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Spontaneous Recovery From Forward and Backward Blocking

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This article demonstrates and analyzes spontaneous recovery of stimulus control following both forward and backward blocking in a conditioned suppression preparation with rats. Experiment 1 found, in first-order conditioning, robust forward blocking and an attenuation of it following a retention interval. Experiment 2 showed, in sensory preconditioning, recovery of responding following both forward and backward blocking. Also, the results of this experiment indicated that response recovery to the blocked stimulus cannot be explained by an impaired status of the blocking stimulus after a retention interval. Experiment 3, also in sensory preconditioning, suggested that spontaneous recovery following both forward and backward blocking in Experiment 2 was due to impaired associative activation of the blocking stimulus' representation during testing with the blocked stimulus. Although no contemporary model of associative learning can explain these results, a modification of R. R. Miller and L. D. Matzel's (1988) comparator hypothesis is proposed to do so.

Few findings in the associative learning literature have encouraged as much research as Kamin's (1968) report of the blocking effect. *Blocking* refers to impaired responding to a conditioned stimulus (CS), X, as a result of its being paired with an unconditioned stimulus (US) in the presence of another CS, A, which receives additional pairings with the US without CS X, relative to a control group that does not receive the additional A–US pairings. Typically, a blocking experiment consists of two different types of training trials (i.e., A–US and AX–US trials) administered in separate phases followed by the presentation of CS X during testing. When the A–US trials precede the AX–US trials, the response impairment to X is called *forward blocking*, whereas when the A–US trials follow the AX–US trials, it is referred to as *backward blocking*. The forward blocking effect is quite robust, and explaining it has become a benchmark test of contemporary models of associative learning (e.g., Rescorla & Wagner, 1972). In addition, the study of blocking has strengthened the link between animal conditioning and human learning by providing evidence of this effect in diverse species. Whereas forward blocking was first found in conditioned suppression with nonhuman animals by Kamin and then demonstrated with humans in a causal learning preparation by Dickinson, Shanks, and Evenden (1984), backward blocking was originally demonstrated by Shanks (1985) in a causal learning preparation with humans and then replicated in a classical conditioning preparation with nonhuman animals (Denniston, Miller, & Matute, 1996; R. R. Miller & Matute, 1996b).

The first explanation of the forward blocking effect was provided by Kamin (1968), who proposed that it occurred because the US was not surprising during the AX–US trials because of its occurrence being fully predicted by the presence of CS A as a consequence of A's prior training with the US. Therefore, CS X was a redundant CS (i.e., it did not add any new information about the occurrence of the US), and, as a result, CS X failed to be encoded as a predictor of the US. This explanation of the forward blocking effect was formally implemented in Rescorla and Wagner's (1972) model. According to this model, the potential for any CS to gain associative strength on a given trial is a direct function of the difference between the total amount of associative strength supportable by the US (λ) and the associative strength of all the CSs that are present on that trial (V_{Total}). In a forward blocking treatment, because of the previous training of CS A with the US, the value of V_{Total} during the AX–US trials already approaches the value of λ, resulting in little associative strength being available for CS X to acquire. This explanation by the Rescorla–Wagner model, like the original explanation provided by Kamin, implies that forward blocking treatment results in a failure of X to enter into an association with the US.

Although this general view of forward blocking as a learning deficit was incorporated in most models of associative learning (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981), it was rejected by R. R. Miller and Matzel's (1988; see also R. R. Miller & Schachtman, 1985) comparator hypothesis (see Figure 1). According to the comparator hypothesis, forward (as well as backward) blocking is due to a failure to express the X–US association during the presentation of CS X at test. During both forward and backward blocking treatments, three different associations are presumed to be learned involving both CSs and the US: an X–US association, an A–US association, and a within-compound X–A association. The presentation of CS X during testing is assumed to directly activate a representation of

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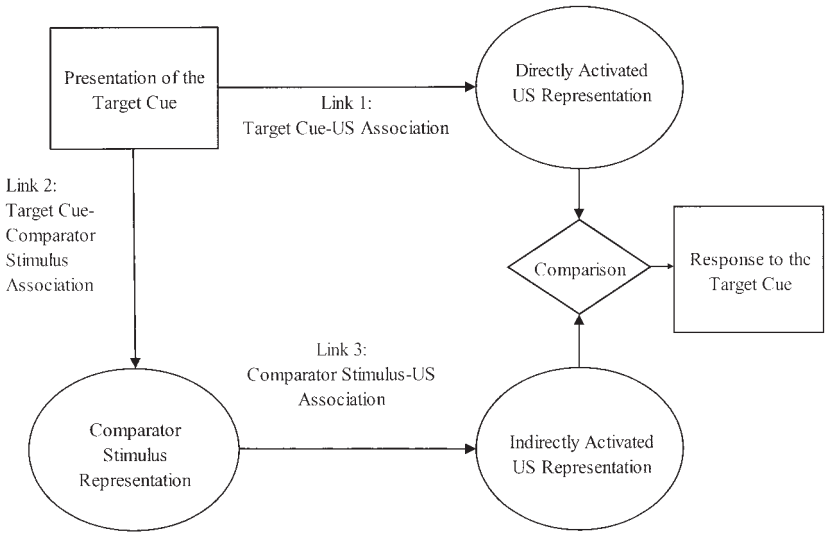

Figure 1. The original comparator hypothesis (after R. R. Miller & Matzel, 1988). US = unconditioned stimulus.

the US through the X–US association (i.e., Link 1 in Figure 1) as well as the representation of A through the X–A within-compound association (i.e., Link 2), which, in turn, indirectly activates a representation of the US through the A–US association (i.e., Link 3). The strength of the conditioned response elicited by CS X during testing is presumed to be a direct function of the strength of Link 1 (i.e., the X–US association) and an inverse function of the product of the strengths of Links 2 and 3 (i.e., the X–A and A–US associations). In other words, responding to X depends on a comparison between the strengths of the direct and indirect activation of the US. In both forward and backward blocking, the strength of the A–US (Link 3) association is greater than the strength of the X–US (Link 1) association because of the additional training with the A–US trials; therefore, less responding to X occurs than in a control condition lacking the A–US pairings. Thus, the comparator hypothesis states that the X–US association is learned but poorly expressed because of its being trained in compound with CS A.

The comparator hypothesis encouraged the view that cue competition effects (e.g., blocking, overshadowing, and relative stimulus validity) represent performance deficits instead of learning deficits as is assumed by traditional models of learning. The performance-deficit view of cue competition has been supported by a number of phenomena. These include recovery of responding to the blocked CS attributable to posttraining manipulations, such as reminder treatments (e.g., US-alone presentations prior to testing; see, e.g., Balaz, Gutsin, Cacheiro, & Miller, 1982; Schachtman, Gee, Kasprow, & Miller, 1983), and extinction of the blocking stimulus following blocking treatment (Arcediano, Escobar, & Matute, 2001; Blaisdell, Gunther, & Miller, 1999; but see Rauhut, McPhee, & Ayres, 1999, for conflicting data). Although recovery from blocking achieved by reminder treatments is exclusively consistent with a performance-deficit view of cue competition, recovery from blocking achieved by posttraining extinction of the blocking CS can be also accounted for by recent revisions of traditional models of learning, such as Dickinson and Burke's (1996) revision of Wagner's (1981) SOP model as well as Van Hamme and Wasserman's (1994) revision of the Rescorla–Wagner (1972) model. According to these models, during extinction training with the blocking CS, the blocked CS gains associative strength. Therefore, this latter phenomenon does not provide exclusive support to either the performance-deficit or acquisition-deficit views of blocking.

