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Temporal Coding in Conditioned Inhibition: Analysis of Associative Structure of Inhibition

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Two experiments with rats as subjects were conducted to investigate the associative structure of temporal control of conditioned inhibition through posttraining manipulation of the training excitor–unconditioned stimulus (US) temporal relationship. Experiment 1 found that following simultaneous Pavlovian inhibition training (i.e., A → US/XA-no US) in which a conditioned stimulus (CS A) was established as a delay excitor, maximal inhibition was observed on a summation test when CS X was compounded with a delay transfer CS. Furthermore, posttraining shifts in the A-US temporal relationship from delay to trace resulted in maximal inhibition of a trace transfer CS. Experiment 2 found complementary results to Experiment 1 with an A-US posttraining shift from serial to simultaneous. These results suggest that temporal control of inhibition is mediated by the training excitor-US temporal relationship.

Students of animal learning have been interested in the phenomenon of behavioral inhibition since the pioneering work of Pavlov (1927). In a typical Pavlovian conditioned inhibition procedure, a conditioned stimulus (CS A) is followed by the unconditioned stimulus (US), except when it is compounded with a putative inhibitor (CS X; A → US/XA-no US). Behavior indicative of inhibition is typically assessed with two assays, the negative summation and retardation tests for inhibition (Hearst, 1972; Rescorla, 1969). A stimulus is said to be a conditioned inhibitor when it passes both tests. That is, it must attenuate responding to an independently trained excitor when presented in compound with it (the summation test), and it must be slow to acquire behavioral control when paired with the US (the retardation test). Since the introduction of this two-test strategy for evidencing conditioned inhibition, much has been learned about the associative structure of conditioned inhibition.

Prior studies of the associative structure of inhibition found that subjecting an inhibitory CS to extinction treatment (i.e., X-no US presentations) prior to inhibition testing does not disrupt inhibitory behavioral control (e.g., Witcher & Ayres, 1984; Zimmer-Hart & Rescorla, 1974). In fact, extinction of the conditioned inhibitor has sometimes been found to enhance inhibitory behavioral control (DeVito & Fowler, 1987; Williams & Overmier, 1988). In contrast, subjecting the training excitor (CS A) with which the inhibitor was established to extinction often disrupts inhibitory behavioral control (e.g., Best, Dunn, Batson, Meachum, & Nash, 1985; Hallam, Matzel, Sloat, & Miller, 1990; Lysle & Fowler, 1985; but see Rescorla & Holland, 1977; Witcher & Ayres, 1984, for conflicting results). On the basis of their findings, Lysle and Fowler suggested that inhibition is a "slave process" to excitation. That is, the inhibitory potential of a CS depends on the excitatory status of the training excitor, such that changes in the associative status of the training excitor produce corresponding changes in inhibitory behavioral control by the conditioned inhibitor. These results suggest that the associative structure of conditioned inhibition includes the excitor with which the inhibitor was established. That is, the inhibitor activates a representation of the training excitor, which in turn activates a representation of the US. Extinction of this latter association tends to disrupt inhibitory behavioral control.

The observation that inhibitory behavioral control is mediated by the associative status of the inhibitor's training excitor is consistent with Miller and Matzel's (1988; also see Denniston, Savastano, & Miller, 2001) comparator hypothesis. The comparator hypothesis views behavior indicative of inhibition as arising from a comparison of the strength of the inhibitor-US association relative to the associative strengths of other stimuli trained in the presence of the inhibitor (i.e., so-called comparator stimuli). In the case of Pavlovian inhibition, the training excitor with which the inhibitor was established is the comparator stimulus. According to the comparator hypothesis, at least three associations are potentially formed during the course of Pavlovian training (see Figure 1). The first association is between the target CS (X) and the US (i.e., X-US, Link 1); the second association is between the target

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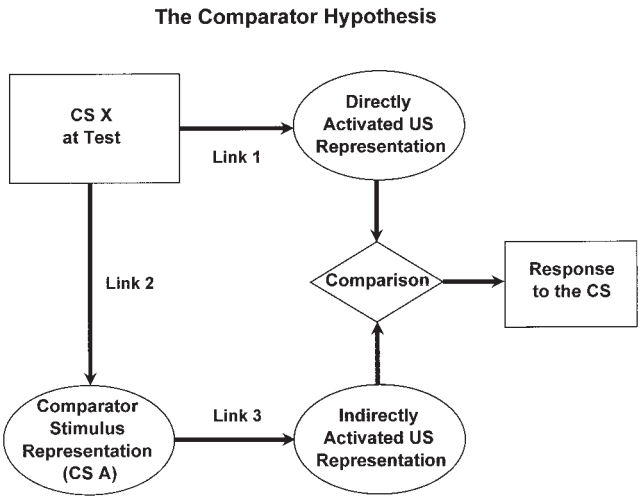

Figure 1. The comparator hypothesis (based on Miller & Matzel, 1988). Conditioned responding to CS X is directly related to the magnitude of the US representation that is directly activated by CS X and is inversely related to the magnitude of the US representation that is indirectly activated by CS X (i.e., mediated by CS X's comparator stimulus through conjoint action of the X-comparator stimulus and comparator stimulus-US associations). CS = conditioned stimulus; US = unconditioned stimulus.

CS (X) and the most salient stimulus present during training, the comparator stimulus (A, i.e., X-A, Link 2); and the third association is between the comparator stimulus (A) and the US (i.e., A-US, Link 3). The strength of the directly activated US representation is determined by the absolute associative strength of the target CS (Link 1), whereas the strength of the US representation indirectly activated through the comparator stimulus is determined by the product of the strengths of Links 2 and 3. Thus, weakening either Link 2 or 3 will result in reduced strength of the indirectly activated US representation. At test, conditioned responding is assumed to reflect a comparison of the US representations directly and indirectly activated by the target CS. Applied to behavior indicative of conditioned inhibition, the comparator hypothesis posits that inhibition arises from the interaction of exclusively excitatory associations. Inhibitory behavioral control (a negative response potential) is predicted to increase with increases in the strength of the indirectly activated US representations (i.e., Link 2 $\times$ Link 3) relative to the strength of the directly activated US representation (Link 1) and to decrease with decreases in the strength of the indirectly activated US representation relative to the strength of the directly activated US representation. Thus, following Pavlovian inhibition training (i.e., A $\rightarrow$ US/XA-no US), inhibitor X generates a negative response potential when it activates a US representation through its comparator stimulus CS A (the training excitor). When the X-A and A-US associations are strong (relative to the X-US association, which is presumably nil), the negative response potential should be robust. In contrast, extinction of the inhibitor's training excitor (CS A) should attenuate the negative response potential because this should weaken the strength of the indirectly activated US representation. This conceptualization of inhibition is entirely consistent with Lysle and Fowler's (1985) suggestion that inhibition is a slave process to excitation.

In contrast to the above account, Rescorla and Holland (1977) failed to obtain a loss of inhibition following extinction of the training excitor (also see Witcher & Ayres, 1984). Their series of experiments were notable because they explicitly attempted to ascertain the associative structure of conditioned inhibition established through A $\rightarrow$ US/XA-no US training. In their experiments, Rescorla and Holland found no reduction in inhibitory behavioral control following extinction of the inhibitor's training excitor, A. This suggests that X-A and X-(A-US) associations do not provide the associative structure of inhibition. They did, however, find that counterconditioning of the transfer excitor, B, used for negative summation testing, resulted in a loss of inhibitory control. This suggests that either X-no US or X-CR (a competing response) associations underlie the associative structure of inhibition. A second experiment seemed to rule out the importance of X-CR associations and, by default, they concluded that the X-no US associative structure is primarily responsible for behavioral inhibition. Thus, it is unclear whether behavior indicative of inhibition is mediated by the inhibitor's training excitor (as suggested by Lysle & Fowler's [1985] findings) or is the result of a direct inhibitor-no US association (as suggested by Rescorla and Holland's [1977] findings).

One potential means of further investigating the associative structure of inhibition is through assessing the informational content of inhibitory behavioral control. Miller and colleagues (Barnet & Miller, 1996; Burger, Denniston, & Miller, 2001; Denniston, Blaisdell, & Miller, 1998; Denniston, Cole, & Miller, 1998) found that conditioned inhibitors exert maximal inhibitory behavioral control at a particular temporal location. That is, following conditioned inhibition training, a conditioned inhibitor produces maximal inhibitory behavioral control when it is compounded with a transfer excitor that signals US presentation at the same temporal location as the inhibitor signals US omission. When these temporal expectancies for US presentation and US omission were inconsistent, inhibitory behavioral control was attenuated. Analogously, Burger et al., using a retardation test for conditioned inhibition, found that retardation pairings in which the US was presented at the same temporal location as the inhibitor signaled US omission produced maximal retardation of acquisition of behavioral control. When the US was presented at a temporal location different from that at which the inhibitor signaled US omission, retardation of behavioral control was attenuated. These results are consistent with the temporal coding hypothesis (Barnet, Arnold, & Miller, 1991; Matzel, Held, & Miller, 1988; Miller & Barnet, 1993; Savastano & Miller, 1998), which states that (a) temporal contiguity is both necessary and sufficient for learning to occur, (b) associations incorporate the temporal relationship between the CS and the US as part of the encoded memory (i.e., subjects form *temporal maps* that link events in memory), (c) the form and timing of the conditioned response are in part determined by these temporal maps, and (d) animals can integrate separate temporal maps when elements common to these separate temporal maps (e.g., a US) are presented together. However, what is unclear is the associative mechanism by which the inhibitor signals US omission. That is, if the inhibitor is never directly paired with the US, then how can it signal the omission of the US?

