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# Thwarting the renewal (relapse) of conditioned fear with the explicitly unpaired procedure: Possible interpretations and implications for treating human fears and phobias<sup>☆</sup>

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## Abstract

In three experiments using the barpress conditioned suppression task with albino rats, we studied the renewal (relapse) of conditioned fear in an ABA fear-renewal paradigm. We found that explicitly unpaired (EU) deliveries of conditioned stimuli (CSs) and unconditioned stimuli (USs) in Context B thwarted fear renewal in Context A. Evidence contraindicated a suggestion by Rauhut, Thomas, and Ayres (2001) that US habituation plays a key role in this effect. For example, renewal was thwarted only when EU CSs and USs were intermingled rather than given in succession. The possibility that EU treatments thwart renewal by creating a CS that inhibits fear in the test context also received no support. Thus, summation and retardation tests in Context A found no evidence that the EU CS became inhibitory, finding instead evidence for a residual excitation. Other possible interpretations of the results and some implications for clinical practice are noted. © 2004 Elsevier Inc. All rights reserved.

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In laboratory rats, fear that is acquired in Context A and extinguished in a different context, B, will return or be renewed when tested in the original acquisition context or in a novel context, C, (e.g., Bouton & Bolles, 1979; Bouton & King, 1983; Bouton & Swartzentruber, 1986). This finding prompted Bouton (1988) to suggest that the context specificity of simple extinction limits its value as a tool for treating conditioned fears, including fears in humans, because any therapeutic benefits are apparently limited to the environment in which they were gained.

Recently, workers using rat subjects have begun to seek treatments that thwart fear renewal. Gunther, Denniston, and Miller (1998) found that renewal of conditioned fear in an ABC paradigm could be reduced by extinguishing it in several contexts rather than just one (for similar results with conditioned taste aversion, see Chelonis, Calton, Hart, & Schachtman, 1999). Denniston, Chang, and Miller (2003) found that both ABC and ABA renewal could be reduced by “massive” extinction exposure. In a direct comparison of ABA, ABC, and AAB renewal, Thomas, Larsen, and Ayres (2003) found AAB renewal to be weaker than the other two forms, which did not differ. They concluded that renewal of conditioned fear can be reduced by extinguishing it in the acquisition context regardless of the nature of the test context. Another technique recently found to thwart fear renewal in ABA paradigms is the explicitly unpaired (EU) procedure (Rauhut et al., 2001). There, the conditioned stimulus (CS) was conditioned by pairing it with a shock unconditioned stimulus (US) in Context A was subsequently explicitly unpaired with the same shock in Context B. Thus, CSs and USs were intermingled during the procedure but kept temporally apart (e.g., Frey & Butler, 1977). Rauhut et al. (2001) reported that this EU procedure not only thwarted the renewal of fear to the treated CS but also weakened fear to another CS previously paired with the same shock in Context A but then left untreated in Context B.

The effect of the EU procedure on untreated cues is of interest for at least two reasons. First, it implies that in clinical practice, an EU treatment might not only weaken a fear or phobia in humans and prevent its relapse but might also have a therapeutic benefit on fears not directly treated in therapy. Second, and more generally, the effect might shed light on the mechanisms responsible for the fear-weakening effects of the EU treatment. In that regard, Rauhut et al. (2001) combined several observations to draw a conclusion about those mechanisms. The observations (see their Experiment 4) were as follows: (a) Relative to a forgetting control procedure, CS-alone exposures in Context B led to weakened fear when the CS was tested in Context A (there was some transfer of extinction across contexts). (b) US-alone exposures in Context B produced a similarly weakened fear in Context A. (c) Both US-alone and EU exposures produced evidence of US habituation. (d) Relative to control procedures, both US-alone and EU procedures weakened fear to an untreated CS. Citing Rescorla’s (1973) finding that US habituation could retroac-

tively weaken conditioned fear based on that US, [Rauhut et al. \(2001\)](#) concluded that their US-alone and EU treatments retroactively weakened fear to their untreated CS because both procedures produced US habituation. They further concluded that their EU treatment completely thwarted renewal because it combined the fear-weakening effects of US habituation and CS-alone extinction. Results to be reported here will challenge the importance of US-habituation in thwarting renewal.

It may be worth noting that we did not set out to test the role of US habituation. Rather, an initial experiment conducted for other reasons yielded unexpected results that cast doubt on the importance of the US-habituation mechanism in thwarting renewal. In trying to understand those results, we were led to further experiments whose results, in turn, provided more definitive evidence against the importance of that mechanism.

Following [Rauhut et al. \(2001\)](#), we measured fear using the barpress conditioned suppression task ([Estes & Skinner, 1941](#)). We conditioned fear by pairing various CSs with shock USs while rat subjects barpressed for food. We took the amount of barpress suppression evoked by the CSs as an index of fear. Suppression occurs because the frightened rat crouches and remains immobile, often away from the bar ([Bevins & Ayres, 1992, 1994](#); [Bouton & Bolles, 1980](#); [Mast, Blanchard, & Blanchard, 1982](#)). That behavior disrupts barpressing.

## Experiment 1

An objection to using an EU procedure to treat human fears and phobias is that it requires the delivery of aversive USs. That objection can be reduced to the extent that the treatment duration can be shortened without limiting its effectiveness. Using an animal model, we hoped in Experiment 1 to gain some preliminary information about the importance of treatment duration. In their Experiment 4, [Rauhut et al. \(2001\)](#) used an EU treatment of 24 sessions. Here, we compared the effects of 24, 12, and 6 EU sessions with those of 24 sessions of CS-alone extinction. In addition, we sought to replicate the fear-weakening effects of the EU treatment on untreated CSs ([Rauhut et al., 2001](#), Fig. 9). To that end, we first conditioned three CSs (X, Y, and Z) in Context A, treated one of them (X) with the EU procedure in Context B, and then tested all three in succession in Context A. [Rauhut et al. \(2001\)](#) tested only one untreated CS. We sought to extend the generality of their findings by testing two. We assumed from their work that EU treatment with CS X should reduce fear to all CSs whose conditioned value was based on the same US used in the original conditioning of CS X.

### *Method*

#### *Subjects*

The subjects were 40 male albino rats of the Holtzman strain from Harlan Industries, Indianapolis, IN. Throughout the study, they were maintained at 80% of their

free feeding weights, which ranged from 339 to 379 g. Rats were housed singly in stainless steel wire-mesh cages with water freely available. The colony was lighted between 06:00 and 22:00 h. The experiment was conducted between 09:00 and 17:00 h.

### *Apparatus*

The apparatus resembled that of [Rauhut et al. \(2001\)](#). Eight identical operant boxes were housed in 0.61-m ventilated cubes of 12.7-mm thick plywood, lined with acoustical tile. One side of each cube was hinged to form a door. Four of the operant boxes were modified to create two sets of four that served as Contexts A and B (counterbalanced). For Set 1, the inside dimensions were 23.2 × 20.3 × 19.5 cm (length × width × height). The end walls were aluminum; the sides were clear Plexiglas. Centered in one end wall was a response bar (5 × 1.5 cm). A weight of 32 g was required to press it enough to close a microswitch located behind it. In the lower left corner of this end wall was a recessed dipper tray (5 × 5 × 5.5 cm). The floor consisted of 18 stainless steel rods (2 mm in diameter, centers spaced 1.3 cm apart). Corrugated gray cardboard lined the catch tray below the rods. A glass furniture coaster containing 10 ml of 2% anise extract (McCormick, Hunt Valley, MD) was placed beside each operant box near the dipper tray. At the start of each session, a clear Plexiglas sound-attenuating door was inserted into the space created by leaving each housing cube's hinged door open. Rats were carried from the colony to this context in a case placed on a cart. The case was divided into cells (21.4 × 24.0 × 15.4 cm), each with a wire-mesh floor, metal walls, and Plexiglas lid.

The second set of four boxes was modified to look, feel, and smell different from Set 1 in several ways. First, Set 2 was separated from Set 1 by a blackout cloth that split the room in half. Second, the fluorescent ceiling light in the half of the room housing Set 2 was removed, making that half much dimmer than the other half. Third, a metal plate was placed in each box in Set 2 so as to form a slanted back wall. It reduced the distance from the work panel to the back wall at floor level from 23.2 to 12.5 cm. Fourth, the work panel was covered with white Formica with vertical black stripes (1 cm wide spaced 1.5–2.0 cm apart). Cutouts in the Formica allowed access to the bar and dipper. Fifth, the outside of the Plexiglas wall to the left of the bar was covered with aluminum screening. Sixth, a glass furniture coaster containing 10 ml of pure vinegar (H.J. Heinz, Pittsburgh, PA) was placed beside each operant box near the dipper tray. Seventh, at the start of the session, the hinged wooden doors of the housing cubes were closed. Eighth, rats were carried from the colony to Set 2 boxes in their home cages, placed within a two-level wooden shelf unit that rested on a cart.

On the lid of each of the eight operant boxes were two speakers (10-cm diameter). One provided a continuous white noise background of 80 dB when superimposed on the noise from the ventilating fans. The other was used to present a tone CS (86 dB, 1000 Hz). Sound intensity was measured using a Radio Shack sound level meter (Catalog No. 33-2050; C scale, slow response) with its microphone about 7 cm from the dipper tray. On the back wall of the housing cube (to the right of the bar) were two white frosted light bulbs (each 7.5 W, 110 V), one above the other. Terminating these

lights provided a second (light) CS. Centered on the outside of the Plexiglas wall to the right of the bar was a relay (Potter & Brumfield KHP17D11, Marion, KY). When it was operated at 10 Hz, it provided a third (click) CS (90 dB). The light, tone, and click CSs will be termed CSs X, Y, and Z, respectively. Throughout this research, CSs were 2 min long and were given independently of barpress responding. Extensive pilot work in our laboratory (see also Rauhut, McPhee, & Ayres, 1999; Thomas & Ayres, 2004) revealed no evidence for generalization of fear or fear extinction among these CSs. Other work (Bevins & Ayres, 1991; Rauhut, McPhee, DiPietro, & Ayres, 2000, p. 105) found no evidence that, for fear conditioning, stimulus termination of this duration is processed differently from stimulus presentation.

The US was a 1-s 0.6-mA (nominal) scrambled grid shock provided by Grason-Stadler shockers (Models E1064 or 700; Grason-Stadler, West Concord, MA). As measured at the grids using a Multimeter (Radio Shack Catalog No. 22-805), the peak shock intensity was about 0.25 mA. A computer in an adjacent room controlled all stimulus exposures and recorded responses.

### *Procedure*

Rats were randomly assigned to five groups ( $ns = 8$ ): Extinction (E), Forgetting Control (FC), Explicitly Unpaired-6 (EU6), Explicitly Unpaired-12 (EU12), and Explicitly Unpaired-24 (EU24). Groups EU6, EU12, and EU24 received, respectively, 6, 12, or 24 sessions of EU treatment.