A third potential type of response recovery from cue competition is spontaneous recovery. Spontaneous recovery refers to an increase in conditioned responding to the target stimulus over increasing intervals between cue competition training and testing. This phenomenon, like the recovery achieved by reminder treatments, supports a performance-deficit view of cue competition while posing a problem to acquisition-deficit models because the blocked CS is assumed to not increase its associative strength without further training trials. Although spontaneous recovery of responding has been demonstrated after overshadowing (Kraemer, Lariviere, & Spear, 1988) and the relative stimulus validity effect (Cole, Gunther, & Miller, 1997), the study of spontaneous recovery following blocking has received little attention in the literature. Spontaneous recovery from the US-preexposure effect, which is often viewed as forward blocking by the context, was demonstrated by Batsell (1997) and J. S. Miller, Jagielo, and Spear (1993) using conditioned taste aversion preparations. But it remains unclear whether spontaneous recovery could be observed when punctate, instead of contextual, cues serve as the blocking stimulus or whether spontaneous recovery from blocking could be observed in a preparation different from the conditioned taste aversion paradigm. In fact, one study of blocking by a punctate stimulus found enhancement, rather than attenuation, of blocking following a retention interval (J. S. Miller, McKinzie, Kraebel, & Spear, 1996). In addition, there have been no reports of spontaneous recovery from backward blocking.

We performed the present experiments to study the impact of the interpolation of a retention interval between blocking treatment and testing on responding to the blocked CS. Experiment 1 as-

sessed spontaneous recovery from forward blocking in a first-order conditioning design. Experiment 2 used a sensory preconditioning design to determine whether not only forward blocking, but also backward blocking, can be attenuated by a retention interval. Also, this experiment assessed whether the recovery of responding to the blocked CS resulted from a weakened status of the blocking CS after the retention interval. Experiment 3 was performed to evaluate additional explanations of the results of Experiments 1 and 2.

Experiment 1

As previously mentioned, some studies demonstrated spontaneous recovery from the US-preexposure effect (i.e., generally viewed as forward blocking by the context, Batsell, 1997; J. S. Miller et al., 1993). However, one study of blocking by a punctate stimulus found enhancement, rather than attenuation, of blocking following a retention interval (J. S. Miller et al., 1996). In Experiment 1, we used a first-order conditioning design in a lick-suppression preparation and attempted to ascertain whether responding to a forward blocked CS was recovered (i.e., attenuation of forward blocking) or reduced (i.e., enhancement of forward blocking) by interpolating an appreciable retention interval between the blocking treatment and testing of the blocked CS.

In the present experiment (see Table 1), subjects in the forward blocking (FB) condition received A–US trials during Phase 1, followed by AX–US trials in Phase 2. Subjects in the control condition were given B–US trials (instead of A–US trials) during Phase 1. Then all groups were tested on X. For subjects in the delay condition, CS X was tested 20 days after Phase 2, whereas subjects in the no-delay condition were tested with CS X 3 days following Phase 2. In this experiment, weaker conditioned suppression to CS X in Group FB-NoDelay than in Group Control-NoDelay would be indicative of forward blocking. More important, for spontaneous recovery of responding to the blocked CS to be observed, (a) less of a difference in responding to CS X should be observed between Groups FB-Delay and Control-Delay, and (b) responding to X should be stronger in Group FB-Delay than in Group FB-NoDelay.

Method

Subjects

Experiment 1 was run in two identical replications (48 subjects in each replication). In total, the subjects were 48 male (190–400 g) and 48 female (150–265 g) Sprague–Dawley, experimentally naïve, young adult rats that were bred in our colony. The animals were handled three times a week for 30 s from weaning until initiation of the study. Subjects were individually housed and maintained on a 16-hr light/8-hr dark cycle, and experimental sessions occurred roughly midway through the light portion. 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 approximately 2 hr after any scheduled treatment.

Table 1
Design of Experiment 1

Group	Phase 1	Phase 2	Phase 3	Test
FB-NoDelay	12 A → US	4 AX → US	—	1 X
Control-NoDelay	12 B → US	4 AX → US	—	1 X
FB-Delay	12 A → US	4 AX → US	Delay	1 X
Control-Delay	12 B → US	4 AX → US	Delay	1 X

Note. *A* and *B* = complex tone and white noise, counterbalanced; X = click train; US = unconditioned stimulus (footshock); numbers in front of treatments = # of trials; → denotes "followed by"; Delay = 20-day retention interval; FB = forward blocking.

Apparatus

The apparatus consisted of 12 identical rectangular chambers, each measuring 30 × 25 × 32 cm (l × w × h). The walls of each chamber were made of Plexiglas, and the floor was constructed of 0.5-cm diameter rods, spaced 2 cm center-to-center, and connected by NE-2 neon bulbs that allowed a 0.675-mA constant-current footshock to be delivered by means of a high-voltage AC circuit in series with a 1.0-MΩ resistor. Each chamber was housed in an environmental isolation chest, which was dimly illuminated by a houselight (#1820 incandescent bulb) mounted on the ceiling of the experimental chamber. Each chamber was equipped with a water-filled lick tube (opening = 0.3 cm in diameter) that extended 1 cm from the rear of a cylindrical niche, 4.5 cm in diameter, that was left-right centered on one wall, with its axis perpendicular to the wall and positioned 4 cm above the grid floor. An infrared photobeam was projected horizontally across the niche, 1 cm in front of the lick tube. In order to drink from the tube, subjects had to insert their heads into the niche, thereby breaking the infrared photobeam. Thus, the amount of time the photobeam was disrupted could be monitored; this served as our dependent measure. A 45-Ω speaker mounted on the interior back side of each environmental chest could deliver a high-frequency complex tone stimulus (a blend of 3000 and 3200 Hz), 8 dB above background. A second 45-Ω speaker mounted on the ceiling of the experimental chamber was used to deliver a click stimulus (6/s) 8 dB above background. A third 45-Ω speaker mounted on the sidewall of the chamber was used to deliver a white noise stimulus 8 dB above background. Ventilation fans in each enclosure provided a constant 72-dB background noise. All auditory cues were measured on the C-scale. A 150-W bulb (nominal at 120 VAC, driven at 90 VAC) mounted on the interior back of the environmental chest could deliver a flashing (0.5 s on/0.5 s off) light. This light was used in Experiments 2 and 3 but not in Experiment 1.

Procedure

The design of Experiment 1 is summarized in Table 1. Subjects were randomly assigned to one of four groups (FB-NoDelay, Control-NoDelay, FB-Delay, and Control-NoDelay), counterbalanced for sex (n = 24). CS X was the click train. Stimuli A and B were the complex tone and the white noise, counterbalanced within groups. The US was a 0.5-s, 0.675-mA footshock. When presented during training, all CSs were 5 s in duration, and the CS presentations took place at 9, 30, 38, and 53 min into the session. All sessions were 60 min in duration.

Acclimation. On Day 1, all subjects were acclimated to the experimental context for 60 min with the lick tubes present. No nominal stimuli were presented during this session. At the end of this session, the lick tubes were removed.

Phase 1. On Days 2–4, Groups FB-NoDelay and FB-Delay received four A–US pairings, whereas Groups Control-NoDelay and Control-Delay received four B–US pairings.

Phase 2. In the session of Day 5, all groups received four AX–US pairings.

