention interval. These subjects were handled for 30 s, 3 times per week.

Phase 3 On Day 26, all subjects received four O–US pairings, starting at 12, 24, 36, and 48 min into a 60-min session. On each of these trials, O and the US coterminated.

Reacclimation On Days 27 and 28, all subjects were reacclimated to their experimental contexts in two daily 30-min sessions. Sessions were separated by an interval of 2 h. Subjects had free access to the water-filled lick tubes, and no nominal stimuli were programmed to occur. The purpose of these sessions was to reestablish stable baseline drinking behavior, which might have been disrupted by the footshock US of Phase 3.

Test On Day 29, testing on X occurred for all groups. All subjects were tested for conditioned lick suppression to CS X. Upon placement of each subject in the test chamber, time spent drinking was recorded. Immediately after completion of an initial 5 cumulative seconds of licking in the absence of any nominal stimulus, subjects were presented with CS X. Thus, all subjects were drinking at the time of CS onset. Time to complete an additional 5 cumulative seconds of licking in the presence of CS X was recorded. The times recorded during the presentation of CS X were interpreted as reflecting subjects' expectancy of the outcome and, in turn, the US following onset of the CS. The test session was terminated after the animals drank for 5 cumulative seconds during the presence of CS X. A ceiling score of 15 min was imposed on the time to complete 5 cumulative seconds of drinking in the presence of CS X. In practice, no animal reached this score. Maximum session duration was 16 min. Following the

convention of our laboratory, all animals that took more than 60 s to complete their first 5 cumulative seconds of licking (i.e., prior to CS onset) during the test session were scheduled to be eliminated from the study because such long latencies may be considered indicative of unusually great fear of the context. One subject from Group Interfere-Short met this criterion and was eliminated. Additionally, 1 subject from Group Spon-Rec was eliminated because of an equipment malfunction.

Results and discussion

The results are represented in Fig. 2. All test scores were converted to log (base 10) seconds to better approximate the within-group normal distributions assumed by parametric statistics. An alpha level of .05 was adopted for all tests of statistical significance.

A one-way analysis of variance (ANOVA) performed on the data recorded prior to the test presentation of CS (i.e., baseline drinking) revealed no significant differences among groups, $p = .45$, indicating that the groups did not differ appreciably with respect to fear of the experimental context.

A one-way ANOVA performed on the data during the CS revealed an effect of treatment $F(3, 42) = 13.70$, $p < .01$, $MSE = .16$, Cohen's $f = 0.91$. Planned comparisons were conducted to test specific hypotheses. Greater suppression was observed in Group Unpaired relative to Group Interfere-Short, $F(1, 42) = 17.37$, $p < .01$, and relative to Group Interfere-Long, $F(1, 42) = 25.45$, $p < .01$, indicating the occurrence of retroactive cue interference. In other words, training another cue with the same outcome as the target cue retroactively interfered with behavioral control by the target cue at test. More suppression was found in Group Spon-Rec than in Group Interfere-Short, $F(1, 42) = 15.46$, $p < .01$, and Group Interfere-Long, $F(1, 42) = 22.87$, $p < .01$, indicating that the imposition of a long delay between Phase 2 and testing diminished retroactive cue interference. A comparison between Groups Spon-Rec and Unpaired did not detect a

significant difference, $p = .88$, suggesting that spontaneous recovery was relatively complete.

These results indicate that retroactive cue interference was present independently of whether subjects received interference treatment immediately after target training (i.e., Interfere-Short) or after a long delay (i.e., Interfere-Long), in that both of these conditions showed weak conditioned suppression to CS X, relative to Group Unpaired. Retroactive cue interference was not appreciably present when a long delay was imposed between Phase 2 and testing (i.e., Spon-Rec), in that subjects in this condition displayed a strong conditioned suppression relative to both control groups (i.e., Interfere-Short and Interfere-Long), and a similar level of suppression to Group Unpaired. Therefore, a retention interval interposed between the interference phase (i.e., Phase 2) and testing produced a recovery of behavior indicative of the first-learned information. This suggests an important parallel between retroactive cue interference and retroactive outcome interference.

