Journal of Experimental Psychology:
Animal Behavior Processes
1998, Vol. 24, No. 4, 453–466

0097-7403/98/$3.00

Comparator Mechanisms and Conditioned Inhibition: Conditioned Stimulus Preexposure Disrupts Pavlovian Conditioned Inhibition but Not Explicitly Unpaired Inhibition

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Three conditioned lick-suppression experiments with rats examined the effects of pretraining exposure to the conditioned stimulus (CS) on behavior indicative of conditioned inhibition. After CS-preexposure treatment, subjects received either Pavlovian conditioned inhibition training or explicitly unpaired inhibition training with the preexposed CS. The inhibitory status of the CS was then assessed with a retardation (Experiment 1) or a summation (Experiment 2) test. Experiment 3 controlled for the unconditioned stimulus-preexposure effect being a potential confound in Experiments 1 and 2. As predicted by the comparator hypothesis (R. R. Miller & L. D. Matzel, 1988), the CS–context association that developed during the CS-preexposure phase disrupted the expression of Pavlovian conditioned inhibition but not the expression of explicitly unpaired inhibition.

Conditioned stimulus (CS)-preexposure effect (also known as latent inhibition, Lubow & Moore, 1959; Lubow, 1973) is the finding that repeated nonreinforced presentations of a CS prior to its being paired with an unconditioned stimulus (US), impairs later acquisition of responding to that CS (i.e., a retardation of behavioral control by the CS). Originally, the label *latent inhibition* was applied to the CS-preexposure effect because the effect was thought to arise from inhibition accrued to the CS as a result of its initially being presented in the absence of the US. However, most current interpretations of the CS-preexposure effect attribute the phenomenon to an attentional decrement accrued to the CS as a consequence of the stimulus predicting nothing of consequence during the pretraining exposure treatment (Lubow, Schnur, & Rifkin, 1976; Lubow, Weiner, & Schnur, 1981; Mackintosh, 1975; Pearce & Hall, 1980; Wagner, 1981). The underlying assumption is that the resultant decrease in attention paid to the preexposed CS as a result of pretraining exposure hinders the subsequent formation of a strong CS–US association during conditioning.

More recently, numerous investigators have demonstrated an apparent release from the CS-preexposure deficit, using posttraining "reminder" treatments (Kasprow, Catterson, Schachtman, & Miller, 1984), delayed testing (Kraemer, Hoffman, & Spear, 1988; Kraemer, Randall, & Carbary, 1991; Kraemer & Roberts, 1984), context switching (Bouton, 1993), or posttraining extinction of the training context (Grahame, Barnet, Gunther, & Miller, 1994) or by presenting a second CS during training (Blaisdell, Bristol, Gunther, & Miller, 1998, see General Discussion). These experiments suggest that CS preexposure does not necessarily render the CS impervious to the acquisition of an effective CS–US association but rather that it interferes with the expression of an adequately acquired association. This conclusion is warranted because the CS-preexposure effect can be reversed in the absence of any posttraining treatment involving presentation of the preexposed CS. Thus, these results are incompatible with the previously stated explanations that assume the CS-preexposure effect arises from an acquisition deficit.

Another avenue with which to explore the CS-preexposure effect is through its effect on conditioned inhibition. Two treatments commonly used to produce an inhibitory CS are Pavlov's (1927) procedure and the explicitly unpaired procedure. In Pavlov's procedure, Stimulus A is reinforced when presented alone (i.e., A → US), but it is not reinforced when presented in compound (usually simultaneous) with the intended inhibitory Stimulus X (i.e., AX−). In the explicitly unpaired conditioned inhibition procedure, unsignaled presentations of the US are interspersed with nonreinforced presentations of Stimulus X, the intended conditioned inhibitor. Both procedures result in the target CS (X) acquiring inhibitory properties, as assessed by passing retardation and summation tests for conditioned inhibition. The retardation test examines the rate at which a putative conditioned inhibitor can be transformed into an excitatory stimulus through X–US pairings. The summation test examines the ability of a putative inhibitor to attenuate excitatory conditioned responding to an independently trained condi-

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Support for this research was provided by National Institute of Mental Health Grant 33881. We thank Daniel C. Burger, James C. Denniston, Lisa M. Gunther, Christopher Randall, Lisa Savage, Hernan Savastano, and David S. Wilson for comments on a preliminary draft of this article. We also thank James Esposito, Lisa Haur, and Nehal P. Vadhan for assistance with the collection of data.

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tioned excitor (B), when the two stimuli are presented simultaneously at test. A comparison is then made between the behavior elicited by the BX-compound stimulus with that elicited by Stimulus B alone. Less conditioned suppression to the BX compound than to B alone is ordinarily interpreted as indicative of X being a conditioned inhibitor.

Both tests of conditioned inhibition are necessary because there are a variety of other processes that can explain a CS "passing" either test alone (Hearst, 1972; Rescorla, 1969; but see Papini & Bitterman, 1993; Savastano, Cole, Barnet, & Miller, in press; Williams, Overmier, & LoLordo, 1992, for criticisms of this dictum). For example, passage of retardation tests may result from decreased attention to the target stimulus (X) because of prior nonreinforced exposures, thereby resulting in retarded development of behavioral control to X. Conversely, passage of a summation test may result from Stimulus X's controlling too much attention at the cost of attention to the summation test excitor. However, passage of both tests would refute these attentional explanations because they are mutually exclusive.

Using the two-test strategy, Rescorla (1971) and Reiss and Wagner (1972) attempted to transform a preexposed target CS into a conditioned inhibition by using Pavlov's conditioned inhibition procedure (i.e., A → US/AX−). They both found that CS preexposure impaired their target CS's potential to pass a summation test for conditioned inhibition. These researchers attributed their results to a decrement in the potential of the preexposed CS to enter into an inhibitory association. More specifically, they hypothesized that the preexposed CS had undergone a loss in its potential to command attention during subsequent inhibitory conditioning. The present research challenges this explanation and provides evidence instead that the observed retardation in acquiring inhibitory potential following CS-preexposure treatment is due to a CS–context association that is formed during CS-preexposure treatment. The basis for this view rests on the comparator hypothesis (Miller & Matzel, 1988; Miller & Schachtman, 1985).

The comparator hypothesis is a qualitative response rule for the expression of acquired Pavlovian associations. In the framework of the comparator hypothesis, conditioned responding is thought to reflect a comparison between the strength of the target CS–US association and the product of the strengths of the target CS–comparator stimulus association and comparator stimulus–US association, with the comparator stimulus for a CS being the stimulus with which the target CS has the strongest association (other than the US). More specifically, the comparator hypothesis posits that there are three associative links that together provide the basis for conditioned responding: Link 1, the direct target CS–US association; Link 2, the target CS–comparator stimulus association; and Link 3, the comparator stimulus–US association (see Figure 1). Excitatory responding is expected under conditions in which Link 1 is strong relative to the product of Links 2 and 3. However, inhibitory responding should increase, whereas excitatory responding should decrease, as a result of either weakening Link 1 or strengthening the product of Links 2 and 3.