Reacclimation. During reacclimation, the lick tubes were returned to the chambers and no nominal stimulus was presented. These sessions served to restabilize baseline drinking following any potential disruption produced by the footshock USs. All the groups were reacclimated to the experimental context during two 60-min sessions, which took place on

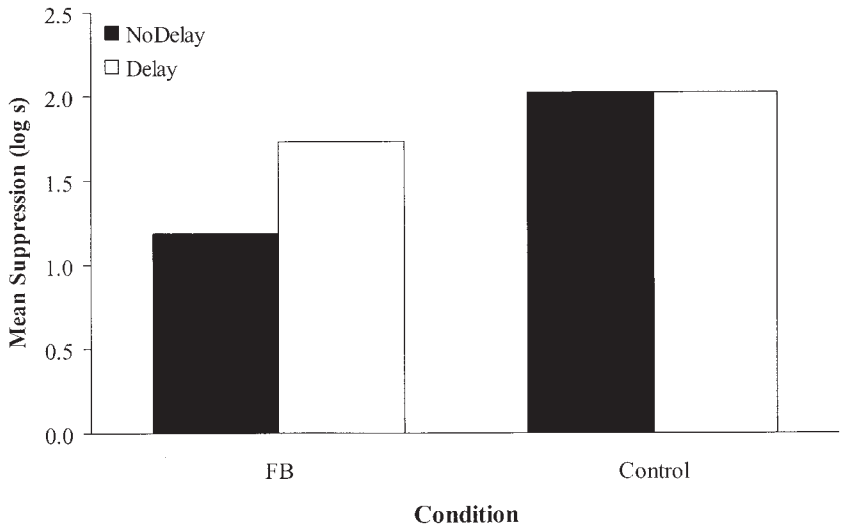

Figure 2. Results of Experiment 1. Analysis of covariance-adjusted mean times to complete 5 cumulative s of drinking in the presence of stimulus X. FB = forward blocking.

Days 6 and 7 for Groups FB-NoDelay and Control-NoDelay and on Days 24 and 25 for Groups FB-Delay and Control-Delay. Between Days 8 and 23, subjects in the delay condition were handled three times per week for 30 s and were maintained on the water deprivation schedule.

Testing. On Day 8, Groups FB-NoDelay and Control-NoDelay were tested for conditioned lick suppression to X. Groups FB-Delay and Control-Delay were tested on suppression to X on Day 26. During this session, subjects were allowed to drink from the lick tubes for 5 cumulative s of drinking in the absence of any CS. Following this initial period of drinking, the target CS, X, was presented. We recorded the time to complete 5 cumulative s of drinking both in the absence of the CS (pre-CS time) and in the presence of the CS (CS time). A 15-min ceiling was imposed on all conditioned lick suppression scores. Subjects that required greater than 60 s to complete the pre-CS time were scheduled to be eliminated from the data analysis because of unusually high fear to the context. No subjects met this criterion in the present experiment. Prior to statistical analysis, all suppression scores and the pre-CS scores were converted to log (base 10) scores to better approximate a within-group normal distribution of scores, as required for the use of parametric statistical analysis. An alpha level of $p < .05$ was adopted for all statistical analyses.

Results and Discussion

As previously mentioned, Experiment 1 was run in two identical replications. Therefore, prior to any analyses on the critical results of Experiment 1, we performed an analysis to ensure the results did not differ between replications. A 2 (replication) × 2 (condition: FB vs. Control) × 2 (delay: No Delay vs. Delay) analysis of covariance (ANCOVA) on the suppression scores in the presence of CS X during testing with the pre-CS scores as a covariate[1] yielded no main effect of replication nor any interaction involving replication as a factor (all $ps > .38$).

The critical results of Experiment 1 are shown in Figure 2. As can be appreciated from the figure, Group FB-NoDelay showed weak conditioned suppression compared with Group Control-NoDelay, thereby indicating that forward blocking occurred. More important, moderate conditioned suppression to X was found in Group FB-Delay. In this group, conditioned suppression was stronger than in Group FB-NoDelay and similar to that of Group Control-Delay. Thus, forward blocking was apparently attenuated when testing was delayed.

We performed a preliminary analysis of variance (ANOVA) in order to examine whether the pre-CS times varied as a function of group. This 2 (condition: FB vs. Control) × 2 (delay: No Delay vs. Delay) ANOVA showed no main effect or interaction (all $ps > .36$).

A 2 (condition: FB vs. Control) × 2 (delay: No Delay vs. Delay) ANCOVA on the suppression scores in the presence of CS X during testing with the pre-CS scores as a covariate yielded main effects of condition, $F(1, 91) = 25.49$, $MSE = 0.29$, $p < .01$, and delay, $F(1, 91) = 6.05$, $MSE = 0.29$, $p < .05$. More important, the Condition × Delay interaction was significant, $F(1, 91) = 6.13$, $MSE = 0.29$, $p < .05$. Planned comparisons using the overall error term from the ANCOVA found conditioned suppression to be weaker in Group FB-NoDelay than in Group Control-NoDelay, $F(1, 91) = 28.16$, $p < .01$. In Group FB-Delay, conditioned suppression was weak relative to Group Control-Delay, but this difference fell short of significance, $F(1, 91) = 3.35$, $p > .07$. Also of importance, conditioned suppression was stronger in Group FB-Delay than in Group FB-NoDelay, $F(1, 91) = 12.18$, $p < .01$, whereas it did not differ between Groups Control-Delay and Control-NoDelay ($p > .98$).

The results of Experiment 1 provided evidence of forward blocking in first-order conditioning using a conditioned lick sup-

[1] Although in Experiments 1 and 3 the pre-CS times did not differ among groups, they were found to marginally differ in Experiment 2. In order to minimize any influence of the pre-CS scores on the analyses with the suppression scores at test, and for the sake of consistency in the analyses of the results across different experiments, we decided to include the pre-CS scores as a covariant in all analyses on the suppression scores at test.

pression preparation (Kamin, 1968) and, more important, demonstrated that conditioned responding elicited by the forward blocked CS can be partially recovered by interpolating a 20-day retention interval between training and testing. The recovery of responding following forward blocking observed after the latter manipulation is consistent with the findings of Batsell (1997; see also J. S. Miller et al., 1993), who reported evidence of spontaneous recovery when a 14-day retention interval was interposed following contextual blocking (i.e., the US-preexposure effect) in a taste-aversion preparation. Our Experiment 1 extends Batsell's findings to forward blocking with a punctate CS in a conditioned lick-suppression preparation. The results of Experiment 1 differ from those of J. S. Miller et al. (1996), who observed enhanced blocking between punctate CSs when testing was delayed, an inconsistency for which we can provide no explanation, although there were a number of procedural differences.

Experiment 2

Experiment 1 found recovery from forward blocking in a first-order preparation. Experiment 2 was designed to replicate the findings of Experiment 1 while extending them to backward blocking. Experiment 2 assessed, in a sensory preconditioning preparation, whether responding to either a forward or backward blocked CS can be recovered by interpolating a retention interval between blocking treatment and testing of the blocked CS. A sensory preconditioning preparation was used in this experiment because previous studies from our laboratory (e.g., Denniston et al., 1996; R. R. Miller & Matute, 1996a, 1996b) showed that backward blocking is difficult to obtain in a first-order conditioning preparation, a result that we attribute to CS X becoming biologically significant in Phase 1 (i.e., during AX–US trials). Therefore, in Experiment 2, instead of directly pairing CSs X and A with the US during training, we paired these CSs with a surrogate US consisting of a neutral stimulus (hereafter referred to as the outcome, O). After training the A–O and AX–O associations, the outcome was paired with a footshock US. Although embedding training within a sensory preconditioning procedure is not necessary to observe forward blocking, we did this for both forward and backward blocking to reduce procedural differences between the two types of blocking. Also, observing spontaneous recovery from forward blocking in a sensory preconditioning preparation would support the generality of Experiment 1's findings.