The present experiments were designed to investigate the associative structure of temporal control of inhibition. The rationale for these experiments follows from two lines of research: (a) the

observation that inhibitory behavioral control by a stimulus is tied to the current associative status of its training excitor (e.g., Lysle & Fowler, 1985); and (b) the observation that inhibitory behavioral control is temporally specific (e.g., Barnet & Miller, 1996). On the basis of this rationale, we hypothesized that inhibitory behavioral control is mediated by the inhibitor's training excitor (as posited by the comparator hypothesis; Miller & Matzel, 1988) and that temporal control of inhibition might also be tied to the training excitor-US temporal relationship in effect at the time of testing. To assess whether temporal control of inhibition is influenced by the temporal relationship between the inhibitor and the training excitor and between the training excitor and the US at the time of testing, we provided rats with Pavlovian conditioned inhibition training (i.e., A → US/XA-no US) followed by posttraining manipulation of the A-US temporal relationship. On the basis of the comparator hypothesis, we expected inhibition training to result in inhibitory behavioral control (a negative response potential) as a consequence of strong inhibitor–training excitor and training excitor-US associations. Furthermore, if these associations also encode the temporal relationship between associates (as posited by the temporal coding hypothesis, Matzel et al., 1988; Savastano & Miller, 1998), then inhibitor X should indirectly activate a representation of the US at a specific moment in time relative to X, as mediated by training excitor A. Posttraining manipulation of the training excitor-US temporal relationship was expected to reveal whether inhibitory behavioral control is mediated by the training excitor-US temporal relationship in effect at the time of testing. That is, changing the training excitor-US temporal relationship should produce a corresponding shift in inhibitory behavioral control if behavior indicative of inhibition is mediated by the training excitor. In contrast, if inhibitory behavioral control is determined by a direct inhibitor-no US association, then posttraining manipulation of the inhibitor's training excitor should have no effect on temporal control of inhibition.

Experiment 1

Prior research investigating the informational content of Pavlovian inhibitors has found that Pavlovian conditioned inhibitors produce maximal negative summation when compounded with a transfer excitor that signals US presentation at the same temporal location as the inhibitor signals US omission (e.g., Barnet & Miller, 1996; Denniston, Blaisdell, & Miller, 1998; Denniston, Cole, & Miller, 1998). However, these experiments did not address the associative structure of inhibitory behavioral control; that is, they did not determine whether the temporal expectancy for US omission is a direct inhibitor-no US association as posited by Rescorla and Holland (1977) or is mediated by the current status of the training excitor (i.e., inhibitor-training excitor-US) as suggested by Lysle and Fowler (1985). Thus, we designed Experiment 1 to contrast these two opposing views.

In Experiment 1 (see Table 1), all subjects received treatment in which CS A was trained as a delay excitor with no gap between CS termination and US onset (i.e., A $\xrightarrow{0}$ US, in which the superscript number represents the interval in seconds between stimuli) and CS X was trained as a simultaneous inhibitor with CS A (i.e., XA-no US). For the purpose of negative summation testing, two transfer excitors (CSs C and D) with different temporal relationships to the US were trained separately. CS C was trained as a delay transfer CS (i.e., C $\xrightarrow{0}$ US), whereas CS D was trained as a trace transfer CS with a 5-s gap between CS termination and US onset (i.e., D $\xrightarrow{5}$ US). Following inhibition training, half of the subjects (shift condition) received posttraining manipulation of the A-US temporal relationship, in which CS A was retrained as a trace CS (with a 5-s gap between CS termination and US presentation, i.e., A $\xrightarrow{5}$ US), whereas the remaining subjects (no-shift condition) received equivalent training with a previously neutral CS (i.e., B $\xrightarrow{5}$ US pairings). Inhibitory behavioral control was assessed during a single summation test, in which the potential of inhibitor X to reduce responding to the transfer excitors was assessed through simultaneous presentations of CS X and transfer excitor C or D depending on group. The magnitude of conditioned inhibition was measured by comparison between these subjects and separate groups tested with C or D alone.

On the basis of the temporal coding hypothesis, we expected subjects in the no-shift condition to demonstrate greater negative summation (indicative of inhibition) when tested with the XC stimulus compound than with the XD stimulus compound because inhibitor X generates a negative response potential (through its

Table 1
Design Summary for Experiment 1

Group	*n*	CI training	Update training	Test	Expected
Shift.C	5			C	CR
Shift.D	5	A $\xrightarrow{0}$ US/XA−/	A $\xrightarrow{5}$ US/	D	CR
Shift.XC	10	C $\xrightarrow{0}$ US/D $\xrightarrow{5}$ US	C $\xrightarrow{0}$ US/D $\xrightarrow{5}$ US	XC	CR
Shift.XD	10	(all shift groups)	(all shift groups)	XD	cr
No Shift.C	5			C	CR
No Shift.D	5	A $\xrightarrow{0}$ US/XA−/	B $\xrightarrow{5}$ US/	D	CR
No Shift.XC	10	C $\xrightarrow{0}$ US/D $\xrightarrow{5}$ US	C $\xrightarrow{0}$ US/D $\xrightarrow{5}$ US	XC	cr
No Shift.XD	10	(all no-shift groups)	(all no-shift groups)	XD	CR

Note. Treatments listed under CI training and update training were provided to all subjects in each condition. Conditioned Stimulus (CS) X was a white noise; CSs A and B were a flashing light or a buzzer, counterbalanced; and CSs C and D were a complex tone and a click train, counterbalanced. All CSs were 5 s in duration, and the US was a 0.5-s, 1.3-mA footshock. Superscript numbers (0 and 5) refer to the interval in seconds between stimulus presentations (i.e., delay and trace, respectively). CI = conditioned inhibition; CR = strong responding expected; US = unconditioned stimulus; cr = weak responding expected.

comparator stimulus, CS A) at the same temporal location as CS C, but not CS D, generates a positive response potential (see Figures 2B and 2D. In effect, inhibition was predicted to be greatest when the transfer excitor predicted the occurrence of the US at the same temporal location as the inhibitor's training excitor, A, predicted the occurrence of the US. Any mismatch in their temporal expectancies would attenuate inhibitory behavioral control, a result that would replicate the prior findings of Miller and his colleagues (Barnet & Miller, 1996; Denniston, Blaisdell, & Miller, 1998; Denniston, Cole, & Miller, 1998). Of central interest were the groups that received posttraining manipulation of the A-US temporal relationship. If inhibition is controlled by an X-no US association (as posited by Rescorla & Holland, 1977), then changes in the A-US temporal relationship should have no effect on the expression of inhibition (i.e., X should maximally inhibit C but not D), just as posttraining extinction of CS A had no effect on inhibitory behavioral control. In contrast, if the inhibitory potential of CS X is modulated by its training excitor (as posited by Lysle & Fowler, 1985), then posttraining manipulation of the A-US temporal relationship should produce a corresponding shift in inhibitory behavioral control. That is, shifting the A-US temporal relationship from delay to trace should result in maximal negative summation when inhibitor X from the shift condition is compounded with the trace transfer CS D but not the delay transfer CS C (see Figures 2A and 2C).

Method

Subjects

The subjects were 30 male and 30 female, experimentally naive, Sprague-Dawley derived rats (*Rattus norvegicus*) from our State University of New York at Binghamton breeding colony. Body weights ranged from 320 to 440 g for males and from 240 to 370 g for females. The rats were individually housed in standard hanging, stainless-steel, wire-mesh cages in a vivarium maintained on a 16:8-hr light–dark cycle. All training occurred approximately midway through the light portion of the cycle. Subjects were allowed free access to food in their home cages, whereas access to water was gradually decreased to 10 min per day prior to the initiation of the experiment. All subjects were handled for 30 s three times per week from weaning until the initiation of the study. Subjects were randomly assigned to one of eight groups (*n*s = 5 or 10), counterbalanced as closely as possible for sex.