In the framework of the comparator hypothesis, Pavlovian conditioned inhibition training (A → US/AX−) produces behavior indicative of conditioned inhibition because the X–US association (Link 1 of Figure 2) is weak due to X having never been paired with the US, whereas the X–A association (Link 2) and the A–US association (Link 3) are both strong as a result of the AX− trials and the A → US trials, respectively, during training. The comparator hypothesis explains X's inhibitory control of behavior as arising from the product of the X–A and A–US associations being strong relative to the X–US association. Extinction of the A–US association (Link 3), by administering nonreinforced presentations of the A stimulus following Pavlovian conditioned inhibition training, has previously been shown to

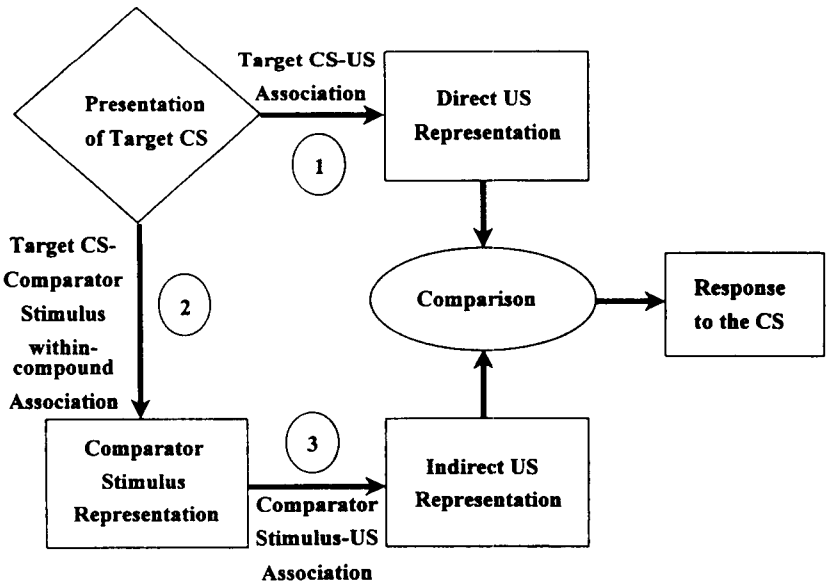

Figure 1. The comparator hypothesis. CS = conditioned stimulus; US = unconditioned stimulus.

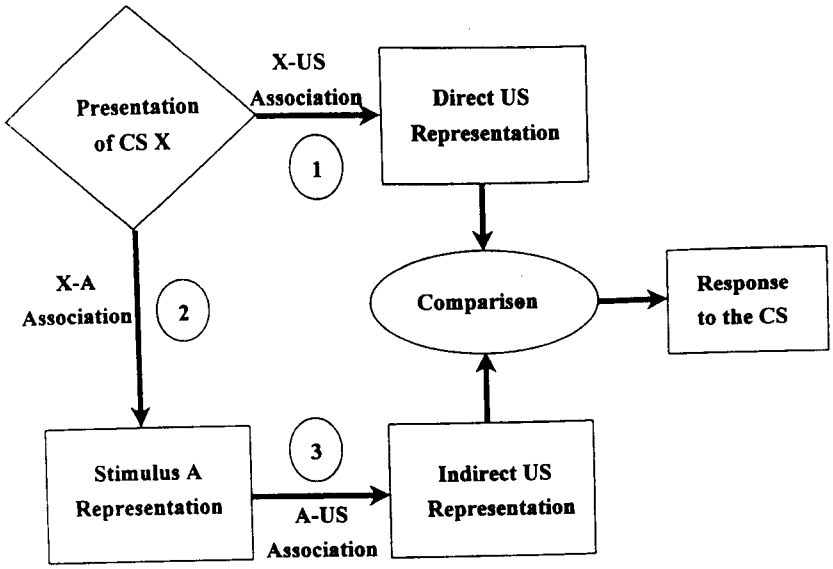

Figure 2. The comparator hypothesis applied to Pavlovian Inhibition. CS = conditioned stimulus; US = unconditioned stimulus; X = conditioned inhibitor; A = conditioned excitor.

result in an attenuation of the behavior indicative of conditioned inhibition (Hallam, Matzel, Sloat, & Miller, 1990; but see Williams, Travis, & Overmier, 1986). According to the comparator hypothesis, this decrement in behavior indicative of conditioned inhibition occurs because the product of Links 2 and 3 has been weakened as a result of devaluing Link 3 (and perhaps Link 2), thereby reducing its value relative to the direct X–US association (Link 1). It is important to note that, in Hallam et al.'s (1990) study, posttraining extinction of the training excitor (A), but not the context in which inhibition training was given, attenuated behavior indicative of conditioned inhibition.

The comparator hypothesis posits that the effective X–A association (i.e., Link 2) will also be devalued when CS-preexposure treatment precedes Pavlovian conditioned inhibition training. The nonreinforced presentations of X during CS-preexposure treatment presumably create a strong X–context association, which renders ineffective (i.e., blocks) the X–A association that would otherwise develop during the AX− trials of Pavlovian conditioned inhibition training. This results in a weak X–A association (Link 2). For evidence that, in a two-event sequence, one subsequent event can block another subsequent event, see Esmoris-Arranz, Matute, and Miller (1997). This blocking of the X–A association presumably resulted in a failure to express conditioned inhibition, that is, the effect originally reported by Rescorla (1971) and Reiss and Wagner (1972).[1]

In contrast to Pavlovian conditioned inhibition training, with explicitly unpaired conditioned inhibition training (i.e., US/X−), the context is the comparator stimulus for X. This view has been supported in studies that found posttraining extinction of the context to attenuate inhibitory behavioral control by the target CS (X; e.g., Kasprow, Schachtman, & Miller, 1987; Schachtman, Brown, Gordon, Catterson, & Miller, 1987). Thus, in the framework of the comparator hypothesis, CS-preexposure treatment (X−) ought to establish a strong X–context association (i.e., Link 2), which, if not already asymptotic, is further strengthened during explicitly unpaired conditioned inhibition treatment (see Figure 3).

According to the comparator hypothesis, the primary difference in the effect of CS preexposure on inhibition produced by these two conditioned inhibition procedures lies in which stimulus is primarily acting as the comparator

[1] An explanation of competition between comparator stimuli, such as that predicted in the present study as well as previously published effects (Blaisdell et al., 1998; Grahame, Barnet, & Miller, 1992; Miller, Esposito, & Grahame, 1992), requires further elaboration. The comparator hypothesis assumes that Link 1 is determined by the absolute strength of the target CS–US association, whereas Links 2 and 3 (the target CS–comparator stimulus and comparator stimulus–US associations, respectively) are "effective" associations (i.e., the comparator value of each association is modulated by its own comparator stimuli). These second-order comparator loops provide the basis for specific predictions regarding the effectiveness of the context and A as comparator stimuli for X. By preexposing subjects to the CS (in the treatment context) prior to Pavlovian conditioned inhibition treatment, the context should be established as the comparator stimulus for X. However, the context–US association (Link 3) presumably was modulated by its comparator stimulus, which was A (we again assume that the target CS and US cannot act as comparator stimuli for the X–US association). Because A was the most salient stimulus present during US presentations and had the greatest contiguity with the US, the A–US association effectively decremented the context's ability to modulate conditioned responding to X (see Blaisdell et al., 1998, for further elaboration of this second-order comparator mechanism). Because of this weak effective context–US association, only a weak indirect representation of the US should have been activated by the Link 2–Link 3 chain on presentation of X. As a strong indirect representation of the US is presumed to be the basis of inhibitory behavior (as well as the CS-preexposure effect) in response to X, conditioned inhibition (or latent inhibition) was not anticipated.