The design of Experiment 2 is summarized in the top half of Table 2. As can be seen in this table, both forward blocking (FB) and backward blocking (BB) conditions were given training with the AX–O trials in Phase 2. The only difference between these conditions consisted of the phase in which training with the A–O trials took place: the FB condition was given training with the A–O trials in Phase 1 whereas the BB condition received these trials in Phase 3. The control conditions received B–O trials in either Phase 1 or Phase 3. By using this design, training of the target X–O association (during the AX–O pairings) occurred at the same times for all groups. In Phase 4, all groups were given pairings of O with the US (i.e., O–US trials) in order to make O a first-order CS and allow assessment of the X–O association. Finally, all groups were tested on X. For subjects in the delay condition, CS X was tested

Table 2
Designs of Experiments 2 and 3

Group	Phase 1	Phase 2	Phase 3	Phase 4	Phase 5	Test
			Experiment 2			
FB-NoDelay	20 A → O	4 AX → O		4 O → US	—	1 X, 1 A
BB-NoDelay		4 AX → O	20 A → O	4 O → US	—	1 X, 1 A
Control-NoDelay	20 B → O	4 AX → O	—	4 O → US	—	1 X, 1 A
	—		20 B → O			
FB-Delay	20 A → O	4 AX → O		4 O → US	Delay	1 X, 1 A
BB-Delay		4 AX → O	20 A → O	4 O → US	Delay	1 X, 1 A
Control-Delay	20 B → O	4 AX → O	—	4 O → US	Delay	1 X, 1 A
	—		20 B → O			
			Experiment 3			
APre-NoDelay	20 A → O	4 AX → O		4 A → US	—	1 X, 1 A
APost-NoDelay		4 AX → O	20 A → O	4 A → US	—	1 X, 1 A
Control-NoDelay	20 A → O	4 BX → O	—	4 A → US	—	1 X, 1 A
	—		20 A → O			
APre-Delay	20 A → O	4 AX → O		4 A → US	Delay	1 X, 1 A
APost-Delay		4 AX → O	20 A → O	4 A → US	Delay	1 X, 1 A
Control-Delay	20 A → O	4 BX → O	—	4 A → US	Delay	1 X, 1 A
	—		20 A → O			

Note. Split treatments in Phases 1 and 3 indicate half the subjects in the group received the treatment above the line and half the subjects in the group received the treatment below the line. A and B = complex tone and white noise, counterbalanced; X = click train; O = flashing light; US = unconditioned stimulus (footshock); numbers in front of treatments = # of trials; → denotes "followed by"; Delay = 20-day retention interval; FB = forward blocking; APre = A-preexposure; APost = A-postexposure.

20 days after Phase 3, whereas subjects in the no-delay conditions were tested with CS X 3 days following Phase 3. In this experiment, weaker conditioned suppression to CS X in the Groups FB-NoDelay and BB-NoDelay than in Group Control-NoDelay would be indicative of forward and backward blocking, respectively. More important, for spontaneous recovery of responding to the blocked CS to be observed, there must be (a) relative to the no-delay condition, less of a difference in responding to CS X between Groups FB-Delay and Control-Delay and less of a difference in responding to CS X between Groups BB-Delay and Control-Delay and (b) stronger responding to CS X in Groups FB-Delay and BB-Delay than in Groups FB-NoDelay and BB-NoDelay, respectively.

Experiment 2 also included a test of CS A. Testing A is theoretically relevant in the framework of the comparator hypothesis (R. R. Miller & Matzel, 1988) because the recovery of responding to the blocked CS observed after the retention interval in Experiment 1 could have been due either to a weakened activation of the indirect O representation during testing (i.e., due to the effective weakening of either the X–A association or the A–O association or both) or to an impairment of the comparator process per se (i.e., both the X–A and A–O associations may have been functional, but the impact of the indirect O representation on the direct O representation may wane with time). According to the comparator hypothesis, the activation of the indirect O representation during testing of X is a function of the product of the strengths of the X–A and A–O associations (i.e., Links 2 and 3, respectively, in Figure 1). Hence, a weakened indirect activation of the O representation could be due to a weakening of either the X–A association (Link 2) or the A–O association (Link 3) or both. Testing A in the present experiment allowed us to assess the role of the A–O association in the spontaneous attenuation of forward and backward blocking.

Method

Subjects and Apparatus

The subjects were 36 male (260–342 g) and 36 female (178–244 g) Sprague–Dawley, experimentally naïve, young adult rats that were bred in our colony. Subjects were maintained and housed as in Experiment 1. The apparatus was the same as in Experiment 1.

Procedure

The procedural aspects of Experiment 2 were identical to those of Experiment 1, with the following two exceptions. First, the outcome (O), instead of the US, was paired with the CSs during the blocking treatment. The outcome consisted of the 5-s presentation of the flashing light together with the houselight being turned off. On all trials during which O was presented as an outcome (i.e., Phases 1–3), the onset of O coincided with the termination of the CS. Subsequently, O was paired with the footshock US. Second, the present experiment used different numbers of trials.

Acclimation. Acclimation on Day 1 proceeded exactly as in Experiment 1.

Phase 1. On both Days 2 and 3, Groups FB-NoDelay and FB-Delay received 10 A–O pairings, whereas Groups BB-NoDelay and BB-Delay received equivalent context exposure with no nominal stimulus being presented. During this phase, half of the subjects in each of Groups Control-NoDelay and Control-Delay received 10 B–O pairings, whereas the other half of the subjects received equivalent context exposure with no nominal stimulus being presented. Pairings occurred at 3, 10, 17, 20, 26, 33, 41, 48, 52, and 57 min into the session.

Phase 2. On both Days 4 and 5, all groups received two AX–O pairings at 17 and 48 min into the session.

Phase 3. On both Days 6 and 7, Groups BB-NoDelay and BB-Delay received 10 A–O pairings, whereas Groups FB-NoDelay and FB-Delay received equivalent context exposure with no nominal stimulus being presented. During this phase, the subjects in Groups Control-NoDelay and Control-Delay that were given context exposure during Phase 1 received 10 B–O pairings, whereas those subjects that received the 10 B–O pairings during Phase 1 received equivalent context exposure with no nominal stimulus being presented. The distribution of pairings was the same as in Phase 1.

Phase 4. On Day 8, all groups received four O–US pairings 9, 30, 38, and 53 min into the session. During this first-order conditioning, the onset of the footshock US coincided with the termination of O.

Reacclimation. Reacclimation proceeded as in Experiment 1. Reacclimation took place on Days 9 and 10 for Groups FB-NoDelay, BB-NoDelay, and Control-NoDelay and on Days 25 and 26 for Groups FB-Delay, BB-Delay, and Control-Delay.

Testing. We tested Groups FB-NoDelay, BB-NoDelay, and Control-NoDelay for conditioned lick suppression to X and A on Days 11 and 12, respectively. We tested Groups FB-Delay, BB-Delay, and Control-Delay for conditioned lick suppression to X and A on Days 27 and 28, respectively. Both test sessions proceeded identically to those of Experiment 1 except that a 10-min ceiling (instead of the 15-min ceiling of Experiment 1) was imposed on all conditioned lick-suppression scores. Also, during the first test, CS X was left on for 10 min for all subjects to equate experience within and across groups so that testing of CS X would not differentially influence the subsequent test of CS A. In this experiment, no subject took longer than 60 s to complete the first 5 cumulative s of drinking prior to the CS presentation on either test.