Experiment 2: Renewal

Experiment 2 was a four-group study that assessed, in a retroactive cue interference paradigm, an analogue of the ABC renewal effect that is commonly observed in retroactive outcome interference (e.g., recovery of responding after extinction when the CS is tested in a context different from that of extinction treatment). Although ABA renewal has been reported in retroactive cue interference (Escobar et al., 2001), it is not clear that a reduction of interference will occur when testing is conducted in a neutral context (e.g., Context C) that does not set the occasion for the cue (X) that has been interfered with (as is the case in ABA renewal). Group ABC received X–O pairings in Context A and Z–O pairings in Context B and was tested on X in Context C; therefore, renewal from retroactive cue interference was expected in this group. Group ABB received X–O pairings in Context A and Z–O

Fig. 2 Results of Experiment 1. Mean times to complete 5 cumulative seconds of drinking in the presence of the target CS X. Higher scores represent stronger responding to the test CS. Error bars represent standard error of the mean. See the text and Table 1 for details

pairings in Context B and was tested on X in Context B; therefore, in this group, the test context did not differ from the context in which the second learning treatment occurred, so strong interference was expected. Group ABC-Unp assessed acquisition without associative cue interference. Group Unp-ABC was similar to Group ABC but received unpaired presentations of X and O in Context A. We expected little fear elicited by X in this group, which would verify that the fear responding in Groups ABC and ABC-Unp is specific to the X–O association and is not due to a nonassociative effect of testing in Context C. All groups additionally received unpaired presentations of a third cue (Y) and the outcome in Context C to equate Contexts B and C for nonassociative experience with cues and the outcome. As in Experiment 1, we used a sensory preconditioning design because retroactive cue interference is weak if the outcome is of high biological significance (Escobar et al., 2001). Since the test was conducted in Context B or C, we controlled for the excitatory status of the test context by pairing the outcome with a biologically significant event (i.e., footshock) in both contexts (see Table 2 for details).

Method

Subjects

The subjects were 24 male and 24 female experimentally naive, Sprague-Dawley-descended rats obtained from our own breeding colony. Body weight ranges were 205–305 g for females and 280–369 g for males. Subjects were randomly assigned to one of four groups ($ns = 12$; ABB, ABC, ABC-Unp, Unp-ABC), counterbalanced within groups for sex. The maintenance and housing of subjects were the same as in Experiment 1.

Apparatus

In addition to the six identical copies of each Chamber V and Chamber R used in Experiment 1, 12 copies of Chamber Modified-R were used. Each Chamber Modified-R was the

same as Chamber R, with four modifications: (1) A different instance of Chamber R was used, (2) a clear Plexiglas floor was added, (3) the houselight was off, and (4) a daily drop of 98% methyl salicylate as an odor context was placed onto a small block of wood located inside the isolation chest.

In addition to the clicks, tone, and white noise of Experiment 1, a 5-s buzzer 10 dB above background was available. CS X was always the click train. Z (the interfering cue) and Y were the complex tone and the buzzer, counterbalanced within groups. The outcome (O) was always the white noise. X, Y, Z, and O were 5 s in duration during treatment. The footshock served as the US. Context A was always Chamber Modified-R. Contexts B and C were Chamber V and Chamber R, counterbalanced within groups. The addition of Cue Y was to equate Contexts B and C for nonassociative experience with cues and the outcome. Note that equivalent nonassociative exposure to cues and the outcome was given in Contexts B and C during each phase of the experiment.

Procedure

Acclimation On Days 1 and 2, all subjects were acclimated to Contexts B and C during daily 30-min sessions in each context. Subjects were exposed to Contexts B and C, with order counterbalanced within groups. During the acclimation phase, subjects had free access to water-filled lick tubes. There were no presentations of any nominal stimuli during this phase. At the end of acclimation, the water tubes were removed until reacclimation. Lick tubes were not present in Context A during acclimation.