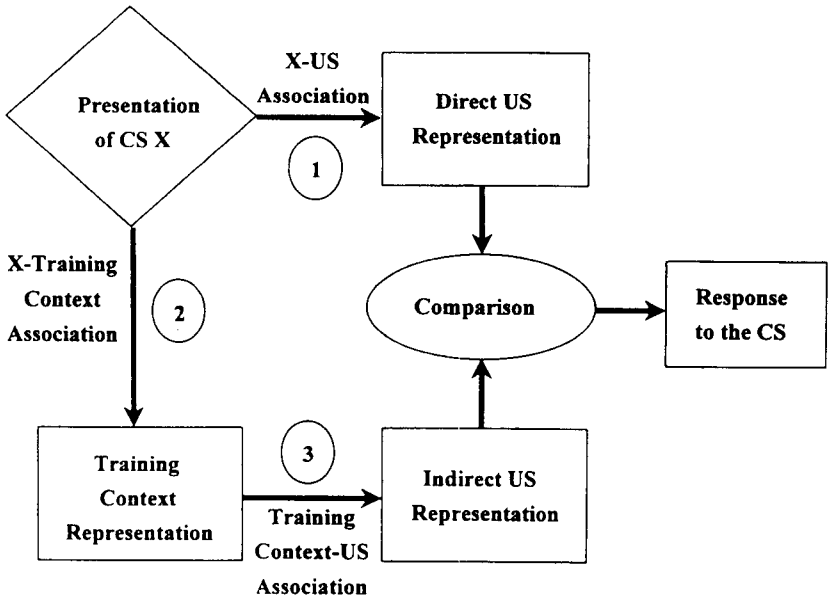

Figure 3. The comparator hypothesis applied to explicitly unpaired inhibition. CS = conditioned stimulus; US = unconditioned stimulus; X = conditioned inhibitor.

stimulus for X at test. CS preexposure establishes the context as X's primary comparator stimulus. Because the context is the comparator stimulus that is normally established during explicitly unpaired conditioned inhibition treatment (i.e., in the absence of CS preexposure), CS preexposure should not degrade inhibitory responding to X. However, Pavlovian conditioned inhibition treatment normally establishes A as the primary comparator stimulus for X. Thus, pretraining establishment of the context as X's primary comparator stimulus ought to degrade behavior indicative of X being a conditioned inhibitor. Consequently, CS-preexposure treatment should impede the development of behavior indicative of conditioned inhibition produced by Pavlovian conditioned inhibition training but not behavior indicative of conditioned inhibition produced by explicitly unpaired conditioned inhibition training. Such an outcome would further challenge the view that CS preexposure results in an inability of the preexposed CS to enter into an association with another stimulus. Rather, confirmation of these predicted outcomes would suggest that the CS-preexposure effect reflects a deficit in expressing, rather than in acquiring, an association. Specifically, these outcomes would provide support for a comparator hypothesis mechanism being responsible for both the CS-preexposure effect and the behavior indicative of conditioned inhibition.

Experiment 1 (Retardation Test)

Experiment 1 used a retardation test to assess conditioned inhibition to examine the effects of CS-preexposure treatment on subsequent inhibition training. According to the comparator hypothesis, conditioned inhibition was expected to develop unimpeded in subjects that received explicitly unpaired conditioned inhibition training, but not in subjects that received Pavlovian conditioned inhibition training.

Method

Subjects

Thirty male (235–360 g) and 30 female (190–240 g) Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to one of five groups (*ns* = 12), counterbalanced for sex. Subjects were individually housed in wire-mesh cages in a vivarium maintained on a 16:8-hr light–dark cycle. Experimental manipulations occurred approximately midway through the light portion of the cycle. A progressive water deprivation schedule was imposed over the week prior to the beginning of the experiment until availability was limited to 10 min per day. All subjects were handled for 30 s three times per week, from weaning until the initiation of the study.

Apparatus

Two types of enclosures were used. Enclosure R was a clear Plexiglas, rectangular chamber measuring 31.5 × 9.5 × 15.5 cm (length × width × height). The floor was constructed of 0.48-cm diameter stainless steel rods, spaced 1.5 cm center-to-center, connected by NE-2 neon bulbs that allowed the delivery of 1.0-mA constant-current footshock by means of a high-voltage AC circuit in series with a 1.0-MΩ resistor. Each of six copies of Enclosure R was housed in a separate light- and sound-attenuating environmental isolation chest. Enclosure R was dimly illuminated by a 2-W (nominal at 120 VAC) incandescent house light driven at 56 VAC. A 25-W incandescent bulb (nominal at 120 VAC) driven at 80 VAC was mounted on the interior back wall of each environmental chest and, when flashed (0.25 s on–0.25 s off), served as CS A. The house light was turned off when the light stimulus was being flashed on and off. Both bulbs were mounted approximately 30 cm from the center of each experimental chamber. Background noise, mostly from a ventilation fan, was 74 db(C) re. SPL.

Enclosure V was a 22.5-cm long box in the shape of a vertical truncated V (30.0 cm high, 21.5 cm wide at the top, and narrowing

to 5.25 cm wide at the bottom). The ceiling was constructed of clear Plexiglas and two side walls of black Plexiglas, whereas the sloping side walls (which gave the chamber its V shape) were composed of stainless steel. The floor consisted of two long parallel metal plates, each 2 cm wide and separated by a 1.25-cm gap. A 1.0-mA constant-current footshock could be delivered through the metal walls and floor of the enclosure. Each of six copies of Enclosure V was housed in a separate light- and sound-attenuating environmental isolation chest. Enclosure V was dimly illuminated by a 7.5-W (nominal at 120 VAC) incandescent house light driven at 56 VAC. A 100-W incandescent bulb (nominal at 120 VAC) driven at 80 VAC was mounted on the back wall of each environmental chest and, when flashed (0.25 s on–0.25 s off), served as CS A. The house light was turned off when the light stimulus was being flashed on and off. Both bulbs were mounted approximately 30 cm from the center of each experimental chamber. Light entered the experimental enclosure primarily by reflection from the roof of the environmental chest. The light intensities of Enclosure V roughly matched those of Enclosure R, because of differences in the opaqueness of the walls of the two types of enclosures compensating for the difference in the luminosity of the bulbs. Background noise, mostly from a ventilation fan, measured 74 db(C).

Enclosures R and V each could 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 narrow Plexiglas walls of the chamber (axis of the recess and of the lick tube were both perpendicular to the wall). Each drinking recess was left–right centered on the chamber wall, with its bottom 1.75 cm above the floor of the apparatus. The recess was 4.5 cm in diameter and 5.0 cm deep. An infrared photobeam was projected horizontally across the recess approximately 1 cm in front of the lick tube. To drink from the lick tube, subjects had to insert their head into the recess, thereby breaking the photobeam. Thus, the times during which the subjects were licking could be recorded.

Each enclosure was equipped with three 45-Ω speakers mounted on the interior walls of the environmental chest. A high-frequency complex tone (3000 and 3200 Hz) and a white-noise stimulus could be emitted from two of the speakers and served as Stimuli X and Y (counterbalanced within groups). The third speaker could deliver Stimulus B, a click train (6/s). All three auditory stimuli were delivered at 8 dB(C) above the background noise of 74 dB(C) and were presented for 10 s in duration during training. The footshock US was always 0.5 s in duration, and it occurred directly after CS termination on trials in which the US was signaled.

Three distinct contexts were used in this study. Context 1 consisted of Enclosures R or V (counterbalanced within groups) illuminated by their house lights. Context 2 consisted of the opposite enclosure (V or R) than Context 1, with the house light also on. Context 3 consisted of a different instance of Enclosure R than was encountered in either of the previous contexts. Context 3 was further differentiated with the addition of a Plexiglas plate covering the grid floor, the house light being turned off, and the presence of a distinctive odor cue. One drop of 98% methyl salicylate was placed onto small wooden blocks located inside of the environmental chests.

Procedure

Specific group names are explained along with a summary of the critical aspects of the training procedure in Table 1.

Acclimation. All subjects were acclimated to Contexts 1, 2, and 3 on Days 1, 2, and 3, respectively. During each daily 30-min session, no punctuate stimuli were presented. The animals had access to the water-filled lick tubes in all three contexts.

Phase 1 (CS-preexposure treatment). Prior to the initiation of Phase 1, the lick tubes were removed from each chamber. Training was conducted on Days 4–11, with 60-min sessions occurring daily in Context 1. Training consisted of 60 pseudorandomly distributed (average intertrial interval of 60 s) nonreinforced presentations of Stimulus X per daily session for Groups CS-preexposure. Pavlovian conditioned inhibition (CSpre.Pav) and CS-preexposure.explicitly unpaired conditioned inhibition (CSpre.EU), and of Stimulus Y for Groups Pavlovian conditioned inhibition (Pav), explicitly unpaired conditioned inhibition (EU), and retardation control (RetardCon).