*Preliminary training.* Preliminary training involved 1 day of training the rats to approach the dipper when raised (magazine training), 3 days of training them to barpress (shaping with each response reinforced), and 2 days of training under a schedule that reinforced responding once every 60 s on average (variable interval [VI] 60-s schedule). In all experiments, reinforcement for barpressing was a 4-s access to a 0.1-ml dipper cup of 32% liquid sucrose. Starting with Day 1 of VI training, the rats barpressed on the VI schedule in every session; all sessions were 60 min long; and the doors of the housing cubes were closed during the sessions because the odor cues were used thenceforth. Following VI training in Experiment 1, all rats received 1 non-reinforced pre-test exposure to each CS on each of 2 days. CSs were always 2 min long. During the 8 days of preliminary training in Experiment 1, rats were placed in Context A on Days 1, 2, 5, and 7 and in Context B on Days 3, 4, 6, and 8, thus gaining familiarity with both contexts.

*Conditioning in Context A.* After preliminary training, each group received in Context A 6 days of conditioning to CS Y, then 6 days to CS Z, then 6 days to CS X. Each day had two CS trials, each coterminating with a 1-s shock US (CS+ trials).<sup>1</sup>

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<sup>1</sup> Because we find no evidence for generalization of acquisition or extinction across our CSs and because forgetting is extremely rare in our procedures, the order in which we condition and test CSs is rather arbitrary. Counterbalancing of stimuli and their orders is technically difficult for us and increases the likelihood of our making procedural errors. Therefore, we tend to counterbalance only when the logic of the experimental design demands it (e.g., Experiment 3, CSs X and Z).

*Treatment in Context B.* Before treatment in Context B, all rats received 1 day of VI training in that context to ensure stable barpressing there at the start of treatment. Beginning on the next day, Group E received a conventional extinction procedure consisting of four nonreinforced CS X trials (X– trials) daily for 24 days. Group EU24 received the same four CSs daily intermingled with two USs. Groups EU12 and EU6 received the same treatment for the last 12 or 6 days, respectively. When not receiving EU treatment, Groups EU12 and EU6 merely barpressed for sucrose on the VI schedule. Group FC, serving as a control for forgetting, received no programmed CSs or USs in Context B but, like the other groups, did receive the VI schedule on each day of treatment. In sum, Group EU24 received a total of 96 X– trials and 48 shock USs. Group EU12 received 48 X– trials and 24 USs. Group EU6 received 24 X– trials and 12 USs. Group E received the same 96 X– trials as Group EU24 but received no USs. Group FC received neither CSs nor USs.

*Testing CSs X, Y, and Z in Context A.* On the day after treatment, each group was returned to Context A for 1 day of VI training to ensure a stable rate of barpressing there in preparation for testing.<sup>2</sup> Next, each group received 2 days of testing to each CS (X, Y, and Z). Each day had two CS– trials. For the 1st 2 days, CS X was tested. Stronger suppression to X on Trial 1 in Context A than on the last treatment trial in Context B would demonstrate renewal. Next, CS Z was tested. Last, CS Y was tested. Testing of the untreated CSs, Y and Z, was intended to see if the EU treatment of CS X would weaken fear to Y and Z as expected from [Rauhut et al. \(2001\)](#).

### *Trial spacing*

Throughout our studies, some sessions had two CS trials, some had three, and some had four. In sessions with two trials, CSs began no sooner than Min 12 and no later than Min 55; the interval between successive CS onsets (the ITI) ranged from 8 to 41 min ( $M = 20.7$ ). In sessions with three trials, CSs began no sooner than Min 15 and no later than Min 46; ITIs ranged from 12 to 16 min ( $M = 13.7$ ). In sessions with four CS trials, including CSs explicitly unpaired with USs, CSs began no sooner than Min 10 and no later than Min 49; ITIs ranged from 4 to 19 min ( $M = 10.4$ ). In EU

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<sup>2</sup> Investigators often equate exactly the total exposure to Contexts A and B before testing for renewal (e.g., Experiment 3 in [Bouton & King, 1983](#)). Here and elsewhere ([Rauhut et al., 2001](#); [Thomas & Ayres, 2004](#); [Thomas et al., 2003](#)), we did not do so. We did, however, ensure that all rats received extensive exposure to each context. We also ensured that all rats received a full session of VI training before beginning renewal testing. We did so to reduce the odds that any unconditioned neophobia or excitation conditioned to the test context might influence the test results. It is not clear why equating experience with the contexts would necessarily eliminate any of these factors. Note too that, in an appetitive situation, [Bouton and Peck \(1989\)](#) directly compared the effects on renewal of equating versus not equating exposure to the contexts and found no difference. Finally, although the renewal paradigm is designed to model the therapeutic situation in humans, it never does so perfectly. For example, the time spent in therapy with humans is never equated with the time spent outside it; and rarely, if ever, are humans exposed to the therapeutic context prior to acquiring their fears, as has been true of almost all animal work; and, finally, humans never spend more time in the therapeutic context (Context B) than they do in the outside world (Context A), as was true here.

treatment sessions, the shortest interval between CS and US onsets was 5 min; that between US onset and onset of the next CS was 4 min.

### *Measure of conditioned fear*

A suppression ratio (SR; *Annau & Kamin, 1961*) was used to index suppression to the CS. The SR was defined as  $D/(D + B)$ , where  $D$  is the number of responses made during a 2-min CS and  $B$  is the number in the 2 min just before CS onset. With this ratio, a score of .5 denotes no effect of the CS, and one of 0 denotes complete suppression during the CS. Occasionally, a rat failed to respond both before and during a CS. On such trials, we estimated the rat's SR by averaging its SRs on the immediately preceding trial and the immediately following trial. If, however, a rat failed to respond before more than two CSs in a row, its data were excluded from all analyses of that particular phase of the experiment. Hence, the degrees of freedom associated with reported statistics will occasionally be smaller than one might expect.<sup>3</sup>

### *Statistical analyses*

We defined renewal as an increase in suppression between the last trial of treatment in Context B and the first trial of testing in Context A. We assessed the significance of that increase using correlated  $t$  tests for each group that received both trial-types. We used analyses of variance (ANOVAs) to assess the significance of differences among groups in the renewal test in Context A and in other phases of the experiment. Throughout, we used a two-tailed critical value of .05.

### *Results*

The main results of Experiment 1 were that all three EU treatments completely thwarted renewal to CS X, as assessed by the increase in suppression from the last trial of treatment to the first trial of the renewal test. The EU6 treatment, however, did not weaken fear as thoroughly as did the longer EU treatments but did do so more effectively than did simple extinction, as measured by suppression during the renewal test. Simple extinction in Context B did transfer to Context A to some extent because Group E suppressed less in renewal testing than did Group FC, whose fear was never treated. None of the treatments of CS X weakened fear to untreated CSs Y and Z, as groups did not differ in their suppression to those CSs.

### *Conditioning in Context A and treatment in Context B*

Fig. 1A shows suppression to CS X on the last trial of acquisition in Context A (far left) and on the first and last trials of each day of treatment in Context B (days separated by gaps in the plots). Suppression was strong on the last trial of acquisition

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<sup>3</sup> In Experiment 1, 0.09, 0, 0, and 0% of the SRs were estimated in the treatment phase, renewal test, Y test, and Z test, respectively. In Experiment 2, 0.001, 0, 0, and 0% of the SRs were estimated in the treatment phase, renewal test, reacquisition phase, and Y test, respectively. In Experiment 3, 0.001, 0, 0, and 0% of the SRs were estimated in the treatment phase, renewal test, summation test, and retardation test, respectively.

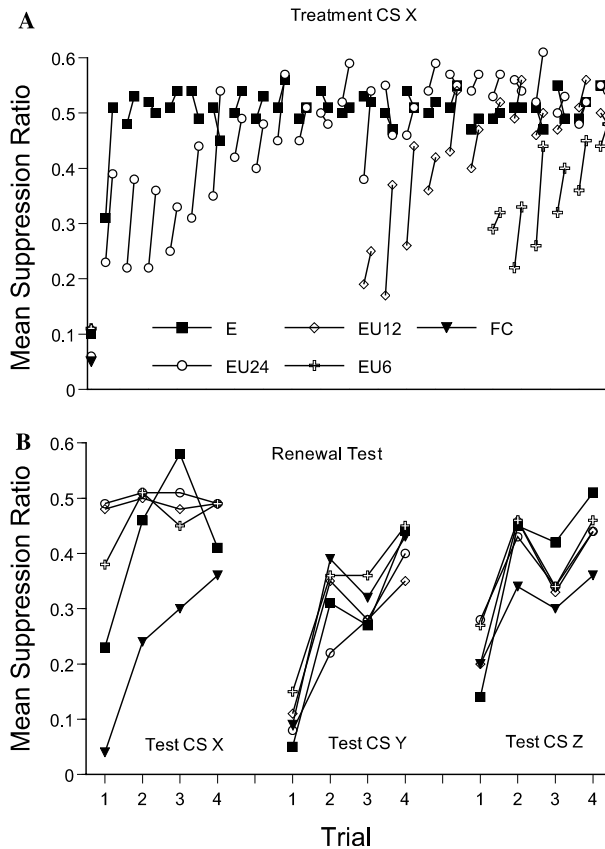


Fig. 1. Experiment 1. (A) Group mean suppression on the last trial of acquisition (far left) and first and last trials of each day of treatment (days separated by gaps in the plots). (B) Group mean suppression during the renewal test of CS X (far left) and tests of the untreated CSs (middle and far right).

to CS X and to CSs Y and Z (Y and Z not shown), where one-way ANOVAs found no group differences,  $F_s(4, 35) < 1.98$ . Suppression then weakened within and across treatment days but recovered between days, especially in the EU groups. By the last treatment trial, suppression was weak in all groups, and a one-way ANOVA revealed no group differences,  $F < 1$ .

Note that early in the treatment phase, conditioned suppression was significantly stronger in the EU groups than in Group E (Fig. 1A). Separate ANOVAs using all the trials from the first 6 days of treatment compared each of the EU groups with Group E. For Group E versus Group EU24, the effects of trial,  $F(23, 322) = 3.60$ , group,  $F(1, 14) = 6.41$ , and Group  $\times$  Trial,  $F(23, 322) = 2.92$ , were significant. For Group E versus Group EU12, the effects of trial,  $F(23, 322) = 7.61$ , group,  $F(1, 14) = 17.83$ , and Group  $\times$  Trial,  $F(23, 322) = 5.11$ , were significant. And for Group E versus Group EU6, the effects of trial,  $F(23, 322) = 4.25$ , group,

$F(1, 14) = 6.29$ , and  $\text{Group} \times \text{Trial}$ ,  $F(23, 322) = 3.58$ , were significant. Of most interest are the effects of group and  $\text{Group} \times \text{Trial}$ , which reflect the stronger suppression and greater resistance to extinction in each of the EU groups relative to Group E.

#### *Testing CSs X, Y, and Z in Context A*

The left part of Fig. 1B shows suppression to the treated CS, X, during the test for renewal in Context A. The middle and right parts show the suppression to untreated CSs, Y and Z, respectively, in that context. Suppression increased from the last trial of treatment to the first trial of renewal testing only in Group E, correlated  $t(7) = 6.53$ , but did not increase significantly for any of the EU groups,  $t_s(7) < 1.44$ . Thus, only Group E showed renewal. (Group FC, which received no treatment trials, provided no data for analysis.)