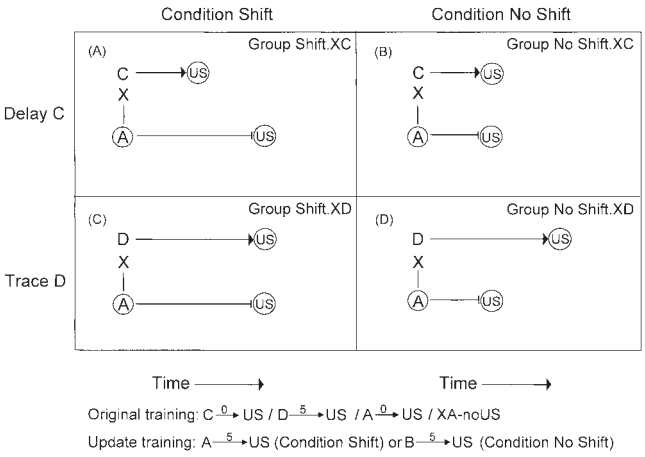

Figure 2. Hypothetical temporal expectancies generated at test, as a result of conditioned inhibition training and transfer excitor training provided in Experiment 1. Horizontal lines indicate a forward expectancy. Horizontal lines with an arrow represent an expected US; horizontal lines ending with a cross bar indicate that an otherwise expected US will be omitted. Vertical lines indicate a simultaneous expectancy. Maximal inhibition is hypothesized to occur when the transfer excitor and the inhibitor's training excitor signal US presentation at the same temporal location. A: Group Shift.XC; B: Group No Shift.XC; C: Group Shift.XD; D: Group No Shift.XD. US = unconditioned stimulus.

Apparatus

Two types of experimental chambers, designated R and V, were used. Chamber R was rectangular in shape and measured 22.75 × 8.25 × 13.0 cm (length × width × height). The walls and ceiling of the chamber were constructed of clear Plexiglas, and the grid floor consisted of stainless-steel rods measuring 0.48 cm in diameter, spaced 1.5 cm center-to-center. The rods were connected by NE2-neon bulbs, which allowed for the delivery of constant-current footshock produced by a high-voltage AC circuit in series with a 1.0-MΩ resistor. Each of six copies of Chamber R was housed in a separate sound- and light-attenuating environmental enclosure. Chamber R could be dimly illuminated by a 2.0-W (nominal at 120-V AC) houselight driven at 60-V AC. The bulb was located on the inside wall of the environmental enclosure, approximately 30 cm from the center of the experimental chamber.

Chamber V was a box 25.5 cm long in the shape of a vertical truncated V. The chamber was 28 cm high, was 21 cm wide at the top, and narrowed to 5.25 cm wide at the bottom. The ceiling was constructed of clear Plexiglas, the front and back end walls were black Plexiglas, and the side walls were stainless steel. The floor consisted of two 25.5-cm-long parallel stainless-steel plates, each 2 cm wide and separated by a 1.25-cm gap. A constant-current footshock could be delivered through the metal walls and floor of the chamber. Each of six copies of Chamber V was housed in a separate sound- and light-attenuating enclosure. Chamber V was illuminated by a 7-W (nominal at 120-V AC) bulb driven at 60-V AC. The bulb was mounted on the inside wall of the environmental enclosure, approximately 30 cm from the center of the experimental chamber, with the light entering the chamber primarily by reflection from the ceiling of the environmental enclosure. The light intensities in Chambers R and V were approximately equal, despite the discrepancy in the lightbulbs used, because of the differences between the chambers in the opaqueness of the walls.

Chambers R and V could each be equipped with a water-filled lick tube. When inserted, the lick tube extended 1 cm into a cylindrical drinking recess that was set into one of the Plexiglas end walls of the chamber. Each drinking recess was left–right centered with its bottom 1.75 cm above the floor of the chamber. The recess was 4.5 cm in diameter and 5 cm deep. An infrared photobeam was projected horizontally across the recess, 1 cm in front of the lick tube. To drink from the lick tube, a subject had to insert its head into the recess, thereby breaking the photobeam. By this means, we could monitor when subjects were accessing the lick tube. Three speakers, mounted on separate walls in each enclosure, could each deliver one of the following auditory cues: a 6 clicks per second click train, a white noise, or a low-frequency tone (compound of 300 and 320 Hz), each 8 dB (C-scale) above the ambient background of 74 dB (C-scale), which was produced primarily by a ventilation fan in each environmental enclosure. Each chamber could also provide a flashing-light stimulus (0.25 s on/0.25 s off). In Chamber R, the flashing light was a 25-W bulb (nominal at 120-V AC) driven at 60-V AC, whereas the flashing light in Chamber V was a 100-W bulb (nominal at 120-VAC) driven at 60-V AC. The bulbs were located on the back wall of each environmental chest. Each enclosure also contained a buzzer that could produce a buzzing sound 8 dB (C-scale) above the

background. All CSs were 5 s in duration, and the US was a 0.5-s, 1.3-mA footshock.

Procedure

In the first phase of training (inhibition training), all subjects received conditioned inhibition training consisting of A $\xrightarrow{0}$ US/XA-no US, in which A represents the training excitor and X represents the conditioned inhibitor. Thus, CS A served as a delay training excitor, and CS X served as a simultaneous inhibitor for CS A. For the purpose of testing, during conditioned inhibition training all subjects also received training intended to establish two transfer excitors (C and D) as signals for US presentation. CS C was trained as a delay CS with no gap between CS termination and US onset (i.e., C $\xrightarrow{0}$ US), whereas CS D was trained as a trace CS with a 5-s gap between CS termination and US onset (i.e., D $\xrightarrow{5}$ US).

In the second phase of training (update training), half of the subjects (shift condition) received conditioning of the previously established delay CS (A) in which the temporal relationship was shifted (i.e., updated) from delay to trace (i.e., A $\xrightarrow{5}$ US) such that there was now a 5-s gap between CS termination and US onset, whereas the other half of the subjects (no-shift condition) merely received equivalent training with a previously novel stimulus, B (i.e., B $\xrightarrow{5}$ US). Additionally, all subjects continued to receive training with transfer excitors C and D. Both phases of training (inhibition and update training) occurred in Context Train, which was Chamber V for half of the subjects in each of the two conditions and Chamber R for the other half of the subjects.

Summation testing was conducted during the third phase of the experiment in Context Test. Context Text was created by switching the type of enclosure (R or V) from that used in inhibition and update training. That is, subjects that received training in Context R were tested in Context V, whereas subjects that received training in Context V were tested in Context R. Different contexts were used for training and testing to ensure that an associatively neutral context was present at the time of testing and that any reduction in conditioned suppression to the test stimuli could be attributed only to inhibition and/or update training and not to contextual associations acquired during training. Extensive prior research had established that rats treat these two contexts as distinctly different (e.g., Gunther, Denniston, & Miller, 1998). Testing occurred during a single test session in which a transfer excitor was presented either alone, or in a simultaneous compound with the putative inhibitor, CS X. For purposes of testing, the subjects in each of the conditions (shift and no shift) were assigned to one of four groups that were tested on C alone ($n = 5$), D alone ($n = 5$), a simultaneous compound of XC ($n = 10$), or a simultaneous compound of XD ($n = 10$). The combination of their prior training (shift vs. no shift) and test procedure defined the eight groups.

Acclimation. Acclimation to the experimental chambers comprising Context Train and Context Test was conducted on Days 1 and 2, respectively, in daily 60-min sessions during which water-filled lick tubes were available and no nominal stimuli were presented. This phase of the experiment served to establish a stable baseline level of drinking behavior, a departure from which would serve as the dependent variable during testing. Following acclimation, the water-filled lick tubes were removed from all chambers.

Conditioned inhibition training. On Days 3–20, conditioned inhibition training was conducted during daily 75-min sessions in Context Train. During each of these 18 sessions, all subjects received interspersed eight A $\xrightarrow{0}$ US, eight XA-no US, two C $\xrightarrow{0}$ US, and two D $\xrightarrow{5}$ US pairings. The buzzer served as CS A for half of the subjects in each group and the flashing light for the other half of the subjects; the click and low-frequency tone served as transfer CSs C and D, counterbalanced within groups; the white noise served as CS X for all subject; and the US was a 0.5-s, 1.3-mA footshock. All sessions began with a reinforced trial, and the remaining reinforced and nonreinforced trials were pseudorandomly distributed within the session. The mean intertrial interval (CS onset to CS onset) for the 20 trials was 3.5 min, with a range from 1.5 to 5.5 min. Three different running schedules, which differed with respect to trial order, were alternated through use of an A, B, C, C, B, A pattern between days. Thus, during inhibition training, CS A was established as a delay excitor in which the US was presented immediately following termination of the CS, CS X was trained as a simultaneous inhibitor through nonreinforced compound presentations of CSs X and A (i.e., XA-no US), and transfer CSs C and D were trained as delay and trace CSs, respectively, in which either there was no gap between termination of CS C and US onset or there was a 5-s gap between termination of CS D and US onset.