Phase 2 (conditioned inhibition training). On Days 12–17, subjects received daily 60-min training sessions in Context 1. Groups CSpre.Pav and Pav received 8 reinforced presentations of A (A → US) interspersed with 8 nonreinforced AX presentations (AX−) per daily session. Group RetardCon received 8 reinforced A presentations (A → US) interspersed with 8 nonreinforced AY presentations (AY−). Groups CSpre.EU and EU received 8 unsignaled footshock presentations interspersed with 8 nonreinforced X presentations (X−) per day. The mean intertrial interval for the 16 trials was 3.5 min. It should be noted that the parameters (numbers of each type of trial) for conditioned inhibition training given to the five groups were less than optimal for either Pavlovian conditioned inhibition or explicitly unpaired conditioned inhibi-

Table 1
Design Summary: Experiment 1 (Retardation Test)

Group	CS preexposure$_1$	Training			Test$_3$	Predicted
		CI$_1$	Transfer$_2$	Excitation$_2$		
CSpre.Pav	X−	A → US/AX−	B → US	X → US	X	CR
Pav	Y−	A → US/AX−	B → US	X → US	X	cr
CSpre.EU	X−	US/X−	B → US	X → US	X	cr
EU	Y−	US/X−	B → US	X → US	X	cr
RetardCon	Y−	A → US/AY−	B → US	X → US	X	CR

Note. X and Y denote two stimuli (tone or white noise), counterbalanced within groups. A was a flashing light. US and a minus sign denote the presence and absence of the US (footshock), respectively. Subscripts denote context. Summation excitor training (B → US) was given to equate stimuli exposure with that of Experiment 2. CS = conditioned stimulus; US = unconditioned stimulus; CI = conditioned inhibition training; CSpre = CS preexposure; Pav = Pavlovian conditioned inhibition; EU = explicitly unpaired conditioned inhibition; RetardCon = retardation control; CR = strong responding; cr = weak responding.

tion. Because of the differential requirements needed to attain maximal conditioned inhibition with the two procedures (Pavlovian and explicitly unpaired; see, e.g., Williams & Overmier, 1988; Williams et al., 1986), a middle ground was taken to match Groups Pav and EU in terms of numbers of nonreinforced trials with X and reinforced trials.

Phase 3 (summation excitor training). On Days 18 and 19, all subjects received four reinforced presentations of Stimulus B per day during a 60-min session in Context 2. The mean intertrial interval was 12 min. This training was given to equate the total stimulus exposure with that of Experiment 2 of this series, which used a summation test for the assessment of conditioned inhibition and hence required a transfer excitor for the summation test.

Phase 4 (retardation excitor training). On Day 20, all subjects received three reinforced presentations of X during a single 60-min session in Context 2. The mean intertrial interval was 15 min.

Reacclimation. On Days 21 and 22, the water-filled lick tubes were reinserted, and daily 30-min reacclimation sessions were conducted in Context 3. This treatment served to restabilize baseline levels of drinking. There were no nominal stimulus presentations during these sessions.

Testing. On Day 23, all subjects were tested for conditioned lick suppression to X by presenting X immediately on completion of 5 cumulative s of licking (as measured by the total amount of time the infrared photobeam was disrupted). Thus, all subjects were drinking at the time of CS onset. Time to complete this initial 5 cumulative s of licking and time to complete an additional 5 cumulative s of licking in the presence of X were recorded. Test sessions were 16 min in duration, with a ceiling score of 15 min being imposed on the time to complete the 5 cumulative s of drinking in the presence of X.

Suppression data were transformed to log (base 10) scores to facilitate the use of parametric statistics. An alpha level of .05 was adopted for all tests of statistical significance. Additionally, following the convention of our laboratory, subjects that took more than 60 s to complete their first 5 cumulative s of licking (i.e., prior to CS onset), thereby exhibiting an unusual reluctance to drink in the test context, were eliminated from all analyses. One subject from Group CSpre.Pav failed to meet this criterion.

Results and Discussion

As can be seen in Figure 4, without CS preexposure, both the Pavlovian conditioned inhibition and the explicitly unpaired conditioned inhibition procedures produced conditioned inhibition as assessed by a retardation test. More important, as measured by a retardation test, conditioned inhibition developed in those subjects that received the explicitly unpaired conditioned inhibition training following CS preexposure, but it did not appear in those subjects that received Pavlovian conditioned inhibition training following CS-preexposure treatment. The following analyses support these conclusions.

A one-way analysis of variance (ANOVA) conducted on the pre-CS times to complete 5 cumulative s of licking found no significant group differences in baseline drinking behavior; that is, no difference in fear of the test context was evident between groups in this experiment (nor in the following two experiments), $Fs < 1.0$. A one-way ANOVA conducted on the suppression scores during the presentation of X revealed a treatment effect, $F(4, 54) = 14.42$, $p < .01$. A 2 × 2 ANOVA with Phase 1 treatment (X or Y; i.e., CS preexposure or no CS preexposure, respectively) and Phase 2 treatment (A → US/AX− or US/X−; i.e., Pavlov's or the explicitly unpaired conditioned inhibition procedure) as factors was conducted on the suppression scores of the first four groups (CSpre.Pav, Pav, CSpre.EU, and EU). This revealed a Phase 1- × Phase 2-treatment interaction, $F(1, 43) = 11.38$, $p < .01$, as well as main effects of Phase 1 treatment and Phase 2 treatment, $Fs(1, 43) = 7.36$ and 31.20, $ps < .01$, respectively.

Planned comparisons were conducted on the suppression to X scores by using the overall error term from the one-way ANOVA. Subjects in Groups Pav and EU suppressed less to X than did subjects in Group RetardCon, thereby demonstrating that, in the absence of CS preexposure, conditioned inhibition was attained with both Pavlov's and the explicitly unpaired procedures, $Fs(1, 54) = 11.27$ and 23.13, $ps < .01$. A comparison between Groups CSpre.Pav and Pav revealed that the CS-preexposure treatment given to the former group had a significant effect on the development of conditioned inhibition attained with Pavlov's procedure, $F(1, 54) = 15.29$, $p < .01$. Moreover, the effect of CS-preexposure treatment on Pavlov's procedure (Group CSpre.Pav) led to behavior highly similar to that of the RetardCon group, revealing that the absence of conditioned inhibition as a result of CS preexposure was nearly complete, $F(1, 54) < 1.0$.

In contrast, a comparison between Groups CSpre.EU and EU revealed no significant effect of CS-preexposure treatment on the attainment of conditioned inhibition obtained with the explicitly unpaired procedure, $F(1, 54) < 1.0$, $p = .67$. Furthermore, comparing Group CSpre.EU with the RetardCon group revealed the acquisition of conditioned inhibition with the explicitly unpaired procedure despite prior CS-preexposure treatment, $F(1, 54) = 27.50$, $p < .01$. Finally, we were not able to detect a difference between Groups Pav and EU within the power provided by the assessment technique, $F(1, 54) = 2.07$, $p > .15$. This last contrast argues against the observed failure of CS-preexposure treatment to attenuate explicitly unpaired conditioned inhibition because of the baseline inhibition being stronger in the case of explicitly unpaired training than in the case of Pavlovian inhibition training.