A  $\text{Group} \times \text{Trial}$  ANOVA on the suppression to CS X in Context A (left part of Fig. 1B) found significant effects of group,  $F(4, 35) = 14.74$ , trial,  $F(3, 105) = 20.17$ , and  $\text{Group} \times \text{Trial}$ ,  $F(12, 105) = 5.39$ . A follow-up ANOVA isolating Groups EU24 and EU12 revealed no significant effects,  $F_s < 1$ . These groups were then pooled and contrasted with Group EU6. That contrast revealed significant effects of trial,  $F(3, 66) = 6.49$  and  $\text{Group} \times \text{Trial}$ ,  $F(3, 66) = 4.15$ . We interpret the interaction to mean that the shortest EU treatment did not reduce fear as thoroughly as did the longer EU treatments. A second follow-up ANOVA isolating Groups EU6 and E revealed significant effects of trial,  $F(3, 42) = 10.74$ , and  $\text{Group} \times \text{Trial}$ ,  $F(3, 42) = 4.12$ . The interaction indicates that fear was initially weaker after 6 days of EU treatment than after 24 days of conventional extinction. A final ANOVA isolating Groups E and FC revealed significant effects of group,  $F(1, 14) = 10.58$ , and trial,  $F(3, 42) = 18.3$ . The effect of group indicates that simple extinction, though less effective than any of the EU treatments, was not entirely specific to its context (i.e., extinction transferred from Context B to Context A).

A  $\text{Group} \times \text{Trial}$  ANOVA was also conducted on the suppression to the untreated CS Y and separately on the suppression to the untreated CS Z. Both ANOVAs revealed significant effects of trial,  $F_s(3, 105) > 24.01$ , reflecting the extinction of fear across nonreinforced test trials, but neither ANOVA revealed a significant effect of group,  $F_s(4, 35) < 1.16$ , or a  $\text{Group} \times \text{Trial}$  interaction,  $F_s(12, 105) < 1.44$ . This means that neither the EU treatments nor the conventional extinction of CS X weakened fear to the untreated CSs, Y and Z. The latter result is consistent with pilot work in our laboratory that found no evidence for generalization of fear or fear extinction among these CSs (see also Rauhut et al., 1999; Thomas & Ayres, 2004, Experiment 4). However, the failure of the EU treatment to weaken fear to an untreated CS is inconsistent with the findings of Rauhut et al. (2001).

A  $\text{Group} \times \text{Trial}$  ANOVA conducted on pre-CS rates during renewal testing of CS X revealed significant effects of group,  $F(4, 35) = 3.58$ , and trial,  $F(3, 105) = 4.38$ . The mean pre-CS rates for Groups E, EU24, EU12, EU6, and FC were 27, 25, 45, 30, and 19 responses per minute, respectively. Although group differences in pre-CS rates are an undesirable complication, the pattern of pre-CS responding seems unrelated to the amounts of conditioned suppression in the groups. Moreover, Group EU12 appears to be the odd group in terms of its pre-CS rates, and its elimination would

not affect our conclusions. In general, it seems unlikely that the differences in conditioned suppression among groups were an artifact of differences in their pre-CS rates.

### Discussion

We interpret the increase in suppression from the last trial of treatment to the first trial of renewal testing as an instance of fear renewal. Such an increase, however, might also be an instance of spontaneous recovery. To rule out spontaneous recovery, it is customary to use an AAA control group, which we did not do here. When we have in the past used AAA control groups with similar time passing between the last extinction trial and first renewal test trial, we have found no spontaneous recovery (e.g., Thomas et al., 2003). This has also been true even when fewer extinction trials were used and when longer periods of time passed between extinction and test (for discussion, see Thomas et al., 2003, p. 418). It seems most unlikely therefore that what we term *renewal* is better described as *spontaneous recovery*.

The finding that EU treatments can completely thwart renewal replicates results of Rauhut et al. (2001). However, our shortest EU treatment (6 days) left a residual fear that was detectable in the renewal test. Still, even 6 days of EU treatment resulted in less fear at test than did 24 days of conventional extinction.

The failure of EU treatments of CS X to weaken fear to untreated CSs Y and Z was unexpected, based on Rauhut et al. (2001). Indeed part of the evidence for their theory that US habituation is key to thwarting renewal and producing weak fear at test was that fear of an untreated CS appeared to be weakened retroactively by EU treatment of another CS (cf. Rescorla, 1973). If the differences among groups in their suppression to CS X at test were caused by differences in US habituation, which in turn led to differences in retroactive weakening of fear to CS X, then we should also predict retroactive weakening of fear to CSs Y and Z due to the same mechanism. Thus, we would predict less suppression to CSs Y and Z in Groups EU24 and EU12 combined than in Group EU6 and less suppression in Group EU6 than in Group E, which had no opportunity for US habituation during treatment. Inspection of Fig. 1B, however, suggests that no such differences exist. (Indeed, over all test trials for CS Y, the mean SR for Groups EU24 and EU12 combined = .26; the mean SR for Group EU6 = .33; the mean SR for Group E = .27. For CS Z, the mean SR for Groups EU24 and EU12 combined = .33; the mean SR for Group EU6 = .38; the mean SR for Group E = .33). Given these results, it is difficult to attribute the thwarting of renewal in the EU groups to any retroactive weakening of fear due to US habituation.

The question remains though: why did Rauhut et al. (2001) find that EU treatment of one CS weakened fear to an untreated CS, whereas we did not? A comparison of our procedures with theirs may explain the discrepancy. In Rauhut et al. (2001), but not here, suppression to an untreated CS was measured after a retardation (reacquisition) test that looked for inhibition possibly conditioned to CS X (Rescorla, 1969b). In that test, Rauhut et al. (2001) paired CS X with shocks in Context A. Those shocks would tend to condition fear to the context in which Rauhut et al.'s (2001) untreated CS was subsequently tested. However, that contextual conditioning would be weaker in their EU and US-alone groups than in their control

groups (E and FC) because the EU and US-alone groups would have had much more opportunity to habituate to the US during treatment in Context B. It is well known that context-fear can reinstate fear to an extinguished CS (e.g., Bouton & King, 1983, 1986). The test of the untreated CS was an extinction test; therefore, fear to the CS was subject to reinstatement during that test. Such reinstatement should have been stronger in the control groups (E and FC, which presumably had the stronger context-fear) than in the EU and US-alone groups. Thus, rather than concluding that their US-alone and EU treatments, by producing US habituation, acted retroactively to weaken fear to an untreated CS, Rauhut et al. (2001) should perhaps have concluded that those treatments acted pro-actively. That is, they reduced subsequent fear conditioning to the ultimate test context and therefore produced less reinstatement of fear to their untreated CS as it underwent extinction in that test. Experiment 2 was designed in part to explore this idea.

## Experiment 2

Before conducting Experiment 2, we sought to reduce the difference between the nominal shock intensity (as read from dial settings) and the intensity actually recorded at the grids (see Experiment 1, Apparatus). To do so, we sprayed the shock scramblers with a conductivity solvent (DeoxIT D5, Caig Laboratories). Measurements taken afterwards revealed peak currents at the grids that corresponded closely to nominal (dial) values (0.6 mA). Because the scramblers had not been cleaned prior to the work of Rauhut et al. (2001), we wanted to see if we could replicate with our higher shock intensity the thwarting of renewal that they found following an EU24 treatment in their Experiment 4.<sup>4</sup> Simultaneously, we reexamined the effect of both EU and US-alone treatments on the fear of previously conditioned but subsequently untreated CSs. For some subjects, we gave this test after the test for renewal and reconditioning of CS X in Context A (as in Rauhut et al., Experiment 4), and for others, we gave it right after the renewal test of CS X in Context A (as in Experiment 1 above). If the reconditioning of CS X results in fear conditioning to the test context, and if such fear helps to reinstate fear to CS Y during its subsequent extinction test in that context, then suppression to CS Y should be stronger in the subjects that receive the Y test following reconditioning to CS X.

### *Method*

#### *Subjects and apparatus*

The subjects were 48 male albino Sprague–Dawley rats about 90 days old from the same supplier as before. Free feeding weights ranged from 350 to 375 g, and all rats

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<sup>4</sup> In Rauhut et al.'s Experiment 4, the dial setting was 0.6 mA; therefore, the actual current at the grids, as measured by our meter, would have been about 0.25 mA.

were kept at 80% of this level. Housing and maintenance were unchanged, as was the apparatus.

### *Procedure*

*Preliminary training.* Before preliminary training, rats were randomly assigned to four groups: Extinction (E,  $n=16$ ), Forgetting Control (FC,  $n=16$ ), Explicitly Unpaired (EU,  $n=8$ ), and US-only (U,  $n=8$ ). Preliminary training itself comprised 2 days of magazine training, 2 days of shaping, 4 days of VI training, and 2 days of pre-testing CSs. Each pre-test session included one nonreinforced CS X (light) and one nonreinforced CS Y (tone). During preliminary training, rats spent odd days in Context A and even days in Context B.

*Conditioning in Context A.* After preliminary training, each group received 5 days of conditioning to CS X in Context A (2 X+ trials daily). CS Y was similarly conditioned over the next 5 days.

*Treatment in Context B.* Before treatment in Context B, all rats received 1 day of VI training in that context to ensure stable barpressing there before treatment. Over the next 24 days, Group EU then received 4 X– trials and 2 USs daily, explicitly unpaired. Group E received only the X– trials of Group EU, and Group U received only the USs. Like the other groups, Group FC was allowed to barpress on the VI schedule in Context B but received neither CSs nor USs.

*Renewal testing and reconditioning in Context A.* After treatment, each group was returned to Context A for 1 day of VI training to ensure stable barpressing there in preparation for renewal testing. Next, each group received 2 days of renewal testing to CS X (2 X– trials daily). Following the renewal test, half the rats in each group were reconditioned to CS X for 6 days. There were 2 X+ trials per day on Days 1 and 2 and 4 X+ trials per day on Days 3 through 6, resulting in a total of 20 trials. During these 6 days, the remaining rats received only VI training.

*Test of CS Y.* Following the 6 days of reconditioning or 6 days of VI training, we tested the untreated CS, Y. There were 2 test days, each with 2 Y– trials. After this test, rats that were not reconditioned to CS X before the test of Y now were reconditioned to X to increase sample size for the analysis of the reacquisition data.

### *Results*

There were five major findings. First, the EU treatment completely thwarted renewal. Second, the 24 days of US-only exposure failed to reduce fear to CS X. Third, reconditioning to CS X was slower in Groups EU and U than in Groups E and FC, suggesting that US habituation had occurred in Groups EU and U. Fourth, rats that had received shocks (X–US pairings) in the test context before CS Y was tested showed more fear of Y than those that had not received such shocks. Fifth,

neither the U nor the EU treatments weakened fear to the untreated CS Y despite the fact that both treatments yielded evidence for US habituation.

*Conditioning in Context A and treatment in Context B*

Fig. 2A shows suppression on the last trial of acquisition to CS X in Context A (far left) and on the first and last trial of each day of treatment in Context B. As in Experiment 1, one-way ANOVAs were conducted on the last trial of acquisition for both CS X and CS Y (Y not shown) and on the first and last treatment trials to CS X. Groups did not differ significantly on these trials;  $F_s(3, 44) < 2.40$  for the last acquisition trial to X and Y, and  $F_s < 1$  for the first and last treatment trials.