Phase 1 On Day 3, subjects in Groups ABB, ABC, and ABC-Unp were presented with four X–O (i.e., click–white noise) pairings in Context A, as in Experiment 1. Subjects in Group Unp-ABC received unpaired presentations of X and O. Presentations of X occurred at 7, 13, 36, and 48 min, whereas presentations of O occurred at 20, 27, 40, and 53 min into a 60-min session.

Table 2 Design summary of Experiment 2

Group	Phase 1	Phase 2	Phase 3	Test
ABC	4 (X–O) _A	4 (Z–O) _B / 4 (Y/O) _C	4 (O–US) _B & 4 (O–US) _C	(X) _C
ABB	4 (X–O) _A	4 (Z–O) _B / 4 (Y/O) _C	4 (O–US) _B & 4 (O–US) _C	(X) _B
ABC Unp	4 (X–O) _A	4 (Z/O) _B / 4 (Y/O) _C	4 (O–US) _B & 4 (O–US) _C	(X) _C
Unp ABC	4 (X/O) _A	4 (Z–O) _B / 4 (Y/O) _C	4 (O–US) _B & 4 (O–US) _C	(X) _C

Note. “X” was a click train. “Y” and “Z” were a tone and a buzzer, counterbalanced. The outcome (O) was a white noise. “US” denotes reinforcement with a brief footshock. Numbers preceding parenthesis indicate total number of presentations in that phase. Subscripts “A”, “B,” and “C” after the parenthesis indicate different contexts. “Unp” denotes unpaired treatment given in either Phase 1 (e.g., Group Unp-ABC) or Phase 2 (e.g., Group ABC-Unp). Slashes denote interspersed presentations within a session. Note that Group ABC-Unp, given the counterbalancing between Contexts B and C as well the counterbalancing between Cues Z and Y, is fully equivalent to a group designated ABB-Unp

Phase 2 On Day 4, Groups ABB, ABC, and Unp-ABC received four Z–O pairings in Context B, starting at 12, 24, 36, and 48 min into a 60-min session. On each of these trials, the onset of the outcome immediately followed the termination of Z. Group ABC-Unp, received unpaired Z and O presentations in Context B with the onset of Z at 7, 13, 36, and 48 min into the session and the onset of O at 20, 27, 40, and 53 min into the session. Additionally, all four groups received unpaired presentations of Y and O in Context C. Presentations of Y began at 7, 14, 33, and 47 min into the 60-min session, whereas O began at 20, 27, 40, and 53 min into the session.

Phase 3 In two separate sessions (one in Context B and one in Context C) on Day 5, all subjects received four O–US pairings in each context, starting at 12, 24, 36, and 48 min into a 60-min session. On each of these trials, O and the US coterminated. The order of these sessions was counterbalanced within groups.

Reacclimation On Days 6 and 7, all subjects were reacclimated to Contexts B and C in two daily 30-min sessions (one for each context). The order of these sessions was counterbalanced within groups. Sessions were separated by an interval of 3 h. Subjects had free access to the water-filled lick tubes, and no nominal stimuli were programmed to occur.

Test On Day 8, testing on CS X occurred for all groups. Groups ABC, ABC-Unp, and Unp-ABC were tested in Context C, while Group ABB was tested in Context B. Testing was procedurally identical to that in Experiment 1.

Results and discussion

The results of Experiment 2 are depicted in Fig. 3. A one-way ANOVA performed on the data recorded prior to the

test presentation of CS (i.e., baseline) revealed no significant differences, $p = .34$. An analogous ANOVA performed on latencies to complete 5 s of licking after onset of the target CS (X) revealed a significance effect, $F(3, 44) = 41.31$, $p < .01$, $MSE = .15$, Cohen's $f = 1.65$. Suppression to the target CS was relatively low when it was tested in the context in which the interfering association was trained (Group ABB), which is indicative of retroactive cue interference. A comparison between Groups ABC and ABB detected greater conditioned suppression when subjects were tested outside the context of interference training, $F(1, 44) = 27.26$, $p < .01$, which is indicative of ABC renewal from retroactive cue interference. A significant difference was found between Groups ABC and ABC-Unp, $F(1, 44) = 10.02$, $p < .01$, which suggests a residual interference effect in Group ABC. Suppression in Group Unp-ABC was not different from that in Group ABB, $p = .18$, and, additionally, was significantly lower relative to the other groups that were tested in Context C, smallest $F(1, 44) = 43.34$, $p < .01$, suggesting that it is not the testing in Context C per se that produced high responding. Furthermore, counterbalancing of both Contexts B and C and Cues Y and Z made Group ABC-Unp identical to a group in which unpaired treatment is provided during Phase 2 and is later tested on X in Context B (e.g., effectively Group ABB-Unp). This counterbalancing further rules out any contribution that the physical identity of Context C per se had on responding.