Update training. On Days 21–28 update training was provided during daily 75-min sessions in Context Train. During each session, subjects in the shift condition received eight trace A → US presentations (i.e., CS A was retrained as a trace CS with a 5-s gap between CS termination and US onset), whereas subjects in the no-shift condition received equivalent training with CS B (i.e., B $\xrightarrow{5}$ US). These 8 trials were interspersed among 2 C $\xrightarrow{0}$ US and 2 D $\xrightarrow{5}$ US trials. The mean intertrial interval (CS onset to CS onset) for the 12 trials was 6 min, with a range from 3 to 9 min. Two different running schedules, which differed with respect to trial order, were alternated through use of an A, B, B, A, A, B, B, A pattern across days.

Reacclimation. On Days 29–31, all subjects were reacclimated to Context Test during daily 60-min sessions. During these reacclimation sessions, the water-filled lick tubes were returned to the chambers to stabilize baseline drinking following any disruption produced by the footshock USs. No nominal stimuli were presented during these sessions.

Summation testing. On Day 32, all subjects were tested for suppression of ongoing drinking in the presence of the test stimuli in Context Test. Subjects in the shift condition were randomly assigned to one of four groups (C and D, ns = 5 per group; XC and XD, ns = 10 per group). Subjects in the no-shift condition were similarly assigned to one of four groups (C and D, ns = 5 per group; XC and XD, ns = 10 per group). The difference in sample size between conditions in which subjects were tested on a transfer excitor alone and an inhibitor–transfer excitor compound was introduced to minimize the number of subjects in the transfer-excitor-alone test conditions (we intended to collapse the C and D data across update treatment conditions [shift and no shift] provided that there was no statistical interaction of condition and C and D test stimuli). On the test day, the subjects were allowed to drink from the lick tubes for 5 cumulative seconds, after which the target stimuli were immediately presented. Thus, all subjects were drinking at the moment of test stimuli onset. The time to complete an additional 5 cumulative seconds of licking in the presence of the test stimuli was recorded. An 11-min ceiling was imposed on the suppression scores.

Data analysis. Prior to statistical analysis in this experiment and Experiment 2, all suppression scores were converted to log (base 10) scores to better normalize the within-group distributions, thereby allowing the use of parametric statistics. An alpha level of .05 was adopted for all statistical tests in each experiment. Also in each experiment, any subject taking more than 60 s to complete its first 5 cumulative seconds of drinking (prior to CS onset), thus showing great reluctance to drink in the test context, was scheduled to be eliminated from the data analysis. In practice, no subjects had to be eliminated for this reason from Experiment 1.

Results and Discussion

The central findings from this study were that subjects that received no shift during the update phase demonstrated less conditioned suppression (i.e., more behavior indicative of conditioned inhibition) when tested with the XC simultaneous compound relative to transfer excitor C alone but not when tested with the XD simultaneous compound relative to transfer excitor D alone (see Figure 3). In contrast, subjects that received posttraining manipulation of the A-US temporal relationship from delay to trace demonstrated less conditioned suppression when tested with the

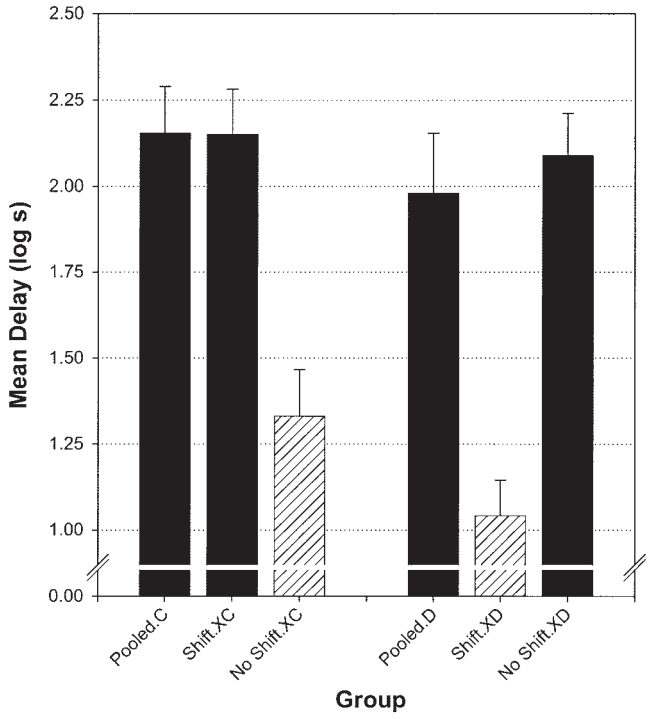

Figure 3. Experiment 1: Group identification refers to whether the A–unconditioned stimulus (A-US) temporal relationship was shifted following conditioned inhibition training and the stimuli presented at test. Bars depict mean times to complete 5 cumulative seconds of drinking in the presence of the test stimulus. Higher scores indicate greater fear (i.e., less inhibition). Groups for which the temporal coding hypothesis predicts strong conditioned inhibition are represented with striped bars. Error brackets denote the standard error of the mean. See Table 1 for procedural details.

XD simultaneous compound relative to transfer excitor D alone but not with the XC compound relative to transfer excitor C alone. Thus, maximal conditioned inhibition was observed in the no-shift condition when the inhibitor (X) produced a negative response potential at the same temporal location as transfer excitor C signaled US presentation. However, if the temporal expectancy for US omission was shifted from delay to trace as a consequence of posttraining manipulation of X's training excitor, CS A (shift condition), then maximal inhibitory behavioral control was observed when testing was conducted with the trace, but not the delay, transfer excitor. These findings were confirmed with the following statistical analyses.

We first analyzed the conditioned suppression scores in the presence of the test stimuli for subjects in Groups Shift.C, Shift.D, No Shift.C, and No Shift.D. A 2 × 2 analysis of variance (ANOVA) with condition (shift vs. no shift) and transfer excitor (C vs. D) as variables was conducted on the suppression scores from the test session and revealed no main effects or interaction of the variables (all Fs < 1). We then pooled the data from subjects tested on transfer excitor C alone across conditions (shift and no shift) and on transfer excitor D alone across conditions to form Groups Pooled.C and Pooled.D, respectively.

Prior to further analysis of the suppression scores from the test session, we analyzed the times to complete 5 cumulative seconds of drinking prior to CS onset (i.e., the pre-CS scores) in order to assess potential differences in baseline drinking behavior. A 2 × 3 ANOVA with transfer excitor (C vs. D) and condition (pooled vs. shift vs. no shift) as variables was conducted on the pre-CS scores from subjects in Groups Pooled.C, Shift.C, No Shift.C, Pooled.D, Shift.D, and No Shift.D. This analysis revealed no main effect of test excitor, $F(1, 54) = 2.41$, $p > .10$, and no main effect of condition, $F(2, 54) = 2.89$, $p > .05$. Although this last effect was of borderline significance, the actual differences between the six conditions were very small, with means ranging from 0.981 to 1.199 log seconds. More important, given the importance of the interaction between transfer excitor and condition in the CS scores, there was no interaction between these variables in the pre-CS scores ($F < 1$). Thus, there were no reliable differences in baseline drinking behavior prior to test stimulus onset.

Analysis of the times to complete 5 cumulative seconds of drinking in the presence of the test stimuli was accomplished with a 2 × 3 ANOVA using the same variables as above. This analysis revealed no main effect of test excitor, $F(1, 54) = 2.49$, $p > .10$, a main effect of condition, $F(2, 54) = 6.57$, $p < .01$; and a Test Excitor × Condition interaction, $F(2, 54) = 23.74$, $p < .001$. Planned comparisons using the error term from the overall ANOVA were conducted on the suppression scores from subjects tested on the delay transfer excitor C. These comparisons revealed attenuated conditioned suppression in Group No Shift.XC, relative to Group Pooled.C, $F(1, 54) = 18.44$, $p < .001$, thereby demonstrating that CS X passed a summation test for conditioned inhibition when the A-US temporal relationship was not shifted after inhibition training. In contrast, no difference in suppression was observed between Group Shift.XC and Group Pooled.C ($F < 1$), thereby demonstrating that posttraining manipulation of the A-US temporal relationship from delay training to trace training attenuated the potential of CS X to inhibit conditioned responding to delay transfer excitor C. Additionally, Group No Shift.XC demonstrated less conditioned responding than did Group Shift.XC, $F(1, 54) = 18.32$, $p < .001$, suggesting that the posttraining manipulation of the A-US temporal relationship attenuated the potential of CS X to inhibit responding to transfer excitor C.