As in Experiment 1, there was more suppression and resistance to extinction in Group EU than in Group E during the first 6 days of the treatment phase.

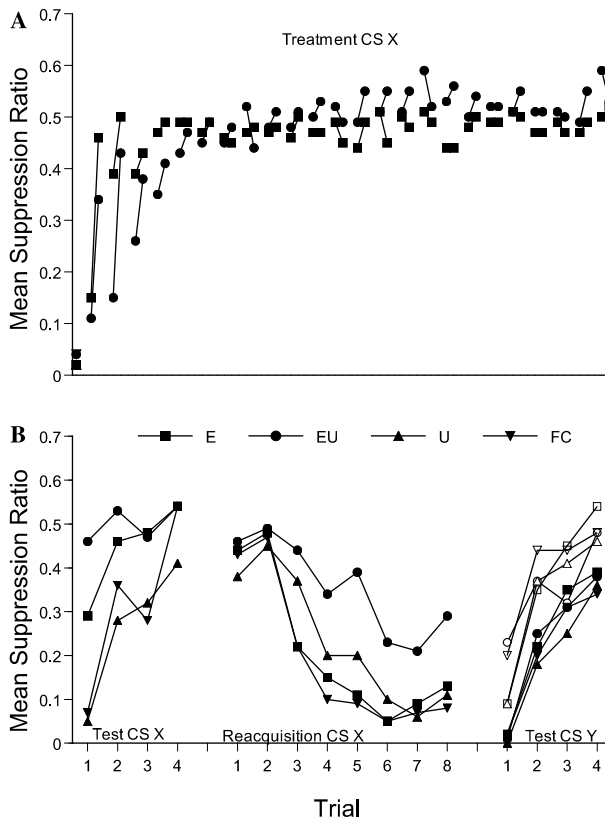


Fig. 2. Experiment 2. (A) Group mean suppression on the last trial of acquisition (far left) and first and last trials of each day of treatment (days separated by gaps in the plots). (B) Group mean suppression in the renewal test (far left), reacquisition (middle), and test of the untreated CS (far right). At the right, closed symbols depict groups that received reacquisition to CS X before the test of the untreated CS, Y, and open symbols depict groups that did not.

A Group  $\times$  Trial ANOVA using all of the trials from those days found effects of group,  $F(1, 21) = 17.79$ , trial,  $F(23, 483) = 22.29$ , and Group  $\times$  Trial,  $F(23, 483) = 3.13$ .

#### *Renewal test of CS X in Context A*

The left part of Fig. 2B shows the results of the renewal test of CS X in Context A. Suppression increased in strength from the last trial of treatment in Context B to the first trial of renewal testing in Context A for Group E,  $t(15) = 5.19$ , but not for Group EU,  $t(7) = 1.97$ . Thus renewal occurred only in Group E. (Groups FC and U provided no data for this analysis, as they received no CSs during treatment.)

A Group  $\times$  Trial ANOVA was conducted on suppression from the four renewal test trials. It revealed significant effects of group,  $F(3, 44) = 18.40$ , trial,  $F(3, 132) = 63.73$ , and Group  $\times$  Trial,  $F(9, 132) = 5.03$ . A follow-up ANOVA isolating Groups E and EU revealed significant effects of trial,  $F(3, 66) = 13.09$ , and Group  $\times$  Trial,  $F(3, 66) = 4.70$ . The interaction suggests that fear was initially greater in Group E than in Group EU. A second follow-up ANOVA isolating Groups E and U also revealed significant effects of group,  $F(1, 22) = 17.10$ , trial,  $F(3, 66) = 47.68$ , and Group  $\times$  Trial,  $F(3, 66) = 5.37$ . The interaction indicates that, unlike Rauhut et al. (2001), fear was initially much greater in Group U than in Group E. A final follow-up ANOVA isolating Groups U and FC revealed only a significant effect of trial,  $F(3, 66) = 54.63$ . Thus, also unlike Rauhut et al. (2001), we found here that 24 days of US-only treatment was entirely ineffectual in weakening fear to CS X.

A Group  $\times$  Trial ANOVA on pre-CS rates from the four renewal test trials revealed significant effects of trial,  $F(3, 132) = 29.90$ , and Group  $\times$  Trial,  $F(9, 132) = 2.47$ . The interaction reflects unsystematic crossings among the groups over the four trials. The mean pre-CS rates for Groups E, EU, U, and FC, respectively, were 23, 20, 18, and 19 responses per minute.

#### *Reconditioning of CS X in Context A*

Reacquisition of suppression to CS X in Context A is shown in the middle part of Fig. 2B. The results are shown for only the first eight of the 20 reacquisition trials. After Trial 8, Groups EU and U showed a strong post-asymptotic performance decrement (cf. Annau & Kamin, 1961; Hendry & Van-Toller, 1965; Prokasy, 1960), probably owing to the US habituation training that they had received during treatment in Context B. During reacquisition trials 9–20, the direction of group differences that were apparent over the first 8 trials (and plotted in Fig. 2B) were maintained. The results plotted include both the rats reconditioned before the test of CS Y and those reconditioned afterwards. A preliminary analysis justified pooling these two sets of rats. In it, the order-of-test factor was not significant and did not interact with any other variables.

A Group  $\times$  Trial ANOVA on the reacquisition data plotted in the figure found significant effects of group,  $F(3, 43) = 6.77$ , trial,  $F(7, 301) = 76.18$ , and Group  $\times$  Trial,  $F(21, 301) = 2.63$ . A similar analysis isolating Groups E and EU revealed significant effects of group,  $F(1, 22) = 17.99$ , trial,  $F(7, 154) = 25.13$ , and Group  $\times$  Trial,  $F(7, 154) = 3.55$ . The effect of group and the interaction reflect weaker fear and slower

reacquisition in Group EU than in Group E. Similarly, a second ANOVA isolating Groups E and U revealed significant effects of trial,  $F(7,154)=38.15$ , and Group  $\times$  Trial,  $F(7,154)=2.24$ . The interaction reflects slower reacquisition in Group U than in Group E. Finally, an ANOVA isolating Groups E and FC revealed only a significant effect of trial,  $F(7,203)=73.36$ .

We interpret the slower reacquisition in Group U relative to Group E as evidence of US habituation in Group U. Note that the slow reacquisition occurred despite the fact that throughout renewal testing, including its last trial, (left part of Fig. 2B) Group U showed stronger fear than Group E and therefore presumably entered reacquisition with a stronger fear residual than did Group E. Other interpretations of the slow reacquisition in Group U, such as context blocking (e.g., Ayres, Bombace, Shurtleff, & Vigorito, 1985) or comparator effects involving comparisons of context fear and CS fear (e.g., Matzel, Brown, & Miller, 1987) are not plausible for two reasons. First, Group U received its USs in Context B, not Context A, where the reacquisition phase took place. Second, prior to the reacquisition phase, half the rats received 3 days of exposure to Context A in the absence of USs (1 day of VI training plus 2 days of testing CS X) and half received 11 such days (1 day of VI training, 2 days of testing CS X, 6 days of VI training, and 2 days of testing CS Y). Such nonreinforced exposure to Context A would be expected to reduce any generalized fear from Context B that might have occurred for Group U as a result of its US-alone treatment in Context B. Because Groups U and EU were equated in terms of the number and spacing of US-alone presentations during treatment, we assume that both of these groups underwent the same US habituation. We assume this despite the apparently weaker suppression in Group EU relative to Group U during reacquisition. The simplest explanation of that weaker suppression is that it reflects the difference in suppression that existed between these two groups during the preceding renewal test phase. That behavioral difference, in turn, was caused by the different treatments of CS X that Groups U and EU received during the treatment phase in Context B.

#### *Test of CS Y in Context A*

The results of testing the untreated CS, Y, are shown in the right panel of Fig. 2B. Because Group U in the reacquisition phase showed evidence of US habituation (and therefore Group EU presumably did as well), one might predict weaker suppression to CS Y in these two groups relative to Groups E and FC. We therefore pooled the U and EU groups and performed a Group  $\times$  Shock history  $\times$  Trial ANOVA on these data. (Shock history refers to whether rats did or did not receive shocks in the test context prior to testing CS Y.) This ANOVA revealed significant effects of Shock history,  $F(1,41)=15.51$ , and trial,  $F(3,123)=110.30$ . As no effects involving group were significant, these results suggest that US habituation did not weaken fear to the untreated CS. Indeed, relative to the forgetting control, none of the treatments weakened fear to the untreated CS, just as in Experiment 1. However, the significant effect of shock history shows that those rats that were shocked in Y's test context before testing CS Y (solid symbols) suppressed more to CS Y than did rats that received only VI training before that test (open symbols).

An identical Group  $\times$  Shock history  $\times$  Trial ANOVA was conducted on the pre-CS rates during the test of CS Y. It revealed significant effects of shock history,  $F(1,42)=6.24$ , trial,  $F(3,126)=13.66$  and Shock  $\times$  Trial,  $F(3,126)=4.06$ . The Trial effect reflects decreasing pre-CS rates across trials. The Shock and Shock  $\times$  Trial effects reflect, respectively, lower pre-CS rates in rats previously shocked in the test context and a faster decrease in rates across trials in those shocked rats than in the nonshocked rats. Mean pre-CS rates for shocked and nonshocked rats, respectively, were 14 and 20 responses per min. In comparison, the corresponding rates before the renewal test in Context A were 20 and 20 responses per minute, respectively. Thus, shocks in the test context prior to testing CS Y reduced pre-CS rates in that test. That fact supports the idea that X–US pairings during the reacquisition phase succeeded in conditioning fear to Y's test context, which was our intent.

### *Discussion*

In Experiment 2, the EU treatment once again thwarted renewal, replicating the results of [Rauhut et al. \(2001\)](#) as well as the results of Experiment 1. This was true even though the shock intensity as measured at the grids was higher than that used in Experiment 4 of [Rauhut et al. \(2001\)](#). Also, like [Rauhut et al. \(2001\)](#), our study found evidence for US habituation in Group U (and presumably Group EU as well) in that reacquisition to CS X following treatment was slower in Group U than in Group E. However, despite this evidence for US habituation, there was no evidence that the U treatment weakened fear to CS X, because suppression to X during renewal testing was similar in Groups U and FC. Nor was there evidence that either the EU or U treatments weakened fear to the untreated CS Y. If US habituation did not weaken fear of either CS X or CS Y in Group U, then it is extremely difficult to see how US habituation could have played a major role in weakening fear to CS X in Group EU relative to the fear of CS X in Group E as measured in renewal testing. Thus, our findings argue strongly against [Rauhut et al.'s \(2001\)](#) conclusion that US habituation is an important factor in the ability of EU treatments to thwart renewal.