Experiment 2 (Summation Test)

Experiment 1 yielded the expected differential effects of CS-preexposure treatment on the expression of conditioned inhibition produced with the explicitly unpaired procedure and Pavlov's procedure, as assessed with a retardation test. Specifically, preexposure to X degraded its ability to act as an inhibitor following Pavlovian conditioned inhibition training, but not following explicitly unpaired conditioned inhibition training. Experiment 2 used a summation test for conditioned inhibition to further examine the effects of CS preexposure on inhibition produced with these two proce-

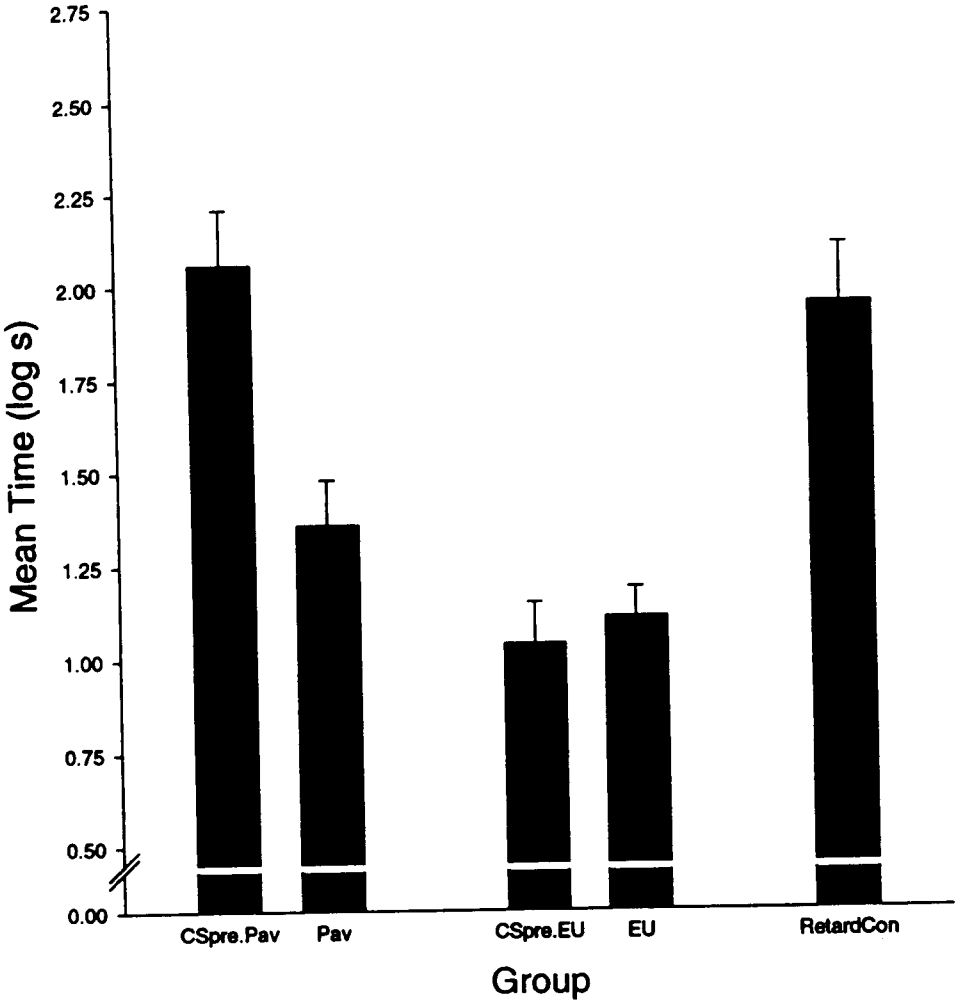

Figure 4. Experiment 1: Mean times (log s) to complete 5 cumulative s of licking in the presence of the target conditioned stimulus (CS). All groups were tested on X. Error bars represent standard errors of means. CSpre = CS preexposure; Pav = Pavlovian conditioned inhibition; EU = explicitly unpaired conditioned inhibition; RetardCon = retardation control.

dures. The predictions outlined in the previous experiment apply to Experiment 2 as well, because the central difference between the two experiments was merely the testing procedure used to assess conditioned inhibition.

Method

Subjects and Apparatus

Thirty male (250–420 g) and 30 female (180–265 g) Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to one of five groups (*ns* = 12), counterbalanced for sex. The animals were housed and maintained as in Experiment 1. The apparatus and stimuli were identical to those used in Experiment 1.

Procedure

Specific group names are explained along with a summary of the critical aspects of the training procedure in Table 2.

Acclimation. On Days 1–3, all subjects were acclimated to Contexts 1–3 as in Experiment 1.

Phase 1 (CS-preexposure treatment). On Days 4–11, all subjects in Groups CSpre.Pav, Pav, CSpre.EU, and EU received training in Context 1 as in Experiment 1. Group summation excitor (SumExcite) received training identical to Group CSpre.Pav.

Phase 2 (conditioned inhibition training). On Days 12–17, Groups CSpre.Pav, Pav, CSpre.EU, and EU received training in Context 1 as in Experiment 1. Group SumExcite received training identical to Group CSpre.Pav.

Phase 3 (summation excitor training). On Days 18 and 19, all subjects received transfer excitor training in Context 2 as in Experiment 1.

Reacclimation. On Days 20 and 21, all subjects were reacclimated to Context 3 as in Experiment 1.

Testing. On Day 22, subjects in Group SumExcite were tested for conditioned lick suppression to B. Subjects in Groups CSpre.Pav, Pav, CSpre.EU, and EU were tested on the compound Stimulus BX. Testing was conducted in Context 3 in the same manner as in Experiment 1. One subject from Group EU was eliminated from

Table 2
Design Summary: Experiment 2 (Summation Test)

Group	CS preexposure$_1$	Training			
		CI$_1$	Transfer$_2$	Test$_3$	Predicted
CSpre.Pav	X−	A → US/AX−	B → US	BX	CR
Pav	Y−	A → US/AX−	B → US	BX	cr
CSpre.EU	X−	US/X−	B → US	BX	cr
EU	Y−	US/X−	B → US	BX	cr
SumExcite	X−	A → US/AX−	B → US	B	CR

Note. X and Y denote two stimuli (tone or white noise), counterbalanced within groups. A was a flashing light. US and a minus sign denote the presence and absence of the US, respectively. Subscripts denote context. CS = conditioned stimulus; US = unconditioned stimulus; CSpre = CS preexposure; Pav = Pavlovian conditioned inhibition; EU = explicitly unpaired conditioned inhibition; SumExcite = summation excitor; CR = strong responding; cr = weak responding; CI = conditioned inhibition training; B = a click train.

the analysis for failing to complete an initial 5 cumulative s of licking (prior to CS onset) within 60 s on the test day.

Results and Discussion

Without CS preexposure, both the Pavlovian conditioned inhibition procedure and the explicitly unpaired conditioned inhibition procedure produced conditioned inhibition as assessed by a summation test. More important, CS preexposure interfered with the expression of Pavlovian conditioned inhibition but not with the expression of explicitly unpaired conditioned inhibition.

A one-way ANOVA conducted on suppression scores during BX (or B) revealed a treatment effect, $F(4, 54) = 7.61, p < .01$ (see Figure 5). A 2 × 2 ANOVA with Phase 1 treatment (X or Y; i.e., CS preexposure or no CS preexposure) and Phase 2 treatment (A → US/AX− or US/X−; i.e., Pavlov's or the explicitly unpaired conditioned inhibition procedure) as factors was conducted on suppression scores from the first four groups (CSpre.Pav, Pav, CSpre.EU, and EU). This revealed an interaction, $F(1, 43) = 7.29, p < .01$, as well as main effects of Phase 1 treatment and Phase 2 training, $Fs(1, 43) = 8.75$ and $20.05, ps < .01$, respectively.

Planned comparisons were conducted on the suppression to BX (or B) scores using the overall error term from the one-way ANOVA. Subjects in Groups Pav and EU suppressed less to BX than subjects in Group SumExcite suppressed to B alone, thereby demonstrating that conditioned inhibition to X was in evidence after either Pavlov's or the explicitly unpaired procedure, in the absence of CS preexposure, $Fs(1, 54) = 3.89, p = .054$, and $8.93, p < .01$, respectively. A comparison between Groups CSpre.Pav and Pav revealed that the CS-preexposure treatment given to the former group impaired the development of conditioned inhibition attained with Pavlov's procedure, $F(1, 54) = 11.88, p < .01$. Moreover, CS-preexposure treatment prior to Pavlov's inhibition procedure (Group CSpre.Pav) resulted in a nonsignificant difference with the SumExcite control group, revealing that conditioned inhibition was essentially eliminated, $F(1, 54) = 2.17, p = .15$.