Comparison of suppression scores from Group No Shift.XD revealed no difference in suppression from Group Pooled.D ($F < 1$), indicative of inhibitor X failing to attenuate conditioned responding to the trace transfer excitor when the A-US temporal relationship (delay) was not changed following inhibition training. In contrast, attenuated conditioned suppression was observed in Group Shift.XD relative to Group Pooled.D, $F(1, 54) = 23.99$, $p < .001$, thereby demonstrating that posttraining shifting of the A-US temporal relationship from delay to trace allowed CS X to inhibit conditioned responding to the trace transfer excitor D. Additionally, Group Shift.XD demonstrated less conditioned responding than did Group No Shift.XD, $F(1, 54) = 29.87$, $p < .001$, suggesting that posttraining manipulation of the A-US temporal relationship enhanced the potential of CS X to inhibit responding to transfer excitor D.

Finally, and most centrally, Group No Shift.XC demonstrated less conditioned responding than did Group No Shift.XD, $F(1, 54) = 15.66$, $p < .001$, whereas Group Shift.XD demonstrated less conditioned responding than did Group Shift.XC, $F(1, 54) = 33.50$, $p < .001$, suggesting that in the absence of posttraining manipulation of the A-US temporal relationship (no-shift condi-

tion), CS X was an effective inhibitor of the delay transfer excitor (C) but not the trace transfer excitor (D) and that following a posttraining shift in the A-US temporal relationship from delay to trace (shift condition), CS X was an effective inhibitor of the trace transfer excitor (D) but not the delay transfer excitor (C).

Thus, in Experiment 1, we found that a simultaneous Pavlovian inhibitor established with a delay training excitor was able to attenuate conditioned responding to a delay, but not a trace, transfer excitor, a finding that replicates prior studies of temporal control of Pavlovian conditioned inhibition (e.g., Barnet & Miller, 1996; Denniston, Blaisdell, & Miller, 1998; Denniston, Cole, & Miller, 1998). More important, posttraining manipulation of the training excitor-US temporal relationship resulted in a shift of temporal control of inhibition. That is, shifting the A-US temporal relationship from delay to trace resulted in inhibitor X maximally attenuating conditioned responding to a trace, but not a delay, transfer excitor. These findings suggest that the temporal expectancy for US omission provided by the conditioned inhibitor is mediated by the current training excitor-US temporal relationship. Hence, the temporal expectancy for US omission does not appear to be determined by a direct inhibitor-no US association; rather, it is dependent on both the inhibitor–training excitor temporal relationship and the prevailing training excitor-US temporal relationship as demonstrated in the current experiment.

Experiment 2

The results of Experiment 1 revealed that a conditioned inhibitor produced maximal negative summation when compounded with a transfer CS that signaled US presentation at the same temporal location as the inhibitor's training excitor signaled the occurrence of the US. That is, lengthening the training excitor-US temporal relationship led to a corresponding shift in inhibitory behavioral control by the target cue. We designed Experiment 2 to investigate the generality of this finding by using serial inhibitors (i.e., established through A $\rightarrow$ US/X $\rightarrow$ A-no US pairings in which $\rightarrow$ signifies "precedes"). In addition, in Experiment 2 we shifted the temporal relationship by shortening the A-US interval, potentially extending the generality of the central finding of Experiment 1 to temporal shifts of the A-US interval in each direction. Lastly, in Experiment 2 we assessed the alternative interpretation of Experiment 1 that CS X is maximally inhibitory when the training and transfer excitors have the same temporal relationship to the US regardless of CS X's superpositioned temporal relationship to the US.

Holland, Hamlin, and Parsons (1997) found that, following serial feature-positive occasion-setting training, modulation by the feature was greatest when the feature-target interval was of the same duration as in occasion-setting training, a result that is consistent with the observations of Denniston, Blaisdell, and Miller (1998), who found similar results with serial inhibition training. Moreover, when Holland et al. varied the feature-target interval from that of training, modulation was attenuated. Their results are consistent with the view that temporal control by the feature is determined in part by the temporal relationships between the feature and the target and between the target and the US. That is, the temporal expectancy for US presentation appears to be mediated by the target.

We designed Experiment 2 to assess whether temporal control of serial inhibition is mediated by the current training excitor-US temporal relationship by providing posttraining shifts in the training excitor-US temporal relationship (see Table 2). As in Experiment 1, all subjects received training intended to establish CS A as a signal for US presentation and CS X as a conditioned inhibitor. CS A was trained as a delay training excitor with no gap between CS termination and US onset (i.e., A $\overset{0}{\rightarrow}$ US), and CS X was trained as a serial inhibitor of CS A (i.e., X $\overset{0}{\rightarrow}$ A-no US) through nonreinforced serial presentations of X and A with no gap between the termination of CS X and the onset of CS A. Additionally, two transfer excitors (CSs C and D) were once again separately established as delay (i.e., C $\overset{0}{\rightarrow}$ US) and trace (i.e., D $\overset{5}{\rightarrow}$ US) transfer excitors for the purpose of summation testing. Following inhibition training, half of the subjects received posttraining manipulation of the A-US temporal relationship in which CS A was retrained as a simultaneous CS with the CS and US being presented coterminously (shift condition), whereas the other half of the subjects merely received equivalent training with a previously

Table 2
Design Summary for Experiment 2

Group	*n*	CI training	Update training	Test	Expected
Shift.C	5			C	CR
Shift.D	5	A $\overset{0}{\rightarrow}$ US/X $\overset{0}{\rightarrow}$ A−/	A−US/	D	CR
Shift.XC	10	C $\overset{0}{\rightarrow}$ US/D $\overset{5}{\rightarrow}$ US	C $\overset{0}{\rightarrow}$ US/D $\overset{5}{\rightarrow}$ US	XC	cr
Shift.XD	10	(all shift groups)	(all shift groups)	XD	CR
No Shift.C	5			C	CR
No Shift.D	5	A $\overset{0}{\rightarrow}$ US/X $\overset{0}{\rightarrow}$ A−/	B−US/	D	CR
No Shift.XC	10	C $\overset{0}{\rightarrow}$ US/D $\overset{5}{\rightarrow}$ US	C $\overset{0}{\rightarrow}$ US/D $\overset{5}{\rightarrow}$ US	XC	CR
No Shift.XD	10	(all no-shift groups)	(all no-shift groups)	XD	cr

Note. Treatments listed under CI training and update training were provided to all subjects in each condition. Conditioned Stimulus (CS) X was a white noise; CSs A and B were a flashing light or a buzzer, counterbalanced; and CSs C and D were a complex tone and a click train, counterbalanced. All CSs were 5 s in duration, and the US was a 5-s, 0.7-mA footshock. Superscript numbers (0 and 5) refer to the interval in seconds between stimulus presentations (i.e., delay and trace, respectively). During update training, subjects received simultaneous A-US (shift condition) or B-US (no-shift condition) presentations. CI = conditioned inhibition; CR = strong responding expected; US = unconditioned stimulus; cr = weak responding expected.

novel CS (no-shift condition; i.e., simultaneous B-US pairings). Inhibitory behavioral control was assessed during a single summation test in which the potential of inhibitor X to reduce responding to the transfer excitors was assessed through simultaneous presentations of CS X and a transfer excitor (C or D) as compared with presentation of the transfer excitor alone.

We expected subjects in the no-shift condition to demonstrate maximal negative summation when CS X was compounded with the trace transfer CS (D; i.e., Group No Shift.XD) relative to the delay transfer CS (C; i.e., Group No Shift.XC) because CS X was expected to generate inhibitory behavioral control 10 s following CS onset, which corresponds to the temporal expectancy for US presentation generated by transfer CS D, but not C (see Figures 4B and 4D). In contrast, we expected to observe greater negative summation by subjects in Group Shift.XC relative to Group Shift.XD because shifting the A-US temporal relationship from delay to simultaneous should shorten the window for inhibitory behavioral control generated by CS X by 5 s, thereby corresponding to the temporal expectancy for US presentation generated by transfer CS C, but not D (see Figures 4A and 4C).

Method

Subjects and Apparatus

The subjects were 30 male and 30 female, experimentally naive, Sprague-Dawley derived rats from our State University of New York at Binghamton breeding colony. Body weights ranged from 266 to 434 g for males and from 201 to 301 g for females. Animal care and deprivation were the same as in Experiment 1. Subjects were randomly assigned to one of eight groups (*n*s = 5 or 10), counterbalanced as closely as possible for sex. The apparatus was the same as in Experiment 1, except that the US was a 5-s, 0.7-mA footshock. US duration and intensity were changed from those of Experiment 1 because Experiment 2 provided simultaneous CS–US presentations (update training) in which both the CS and the US were 5 s in duration.