Suppression to the untreated CS Y was stronger for rats that received shocks in the test context before testing Y than for rats that did not. This result supports the suggestion we made earlier that fear conditioned to Y's test context during the reconditioning of CS X could have reinstated or augmented fear to CS Y during its extinction test. This reinstatement effect ([Bouton & King, 1983, 1986](#)) could explain why [Rauhut et al. \(2001\)](#) found weaker suppression to their untreated CS Y in their EU and U groups than in their E and FC groups. US habituation in their EU and U groups could have reduced the conditioning of fear to Y's test context and thus have reduced reinstatement of fear to CS Y in its extinction test. So US habituation in their work might have had a proactive effect in reducing context-fear and reinstatement rather than a retroactive effect in weakening fear to a previously conditioned CS Y or CS X. The only evidence against this possibility in our work is that rats in Groups U and EU that received shocks in Y's test context did not suppress less to Y than did Groups E and FC that also received similar shocks. This result, like the

failure of un signaled shocks in Group U to weaken fear to CS X relative to that in Group FC, fails to replicate the findings of Rauhut et al. (2001).

It seems possible that the two findings that fail to replicate Rauhut et al. (2001) may reflect weaker US habituation here than there. The discrepancy could reflect a difference in US intensity, as the degree of habituation tends to vary inversely with stimulus intensity (e.g., Donahoe & Wessells, 1980, p. 50). Our US intensity, as measured directly at the grids, was higher than in Rauhut et al. (2001). Supporting this suggestion are experiments using intense USs in post-conditioning US-alone treatments. Those experiments found no retroactive weakening of fear to a previously conditioned CS (Ayres & Benedict, 1973; Ayres, Mahoney, Proulx, & Benedict, 1976). However, using a weak US, Rauhut et al. (2001), like Rescorla (1973), found evidence that US habituation did indeed weaken fear to a previously conditioned CS. If US habituation was in fact weaker in the present work than in Rauhut et al., as we've just proposed, then that further challenges the idea that US habituation played a major role in thwarting renewal in Group EU. That is, even though US habituation was presumably weaker here than in Rauhut et al., the thwarting of renewal by EU treatment was complete in both cases.

### Experiment 3

In Experiment 3, we reexamined Rauhut et al.'s (2001) hypothesis that the efficacy of the EU treatment in thwarting ABA renewal reflects a combination of the retroactive fear-weakening effects of US habituation and transfer of extinction from Context B to Context A. As noted by Rauhut et al., that idea implies that one should be able to thwart renewal by giving USs alone and CSs alone in succession as well as by intermingling them in an EU fashion. In Experiment 3, we tested this prediction by giving Group CU in the treatment phase a series of CSs alone, followed by a series of USs alone. To Group UC, we gave these same events in reverse. To Group EU, we gave the same number of CSs and USs but intermingled within sessions. For comparison, we included a forgetting control group, Group FC, and a conventional extinction group, Group E. It had two subgroups, Subgroups F–E and E–F. These two subgroups received CSs alone when Groups UC and CU, respectively, did but received only forgetting procedures when Groups UC and CU, respectively, received USs alone. We assumed these subgroups would behave similarly and planned to pool them if they did.

We also tested a second hypothesis. It attributes the EU treatment's efficacy to inhibition conditioned to the CS during the treatment phase in Context B (Rescorla, 1969a). It assumes further that the inhibition transfers from Context B to Context A, where the CS is ultimately tested for renewal. Rauhut et al. (2001) found no evidence for the idea, but we tested it again by using summation and retardation tests in Context A for any inhibitory effects of CS X in that context. Assuming that CS X is found to be inhibitory in Context A after EU treatment in Context B, at least one theory (Rescorla & Wagner, 1972) predicts that it should be less inhibitory in Group UC, and not inhibitory at all in Group CU. According to that theory, inhibition condi-

tioned to a CS increases with the excitation conditioned to the background in which the CS is nonreinforced. That background should be excitatory throughout treatment in Group EU but only at the start of CS exposure in Group UC and should have little excitatory value at all in Group CU. So if conditioned inhibition is key in thwarting renewal, then renewal should be weakest in Group EU, next weakest in Group UC, and strongest in Group CU. In contrast, if thwarting renewal reflects a combination of US habituation and transfer of extinction across contexts (Rauhut et al., 2001), then renewal should be equally weak in all three of these groups.

After the study was completed at the University of Massachusetts, the laboratory was moved to Baldwin-Wallace College, Berea, Ohio, and the experiment was replicated there in its entirety. Results from the pooled replications are described.

### *Method*

#### *Subjects*

In each replication, the subjects were 48 male Sprague–Dawley rats about 90 days old from the same supplier as before. Free feeding weights ranged from 324 to 351 g in the first replication and from 305 to 355 g in the second. In both replications, all rats were kept at 80% of their free feeding levels and were housed and maintained as before.

#### *Apparatus*

In the first replication, the apparatus was unchanged. In the second replication, the wooden housing cubes were replaced by cubes (internal dimensions = 55.9 × 55.9 × 48.9 cm) made of Ultra Plus foam core with a 1 mm PVC facer (United Industries, Bentonville, AR). Eight cubes were placed in each of two rooms. In each room, four of the cubes were stacked 2 × 2 on a bench top affixed to the north wall, and four were similarly stacked on the adjacent west wall. A 2.5-cm sheet of foam insulation separated top and bottom boxes to reduce any cross talk between them. Adjacent cubes were a minimum of 20 cm apart. The operant chambers were identical to those previously described except that no aluminum screening was used on the outside of any of them. A single transport cart was used to carry the rats from the colony to the operant chambers. Four boxes in each room were scented with anise, and four were scented with vinegar, and half the rats in each group were run in each room. In both replications, the shock US intensity was 0.6 mA on the dials and at the grids. Shock was produced by Grason-Stadler shocker scramblers (eight in the first replication and 16 in the second).

#### *Procedure*

*Preliminary training.* Before preliminary training, rats were randomly assigned to five groups E, FC, EU, UC, and CU. In each replication, there were eight rats in each group except Group E, which had eight rats in each of its subgroups (F–E and E–F). Preliminary training itself resembled that of Experiment 2 except that, in addition to the other CSs, a noise-off (noise) CS was given in the pre-test phase. During the 10

days of preliminary training, rats spent odd days in Context A and even days in Context B.

*Conditioning in Context A.* After preliminary training, each group received 6 days of conditioning to tone (CS Y) in Context A, each day with 2 Y+ trials. Next came identical conditioning to CS X, which for half the rats in each group was noise and for half was light.

*Treatment in Context B.* Before treatment in Context B, all rats received 1 day of VI training there to ensure stable responding at the start of treatment. Treatment itself lasted 24 days. On the odd-numbered of these 24 days, Group EU received 4 X– trials intermingled with 2 USs. On even days, Group EU received only VI training. Group CU received 4 X– trials daily for the 1st 12 days and 2 USs daily for the last 12 days. Group UC received the reverse. In Group E, Subgroup E–F received 4 X– trials daily for the 1st 12 days and no exposures on the last 12 days, and Subgroup F–E received the reverse. Finally, Group FC received no exposures on any day. All groups received VI training on every day.

*Renewal, summation, and retardation testing in Context A.* After treatment, each group was returned to Context A for 1 day of VI training to ensure stable barpressing there in preparation for renewal testing. Next, each group received renewal testing to CS X for 3 days, each with 2 X– trials. On the 2 days after renewal testing, a transfer summation test was used to assess inhibition possibly conditioned to CS X. Each day had 1 XY– trial and 1 Y– trial. The order was XY–, Y– on Day 1 and Y–, XY– on Day 2. Recall that Y had been conditioned but was then left untreated. So weaker suppression to XY than to Y would suggest inhibition conditioned to X. For the final 5 days, a retardation test was used as a second measure of inhibition to X. Day 1 had four trials in the following order: Z–, X–, Z+, X+. Days 2, 3, 4, and 5 had two reinforced trials each. The order was Z+, X+ on Day 2, and that order then alternated daily. CS Z was light for rats for which X was noise and was noise for rats for which X was light. CS Z had been left neutral before this test. Slower acquisition to X than to Z would suggest inhibition conditioned to X.

## Results

The main result of Experiment 3 was that only the EU treatment completely thwarted renewal. The UC and CU treatments failed to do so, even though rats in these groups received the same number of CSs and USs as rats in Group EU. In fact, the CU treatment actually increased fear in the renewal test relative to the conventional extinction treatment. Summation and retardation tests in Context A found no evidence that the EU treatment made the CS inhibitory in that context. Indeed, evidence suggested that the CS retained some excitation there. That residual excitation was stronger in Group E than in Group EU.

Despite the fact that the two replications of the experiment involved differences in location and apparatus, and despite the fact that pre-CS rates were much higher in

the second replication than the first, the results in terms of conditioned suppression were highly similar, and so we describe below the pooled data. To make sure this pooling was justified, however, we first conducted a Group  $\times$  Replication  $\times$  Trial ANOVA on the data from renewal testing. The only factor to interact with replication was trial (Replication  $\times$  Trial,  $F(5,415) = 7.07$ ), which reflected faster extinction on the nonreinforced test trials of the second replication than the first.

*Conditioning in Context A and treatment in Context B*

Fig. 3 shows, for CS X, the data from the last trial of acquisition (A), the first (F), and last (L) treatment trials for the 1st, 2nd, 3rd, 4th, and 12th days in which CSs were given, and during the six renewal test trials (far right). One-way ANOVAs, conducted for the last trial of acquisition to CS X and CS Y (Y not shown) and for the first and last treatment trial of CS X, revealed no significant differences among groups,  $F_s < 1$ . Thus, all groups behaved similarly to CS X both at the end of acquisition and just before the renewal test.

As in the previous experiments, Group EU showed greater resistance to extinction than Group E during the treatment phase. A Group  $\times$  Trial ANOVA using all of the trials from the first 6 days of treatment found a main effect of trial,  $F(23,1012) = 41.96$ , as well as a Group  $\times$  Trial interaction,  $F(23,1012) = 2.87$ . The effect of group was not significant,  $F(1,44) = 2.72$ . The interaction indicates differential rates of extinction during the first 6 days with Group EU extinguishing more slowly than Group E. Similar comparisons using Groups CU or UC and Group E found only significant main effects of Trial,  $F_s(23,1035) > 34.58$ . Thus, there was no evidence that rate of extinction differed between Groups UC and E or between Groups CU and E.

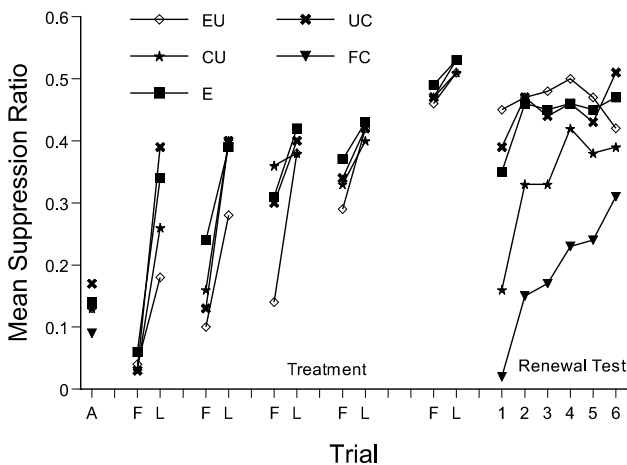


Fig. 3. Experiment 3. Group mean suppression from the last trial of acquisition (A), first (F) and last (L) trials on Days 1, 2, 3, 4, and 12 of treatment (middle), and renewal test trials (right).