In contrast, a comparison between Groups CSpre.EU and EU revealed no significant effect of CS-preexposure treatment on the expression of conditioned inhibition obtained with the explicitly unpaired procedure, $F(1, 54) < 1.0$. Furthermore, comparing Group CSpre.EU with the SumExcite control group revealed the retainment of conditioned inhibition despite CS-preexposure treatment preceding the explicitly unpaired procedure, $F(1, 54) = 8.40, p < .01$. Finally, we were not able to detect a difference between Groups Pav and EU with the power provided by the test, $F(1, 54) < 1.0$. This last contrast argues against the observed failure of the CS-preexposure treatment to attenuate explicitly unpaired conditioned inhibition because of the baseline inhibition being stronger in the case of explicitly unpaired training than in the case of Pavlovian inhibition training.

Experiment 3 (Control for the US-Preexposure Effect)

In Experiments 1 and 2, we failed to control for an alternative interpretation of the uniformly low suppression observed in Groups CSpre.EU and EU of Experiments 1 and 2. Specifically, Groups CSpre.EU and EU received the US and X in an explicitly unpaired fashion during Phase 2 conditioned inhibition treatment. Such unsignaled exposure to the US could impair subsequent acquisition of (or behavioral control by) an X–US association (i.e., retardation treatment) in Experiment 1 and acquisition of (or behavioral control by) a B–US association (i.e., transfer excitor training) in Experiment 2. Thus, the low suppression to X by these two groups observed in Experiment 1 (Figure 4) and to the BX compound in Experiment 2 (Figure 5) could reflect a US-preexposure effect rather than conditioned inhibition of X. The US-preexposure effect has been shown to be disrupted by context switches (Matzel, Brown, & Miller, 1987; Randich & LoLordo, 1979). To reduce the likelihood of obtaining a US-preexposure effect in Experiments 1 and 2, we imposed a context switch between conditioned inhibition training (Phase 2) and X–US pairings (Phase 4) in Experiment 1, and the transfer excitor training (Phase 3) in Experiment 2. However, we did not include an appropriate group to document the extent to which the context switch adequately disrupted a potentially real US-preexposure

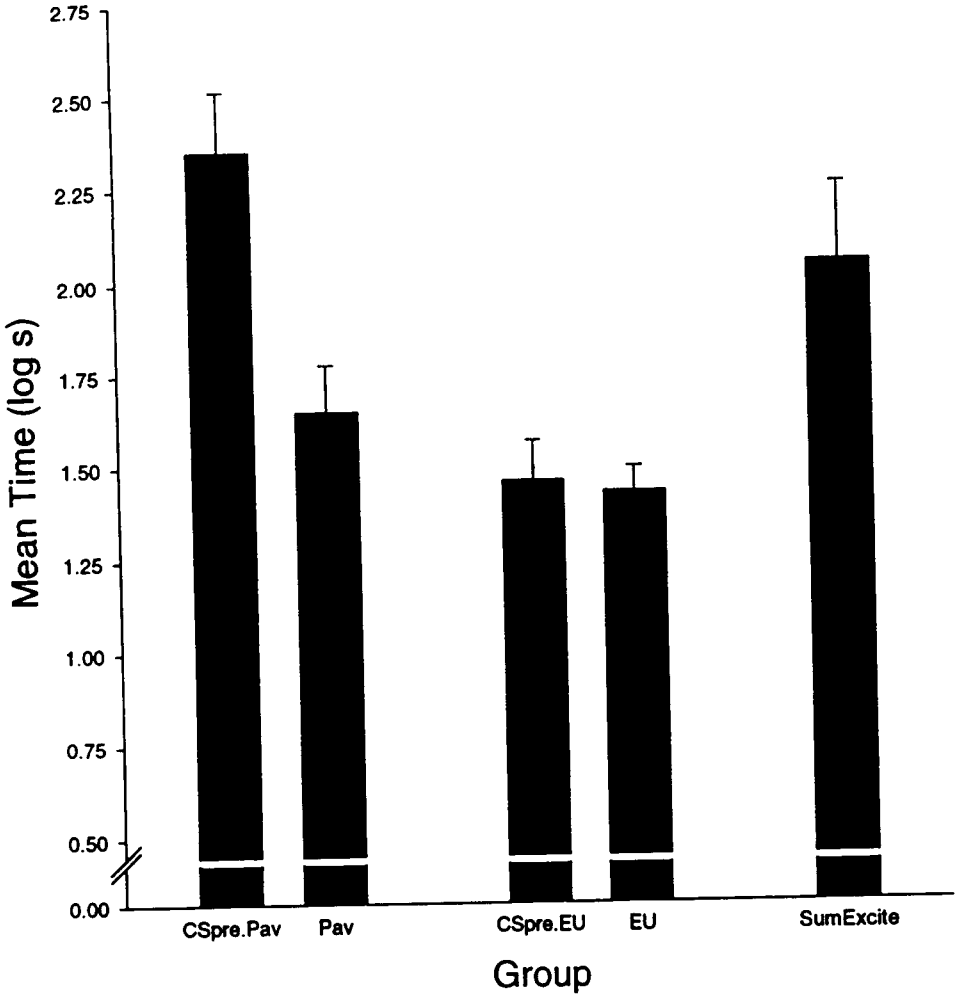

Figure 5. Experiment 2: Mean times (log s) to complete 5 cumulative s of licking in the presence of the target conditioned stimulus (CS). All groups were tested on BX, except for Group SumExcite, which was tested on B. Error bars represent standard errors of means. CSpre = CS preexposure; Pav = Pavlovian conditioned inhibition; EU = explicitly unpaired conditioned inhibition; SumExcite = summation excitor.

effect. Thus, Experiment 3 was conducted to rule out an alternative interpretation of Experiments 1 and 2 in terms of the US-preexposure effect by providing such a control condition.

Method

Subjects and Apparatus

Thirty-six male (250–460 g) and 36 female (221–285 g) Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to one of six groups (*ns* = 12), counterbalanced for sex. The animals were housed and maintained as in Experiments 1 and 2. The apparatus and stimuli were identical to those used in Experiments 1 and 2.

Procedure

Specific group names are explained along with a summary of the critical aspects of the training procedure in Table 3.

Acclimation. On Days 1–3, all subjects were acclimated to Contexts 1–3 as in Experiments 1 and 2.

Phase 1 (CS-preexposure treatment). On Days 4–11, all subjects were given nonreinforced presentations to X (Groups CS-preexposure.explicitly unpaired.retardation [CSpre.EU.Ret], conditioned inhibition.control.retardation [CI.Control.Ret], CS-preexposure. explicitly unpaired.summation [CSpre.EU.Sum], and conditioned inhibition.control.summation [CI.Control.Sum]) or Y (Groups explicitly unpaired conditioned inhibition.retardation [EU.Ret] and explicitly unpaired conditioned inhibition.summation [EU.Sum]) in Context 1 as in Experiments 1 and 2.

Phase 2 (conditioned inhibition training). On Days 12–17, all subjects received explicitly unpaired conditioned inhibition treatment with X (Groups CSpre.EU.Ret, EU.Ret, CSpre.EU.Sum, and EU.Sum) or Y (Groups CI.Control.Ret and CI.Control.Sum) in Context 1 as in Experiments 1 and 2.

Phase 3 (summation excitor training). On Days 18 and 19, all subjects received transfer excitor training in Context 2 as in Experiments 1 and 2.