The results indicate that recovery from retroactive cue interference in the form of renewal was observed in the ABC group, which was tested in a context different from that in which the second-learned interfering association was acquired. This is analogous to the ABC renewal effect observed in retroactive outcome interference (e.g., extinction, Bouton & Bolles, 1979; latent inhibition, Wheeler, Chang, & Miller, 2003), thereby suggesting further parallels between retroactive cue interference and retroactive outcome interference.

Fig. 3 Results of Experiment 2. Mean times to complete 5 cumulative seconds of drinking in the presence of the target CS X. Higher scores represent stronger responding to the test CS. Error bars represent standard error of the mean. See the text and Table 2 for details

General discussion

The present series of experiments provides two demonstrations of recovery from retroactive cue interference within a Pavlovian situation. Experiment 1 is, to our knowledge, the first demonstration in a Pavlovian preparation of spontaneous recovery from retroactive cue interference (i.e., attenuated acquired behavior produced by a second cue trained apart from the target cue, but with a common outcome). Experiment 2 demonstrated recovery from retroactive cue interference when testing occurred outside of the contexts in which either the first-learned or the second-learned associations were formed (e.g., ABC renewal of Pavlovian retroactive cue interference).

In Experiment 1, we found that interposing a long delay between interference treatment (i.e., Phase 2) and testing produced a stronger response, relative to groups that did not receive any appreciable delay between interference and testing. Moreover, subjects that experienced a long delay interposed between interference training and testing did not differ significantly from subjects that received unpaired presentations of the potentially interfering cue and the same outcome that was used to form the target association in Phase 1. This indicates that spontaneous recovery was relatively complete. The results of Experiment 1 are congruent with Miller and Escobar's (2002) model, which can be viewed as an extension of Bouton's (1993, 1997) account of renewal. In that framework, spontaneous recovery is viewed as an instance of renewal by considering time as a temporal component of the context (Bouton, 1993). Although Bouton's account of spatial and temporal renewal was developed to explain outcome interference produced by extinction of prior excitatory learning, Miller and Escobar (see also Matute & Pineño, 1998a,b) suggested that this mechanism could also be applied to interference between two cues that share a common outcome but are trained in different sequential phases. In both cases (i.e., spontaneous recovery from retroactive cue interference and renewal from retroactive cue interference), the context of the second-learned association formed in Phase 2 modulates the expression of this association. Thus, if the test context consists of a spatiotemporal context that is the same as or similar to that in which Phase 2 occurred, then the test context will occasion set the retrieval of the second-learned association, allowing it to interfere with the expression of the first-learned association (i.e., that between the target CS and the outcome), as happened for both Groups Interfere-Short and Interfere-Long in Experiment 1 and Group ABB in Experiment 2. Alternatively stated, Miller and Escobar's framework explains the spontaneous recovery from interference observed in Experiment 1 by positing an occasion-setting mechanism in which the occasion setter is the temporal test context that, with a short Phase 2–test