Results and Discussion

As can be observed in the left panel of Figure 3, the results of testing of X showed that both forward and backward blocking effects were found in the present experiment. Also, the results of this experiment show that interposing a retention interval between training and testing resulted in spontaneous recovery of responding following both forward and backward blocking effects. Therefore, this experiment replicated the findings of Experiment 1. Moreover, the results of the test of A in this experiment (see right panel of Figure 3) showed that responding to this CS did not change in the forward and backward blocking conditions because of the retention interval. Therefore, to the extent that one can draw conclusions from null results, the recovery of responding observed during the test of X cannot be accounted for by a decrease in the effectiveness of A as a predictor of O (i.e., the A–O association remained functional).

We performed two preliminary analyses in this experiment. The first of them compared responding between the subjects in the control condition that received the B–O trials in Phase 1 and Phase 3. A 2 (group: Control-NoDelay vs. Control-Delay) $\times$ 2 (phase of B–O trials: 1 vs. 3) ANOVA on X test scores showed no main effect of the phase of B–O trials, $p > .67$, nor any Group $\times$ Phase of B–O Trials interaction, $p > .66$. An analogous ANOVA on suppression to A also found no main effect of the phase of B–O trials, $p > .91$, nor any Group $\times$ Phase of B–O Trials interaction, $p > .97$. Therefore, this analysis shows that responding during testing of X and A in the control groups was not appreciably affected by the order in which the B–O and AX–O trials were

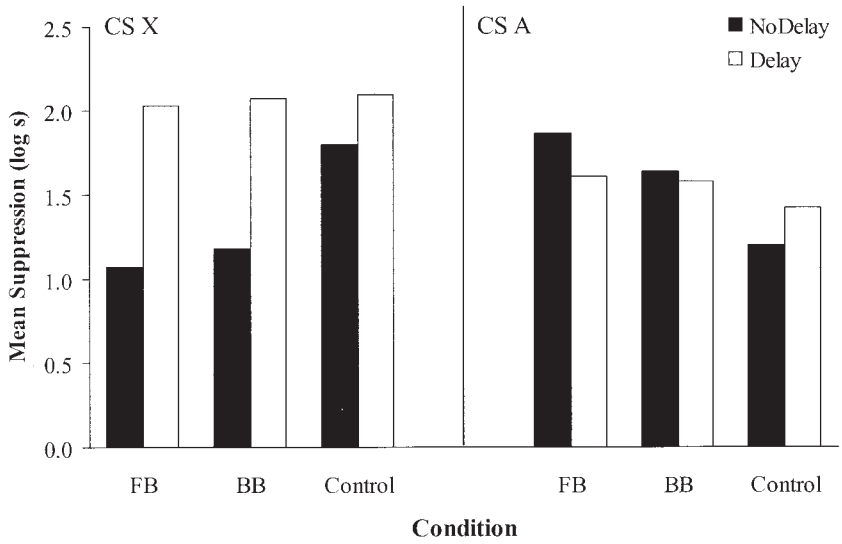

Figure 3. Results of Experiment 2. Analysis of covariance-adjusted mean times to complete 5 cumulative s of drinking in the presence of the blocked stimulus X (left panel) and the blocking stimulus A (right panel). FB = forward blocking; BB = backward blocking.

given. Thus, in the following analyses, data from those subjects in Group Control-NoDelay that received the B–O trials in Phases 1 or 3 were pooled across subjects, as were data from Group Control-Delay group.

The second preliminary analysis was performed in order to examine whether the pre-CS times differed among groups on each test trial. A 3 (condition: FB vs. BB vs. Control) × 2 (delay: No Delay vs. Delay) ANOVA on the mean time to complete 5 cumulative s of drinking during the pre-CS period for the test of X showed a marginal Condition × Delay interaction, $F(2, 66) = 2.78$, $MSE = 0.03$, $p < .07$. The main effects of condition and delay were not significant, ps $> .31$. An identical ANOVA performed on the mean time to complete 5 cumulative s during the pre-CS period for the test of A also showed a marginal Condition × Delay interaction, $F(2, 66) = 2.85$, $MSE = 0.05$, $p < .07$, but no main effects of condition and delay, ps $> .31$. As in Experiment 1, in order to make sure that these different pre-CS scores did not affect conditioned suppression to X and A during test, the analyses of the suppression scores during the test CS presentations included the pre-CS scores as a covariant.

A 3 (condition: FB vs. BB vs. Control) × 2 (delay: No Delay vs. Delay) ANCOVA on the suppression scores during the presentation of CS X at test with the pre-CS X scores as a covariate showed a main effect of both condition, $F(2, 65) = 7.95$, $MSE = 0.13$, $p < .01$, and delay, $F(1, 65) = 70.20$, $MSE = 0.13$, $p < .01$, as well as a Condition × Delay interaction, $F(2, 65) = 5.98$, $MSE = 0.13$, $p < .01$. Planned comparisons using the overall error term from the ANCOVA found conditioned suppression to be weaker in Group FB-NoDelay than in Group Control-NoDelay, $F(1, 65) = 23.71$, $p < .01$, as well as in Group BB-NoDelay than in Group Control-NoDelay, $F(1, 65) = 16.52$, $p < .01$. Thus, both forward and backward blocking were found in the present experiment. In the delay condition, neither forward (FB-Delay vs. Control-Delay) nor backward blocking (BB-Delay vs. Control-Delay) were found, ps $> .65$. Of importance, conditioned suppression to X increased because of the interpolation of the long retention interval before testing. Conditioned suppression to X was stronger in Group FB-Delay than in Group FB-NoDelay, $F(1, 65) = 39.42$, $p < .01$, as well as in Group BB-Delay than in Group BB-NoDelay, $F(1, 65) = 35.53$, $p < .01$. Suppression to X also increased somewhat after the retention interval in the control condition, as revealed by a comparison between Groups Control-NoDelay and Control-Delay, $F(1, 65) = 4.10$, $p < .05$. Another important finding was that suppression to X in Group Control-Delay did not increase nearly as much as in Groups FB-Delay and BB-Delay. That is, despite the increase in suppression to X in Group Control-Delay, there was no evidence of blocking in Groups FB-Delay and BB-Delay, $p > .65$. Moreover, the similarity of the delay groups was not due to a ceiling effect (ceiling was 2.95 log s).

A 3 (condition: FB vs. BB vs. Control) × 2 (delay: No Delay vs. Delay) ANCOVA on the suppression scores during the presentation of CS A at test with the pre-CS A scores as a covariate showed a main effect of condition, $F(2, 65) = 10.91$, $MSE = 0.10$, $p < .01$, and a Condition × Delay interaction, $F(2, 65) = 3.39$, $MSE = 0.10$, $p < .05$. The main effect of delay was not significant, $p > .71$. Planned comparisons using the ANCOVA's overall error term showed that the conditioned suppression to CS A was weaker in Group Control-NoDelay than in Groups FB-NoDelay, $F(1, 65) = 25.70$, $p < .01$, and BB-NoDelay, $F(1, 65) = 11.16$, $p < .01$, which is consistent with Group Control-NoDelay having received fewer A–O pairings. Suppression to CS A did not differ between Groups FB-NoDelay and BB-NoDelay, $p > .09$. When the test of A took place after the retention interval, suppression to CS A in Group Control-Delay did not differ from either Groups FB-Delay, $p > .17$, or BB-Delay, $p > .24$. Also, suppression to A in Groups FB-Delay and BB-Delay was statistically equivalent, $p > .84$. Suppression to A did not vary as a function of the retention interval within the BB condition, $p > .66$. The Condition × Delay interaction arose because there was a tendency for suppression to A to

decrease over time in the FB condition and to increase in the control condition, although neither difference was significant ($ps > .06$).