Phase 4 (retardation excitor training). On Day 20, subjects in Condition Ret received retardation excitor training in Context 2 as

Table 3
Design Summary: Experiment 3 (Control for the Unconditioned Stimulus [US]-Preexposure Effect)

Group	CS preexposure$_1$	Training			Test$_3$	Predicted
		CI$_1$	Transfer$_2$	Excitation$_2$		
CSpre.EU.Ret	X–	US/X–	B → US	X → US	X	cr
EU.Ret	Y–	US/X–	B → US	X → US	X	cr
CI.Control.Ret	X–	US/Y–	B → US	X → US	X	CR
CSpre.EU.Sum	X–	US/X–	B → US	—	BX	cr
EU.Sum	Y–	US/X–	B → US	—	BX	cr
CI.Control.Sum	X–	US/Y–	B → US	—	B	CR

Note. X and Y denote two stimuli (tone or white noise), counterbalanced within groups. B was a click train. US and a minus sign denote the presence and absence of the US (footshock), respectively. Subscripts denote context. CS = conditioned stimulus; CI = conditioned inhibition; CSpre = CS preexposure; EU = explicitly unpaired conditioned inhibition; CI.Control = conditioned inhibition control; Ret = retardation; Sum = summation; CR = strong responding; cr = weak responding; — = no treatment.

in Experiment 1. Subjects in Condition Sum remained in their home cages.

Reacclimation. On Days 21 and 22, all subjects were reacclimated to Context 3 as in Experiments 1 and 2.

Testing. On Day 23, subjects in Groups CSpre.EU.Ret, EU.Ret, and CI.Control.Ret were tested for conditioned lick suppression to X as in Experiment 1. Subjects in Groups CSpre.EU.Sum and EU.Sum were tested for conditioned lick suppression to a compound of BX as in Experiment 2. Subjects in Group CI.Control.Sum were tested for conditioned lick suppression to B as in Experiment 2. The data from 1 subject (Group CI.Control.Sum) were eliminated from analyses because the rat did not complete the 5 cumulative s of licking within 60 s on the test day. One subject from Group CSpre.Eu.Sum was also eliminated from analyses because of an apparatus failure.

Results and Discussion

Without CS preexposure, the explicitly unpaired conditioned inhibition procedure produced conditioned inhibition as assessed by both retardation and summation tests. Furthermore, CS preexposure did not interfere with the acquisition or expression of explicitly unpaired conditioned inhibition. The critical observation was that both control groups demonstrated strong conditioned suppression. That is, with the present parameters, explicitly unpaired conditioned inhibition treatment failed to produce a US-preexposure effect with respect to either an X–US association or a B–US association.

Retardation Test

A one-way ANOVA conducted on suppression scores obtained during the presentation of X on Day 23 revealed a treatment effect, $F(2, 33) = 22.20, p < .0001$ (see Figure 6). Planned comparisons were conducted on the suppression to X scores using the overall error term from the one-way ANOVA. Group EU.Ret suppressed less than Group CI.Control.Ret, demonstrating conditioned inhibition to X after explicitly unpaired conditioned inhibition treatment with a retardation test, $F(1, 33) = 31.48$, $p < .0001$. Furthermore, CSpre.EU.Ret did not differ significantly from Group EU.Ret ($F < 1.0$), thereby again suggesting no effect of CS preexposure on the expression of explicitly unpaired conditioned inhibition.

Summation Test

A one-way ANOVA conducted on suppression scores during BX (or B) revealed a treatment effect, $F(2, 31) = 13.26, p < .0001$ (see Figure 6). Planned comparisons were conducted on the suppression to BX (or B) scores using the overall error term from the one-way ANOVA. Group EU.Sum suppressed less than Group CI.Control.Sum, indicating conditioned inhibition to X after explicitly unpaired conditioned inhibition treatment with a summation test, $F(1, 31) = 18.96, p < .001$. Furthermore, CSpre.EU.Sum did not differ significantly from Group EU.Sum ($F < 1.0$), thereby again suggesting no effect of CS preexposure on the expression of explicitly unpaired conditioned inhibition.

This experiment replicated Experiments 1 and 2 in demonstrating that CS preexposure, which according to the comparator hypothesis should establish the context as the comparator stimulus for the target inhibitor, had no detrimental effect on the expression of explicitly unpaired conditioned inhibition. More important, explicitly unpaired conditioned inhibition treatment, which involves the presentation of unsignaled USs, failed to interfere with acquisition (or expression) of a strong X–US (Group CI.Control.Ret) or B–US (Group CI.Control.Sum) association. That is, the weak suppression to X in Groups EU.Ret and CSpre.EU.Ret and to BX in Groups EU.Sum and CSpre.EU.Sum cannot be attributed to a US-preexposure effect.

It is important to note that, although no statistical difference was detected between Groups Pav and EU in either Experiment 1 or 2, there is no direct way to show that these groups were equivalent without resorting to cross-experiment comparisons (e.g., a comparison of the difference between Groups Pav and RetardCon of Experiment 1 and the difference between Groups EU.Ret and CI.Control.Ret of Experiment 3). The comparison of simple performance in Groups Pav and EU in Experiment 1 or 2 is only a rough

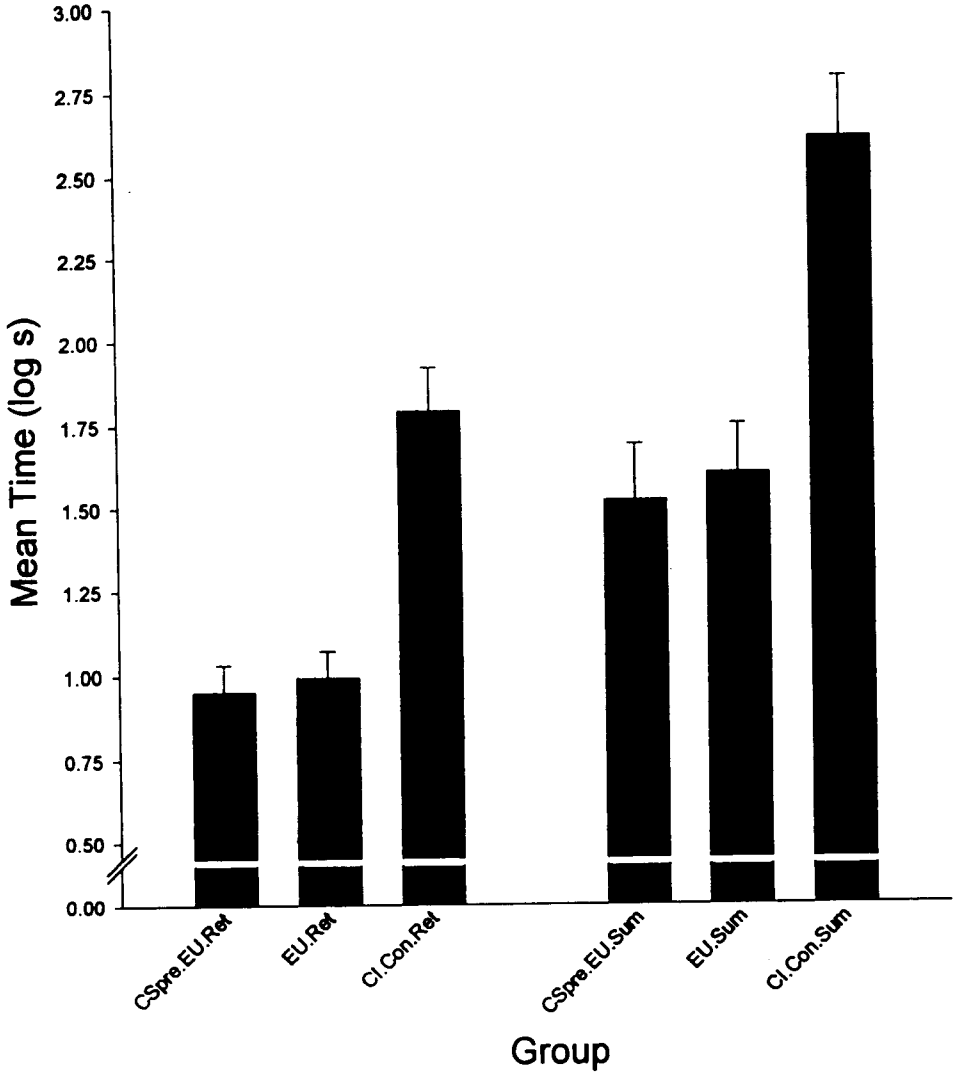

Figure 6. Experiment 3: Mean times (log s) to complete 5 cumulative s of licking in the presence of the target conditioned stimulus (CS). All subjects in Condition Ret were tested on X. Subjects in Groups CS preexposure.explicitly unpaired.conditioned stimulus.summation (CSpre.EU.Sum) and EU.Sum were tested on BX. Subjects in Group CI.Control.Sum were tested on B. Error bars represent standard errors of means. (See text for meanings of abbreviations.)