interval, positively occasion sets the Phase 2 association that was acquired in a temporal context similar to the temporal context of testing. With a long retention interval following Phase 2, the temporal context of testing becomes different from the temporal context of Phase 2, thereby reducing retrieval of Phase 2 information and, consequently, reducing interference with retrieval of Phase 1 information. Thus, Miller and Escobar's negative occasion-setting mechanism is reduced by a long delay, but there is no positive occasion-setting mechanism involved in the recovery of the response to the target CS X. Thus, it is unclear how Miller and Escobar can account for the similar levels of responding between the Groups Spon-Rec and Unpaired of Experiment 1 without making further assumptions. Instead, the Miller and Escobar account would seem to predict an averaging of the impact of Phases 1 and 2 in responding to CS X in Group Spon-Rec, because the new temporal context does not specifically occasion set either the Phase 1 or Phase 2 association. An alternative account of the present spontaneous recovery that can explain the nearly complete reversion of behavior to Phase 1 training is provided by Devenport (1998) and Devenport and Devenport (1994), who hypothesized that the weight of a training phase diminishes hyperbolically with increasing passage of time. However, this model fails to account for the renewal effect that we saw in Experiment 2. The occasion-setting mechanism of Miller and Escobar is an extension of Bouton's (1997) and McGeoch's (1932) conception of a priming mechanism, and it is similar to the cognitive construct of *retrieval-induced forgetting* (e.g., Anderson, Bjork, & Bjork, 1994), which refers to decreased accessibility of items stored in memory when the items have recently been retrieved as part of a different association.

Interestingly, the results of Group Interfere-Long in Experiment 1 suggest that a long delay between Phases 1 and 2 does not impair retroactive cue interference, as suggested by Escobar et al. (2001), who reported that a short interval between Phase 1 and Phase 2 decreased responding due to interference to the target CS more than did a long interval between these phases (Escobar et al., 2001, Experiments 1A and 1B). Their finding seems counterintuitive in that a retention interval between the two phases of training should make the temporal context of Phase 2 more dissimilar to that of Phase 1 and, consequently, relatively more similar to that of the test phase. By analogy to renewal, a short Phase 1–Phase 2 interval should be like AAC renewal (which is usually weak), and a long interval should be like ABC renewal (which is usually strong). Therefore, the second-learned association (the interfering one in retroactive cue interference) should be better occasion set by the temporal context at the moment of testing, relative to the first-learned association. In contrast, in the present Experiment 1, retroactive cue

interference was similar regardless of the interval between Phase 1 and Phase 2. One possible explication of the discrepancy between the present study and Escobar et al.'s study, despite the use of similar procedures and apparatus, is a difference in the number of days of the delay between Phase 1 and Phase 2. Escobar et al.'s study used a 14-day retention interval between the interfering phases, while the present research used a longer interval of 21 days. Considering that the two studies obtained similar levels of interference with 24 h between the phases and taking together the present results and those from Escobar et al., one might suppose that the time between phases does not follow a monotonic function to produce interference. However, a parametric study is required to determine the features of that function. Nevertheless, the results of Experiment 1 are consistent with the view that second-learned associations become less accessible with increasing retention intervals between Phase 2 and testing (Bouton, 1993, 1997), as has been demonstrated for excitation and extinction (Bouton & Bolles, 1979), for excitation and inhibition (Sissons & Miller, 2009), and for counterconditioning (Bouton & Peck, 1992), as well as when the interference is created by introducing conflicting temporal relationships between cue–outcome associations across phases (Molet, Urcelay, Miguez, & Miller, 2010) or when interference is produced in serial list learning (e.g., Wright et al., 1985).

Experiment 2 demonstrated recovery from retroactive cue interference when the test context physically differs from that used for either target training (Phase 1) or interference treatment (Phase 2)—that is, an analogue to ABC renewal from extinction. Although Escobar et al. (2001) reported evidence of ABA renewal following retroactive cue interference, ABC renewal should be weaker than ABA renewal because, in an ABA renewal design, the context of testing can set the occasion for the expression of the target association. In an ABC renewal design, the physical context of testing presumably does not set the occasion for the expression of the target CS–outcome association; it merely fails to prime the interfering association. The decrease in responding to X in the ABB condition relative to the Group ABC is explained by Miller and Escobar's (2002) modification of Bouton's (1993) model by assuming that Context B set the occasion for the activation of the Z–O association and, consequently, inhibited retrieval of the target association (i.e., X–O). Bouton's (1997) retrieval model explains recovery from extinction (i.e., retroactive outcome interference) by assuming that extinction learning involves the creation of ambiguity because treatment during the extinction phase contradicts the prior knowledge of reinforcement, and this ambiguity fosters context-specific encoding of the extinction information, which is later used to reduce ambiguity. Therefore, by applying Miller and Escobar's variant of