### *Renewal Test in Context A*

Before conducting our main analyses, we first performed a preliminary analysis to ensure that we were justified in pooling the E–F and F–E subgroups and calling them *Group E*, as originally intended. Here, we compared the suppression in the two subgroups over the course of the six renewal test trials using a Group  $\times$  Trial ANOVA. This analysis revealed a significant effect of trial,  $F(5, 150) = 7.49$ , but no effect of group or Group  $\times$  Trial,  $F_s < 1$ . As the subgroups did not differ and behaved similarly over trials, they were pooled for other analyses and, together, are termed *Group E*.

Suppression increased significantly from the last trial of treatment in Context B to the first trial of renewal testing in Context A for Groups UC,  $t(15) = 2.85$ , E,  $t(31) = 6.72$ , and CU,  $t(15) = 9.24$ , but not for Group EU,  $t(15) = 1.87$ . Thus, renewal was thwarted completely only in Group EU. (Group FC could not be used in this analysis, as it received no CSs during the treatment phase.)

A Group  $\times$  Trial ANOVA conducted on the six renewal test trials revealed significant effects of group,  $F(5, 89) = 13.54$ , trial,  $F(5, 445) = 26.66$ , and Group  $\times$  Trial,  $F(20, 445) = 2.63$ . Follow-up Group  $\times$  Trial ANOVAs contrasted each of Groups E, UC, and CU against Group EU. In each case, the Group  $\times$  Trial interaction was significant (for E vs. EU,  $F(5, 230) = 3.31$ ; for UC vs. EU,  $F(5, 150) = 3.25$ ; for CU vs. EU,  $F(5, 150) = 7.64$ ), reflecting more fear at the start of testing in Groups E, UC, and CU than in Group EU. Similar ANOVAs then contrasted each of Groups UC, CU, and FC against Group E. The contrast of Groups E and UC revealed only an effect of trial,  $F(5, 230) = 7.94$ , reflecting the similar initial fear in these groups, which then extinguished across test trials. The contrast of Groups E and CU, however, found both the effects of group,  $F(1, 46) = 9.81$ , and trial,  $F(5, 230) = 18.42$ , to be significant. Of most interest is the effect of group, which reflects the stronger suppression in Group CU than in Group E. The contrast of Groups E and FC revealed significant effects of group,  $F(1, 45) = 38.89$ , trial,  $F(5, 225) = 17.45$ , and Group  $\times$  Trial,  $F(5, 225) > 2.37$ . Again, the effects of group and group  $\times$  trial are of most interest, as they reflect the weaker suppression in Group E than in Group FC. This difference shows, once again, that extinction in Context B can transfer to Context A.

A Group  $\times$  Trial ANOVA of the pre-CS rates during renewal testing in Context A revealed no significant effects involving group. The mean pre-CS rates for Groups EU, CU, UC, E, and FC were 40, 38, 53, 46, and 35 responses per minute, respectively.

### *Summation and retardation testing in Context A*

Fig. 4 shows responding to the transfer excitator (CS Y) and to a compound of this excitator and the treated CS, X, averaged over the 2 days of summation testing. Weaker suppression to the XY compound than to the Y element may be taken as evidence for inhibition conditioned to CS X. However, the figure suggests the opposite outcome in all groups. Indeed, a Group  $\times$  Stimulus (Y vs. XY) ANOVA of these data found significant effects of both stimulus,  $F(1, 89) = 68.18$ , and group,  $F(4, 89) = 5.18$ , but no Group  $\times$  Stimulus interaction,  $F(4, 89) = 1.55$ ,  $p = .195$ . The effect of stimulus reflects stronger suppression to the XY compound than to the Y element, and this result suggests that treatment in Context B did not transform CS X to a fear inhibitor

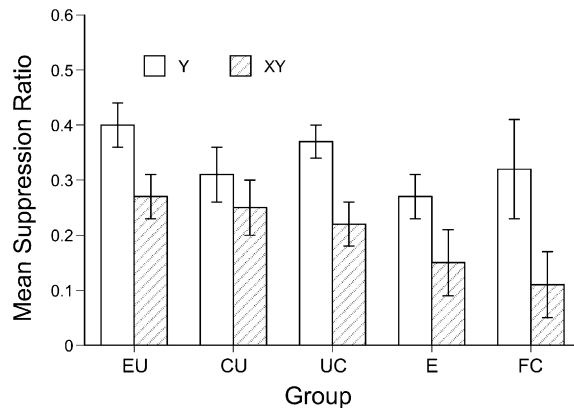


Fig. 4. Experiment 3. Group mean suppression to the transfer excitator (CS Y) and to the compound of Y plus the treated CS, X. Error bars depict  $\pm 1$  SEM.

in any group, at least as assessed in Context A. Rather, the CS in each group appeared to have retained some excitatory value in that context.

Fig. 5 shows the results of the retardation test (i.e., reconditioning to the treated CS X and initial conditioning to CS Z for each group). The results for Group E, labeled E–X and E–Z, are shown in each panel to provide a common comparison for each of the other groups. Slower reconditioning to CS X relative to the initial conditioning of CS Z may be taken as evidence for the inhibitory qualities of CS X. Conversely, faster reconditioning of CS X relative to initial conditioning of CS Z would suggest residual excitation to CS X.

For the retardation test, Group  $\times$  Trial  $\times$  Stimulus (X vs. Z) ANOVAs were conducted on the data in each of Figs. 5A–D. The first ANOVA (A) found significant effects of group,  $F(1,46) = 13.34$ , trial,  $F(5,230) = 90.59$ , Group  $\times$  Trial,  $F(5,230) = 5.23$ , and Stimulus  $\times$  Trial,  $F(5,230) = 3.25$ . Respectively, these effects indicate that Group EU suppressed less than Group E, that suppression increased across trials, increased faster in Group E than Group EU, and faster to CS X than to CS Z.

The second ANOVA (B) found similar results. The effect of group was significant,  $F(1,46) = 12.11$ , as were the effects of trial,  $F(5,230) = 85.04$ , Group  $\times$  Trial,  $F(5,230) = 5.36$ , and Stimulus  $\times$  Trial,  $F(5,230) = 3.57$ . Respectively, these effects indicate that Group CU suppressed less than Group E, that suppression increased across trials, and increased faster in Group E than Group CU, and faster for CS X than CS Z.

The third ANOVA (C) found similar results yet again. It found significant effects of group,  $F(1,46) = 9.19$ , trial,  $F(5,230) = 97.52$ , and Stimulus  $\times$  Trial,  $F(5,230) = 5.02$ . Respectively, these effects indicate that Group UC suppressed less than Group E, that suppression increased over trials, and increased faster for CS X than for CS Z.

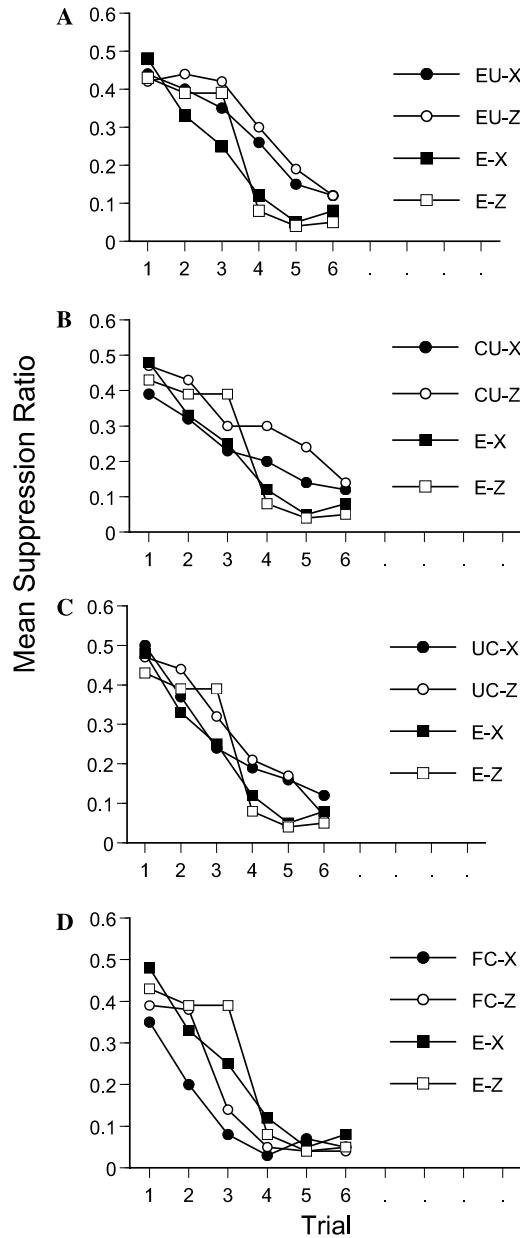


Fig. 5. Experiment 3. Group mean suppression for each group during reconditioning to the treated CS, X, and initial conditioning to CS Z.

Finally, the last ANOVA (D) found significant effects of group,  $F(1,45) = 6.04$ , trial,  $F(5,225) = 2.51$ , Group  $\times$  Trial,  $F(5,225) = 2.51$ , and Stimulus  $\times$  Trial,  $F(5,225) = 6.88$ . Respectively, these effects indicate that Group E suppressed less

than Group FC, that suppression increased across trials, increased faster in Group FC than in Group E, and faster for CS X than CS Z.

In summary, these four analyses found that all but Group FC showed retarded acquisition of suppression relative to Group E. In addition, all the analyses found evidence for faster acquisition of suppression to CS X than to CS Z. That finding suggests that CS X retained some excitatory value after treatment and is consistent with the results of summation testing.

Both the summation and retardation tests suggested that CS X retained some excitatory residual following treatment and renewal testing. A question of interest is whether the size of that residual differed across groups. Consider, for example, Groups EU and E. By the end of renewal testing, those two groups showed no suppression, suggesting no fear residual in either group. Clearly, though, suppression was so weak in Group E that it could hardly be weaker in Group EU. So a floor effect may have obscured potential differences between groups. A summation test or a reacquisition (retardation) test might remove that floor effect and reveal a potential difference. Although we know of no report that a summation test can do so, we have indeed seen such a demonstration in a reacquisition test (see Fig. 1 of Ayres et al., 1976). In search of similar effects here, we contrasted the groups' suppression to the XY compound in the summation test and separately contrasted their suppression to CS X in the reacquisition (retardation) test. In the summation test, the group mean SRs to the XY compound for Groups EU, CU, UC, E, and FC, respectively, were .27, .24, .22, .14, and .11 (cf. Fig. 4). A one-way ANOVA found significant differences among groups,  $F(4, 89) = 4.18$ . Contrasts of Groups CU, UC, and E against Group EU showed only the difference between Groups EU and E to be significant,  $F(1, 45) = 7.84$ . In the reacquisition (retardation) test, the mean SRs to CS X for Groups EU, CU, UC, E, and FC were .29, .27, .28, .20, and .13. A one-way ANOVA again found significant differences among groups,  $F(4, 90) = 6.66$ . And contrasts of Groups CU, UC, and E against Group EU again revealed only the difference between Groups EU and E to be significant,  $F(1, 46) = 8.98$ . Thus, despite the fact that the renewal test involved a procedure designed to minimize differences among groups by reducing fear in all of them, both the summation and retardation tests, which followed renewal testing, showed that at least the difference between Groups EU and E remained intact. The tests succeeded in revealing a difference between these groups that had been obscured by a floor effect at the end of renewal testing. The direction of the difference strongly supports the idea that the EU treatment leaves a much weaker fear residual relative to conventional extinction.