estimate of the relative strengths of conditioned inhibition. Although Experiment 3 demonstrated that the explicitly unpaired procedure for conditioned inhibition is producing something more than just a US-preexposure effect, the latter effect could arguably still have contributed to performance by Group EU in Experiments 1 and 2. Thus, true inhibition may have been weaker in the explicitly unpaired condition than in the Pavlovian condition. Such a difference seems inconsistent with the resistance to disruption of explicitly unpaired conditioned inhibition by preexposure to CS X. That is, if explicitly unpaired inhibition is truly weaker than Pavlovian conditioned inhibition, then CS preexposure should have more readily disrupted behavior indicative of explicitly unpaired conditioned inhibition than behavior indicative of Pavlovian conditioned inhibition. However, Experiments 1 and 2 yielded the opposite results; Pavlovian conditioned inhibition but not explicitly unpaired inhibition was disrupted by CS-preexposure treatment.

General Discussion

Using both a retardation (Experiment 1) and summation (Experiment 2) test to assess conditioned inhibition, we demonstrated the differential effects that CS preexposure has on conditioned inhibition produced with the Pavlovian and explicitly unpaired conditioned inhibition procedures. Additionally, Experiment 3 ruled out a possible US-preexposure effect as an alternative explanation for the weak suppression to X (Experiment 1) and BX (Experiment 2) in Groups CSpre.EU and EU. Furthermore, the results here serve as additional evidence (Williams, & Overmier, 1988; Williams et al., 1986) that conditioned inhibition produced by differ-

ent procedures is not equivalent. That is, in contrast to most models of associative acquisition (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972), conditioned inhibitors produced by different procedures result in inhibitors that differ both qualitatively and quantitatively.

For example, Williams and Overmier (1988) found that five different types of conditioned inhibition procedures (i.e., differential, explicitly unpaired, Pavlovian conditional, trace, and backward) established conditioned inhibitors that differed in several dimensions. Specifically, with a few training trials with the conditioned inhibitor, differential and explicitly unpaired procedures established an effective conditioned inhibitor, but Pavlovian, trace, and backward procedures failed to produce an effective inhibitor. Williams and Overmier reasoned that these latter three procedures were capable of producing a conditioned inhibitor but that inhibitory responding was masked by collateral excitatory associations either to the excitatory CS (Pavlovian procedure, see Williams et al., 1986) or to the US (trace and backward procedures). Their evidence for this interpretation was that posttraining extinction of the inhibitor prior to testing enhanced behavior indicative of inhibition following training with each of the three latter procedures. Furthermore, extensive inhibitory training caused the Pavlovian trained stimulus to act as an effective inhibitor. Thus, different types of conditioned inhibition procedures are not equally effective in establishing a stimulus as a conditioned inhibitor. Williams and Overmier (see also Williams et al.) attributed this to the fact that some conditioned inhibition procedures also imbue the inhibitor with excitatory associations that may interfere with the expression of its inhibitory value.

The comparator hypothesis views all associations (Links 1, 2, and 3 in Figures 1–3) as excitatory and explains behavior indicative of conditioned inhibition as arising from inhibitory test procedures when Link 1 is small relative to the product of Links 2 and 3. In the present situation, the comparator hypothesis predicts a disparity in expressed conditioned inhibition because of the relative differences in strength of Link 2 (i.e., the target CS–comparator stimulus association) that is activated at test after CS preexposure followed by Pavlovian or explicitly unpaired inhibition training. With Pavlov's procedure (A → US/AX−), Stimulus A is X's comparator stimulus at test (Link 2 of Figure 2). CS-preexposure treatment interferes with the expression of this association because the nonreinforced presentations of the X stimulus in Phase 1 results in the context, rather than Stimulus A, becoming X's primary comparator stimulus. Blaisdell et al. (1998) used this same comparator mechanism to explain their finding that the effects of CS-preexposure and overshadowing treatments counteract each other. Specifically, they preexposed the intended overshadowed stimulus (X) prior to overshadowing training (AX → US, in which the more salient Stimulus A normally overshadows the less salient Stimulus X) and found strong conditioned suppression to X at test. This conditioned suppression was strong relative to a group that received preexposure to X followed by X → US pairings (i.e., impaired responding due to the CS-preexposure effect) and a group that received no preexposure treatment followed by AX → US pairings (i.e., overshadowing). They explained this outcome as arising from the preexposure treatment establishing the context, and not A, as the primary comparator stimulus for X, thus preventing the expression of overshadowing, which depends on a strong effective X–A association and a strong effective A–US association. Furthermore, the presence of A during overshadowing training prevented the context from forming a strong effective association with the US, thereby attenuating the CS-preexposure effect. Thus, the same comparator mechanism explains the attenuating effects of target CS preexposure on both overshadowing and Pavlovian conditioned inhibition.

In contrast to Pavlovian conditioned inhibition, with explicitly unpaired conditioned inhibition training (US/X−), X's comparator stimulus is the context (not Stimulus A; see Figure 3), and the X–context association is strengthened during CS preexposure. The comparator hypothesis predicts that inhibitory responding will be expressed when the product of Links 2 and 3 (Link 3 is the comparator stimulus–US association) is strong relative to Link 1 (the direct CS–US association). The development of the CS–context association is presumably facilitated by CS-preexposure treatment; thus, CS-preexposure treatment might be expected to facilitate the development of explicitly unpaired conditioned inhibition. However, the 48 presentations of the US interspersed with 48 presentations of X over the 6 days of explicitly unpaired conditioned inhibition training presumably incremented the X–context association to an asymptotic level with respect to production of inhibitory behavior. Thus, the comparator hypothesis's prediction that conditioned inhibition would be increased when explicitly unpaired conditioned inhibition training is preceded by CS-preexposure treatment is mitigated by the likelihood of the CS–context association (Link 2) being asymptotic at the conclusion of conditioned inhibition training, regardless of whether CS-preexposure treatment is given. Conversely, with Pavlovian conditioned inhibition training, the effective Link 2 (i.e., X–A) was presumably weakened when CS-preexposure treatment preceded the Pavlovian conditioned inhibition training procedure because of competition between A and the context for serving as the primary comparator stimulus for the target CS (X). Consequently, Link 1 should have exceeded the product of Links 2 and 3, and hence behavior indicative of inhibition should not have been observed during testing of the Pavlovian conditioned inhibition group that received CS-preexposure treatment. Indeed, exactly this was observed.

The present results constrain Rescorla's (1971, Experiment 2) and Reiss and Wagner's (1972, Experiment 2) conclusions that a preexposed CS is "less trainable as a conditioned inhibitor" (Reiss & Wagner, 1972, p. 244). Their results were apparently a consequence of the particular method with which they attempted to produce conditioned inhibition, that is, Pavlov's procedure. Though their specific findings were replicated in this study (i.e., CS-preexposure treatment does inhibit a cue from expressing inhibitory properties acquired through Pavlov's procedure), their ubiquitous assertion, that a preexposed cue will encounter difficulty in entering into an inhibitory association regard-

less of the inhibitory training procedure that is used, was not confirmed.

Moreover, Rescorla's view that his findings were consistent with Carlton and Vogel's (1967) notion of the loss of fundamental impact of a preexposed stimulus is also rebuked. The present results, as well as those of Rescorla (1971) and Reiss and Wagner (1972), are better interpreted within the framework of the comparator hypothesis, specifically its response rules for the expression of conditioned inhibition, and not as the result of an attentional deficit.

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Received October 2, 1997
Revision received March 30, 1998
Accepted April 7, 1998 ■