Bouton's retrieval model of retroactive outcome interference to retroactive cue interference, we speculate that in our preparation the second-learned association (i.e., Z–O) arguably introduces ambiguity because there was already an association with the outcome formed during the first phase (i.e., X–O). Consequently, the second-learned association is more context specific than the target CS–outcome association. This results in the target association generalizing better to a neutral context as in ABC renewal than to the context of second learning. Note that Bouton's account depends on there being a common cue; however, in our design, we used a common outcome, a critical distinction addressed by Miller and Escobar. Additionally, the results of Experiment 2 cannot be accounted for in terms of generalization between the auditory cues X and Z. If generalization had occurred, we would expect similar levels of responding between Group Unp-ABC and Group ABC, which is not what the data indicated.

In both Experiments 1 and 2, we used a sensory preconditioning design to produce the retroactive cue interference effect. The sensory preconditioning design aspect left us with three possible scenarios regarding whether interference occurred at the time of test, Phase 3, or Phase 2. Interference could have occurred during testing if the Z–O–US associative linkage interferes with the expression of the X–O–US associative linkage. Presumably, the activation of the Z–O–US associative linkage is produced by the indirect activation of the representation of O by X at test. Interference could also have occurred during Phase 3, at the moment(s) that O was paired with the US. In this framework, the Z–O interferes with the X–O associative linkage during the presentation of O during the O–US pairings. This interference would down modulate the O–US components of the X–O–US associative linkage that is activated at test. Finally, interference could also have occurred during Phase 2. This mechanism would involve a down modulation of the X–O associative strength due to the interfering Z–O association. Note that the hypothetical mechanism here cannot be a degradation of the contingency underlying the X–O association at Phase 2, given that the number of unpaired presentations of Z and O in the Phase 2 Unpaired condition matched the frequency of the paired Z–O presentations in the paired Phase 2 condition. Any of these three scenarios, or a combination of them, could underlie our data. But in any case, performance was indicative of a decrement produced by pairing two CSs with a common outcome in different sequential phases.

Associative theories have been slow to consider the possibility that similar processes might underlie the phenomena in all four cells of the matrix presented in Fig. 1. Further comparison among cells is needed to determine whether the same phenomena can be observed in each cell. Experiments 1 and 2 contribute to the list of

similarities among the cells of the matrix by providing evidence of further commonalities between interfering cues and interfering outcomes as interacting stimuli (i.e., Cells 3 and 4 of Fig. 1). Recovery from interference in the specific setting of Pavlovian conditioning seems to occur in both retroactive outcome interference (e.g., Cell 4) and retroactive cue interference (e.g., Cell 3). Other phenomena that decrease Pavlovian response could and should be compared to determine whether similar mechanisms govern all kinds of associative reductions in acquired behavior. One example of this is provided by Miguez, Laborda et al. (2011), who have shown that retrospective reevaluation occurs in retroactive cue interference (Cell 3; i.e., extinction of the Phase 2 association prior to testing decreases the interference), as has been reported in cue competition (Cell 1; e.g., Kaufman & Bolles, 1981; Miguez, Witnauer, & Miller, 2011). Comparisons between cue competition (Cell 1) and outcome competition (Cell 2) are scarce. Notably, recovery from outcome overshadowing and blocking has been reported (Burger, Mallemat, & Miller, 2000; Wheeler & Miller, 2005), which is analogous to numerous reports of recovery from cue overshadowing and blocking (e.g., Blaisdell, Gunther, & Miller, 1999; Matzel, Schachtman, & Miller, 1985). Thus, retrospective reevaluation has been observed in all four cells. One can hypothesize that procedures that produce recovery from cue competition can produce recovery from outcome competition as well; however, spontaneous recovery and renewal (observed now in Cells 3 and 4) should not be expected in competition situations (Cells 1 and 2), because training of the interacting stimuli occurs together and, hence, in the same spatiotemporal context.

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