Note that in both the summation and reacquisition tests, Groups CU and UC performed similarly and at a level more like that of Group EU than Group E. This pattern is consistent with the idea that the much stronger suppression in Group CU versus Groups UC and E at the start of renewal testing was due to some factor operating there on Group CU. We shall discuss that factor in the Discussion section below.

Suppression to CS Z during the retardation test would seem to offer the best measure of US habituation in Groups EU, UC, and CU, which received USs during treatment, relative to Groups E and FC, which did not. Unlike CS X, CS Z was asso-

ciatively neutral at the start of the retardation test and had not been treated differently in the groups during the treatment phase. Using the suppression to CS Z, we can ask two important questions about US habituation. First, did Groups EU, UC, and CU combined show weaker suppression to CS Z than Groups E and FC combined? Second, did Groups EU, UC, and CU differ in their suppression to CS Z? If Group EU underwent more US habituation (accounting for its ability to thwart renewal?), then it should suppress less to CS Z than should Groups UC and CU. With regard to the first question, the mean SR for CS Z (averaged over the six retardation test trials) for groups EU, UC, and CU combined was .30; the corresponding mean for Groups E and FC combined was .20,  $t(92) = 2.82$ ,  $p < .01$ . The difference is consistent with the idea that Groups EU, UC, and CU underwent US habituation during treatment. With regard to the second question, the mean SRs for CS Z for Groups EU, UC, and CU, respectively, were .30, .28, and .31. These means did not differ significantly,  $F < 1$ . Thus, the suppression to CS Z revealed evidence of US habituation in Groups EU, UC, and CU but showed no differences among the three groups, even though these groups differed in their fear during renewal testing. Once again, this evidence offers no support for the hypothesis that US habituation played a major role in thwarting renewal following EU treatment.

### *Discussion*

As in Experiments 1 and 2, renewal was thwarted completely only in Group EU. In contrast, renewal did occur following conventional extinction, as before, and it did not matter that 12 days of VI training preceded CS-alone trials (Subgroup F–E) or followed them (Subgroup E–F). Finally, Group CU showed significantly more suppression in the renewal test than either Group UC or Group E.

These results further challenge the hypothesis that the efficacy of the EU treatment in thwarting renewal reflects the combination of the transfer of extinction across contexts and the retroactive fear-reducing effects of US habituation. If that hypothesis were correct, then one would expect the UC and CU treatments to thwart renewal just as well as the EU treatment because all three involved the same number of unpaired CSs and USs. Yet, this was clearly not the case, as renewal occurred in both the CU and UC treatments, and fear in the renewal test was stronger in these groups than in Group EU.

A measure of US habituation, the suppression to CS Z during retardation testing, found weaker suppression in the groups that had received USs during treatment (Groups EU, UC, and CU combined) relative to those that did not (Groups E and FC combined), suggesting that US habituation had occurred in the former groups. Yet Groups EU, UC, and CU did not differ on this measure, suggesting that different levels of habituation, which were unexpected theoretically in any case, did not contribute to differences in the groups' fear in the renewal test. Thus US habituation does not appear to be key in the EU treatment's ability to thwart renewal.

An alternative hypothesis was that the EU treatment might cause the CS to become inhibitory and that such fear-inhibition in Context A could thwart renewal. Like Rauhut et al. (2001), we found no support for that hypothesis. That is, in Con-

text A, the CS, X, passed neither a summation test nor a retardation test for conditioned inhibition. Indeed, both tests suggested that the CS retained some excitation following treatment. This latter finding differs somewhat from the results of Rauhut et al. There, the CS in the summation test did not produce an excitatory summation effect as it did here. Presumably, the discrepancy reflects the difference in the length of the EU treatment and a difference in shock intensity. The EU treatment was twice as long in Rauhut et al., as it was here; and the shock here, as measured at the grids, was more intense. The present Experiment 1 showed the length of treatment to be key in weakening fear as assessed in the renewal test; and it is well known that the strength of conditioned suppression increases with US intensity, as does the difficulty in weakening that suppression with extinction procedures (e.g., [Annau & Kamin, 1961](#)). It is reasonable to believe, therefore, that shock intensity and treatment duration should also be important determinants of a CS's effects in post-extinction tests, such as the summation and retardation tests used here and in Rauhut et al.

An unanticipated finding was that suppression during the renewal test was stronger in Group CU than in Group E. One possible explanation of that result is that the USs that occurred in the last 12 days of the CU treatment conditioned fear to Context B that generalized to Context A. Such fear would not be expected in Group E because that group received no USs during treatment. Nor would such fear be expected in Group UC. In that group, the fear conditioned to Context B during the 12 days of US-alone presentations should have extinguished during the following 12 days of CS-alone presentations. If context-fear was indeed greater in Context A for Group CU than for Group E during renewal testing, then that context fear might augment or reinstate the partially extinguished CS-fear ([Bouton & King, 1983, 1986](#)), giving the appearance of strengthened renewal. Though one might argue that the same sort of mechanism should operate in Group EU, we believe that other mechanisms that operate in the EU procedure (see General discussion) would eliminate that effect.

In comparison to previous renewal effects seen in our laboratory ([Rauhut et al., 2001](#); [Thomas & Ayres, 2004](#); [Thomas et al., 2003](#); present Experiments 1 and 2), the renewal that occurred in Group E in Experiment 3 seemed quite weak, albeit still highly significant. Yet that weak renewal was consistent across both replications of the experiment and in both the F–E and E–F subgroups that composed Group E. The curve plotted for Group E in [Fig. 3](#) is based on a sample size of 32 rats. Perhaps the combination of 12 days of conventional extinction and 12 additional days of exposure to the treatment context combined in some way to reduce fear as assessed in Context A. We certainly did not anticipate this possibility. It seems to warrant future systematic study.

## General discussion

### *Interpretations of the EU treatment's effects*

Our work has replicated [Rauhut et al.'s \(2001\)](#) finding that EU treatments thwart renewal of conditioned fear in an ABA fear renewal paradigm. More

importantly, it has challenged [Rauhut et al.'s \(2001\)](#) conclusions about the mechanisms responsible for that effect. Based on evidence outlined in our introduction, [Rauhut et al. \(2001\)](#) attributed the EU treatment's efficacy to a combination of two separate effects: (a) the transfer of CS-alone extinction from Context B to Context A, and (b) the ability of US habituation to weaken fear retroactively to a previously conditioned CS ([Rescorla, 1973](#)). Though not disagreeing with the first part of that conclusion, our results have provided three bits of evidence against the second part. First, all of the EU treatments in Experiment 1 completely thwarted renewal to the treated CS (although 6 days was insufficient to completely eliminate the original suppression) but did not weaken fear of other previously conditioned but subsequently untreated CSs. If the EU treatments thwarted renewal because they produced US habituation that retroactively weakened the fear to the treated CS, then they should also have retroactively weakened fear to other, untreated CSs by the same mechanism. Second, the EU treatment and the US-alone treatment of Experiment 2 contained the same number and temporal distribution of USs. US habituation should have been similar in these two groups. Yet, the EU treatment completely thwarted renewal to the treated CS, resulting in negligible fear during renewal testing, but the US-alone treatment did not weaken fear to that CS. If US habituation in Group EU was a major factor in producing the weak fear to the treated CS during renewal testing, then fear should also have been weakened to that CS after the US-alone treatment. Moreover, neither the EU treatment nor the US-alone treatment in Experiment 2 weakened fear to a second CS that for both groups had been conditioned but was subsequently left untreated. Both treatments should have weakened fear to this second CS if US habituation in Group EU was largely responsible for the weak fear of the treated CS. Third, the EU procedure of Experiment 3 once again completely thwarted renewal. In contrast, the CU and UC procedures, which contained the same number of unpaired CSs and USs but in different, not intermingled, orders did not thwart renewal. US habituation should have been (and was by our measures) similar in all three of these groups. Given that the three groups performed so differently during renewal testing, it seems highly unlikely that the efficacy of the EU procedure in thwarting renewal reflects its ability to weaken conditioning retroactively by producing US habituation.

An alternative hypothesis as to the EU treatment's efficacy is that it transforms the treated CS from a conditioned excitor to a conditioned inhibitor of fear and that the inhibition operates in Context A. [Rauhut et al. \(2001\)](#) rejected that idea because they found no evidence in Context A for inhibition conditioned to the treated CS. In that context, the CS failed both a retardation test and a summation test for conditioned inhibition. Based on our own summation and retardation tests, we too reject that hypothesis. In our tests, however, the evidence suggested that not only was the EU CS not inhibitory in Context A, it actually retained some excitatory value there that was not detectable in renewal testing. In this respect, our results resembled those of [Reberg \(1972\)](#) and [Hendry \(1982\)](#) who found that summation tests given after a long series of extinction trials detected excitation that was not apparent at the end of the extinction series itself.

It is interesting that we found evidence for residual excitation in our summation test but [Rauhut et al. \(2001\)](#) did not. During conditioning in their Experiment 4, [Rauhut et al. \(2001\)](#) paired their CS with a weaker US (0.25 mA vs. 0.6 mA here), and their treatment duration was twice as long as ours (24 days vs. 12 days here). We believe that the efficacy of the EU treatment varies inversely with the strength of the initial conditioning and directly with the length of treatment. Experiment 1 supported the second part of that belief by showing that the EU treatment's efficacy in weakening fear as assessed in a renewal test increased with treatment duration. The belief is also consistent with the well known finding that conditioned suppression increases as a function of shock intensity, as does the number of extinction trials required to eliminate that suppression ([Annau & Kamin, 1961](#)).

If the efficacy of the EU treatment does not reflect the combined effects of CS-alone extinction and US habituation, and if it does not reflect the transformation of a fear-excitor into a fear-inhibitor that operates in Context A, then what does it reflect? Let us consider first the best-known theories of renewal, those proposed by [Bouton](#) and his collaborators ([Bouton, 1988, 1991, 1993](#); [Bouton & Nelson, 1998](#); [Bouton & Ricker, 1994](#)).

[Bouton \(1988, 1991, 1993\)](#) has suggested that a CS in an ABA fear renewal paradigm acquires ambiguous meaning. During conditioning in Context A, it comes to mean danger. Then during extinction in Context B, it comes to mean safe. It now has ambiguous meaning, which can be "disambiguated" by a return to Context A. There, the CS means danger, and so fear is renewed. The original meaning of the CS was not lost during the extinction process just as the meaning of the word "fire," meaning to fire a gun on the firing range is not lost if we later learn in a different context that the word "fire" means "there's a fire in the house." The word "fire" has two meanings, and our interpretation of its current meaning depends on the context. The process should be the same in a fear renewal paradigm with an EU treatment. The CS should come to mean danger after conditioning in Context A and come to mean safety during the EU treatment, where each CS presentation predicts a time free of USs ([Rescorla, 1969a](#)). A return to Context A should disambiguate the meaning. The CS should once again mean danger, and fear should be renewed. Knowing that fear renewal is instead thwarted after EU treatment, we can see that this way of talking about renewal is not helpful in understanding the EU treatment's efficacy.