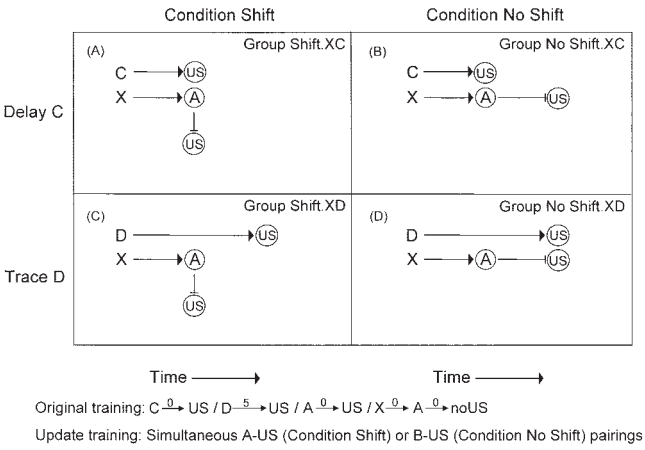

Figure 4. Hypothetical temporal expectancies generated at test, as a result of conditioned inhibition training and transfer excitor training provided in Experiment 2. Horizontal lines indicate a forward expectancy. Horizontal lines with an arrow represent an expected US; horizontal lines ending with a cross bar indicate that an otherwise expected US will be omitted. Vertical lines indicate a simultaneous expectancy. Maximal inhibition is hypothesized to occur when the transfer excitor and the inhibitor's training excitor signal US presentation at the same temporal location. A: Group Shift.XC; B: Group No Shift.XC; C: Group Shift.XD; D: Group No Shift.XD. US = unconditioned stimulus.

Procedure

The design and procedure of Experiment 2 were similar to those of Experiment 1, with the following exceptions. In the first phase of treatment, all subjects received conditioned inhibition training consisting of A $\xrightarrow{0}$ US/X $\xrightarrow{0}$ A-no US. Thus, CS A served as a delay training excitor, and CS X served as a serial inhibitor of CS A. As in Experiment 1, all subjects also received training intended to establish two transfer excitors, one delay (C) and one trace (D) as signals for US presentation. In the second phase of treatment, subjects in the shift condition received simultaneous CS A-US pairings in which the temporal relationship was shifted (i.e., updated) from delay in Phase 1 to simultaneous (i.e., A-US) in Phase 2, whereas subjects in the no-shift condition merely received equivalent training with a previously novel stimulus, B (i.e., B-US). Testing occurred during a single test session in which each transfer excitor was presented either alone or in a simultaneous compound with the putative inhibitor, X. As in Experiment 1, the subjects in each update condition (shift and no shift) were divided into four groups that were tested on C alone (n = 5), D alone (n = 5), a simultaneous compound of XC (n = 10), or a simultaneous compound of XD (n = 10).

Acclimation. Acclimation to the experimental chambers comprising Context Train and Context Test were conducted on Days 1 and 2, respectively, with the same procedure as in Experiment 1.

Conditioned inhibition training. On Days 3–20, conditioned inhibition training was conducted during daily 75-min sessions in Context Train. During each of these 18 sessions, all subjects received eight A $\xrightarrow{0}$ US, eight X $\xrightarrow{0}$ A-no US, two C $\xrightarrow{0}$ US, and two D $\xrightarrow{5}$ US pairings. The physical stimuli serving as A, X, C, and D, as well as trial order and stimulus spacing, were the same as in Experiment 1. Thus, CS A was established as a delay excitor in which the US was presented immediately following termination of the CS, and CS X was trained as a serial inhibitor in which termination of CS X was followed immediately by onset of CS A, which on these trials was nonreinforced (i.e., X $\xrightarrow{0}$ A-no US).

Update training. Update training (Days 21–28) was the same as in Experiment 1, except that subjects in the four shifted groups received eight simultaneous reinforced CS A-US presentations in which CS A and the US were presented coterminously (i.e., A-US). Subjects in the four unshifted groups received equivalent training with CS B (i.e., B-US).

Reacclimation. On Days 29 and 30, all subjects were reacclimated to Context Test during daily 60-min sessions, as in Experiment 1. No nominal stimuli were presented during these sessions. In Experiment 2, only two reacclimation sessions were needed to restabilize baseline drinking behavior.

Summation testing. On Day 31, all subjects were tested for suppression of ongoing drinking in the presence of the test stimuli in Context Test, as in Experiment 1. At test, the time to complete 5 cumulative seconds of drinking in the presence of the test stimuli was recorded.

Data analysis. As in Experiment 1, any subject requiring more than 60 s to complete the initial 5 cumulative seconds of drinking prior to test stimulus onset was scheduled to be eliminated from the data analysis. One subject from Group Shift.C had to be eliminated for this reason.

Results and Discussion

The primary findings from this study were that when the A-US interval was not shifted, the summation test to D provided good evidence of inhibitory control to X, but no such evidence was found in the summation test to C (see Figure 5). In contrast, when the A-US temporal relationship was shifted from delay to simultaneous, the summation test to C provided good evidence of

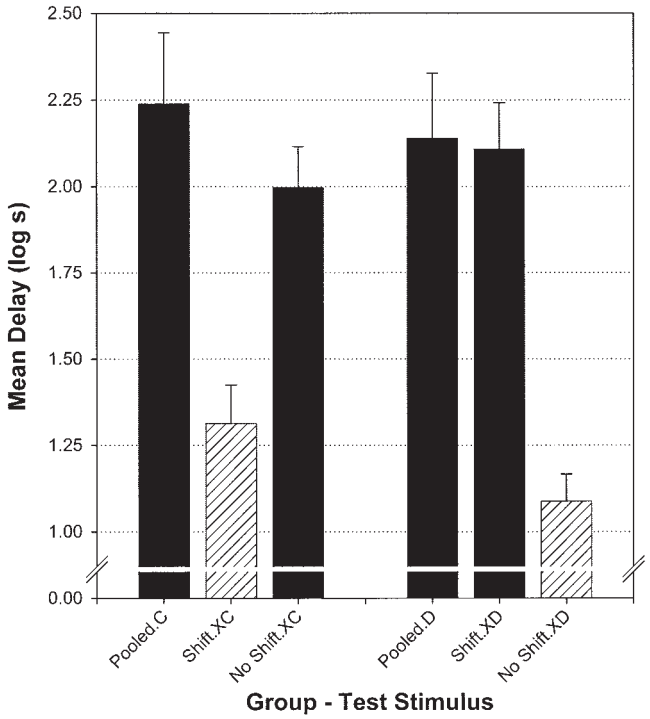

Figure 5. Experiment 2: Group identification refers to whether the A–unconditioned stimulus (A-US) temporal relationship was shifted or maintained following conditioned inhibition training and the stimuli presented at test. Bars depict mean times to complete 5 cumulative seconds of drinking in the presence of the test stimulus. Higher scores indicate greater fear (i.e., less inhibition). Groups for which the temporal coding hypothesis predicts strong conditioned inhibition are represented with striped bars. Error brackets denote the standard error of the mean. See Table 2 for procedural details.

inhibitory control to X, but no such evidence was found in the summation test to D. Thus, greater conditioned inhibition was observed in the no-shift condition when inhibitor X generated inhibitory behavioral control at the same temporal location as transfer CS D signaled US presentation. More centrally, when the training excitor-US temporal relationship was shifted from delay to simultaneous (shift condition), maximal inhibition was then observed when testing was conducted with delay transfer CS C and conditioned inhibitor X. These findings were confirmed with the following statistical analyses.

As in Experiment 1, we first analyzed the conditioned suppression scores in the presence of the test stimuli for subjects tested on the transfer excitors (C and D) alone. A 2 × 2 ANOVA with condition (shift vs. no shift) and transfer excitor (C vs. D) as factors was conducted on the suppression scores from the test session. This analysis revealed no main effects or interactions (all Fs < 1). Therefore, we pooled the transfer excitor data (C and D separately) across conditions. Using the same analysis as that used in Experiment 1, we next analyzed the pre-CS scores to assess potential differences in baseline drinking behavior. A 2 × 3 ANOVA with transfer excitor (C vs. D) and condition (pooled vs. shift vs. no shift) as factors revealed no main effects or interactions (all Fs < 1). Thus, there were no reliable differences in baseline drinking behavior prior to test stimulus onset.

Analysis of the times to complete 5 cumulative seconds of drinking in the presence of the test stimuli was accomplished with a 2 × 3 ANOVA that used the same factors as were used for the pre-CS score analysis. This 2 × 3 ANOVA revealed no main effect of transfer excitor ($F < 1$); a main effect of condition, $F(2, 53) = 10.73$, $p < .001$; a Transfer Excitor × Condition interaction, $F(2, 53) = 17.88$, $p < .001$. Planned comparisons using the error term from the overall ANOVA were conducted on the suppression scores from subjects tested on trace transfer CS D. These comparisons revealed less conditioned suppression in Group No Shift.XD relative to Group Pooled.D, $F(1, 53) = 27.25$, $p < .001$, thereby demonstrating the passage of a summation test for conditioned inhibition by CS X when the A-US temporal relationship was not shifted between inhibition training and testing. More important, there was no difference in conditioned suppression between Groups Shift.XD and Pooled.D ($F < 1$), thereby evidencing that posttraining shifting of the A-US temporal relationship from delay to simultaneous virtually eliminated inhibitory behavioral control by CS X when compounded with trace transfer CS D. Furthermore, Group No Shift.XD demonstrated less conditioned responding than did Group Shift.XD, $F(1, 53) = 25.66$, $p < .001$, suggesting that posttraining manipulation of the A-US temporal relationship reduced the effectiveness of CS X to inhibit responding to transfer excitor D.