In summary, the results of Experiment 2 showed evidence of both forward and backward blocking; in addition, the results demonstrated that both forward and backward blocking effects were attenuated by interposing a retention interval between training and testing (i.e., suppression to the blocked CS was spontaneously recovered). Experiment 2 also showed that the recovery of suppression to the blocked CS, X, in the delay condition cannot be explained as arising from a decrease in suppression to the blocking CS, A. These results clearly show that, after a retention interval, stimulus control by both A and X was strong.

As previously mentioned, the comparator hypothesis (R. R. Miller & Matzel, 1988) could explain the recovery of suppression to X observed in the delay condition as a decrease in the effectiveness of A as a comparator stimulus. This decreased effectiveness of A as a comparator stimulus could have been due to CS A having lost its potential to control behavior during the retention interval (i.e., a weakening of the A–O association). In fact, previous studies achieved a recovery of responding to a blocked CS, X, through posttraining extinction of the comparator stimulus, A (Arcediano et al., 2001; Blaisdell et al., 1999). Clearly, the observed robust suppression to A argues against a similar mechanism in the present case.

According to the comparator hypothesis, however, a reduced effectiveness of CS A as a comparator stimulus can be achieved with posttraining manipulations other than the nonreinforced postexposure (i.e., extinction) of CS A. Because the indirect activation of the O representation during testing depends on the product of the strengths of Links 2 and 3, a weakening of either of these links would influence responding to CS X. That is, as the posttraining extinction of CS A involves not only the presentation of A without O but also without CS X, the presentation of A-alone trials will produce, in addition to a weakening of Link 3 (i.e., the A–O association), a weakening of Link 2 (i.e., the X–A association). Although the present experiment demonstrates that the A–O association remained functional through the retention interval (i.e., a weakening of Link 3 cannot explain recovery of responding to CS X), this experiment did not assess the activation of CS A's representation during the presentation of CS X at test. Although speculative, if the effectiveness (i.e., retrieval or expression) of the X–A association waned during the retention interval in the delay condition, this should have released X from blocking by A.

Another alternative explanation of the results of Experiment 2 would be based on a flattening of generalization gradients. This explanation is based on evidence showing that generalization among different stimuli can increase over time, or, in other words, that the animal might fail to discriminate between the different stimuli after a retention interval (e.g., Riccio, Ackil, & Burch-Vernon, 1992; Riccio, Rabinowitz, & Axelrod, 1994; Riccio, Richardson, & Ebner, 1984). If generalization increases over time, then responding to the different stimuli will become more similar (e.g., McAllister & McAllister, 1963; McAllister, McAllister, & Franchina, 1965; Perkins & Weyant, 1958; Thomas & López, 1962). In this alternative view, our present results would merely reflect a failure in the delay condition to differentiate CS X from CS A. Simply put, if the subjects of Groups FB-Delay and BB-Delay could not differentiate the different CSs, they would respond to the blocked CS as if it was the blocking CS, and vice versa. Moreover, because of the different number of pairings with O received by CSs A and X (i.e., 24 A–O trials and 4 X–O trials) during training, the effect of increased generalization should have affected responding to X and A asymmetrically. That is, the enhancement of responding to X should have been greater than the reduction of responding to A. Alternatively, the results of Experiments 1 and 2 could be seen as showing an increase of fear over the retention interval (i.e., incubation of fear; Eysenck, 1968). Although incubation of fear should similarly affect all the animals tested after the long delay, this increase of fear might have been observed in Groups FB-Delay and BB-Delay but not in Group Control-Delay due to responding in this last group being subject to a ceiling effect (i.e., fear responding in this group was already strong). The next experiment assessed the adequacy of these alternative explanations for the results of Experiments 1 and 2.

Experiment 3

Experiment 3 contrasted the alternative accounts of the results of Experiments 1 and 2 by using a variation of the design of Experiment 2 (see Table 2). Specifically, as occurred in the FB and BB conditions of Experiment 2, in Experiment 3 subjects in the A-preexposure (APre) and A-postexposure (APost) conditions were exposed to AX–O trials in Phase 2 and A–O trials in either Phase 1 (APre condition) or Phase 3 (APost condition). However, in Experiment 3 the presentation of O in these trials was irrelevant and was maintained only to minimize differences with Experiment 2 (see bottom half of Table 2). The critical difference between Experiments 2 and 3 consisted of the pairings of CS A (instead of O) with the US during Phase 4 of Experiment 3. Therefore, although the APre and APost conditions in the present experiment were exposed to the same training with the A–O and AX–O trials as the FB and BB conditions in Experiment 2, the pairings of CS A (instead of O, as in Experiment 2) with the US converted the forward and backward blocking design of Experiment 2 into a sensory preconditioning design. In Experiment 3, the effect of a retention interval on preexposure and postexposure to CS A was assessed within a sensory preconditioning procedure designed to maximize similarities to the design of Experiment 2.

Experiment 3 included not only a test of CS X, but also a test of CS A in order to ascertain whether suppression to the first-order CS is affected by the retention interval. If sensory preconditioning (as assessed by responding to CS X) dissipates with time and first-order conditioning (as assessed by responding to CS A) is not affected by the retention interval, then these results could be viewed as indicative of a weakening in the effectiveness of the X–A within-compound association with the passage of time.

In addition, the present experiment allowed us to evaluate two additional explanations of the results of Experiments 1 and 2, namely, that the enhanced response to the blocked CS observed in the delay condition arose from either a flattening of the generalization gradients of CSs A and X or incubation of fear over the retention interval. As previously mentioned, these accounts could both explain the strong suppression produced by CS X in Groups FB-Delay and BB-Delay of Experiment 2 by assuming that either CS X became more similar to CS A after the retention interval (i.e., flattening of the generalization gradients) or that fear to the CSs increased over the retention interval (i.e., incubation of fear).

Therefore, both of these accounts of spontaneous attenuation of blocking predict that, in the present experiment, suppression to X will increase over the retention interval. By contrast, if the results of Experiments 1 and 2 are due to the effectiveness of the X–A within-compound association waning over the retention interval (as in our proposed account based on the comparator hypothesis), suppression to CS X should decrease with time.

Method

Subjects and Apparatus

The subjects were 36 male (175–320 g) and 36 female (175–205 g) Sprague–Dawley, experimentally naïve, young adult rats that were bred in our colony. Subjects were maintained and housed as in Experiments 1 and 2. The apparatus was the same as in Experiments 1 and 2.

Procedure

Unless otherwise stated, the procedures of Experiment 3 were identical to those of Experiment 2.

Acclimation. Acclimation on Day 1 proceeded exactly as in Experiments 1 and 2.

Phase 1. On both Days 2 and 3, Groups APre-NoDelay and APre-Delay as well as half of the subjects in Groups Control-NoDelay and Control-Delay received 10 A–O pairings, whereas Groups APost-NoDelay and APost-Delay as well as the other half of the subjects in Groups Control-NoDelay and Control-Delay received equivalent context exposure with no nominal stimulus being presented.