[Bouton \(e.g., 1993; Bouton & Nelson, 1998; Bouton & Ricker, 1994\)](#) also proposed other concepts to explain renewal—the concepts of excitation, inhibition, and generalization decrement. During conditioning in Context A, the CS acquires the ability to excite fear; it becomes excitatory. During its subsequent extinction in Context B, it retains that excitation, but simultaneously acquires inhibition (or a CS–no-US association). The inhibition grows, eventually balancing the excitation. At that point, responding ceases. The inhibition is postulated to be more sensitive to context than is the excitation. So when the animal is moved out of Context B, the inhibition suffers generalization decrement, allowing the still present excitation free to manifest itself. If the inhibition that accrues on CS-alone trials in an EU procedure is like the inhibition that accrues in a CS-alone extinction procedure, then this theory cannot explain why EU procedures thwart renewal. It could, however, do so if it added the proviso

that the inhibition that builds up in EU procedures is less sensitive to context than is the inhibition that accrues during extinction. With that proviso, one could imagine that the inhibition that built up in Context B during an EU treatment generalizes fully to Context A, with the result that the still fully intact excitation and the inhibition continue to balance out and, therefore, responding does not occur. Renewal is thwarted. This proviso alone, however, would not explain why we found evidence for residual excitation in our summation test, for if the excitation and inhibition continued to balance out, leaving a net value of zero, there would be no value to summate with the transfer excitator's value and hence no excitatory summation effect. One might then have to argue that the transfer excitator weakened the inhibition, leaving two excitatory values to sum. Likewise, in the retardation test, the USs paired with the target CS, CS X, might quickly abolish the inhibition previously conditioned to that CS in the EU procedure, leaving only the still fully intact excitation. The rapid abolition of the inhibition would thus produce our finding, that is, make CS X "appear" to acquire excitation faster than the neutral CS, CS Z.

Although this amended version of Bouton's theory can account in principle for our present findings, it has difficulty with earlier findings from our laboratory (Thomas & Ayres, 2004). In that work, fear in an ABA renewal test tended to vary inversely with the values of co-present cues during extinction of the target CS in Context B. Specifically, three experiments found that, compared to ordinary extinction in Context B, the extinction of the target CS in compound with a putative conditioned inhibitor of fear in Context B led to enhanced fear in a renewal test in Context A. A fourth experiment found that, compared with ordinary extinction of three separate excitators in Context B, the extinction of all three in compound led to weaker fear to two of the three elements and to the compound in a renewal test in Context A. These findings are consistent with theories that hold that CSs actually lose conditioned value during nonreinforced CS exposure and, moreover, that the degree of such loss is directly related to the values of co-present cues during extinction (e.g., Rescorla & Wagner, 1972). The findings are less consistent with theories, such as Bouton's, that hold that conditioned value remains fully intact during extinction procedures. Thus, although Bouton's theory, as amended here, can explain our present findings, a theory that could simultaneously explain both our present findings and those of Thomas and Ayres (2004) would seem desirable. We return to this issue below.

Another way of explaining why EU procedures thwart renewal is to note that shocks intermingled among explicitly unpaired CSs in the same session might make the treatment context, Context B, resemble the acquisition context, Context A, where shocks and CSs also occurred in the same session. On this view, Group EU's paradigm might be relabeled AAA in contrast to Group E's paradigm labeled ABA. As renewal does not occur in AAA paradigms (e.g., Bouton & Bolles, 1979; Bouton & King, 1983; Thomas et al., 2003), this relabeling easily and parsimoniously explains why the EU treatment thwarts renewal. However, like the theory considered in the previous paragraph, it too is silent about the results of Thomas and Ayres (2004) and even about other findings in the present work, such as why so many of Group EU24's suppression ratios exceeded .50 during treatment in Context B (see Figs. 1 and 2). In addition, the theory relies on the ad hoc and dubious assumption that the similarity

of a few discrete events scattered widely in time in intentionally discriminable contexts can outweigh those intentional context differences. And finally, the theory assumes that the ability to outweigh those context differences is virtually complete. That assumption is necessary because significant renewal has been demonstrated with only a few differences between contexts: odor, location in a small room, and unintended differences between copies of the same operant chamber (Thomas et al., 2003).

Thomas and Ayres (2004) proposed a theory of renewal that combines aspects of Bouton's theory (e.g., Bouton, 1993; Bouton & Nelson, 1998) with aspects of the model of Rescorla and Wagner (1972). Though possibly more complex than other theories we have considered here, it appears to have the virtue of being far more comprehensive. It can explain the present findings, the findings of Thomas and Ayres (2004) about the role of co-present cues during extinction, the findings of Thomas et al. (2003) that AAB renewal is weaker than ABA and ABC renewal, and many other results in the renewal literature (for discussion of those results, see Thomas & Ayres, 2004). Like Bouton's theory, it explains renewal by assuming that some inhibitory process builds up during extinction to balance existing excitation; it assumes that the excitation and inhibition can coexist; and it assumes that inhibition weakens more than does excitation with a change in context. Unlike Bouton's theory, but like the Rescorla–Wagner model, it does not assume that excitation remains fully intact on nonreinforced trials. Rather, like the Rescorla–Wagner model, it assumes that CSs lose value on nonreinforced trials. Moreover, it assumes that the loss increases with (a) the degree to which responding is evoked in CS presence (Rescorla, 2001; Solomon & Wynne, 1954) or (b) the value of co-present stimuli (Rescorla & Wagner, 1972).

In all three experiments of the present report, it was clear that more responding (conditioned suppression) was evoked in CS presence during the first several days of treatment for the EU procedure than for the conventional extinction procedure. According to Assumption *a*, this stronger responding should have caused a greater loss in CS value in Group EU than in Group E.

The stronger responding in the EU groups that was just mentioned may have reflected greater reinstatement in those groups. As previously discussed, reinstatement refers to the tendency for excitatory contexts to augment responding to an extinguished CS or one that is undergoing extinction (Bouton & King, 1983, 1986). That should clearly happen in EU groups because CS-alone trials occur in a context presumably kept excitatory by US-alone trials. Thus, Assumption *b* also predicts that the CS should have lost more value during the EU procedure than during the conventional extinction procedure.

According to Thomas and Ayres' (2004) theory, as the CS loses excitatory value, it simultaneously gains an inhibitory value that increasingly balances the decreasing residual excitation. If it loses enough excitatory value and gains enough inhibitory value, it may appear to be inhibitory when tested in Context B. Evidence for this possibility is suggested by the finding that SRs often increase above .50 during EU treatments in Context B (Rauhut et al., 2001; present work as seen in Figs. 1 and 2). When the context is changed, however, the inhibition suffers more generalization decrement than the residual excitation does. This allows responding to be renewed in proportion

to the size of the excitatory residual. Although [Thomas and Ayres \(2004\)](#) did not discuss the role of the length of the EU treatment, it seems reasonable to suggest that if the EU treatment is long enough, there may be no excitatory residual at all, as suggested by the renewal and summation test data of [Rauhut et al.'s \(2001\) Experiment 4](#) following 24 sessions of EU treatment. If the EU treatment is shorter, the residual excitation, though still not detectable in a renewal test, may be detected in a summation test (e.g., [Hendry, 1982](#); [Reberg, 1972](#)) as it was following 12 days of EU treatment in our present Experiment 3. If the EU treatment is shorter still, the residual excitation may even be detectable in a renewal test, as it was following 6 days of EU treatment in our present Experiment 1.

The idea that the strength of the excitatory residual is inversely related to the associative strength of co-present stimuli during treatment may help to explain [Thomas et al.'s \(2003\)](#) finding that AAB renewal is weaker than ABA and ABC renewal. Thus, in the AAB case, the extinction (or treatment) context is the same context that has just been paired with USs during the acquisition phase. Therefore, it should be more excitatory during nonreinforced CS trials than should Context B in the ABA and ABC cases.

[Thomas and Ayres' \(2004\)](#) theory is also consistent with [Rauhut et al.'s \(2001\)](#) finding that conditioned inhibition and differential conditioning treatments also completely thwart renewal. In the conditioned inhibition treatment, the CS is always nonreinforced in compound with another CS whose excitatory value is deliberately maintained during treatment. In the differential conditioning treatment, the CS is nonreinforced in a context that can acquire excitatory value when a second CS is reinforced in its presence. [Thomas and Ayres'](#) theory expects the treated CS to lose more value in these procedures than it would during conventional extinction. It further expects the treated CS to gain inhibitory value as it loses excitatory value. If it loses enough excitatory value and gains enough inhibitory value, it may, as mentioned earlier, appear to be inhibitory in Context B. [Rauhut et al. \(2001\)](#) found some evidence for such inhibition in their summation test conducted in Context B in their Experiment 3. However, in their summation test conducted in Context A in their Experiment 4, they detected no evidence for inhibition. Those results are consistent with the assumption that inhibition shows considerable generalization decrement when the context is changed. [Rauhut et al. \(2001\)](#) also detected no evidence for residual excitation in either their renewal test or their summation test of their Experiment 4, suggesting that their (lengthy) conditioned inhibition and differential conditioning treatments drove their CS's excitatory value close to zero.

The only evidence we know of that does not support these ideas is the failure of the UC treatment in the present Experiment 3 to reduce fear in the renewal test relative to conventional extinction. We might have expected the USs that were given in the 1st 12 days of treatment to have strongly conditioned the treatment context. Nonreinforced CS exposures in that context over the next 12 days should have weakened the CS's value more than 12 days of exposure in a more neutral context (e.g., Subgroup F–E). That result did not occur. Yet, this test of the theory was not optimal because there was nothing to prevent the treatment context from losing its excitatory value during the 12 days of nonreinforced CS exposure in Group UC.

### *Implications for treating human fears and phobias*

Rauhut et al. (2001) first showed that 24 days of EU treatment could prevent renewal (relapse). They noted, however, that using aversive stimuli during therapy with humans would be distasteful to therapists and clients alike and wondered if fewer EU sessions might be as effective, while minimizing exposure to aversives. We have seen here that as few as 6 days of EU treatment weakened fear in a renewal test significantly more than did 24 days of conventional extinction. However, such a short EU treatment still left significant fear in that test.

Rauhut et al. (2001) suggested that a possibly unique advantage of the EU treatment was its ability to weaken fear to other CSs besides the one undergoing treatment. They attributed that ability to the retroactive fear-weakening effects of US habituation (Rescorla, 1973). We have found little evidence for such effects here and believe that if they are real, they occur only with very weak USs and/or very long treatments.

These considerations make the use of EU treatments in therapy even less likely than they were before we began this project. However, the theoretical mechanisms that Thomas and Ayres (2004) use to explain the ability of EU treatments to prevent relapse suggest other ways of achieving that goal without presenting unconditioned aversive stimuli in therapy. They suggest that feared cues should be nonreinforced in therapy with other co-present feared cues and not in the presence of cues that might be fear inhibitors. Thomas and Ayres (2004) have presented empirical support for that suggestion using animal models in ABA renewal paradigms and have discussed in some detail how that approach could be applied to therapy with humans.

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