Comparison of suppression scores from Group No Shift.XC revealed no difference in suppression relative to Group Pooled.C, $F(1, 53) = 1.38$, $p > .20$, indicative of inhibitor X failing to attenuate conditioned responding to the delay transfer excitor when the A-US temporal relationship (delay) was maintained between inhibition training and testing. Of greater interest, attenuated conditioned suppression was observed in Group Shift.XC relative to Group Pooled.C, $F(1, 53) = 19.99$, $p < .001$, thereby evidencing that posttraining shifting of the A-US temporal relationship from delay to simultaneous allowed CS X to better inhibit responding to delay transfer CS C. Furthermore, Group Shift.XC demonstrated less conditioned responding than did Group No Shift.XC, $F(1, 53) = 11.48$, $p < .01$, suggesting that posttraining shifting of the A-US temporal relationship resulted in greater inhibitory behavioral control by CS X when compounded with delay transfer CS C.

Finally, greater attenuation of conditioned responding was observed in subjects in Group No Shift.XD relative to Group No Shift.XC, $F(1, 53) = 20.33$, $p < .001$, whereas greater attenuation of conditioned responding was observed in Group Shift.XC relative to Group Shift.XD, $F(1, 53) = 15.56$, $p < .001$. These findings suggest that when the A-US temporal relationship was maintained between conditioned inhibition training and testing (no-shift condition), CS X was a more effective inhibitor of the trace transfer excitor (D) than of the delay transfer excitor (C), and that when the A-US temporal relationship was shifted (from delay to simultaneous) between inhibition training and testing (shift condition), CS X was a more effective inhibitor of the delay transfer excitor (C) than of the trace transfer excitor (D).

In summary, in Experiment 2 we found that a serial Pavlovian inhibitor established with a delay training excitor produced greater conditioned inhibition when tested with a trace, but not a delay, transfer CS. However, posttraining shifting of the training excitor-US temporal relationship resulted in a corresponding shift of temporal control of inhibition. That is, changing the A-US

temporal relationship from delay to simultaneous allowed inhibitor X to inhibit conditioned responding to the delay, but not the trace transfer CS. These findings provide a conceptual replication of the results from Experiment 1 and additional evidence for the temporal control of inhibition being mediated by the training excitor-US temporal relationship in effect at the time of testing. Additionally, they extend the generality of the effect of lengthening the A-US interval after conditioned inhibition training that was seen in Experiment 1 to shortening of the A-US interval as well.

General Discussion

The results of Experiments 1 and 2 indicate that the associative structure of behavior indicative of inhibition is modulated by the inhibitor's training excitor (i.e., inhibitor-training excitor-US [X-A-US]). In Experiment 1 we found that a cue paired in simultaneous compound with a delay training excitor produced greater negative summation when compounded with a delay transfer CS relative to a trace transfer CS but that when the training excitor-US temporal relationship was shifted from delay to trace, the cue produced greater negative summation when tested with the trace transfer CS relative to the delay transfer CS. Similarly, in Experiment 2 we found that a cue paired in serial compound with a delay training excitor maximally inhibited a trace transfer CS relative to a delay transfer CS but that when the training excitor-US temporal relationship was shifted from delay to simultaneous, maximal inhibition was observed when the cue was compounded with the delay relative to the trace transfer CS.

These results are consistent with the temporal coding hypothesis (Barnet et al., 1991; Matzel et al., 1988; Miller & Barnet, 1993; Savastano & Miller, 1998), which posits that animals form temporal maps linking events in memory and use these integrated maps to determine the form and timing of the conditioned response. It is important that these integrated temporal maps appear to include the training excitor with which the inhibitor was established. For example, in Experiment 1, CS X was established as a simultaneous inhibitor for CS A. Assuming that behavior indicative of inhibition is maximal when the inhibitor generates a negative response potential at the same temporal location that the transfer CS signals US presentation, greater inhibition should have been observed when CS X was compounded with the delay transfer CS (C) than with the trace transfer CS (D). Our results confirmed this prediction. Toward investigating the associative structure of temporal control of inhibition, we then shifted the A-US temporal relationship. If the associative structure of inhibition is based on an inhibitory association between the CS and the time of US absence experienced during the nonreinforced XA trials, then changing the A-US temporal relationship after completion of inhibition training should have had no effect on temporal control of inhibition. In contrast to this prediction, we observed a shift in inhibitory behavioral control that paralleled the shift in the training excitor-US temporal relationship. That is, shifting the A-US temporal relationship from delay to trace resulted in the inhibitor producing negative summation when compounded with the trace, but not the delay transfer excitor. These results (as well as the results of Experiment 2) strongly support the view that the temporal location of maximal inhibition following onset of a CS is mediated by the training excitor-US temporal relationship existing at the time when the test is conducted (see Figures 2 and 4).

Although our present results are consistent with those of Lysle and Fowler (1985; see also Hallam et al., 1990) who found that the inhibitory potential of a CS is dependent on the current excitatory associative status of its training excitor, they are inconsistent with those of Witcher and Ayres (1984) and Rescorla and Holland (1977). Witcher and Ayres provided Pavlovian inhibition training (A $\rightarrow$ US/XA-no US) followed by conjoint extinction of CSs A, X, and XA and failed to observe an attenuation of inhibition, relative to control subjects that received no extinction treatment. Applied to the results of Witcher and Ayres, the comparator hypothesis would anticipate that extinction of the training excitor (CS A) should decrease the strength of Link 3, the comparator stimulus-US association, thereby leading to attenuated inhibitory behavioral control. However, Witcher and Ayres additionally provided extinction of the XA compound which might have further strengthened Link 2 (the X-A association), thereby counteracting the effect of weakening Link 3. (Although it should be acknowledged that presentations of X and A alone might have offset any strengthening of the X-A association.)

In a different line of research, Rescorla and Holland provided Pavlovian inhibition training (i.e., A $\rightarrow$ US/XA$-$) followed by extinction of the training excitor (CS A) and found no loss of inhibition, a result inconsistent with the associative structure of inhibition being mediated by the training excitor. However, they provided fewer extinction trials with CS A (24) than have previous studies that observed a loss of inhibition following extinction treatment of the training excitor (e.g., Lysle & Fowler, 1985, provided 96 extinction trials and Hallam et al., 1990, provided 48 extinction trials). Thus, it is possible that had Rescorla and Holland provided more extinction trials, they would have observed a loss of inhibition. More important, Rescorla and Holland found a loss of inhibition following counterconditioning treatment of the transfer excitor. During their counterconditioning treatment, the previously feared transfer excitor that had been 30 s in duration was then presented for 10.5 s and followed by an appetitive US. In light of the present findings, the loss of inhibition following counterconditioning of the transfer excitor might have been due to the change in the valence of the US paired with the transfer excitor and/or the shift in the transfer excitor-US temporal relationship. That is, at test, the inhibitor, which was established with a training excitor that signaled shock at a specific temporal location in the absence of the inhibitor, was compounded with a transfer excitor that signaled the presentation of a different US (food) at another temporal location. The present results suggest that the change in temporal relationship between the transfer excitor and the US might have been sufficient to attenuate inhibitory behavioral control.

In Experiment 2, we investigated the associative structure of an inhibitor trained serially with its training excitor. Following serial inhibition training (i.e., A $\xrightarrow{0}$ US/X $\xrightarrow{0}$ A-no US), CS X produced greater negative summation when compounded with a trace transfer CS than when compounded with a delay transfer CS. Of greater importance, inhibitory behavioral control was shifted following posttraining manipulation of the A-US temporal relationship from delay to simultaneous. This training allowed CS X to better inhibit responding to the delay transfer CS than to the trace transfer CS. The observed potential of a serial inhibitor to attenuate responding to a stimulus when presented in a simultaneous compound is

inconsistent with the findings of Holland and colleagues (Holland, 1984; Holland & Lamarre, 1984; Lamarre & Holland, 1987), who have consistently reported that serial and simultaneous discrimination training result in two different types of inhibitors (negative features). They found that serial negative features generally fail to transfer to simple CSs and instead tend to be specific to excitors that were trained as targets in other feature-negative discriminations (Holland & Lamarre, 1984; Lamarre & Holland, 1987). In contrast, simultaneous negative features readily transfer to simple excitatory stimuli. Thus, our observation that a serial inhibitor (feature) attenuated responding to an independently trained excitor (provided that the temporal expectancies for US presentation and the inhibitor's negative response potential were consistent) is somewhat surprising. However, there are numerous procedural differences between our experiments and those of Holland and his colleagues (see Denniston, Blaisdell, & Miller, 1998, for a review); perhaps the most important difference is that Holland and his colleagues typically presented a gap between their serial negative feature and the target, whereas in the present Experiment 2, there was no gap. Regardless of the source of this discrepancy, Holland et al. (1997) reported a series of experiments in which they observed temporal control of occasion setting similar to our temporal control of inhibition. That is, they found that following serial feature-positive occasion setting training, modulation by the feature was greatest when the feature-target interval was the same as that established during occasion-setting training, a result that is consistent with the present results as well as those of Denniston, Blaisdell, and Miller.