Phase 2. On both Days 4 and 5, Groups APre-NoDelay, APost-NoDelay, APre-Delay, and APost-Delay received two AX–O pairings, whereas Groups Control-NoDelay and Control-Delay were given two BX–O pairings.

Phase 3. On both Days 6 and 7, Groups APost-NoDelay and APost-Delay received 10 A–O pairings, whereas Groups APre-NoDelay and APre-Delay received equivalent context exposure with no nominal stimulus being presented. During this phase, the subjects in Groups Control-NoDelay and Control-Delay that were given context exposure during Phase 1 received 10 A–O pairings, whereas those subjects that received the 10 A–O pairings during Phase 1 received equivalent context exposure with no nominal stimulus being presented.

Phase 4. On Day 8, all groups received four A–US pairings.

Reacclimation. Reacclimation proceeded as in Experiments 1 and 2. Reacclimation took place on Days 9 and 10 for Groups APre-NoDelay, APost-NoDelay, and Control-NoDelay and on Days 25 and 26 for Groups APre-Delay, APost-Delay, and Control-Delay.

Testing. Groups APre-NoDelay, APost-NoDelay, and Control-NoDelay were tested for conditioned lick suppression to X and A on Days 11 and 12, respectively, using the procedures of Experiment 2. Groups APre-Delay, APost-Delay, and Control-Delay were tested for conditioned lick suppression to X and A on Days 27 and 28, respectively. On the basis of the preconditioned stimulus rejection criterion used in all of the experiments, in this study 2 subjects from Group Control-Delay were removed from the analyses because they took longer than 60 s to complete the first 5 cumulative s of drinking prior to the presentation of X. In addition, the data of 6 subjects (one from each group) were eliminated from the analyses because of an equipment failure.

Results and Discussion

Figure 4 shows the results of Experiment 3. As can be observed in the left panel of this figure, sensory preconditioning took place in both APre and APost conditions when X was tested soon following training (i.e., the no-delay condition). However, when X was tested following a long retention interval (i.e., the delay condition), no evidence of sensory preconditioning was found. An important finding was that the weaker conditioned suppression to CS X in the delay condition could not be explained as arising from a weakening of the A–US association, as shown by the results of the test of A (see right panel of Figure 4). Responding to CS A did not appear to differ among groups within either the no-delay or delay conditions. Also, responding to CS A was similar across the no-delay and delay conditions. These impressions were confirmed by the following analyses.

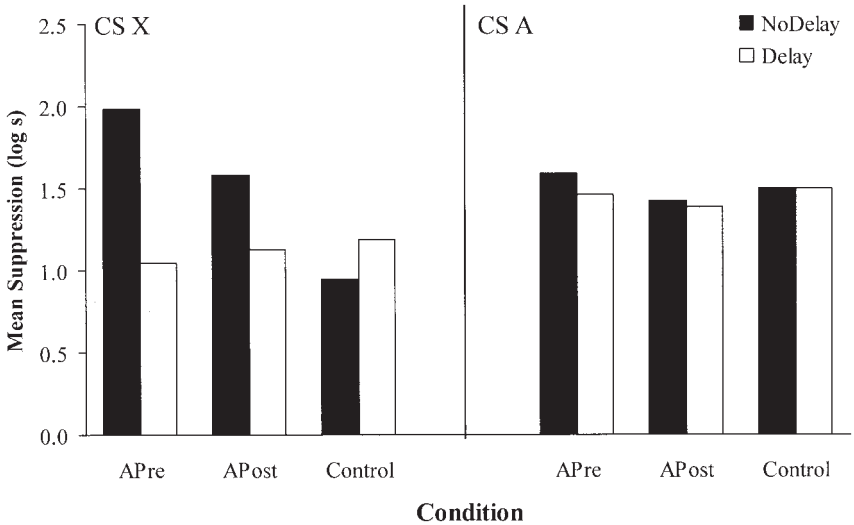

Figure 4. Results of Experiment 3. Analysis of covariance-adjusted mean times to complete 5 cumulative s of drinking in the presence of the sensory preconditioned stimulus X (left panel) and the first-order conditioned stimulus A (right panel). APre = A-preexposure; APost = A-postexposure.

As in Experiment 2, we performed two preliminary analyses. The first of them compared responding to X between the subjects of the control groups that received the A–O trials in Phase 1 and Phase 3. A 2 (group: Control-NoDelay vs. Control-Delay) × 2 (phase of A–O trials: 1 vs. 3) ANOVA on X test scores showed no main effect of the phase of A–O trials, $p > .95$, nor any Group × Phase of A–O Trials interaction, $p > .38$. An analogous ANOVA on the test of A also found no main effect of the phase of A–O trials, $p > .54$, nor any Group × Phase of A–O Trials interaction, $p > .74$. Therefore, this analysis shows that responding during testing of X and A in the control groups was not affected by the order in which the A–O and BX–O trials were given. Thus, in the following analyses, data from those subjects in Group Control-NoDelay that received the A–O trials in Phases 1 or 3 were pooled across subjects, as were data from the Control-Delay group.

The second preliminary analysis was performed to examine whether the pre-CS scores differed among groups on each test trial. A 3 (condition: APre vs. APost vs. Control) × 2 (delay: No Delay vs. Delay) ANOVA on the mean time to complete 5 cumulative s of drinking during the pre-CS period at test of X showed no main effect or interaction, ps $> .42$. An analogous ANOVA performed on the mean time to complete 5 cumulative s during the pre-CS period for the A test also showed no main effect or interaction, ps $> .14$.

A 3 (condition: APre vs. APost vs. Control) × 2 (delay: No Delay vs. Delay) ANCOVA on the suppression scores during the presentation of CS X at test with the pre-CS X scores as a covariate showed a main effect of both condition, $F(2, 57) = 7.85$, $MSE = 0.13$, $p < .01$, and delay, $F(1, 57) = 17.29$, $MSE = 0.13$, $p < .01$, as well as a Condition × Delay interaction, $F(2, 57) = 13.51$, $MSE = 0.13$, $p < .01$. Planned comparisons between Groups APre-NoDelay and Control-NoDelay using the overall error term from the ANCOVA revealed that sensory preconditioning had occurred despite preexposure of CS A during Phase 1, $F(1, 57) = 43.50$, $p < .01$. Sensory preconditioning was also observed despite postexposure of CS A during Phase 3, as shown by the comparison between Groups APost-NoDelay and Control-NoDelay, $F(1, 57) = 16.60$, $p < .01$. In the delay condition, these differences between Groups APre-Delay and Control-Delay, as well as between Groups APost-Delay and Control-Delay were not observed, ps $> .39$. Moreover, responding to X decreased because of the interpolation of the retention interval between training and testing in both the APre and APost conditions. Conditioned suppression to X was weaker in Group APre-Delay than in Group APre-NoDelay, $F(1, 57) = 35.81$, $p < .01$, as well as in Group APost-Delay compared with Group APost-NoDelay, $F(1, 57) = 8.40$, $p < .01$, but it did not differ between Groups Control-NoDelay and Control-Delay, $p > .14$. Moreover, responding to CS X was stronger in Group APre-NoDelay than in Group APost-NoDelay, $F(1, 57) = 6.41$, $p < .05$, thereby suggesting that presenting A–O trials following AX–O trials partially extinguished the X–A association. In contrast, responding to X did not differ between Groups APre-Delay and APost-Delay, $p > .60$.