At the theoretical level, the present results are problematic for Schmajuk, Lamoureux, and Holland's (1997) neural network model of inhibition and occasion setting. According to their model, a feature can exert behavioral control through a direct link with the US, an indirect link with the US (mediated by a hidden unit), or both. Typically, serial (occasion-setting or inhibition) training results in a pattern of connections in which both the feature and the target become most strongly linked with a hidden unit that is in turn linked to the US. Each of these links exerts temporal control as a consequence of multiple memory traces (with different onsets and durations) evoked by the CS. Thus, both the feature and the target have separate connections to a common hidden unit, which in turn is linked with the US. One interesting prediction of this model is that posttraining manipulation of the target CS–US temporal relationship might result in the strengthening of different hidden unit-US memory traces (and the extinction of the original hidden unit-US memory traces), which could then allow the feature to exert behavioral control at a different temporal location than that at which it had been originally trained. This prediction is consistent with the results of Experiment 2 in that changes in the A-US temporal relationship apparently resulted in changes in the X-no US temporal expectancy.

Problematic for Schmajuk et al.'s (1997) theory are the results of Experiment 1. In Experiment 1 we found a similar shift in temporal control of inhibition when the training excitor-US temporal relationship was manipulated. According to Schmajuk et al.'s model, simultaneous features (inhibitors) generally form a direct inhibitory CS–US connection. This direct CS–US connection should not be influenced by changes in the training excitor-US temporal relationship, unlike serially trained stimuli, as the patterns of connections differ (i.e., direct vs. mediated, respectively). On the basis of our present findings, it appears that both simultaneous and serial inhibitors exert behavioral control through a common associative structure (X-A-US), rather than different associative structures, a result more consistent with Buhusi and Schmajuk's (1999) model that has multiple memory traces for each CS.

The present results can be illuminated by the comparator hypothesis's (Denniston et al., 2001; Miller & Matzel, 1988) conceptualization of behavior indicative of inhibition. According to the comparator hypothesis, the strength of the inhibitor-US association (which is nil) is compared with the strength of the training excitor (comparator stimulus)-US association. As the strength of the comparator stimulus-US association increases relative to the strength of the inhibitor-US association, inhibitory behavioral control is predicted to increase (and excitatory behavioral control is predicted to decrease). Conversely, as the strength of the comparator stimulus-US association decreases, inhibitory behavioral control should decrease (and excitatory behavioral control should increase). Thus, the comparator hypothesis can anticipate the occurrence of behavior indicative of inhibition and can explain the attenuation of inhibitory behavioral control following extinction of the training excitor.

Notably, the comparator hypothesis alone is unable to explain the effect of posttraining manipulation of the training excitor-US temporal relationship on the temporal control of inhibition in that it has no mechanism for explaining temporal control of behavior. However, integration of the comparator and temporal coding hypotheses provides a more complete account of the present results (see Blaisdell, Denniston, & Miller, 1999; Denniston, Blaisdell, & Miller, 1998). According to this conjoint application, the inhibitory strength of CS X is derived from its association with CS A (X's training excitor, because CS X was never directly paired with the US) and is directly proportional to the strengths of the X-A and A-US associations. Likewise, temporal expectation for US omission is provided primarily by the conditioned inhibitor's (CS X's) training excitor (CS A, its comparator stimulus). In other words, the conditioned inhibitor does not directly signal US omission; rather, it indirectly activates a strong US representation at a particular temporal location through its comparator stimulus (the training excitor). Thus, the inhibitor–training excitor and the training excitor-US temporal relationships determine the temporal location at which inhibitory behavioral control will be maximal. Hence, on a summation test, behavior indicative of inhibition reflects the sum of the effective associative strengths of the transfer excitor and the conditioned inhibitor. Alternatively stated, inhibitory behavioral control arises from the comparator process producing a negative effective response potential that subtracts from the positive response potential of the transfer excitor.

It is important that negative summation should be maximal when the transfer excitor and the conditioned inhibitor's comparator stimulus (CS A) activate representations of the US that share common temporal (as well as quantitative and qualitative) attributes. When there is a mismatch in any of these US attributes, attenuated inhibitory behavioral control should be observed. For example, on the basis of the comparator hypothesis alone, one might expect that the additional training excitor-US pairings provided during update training should have enhanced inhibition overall (i.e., a form of associative inflation that should have enhanced inhibition regardless of which transfer excitor the inhib-

itor was compounded with at test). In contrast to this prediction, specific temporal control of inhibition at a new temporal location was observed, a result that highlights the importance of temporal variables in determining the output of the comparator process.

In the present experiments, behavior indicative of inhibition was maximal when the conditioned inhibitor's comparator stimulus activated a US representation at the same temporal location as the transfer excitor activated a US representation. When there was a mismatch in temporal expectancies, as signaled by the inhibitor's training excitor and the transfer excitor presented at test, inhibitory behavioral control was reduced. Of greater importance, posttraining manipulation of the training excitor-US temporal relationship produced a corresponding shift in inhibitory behavioral control. On the basis of the above account, this shift in inhibitory behavioral control is thought to result from the inhibitor indirectly activating a strong US representation at a new temporal location (see Figures 2 and 4). At test, this allowed the conditioned inhibitor to pass a negative summation test with a transfer excitor that signaled US presentation at this new temporal location, but not with a transfer excitor that signaled US presentation at the original temporal location.

As previously discussed, both the temporal coding and comparator hypotheses are required to more fully account for the present results. However, some aspects of the current results and theoretical integration warrant comment. First, it is somewhat surprising that our posttraining manipulation of the training excitor-US temporal relationship was as effective as it was, particularly after only 64 training trials at the new temporal interval, in that conceptually similar research has required far more training to obtain analogous comparator effects. For instance, both overshadowing and blocking can be attenuated when the comparator stimulus is subjected to posttraining devaluation (i.e., extinction), which presumably weakens the effective strength of the indirectly activated US representation. However, these effects typically require extensive extinction treatment (e.g., 214 trials in the case of overshadowing, Denniston, Savastano, Blaisdell, & Miller, 2003; and 800 trials in the case of blocking, Blaisdell, Gunther, & Miller, 1999). One potential explanation for the increased effectiveness of the temporal shifting manipulation, relative to the posttraining devaluation manipulation, is that US presentation might be more salient than US omission. That is, in the present experiments we manipulated the status of the inhibitor's comparator stimulus by pairing it with the US at a different temporal location than had been in effect during original inhibition training. Studies of recovery from cue competition, however, manipulated the associative status of the overshadowed or blocked CS's comparator stimulus by presenting the comparator stimulus in the absence of the US. If US presentation is more salient than US omission, the former treatment might produce more rapid changes in associative strength than the latter. Such a view has been incorporated into many contemporary theories of associative acquisition. For example, the Rescorla–Wagner model (Rescorla & Wagner, 1972) posits different values for beta (a learning rate parameter) depending on whether the US is present or absent.

An additional potential concern regarding the current theoretical integration is that both the comparator and temporal coding hypotheses are qualitative in nature (i.e., they lack quantitative rules for acquisition and performance), potentially reducing their predictive power. However, many so-called quantitative models of associative learning (e.g., Rescorla & Wagner, 1972) ultimately reduce to ordinal predictions because they lack precise rules for transforming learning into performance. Thus, the comparator and temporal coding hypotheses might not be qualitatively different than their quantitative peers.

In summary, the present experiments found that temporal control of inhibition is mediated by the temporal associative status of the inhibitor's training excitor at test. This suggests that the associative structure of temporal control of inhibition is X-A-US rather than a direct X-US association. Supportive of this conclusion is the observation that posttraining shifts in the training excitor-US temporal relationship resulted in a corresponding shift in the negative response potential generated by the inhibitor. When this updated temporal expectancy for US omission matched the temporal expectancy for US presentation signaled by the transfer excitor, robust inhibitory behavioral control was observed. These findings are problematic for theories of inhibition that posit a direct inhibitor-US association but are fully consistent with an integration of the temporal coding and comparator hypotheses.

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