A 3 (condition: APre vs. APost vs. Control) × 2 (delay: No Delay vs. Delay) ANCOVA on the suppression scores during the test presentation of CS A with the pre-CS A scores as a covariate showed no main effect or interaction (all ps $> .41$). Therefore, this ANCOVA shows that responding to CS A was unaffected by its being tested after the retention interval.

It may seem strange that suppression in some groups was stronger to X than to A when A rather than X was directly paired with the US, but X (clicks) was seemingly more salient than A (tone or white noise). Moreover, X was always tested first and likely there was some generalization of extinction from the X test trial to the A test trial.

Overall, these results demonstrate that sensory preconditioning wanes with long retention intervals. Thus, the results of this experiment refute an explanation of the results of Experiments 1 and 2 based on either a flattening of the generalization decrements of CSs X and A or incubation of fear because both of these views predicted that responding to CS X, if anything, should increase over the retention interval. Moreover, because suppression to the first-order CS was not affected by the retention interval, to the extent that we can draw a conclusion from a null result, the weak responding to CS X after a retention interval cannot be explained as arising from an impaired potential of CS A to activate the representation of the US. Therefore, the most plausible explanation of these results is that CS X was impaired in its potential to activate the representation of CS A during delayed testing. In other words, the effectiveness of the X–A within-compound association seemed to wane over the retention interval.

General Discussion

The present series of experiments demonstrates forward and backward blocking as well as spontaneous recovery from both forward and backward blocking. Experiment 1 used a first-order conditioning design and showed that suppression to a CS, X, that previously underwent forward blocking was recovered when testing was delayed 20 days. Experiment 2 used a sensory preconditioning design to replicate the finding of Experiment 1 while also extending it to backward blocking. This experiment additionally suggested that the recovery of suppression to the blocked stimulus was not due to an impaired association between the blocking stimulus, A, and O after a retention interval because suppression to CS A at test was not appreciably affected by the retention interval. Finally, Experiment 3 showed that sensory preconditioning is attenuated over a retention interval, a result that allowed us to reject alternative accounts for the results of Experiments 1 and 2 based on a flattening of the generalization gradients of CSs X and A as a function of passage of time (e.g., McAllister & McAllister, 1963; McAllister et al., 1965; Perkins & Weyant, 1958; Thomas & López, 1962) or incubation of fear over the retention interval (Eysenck, 1968). This experiment also found that responding to the first-order CS, A, was not significantly affected by the retention interval. Therefore, the decrease of responding to CS X suggests that the effectiveness of the within-compound X–A association waned during the retention interval.

The results of Experiments 1 and 2 are problematic for all contemporary models of associative learning. First, some of these models are able to explain forward but not backward blocking (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981). Second, according to these models forward blocking consists of an acquisition deficit; therefore, spontaneous recovery from forward blocking cannot be explained by these models. Even those models that were developed in order to explain backward blocking, such as those of Dickinson and Burke (1996) and Van Hamme and Wasserman (1994), are unable to explain

spontaneous recovery of responding following either forward or backward blocking. The attentional explanation of forward and backward blocking proposed by Kruschke and Blair (2000) can explain spontaneous recovery from backward blocking, but not from forward blocking. According to this model, backward blocking is due to learned inattention to CS X after the acquisition of the X–O association, whereas forward blocking is due to impaired learning of the X–O association. Therefore, if attention to CS X is assumed to increase over the retention interval (Robbins, 1990), based on Kruschke and Blair's model one could expect conditioned responding elicited by X to recover after backward blocking but not after forward blocking. Finally, the retrieval-failure view (e.g., Bouton, 1993; R. R. Miller, Kasprow, & Schachtman, 1986; Spear, 1971) predicts a shift from recency to primacy following a retention interval. This shift to primacy was expected to have opposite effects on forward and backward blocking. In forward blocking, the blocking A–O association should be retrieved following the retention interval, therefore causing enhanced blocking of the X–O association (see J. S. Miller et al., 1996). However, in backward blocking, both the A–O and X–O associations would be retrieved following the retention interval (i.e., a shift to primacy should favor retrieval of the memory of the AX–O trials), therefore allowing a stronger expression of the X–O association during testing.

Therefore, the above models are unable to explain the recovery of suppression to the forward and backward blocked CS observed following the long retention interval. Moreover, it is hard to see how these models could be readily adapted in order to explain the results of Experiments 1 and 2. Although speculative, one theory that might be adapted to explain these results is the comparator hypothesis (R. R. Miller & Matzel, 1988). According to this theory, both forward and backward blocking are due to the blocking association (A–O) impairing the expression of the X–O association during testing through the processes described in the introduction. A priori, it is difficult to see how a manipulation like the interpolation of a retention interval following a blocking treatment can weaken the X–A (Link 2) or A–O (Link 3) associations that presumably underlie blocking. However, although the results of Experiment 2 show that Link 3 was not affected by the retention interval (i.e., responding to CS A at test did not differ as a function of the delay), the results of Experiment 3 indicated that interposing a retention interval weakened the effectiveness of the within-compound association between CSs X and A. Therefore, the finding suggesting a weakened effectiveness of the X–A association in the delay condition that was found in Experiment 3, in the framework of the comparator hypothesis, could be viewed as the basis of spontaneous recovery from forward blocking in Experiment 1 and from both forward and backward blocking in Experiment 2.

Regardless of their theoretical interpretation, the empirical results of Experiments 1 and 2 can be taken as the first systematic demonstration of spontaneous recovery from both forward and backward blocking. The observation that responding to the blocked conditioned stimulus recovered over a retention interval is consistent with demonstrations of spontaneous recovery from other instances of competition between CSs, such as overshadowing (Kraemer et al., 1988) and relative validity (Cole et al., 1997). Response recovery from forward blocking has also been achieved by giving US-alone trials prior to testing (Balaz et al., 1982; Schachtman et al., 1983) and by extinguishing the blocking CS following blocking treatment (Arcediano et al., 2001; Blaisdell et al., 1999). Therefore, Experiments 1 and 2 provide further support for the view that forward and backward blocking arise from a failure to retrieve and/or express the target association during testing, instead of a failure to acquire the target association during training. Previous experiments have demonstrated spontaneous recovery from forward blocking (e.g., Batsell, 1997; J. S. Miller et al., 1993). However, these experiments were performed in a conditioned taste aversion preparation and studied the US-preexposure effect, which might or might not be viewed as an instance of forward blocking by the context. Therefore, it was not clear whether these results could be replicated using (a) a preparation other than taste aversion and (b) blocking between punctate CSs. Our Experiments 1 and 2 show that spontaneous recovery from both forward and backward blocking between punctate CSs can be observed in a conditioned suppression preparation.

The present results are particularly important because they are opposite to those obtained in a conditioned suppression preparation by J. S. Miller et al. (1996). That is, they found that blocking was enhanced when testing was delayed: forward blocking was not apparent when testing occurred after a 1-day retention interval, whereas blocking was observed following both 6-day and 11-day retention intervals. An important question to be addressed by future research is why the study by J. S. Miller et al. (1996) found results contrary to those of our Experiments 1 and 2, all of which used a punctate blocking stimulus. However, J. S. Miller et al.'s findings are diametrically opposed not only to our Experiments 1 and 2, but also to those of Batsell (1997) and J. S. Miller et al. (1993), who both used the context as a blocking CS. Therefore, the results of the present experiments indicate that the results of J. S. Miller et al. (1996) are the ones at odds with the growing set of evidence showing that the blocking effect can be attenuated by performing a great variety of posttraining manipulations, among which is the interpolation of a retention interval.

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