CSs and USs: What's the Difference?

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Differences in processing representations of conditioned and unconditioned stimuli (CSs and USs) may result from either their temporal order in training (i.e., CSs precede USs) or the greater biological significance of USs. The CS- and US-preexposure effects were used to probe this question. These effects are similar except that context extinction between preexposure and training more readily attenuates the US- than the CS-preexposure effect. In Experiments 1, 2, and 3, context extinction following preexposure to the stimulus that later served as Event 1 in Event 1-Event 2 pairings alleviated the response deficit due to Event 1 preexposure if Event 1 was biologically significant. In Experiments 3 and 4, context extinction alleviated the response deficit due to Event 2 preexposure if Event 2 was biologically significant. Thus, biological significance and not temporal order determines how a representation will be processed.

There are two families of theories concerning acquisition in Pavlovian conditioning: one positing that prior experience alters processing of conditioned stimuli (CSs; e.g., Pearce & Hall, 1980; Mackintosh, 1975) and one positing that prior experience alters processing of unconditioned stimuli (USs; e.g., Rescorla & Wagner, 1972). A third view, perhaps less parsimonious but potentially more realistic, might incorporate both principles (e.g., Wagner, 1981). Thus, most models of conditioning assume that CSs and USs are subject to different processes during learning. The present research asked which differences between CSs and USs are the basis of differential processing of CSs and USs. There are two dimensions on which CSs ordinarily differ from USs, temporal order (i.e., CSs usually precede USs) and biological significance (i.e., CSs are usually of lower biological significance than are USs). Alternatively stated, CSs are commonly presented as antecedents to USs and are of relatively low biological significance, whereas USs are commonly presented subsequent to CSs and are of relatively high biological significance.

In the present research, we use as a tool to examine this issue the differential effects on the CS- and US-preexposure effects of extinction of associations to the context that occurs between preexposure and conditioning. That is, extensive context exposure (presumably extinguishing the association between the context and the preexposed stimulus) following US-preexposure and prior to the conditioning phase has been observed to readily attenuate the performance deficit typically seen after US preexposure (Randich, 1981). However, similar extinction of associations to the context between CS preexposure and subsequent training does not ordinarily attenuate the deleterious effect of CS preexposure (e.g., Hall & Minor, 1984).

Temporal Order

Although many studies have used simultaneous and backward conditioning procedures, the CS precedes the US in the vast majority of conditioning experiments because this temporal ordering results in superior behavioral control by the CS when anticipatory behavioral measures are used (which is usually the case). Moreover, all of the reports concerning the effects of CS or US preexposure and particularly all of the published studies of the effect of extinction of associations to the context between preexposure and conditioning have administered forward pairings (i.e., CS-US) during the conditioning phase. Thus, in the CS-preexposure effect, it is always the first element of the CS-US sequence to which the subject is preexposed; whereas, in the US-preexposure effect, it is always the second element of the CS-US sequence to which the subject is preexposed. Consequently, the differential consequences of extinguishing contextual associations after CS and US preexposure are perfectly correlated with the temporal position of the preexposed cue during the subsequent CS-US pairings.

Biological Significance

We define biological significance in terms of the behavioral control a stimulus exercises over a subject, with greater
responding corresponding to higher biological significance (i.e., either unconditioned or conditioned responses). USs are typically assumed to be events of inherently high biological significance (e.g., food, water, painful stimulation, or most any very intense stimulus). More empirically, a US is ordinarily regarded as a stimulus that elicits a strong unconditioned response from the subject. Food, water, painful stimuli, and any very intense stimuli are examples of stimuli with inherently high biological significance. With respect to the intensity dimension, presumably the biological significance of a stimulus increases with the intensity of that stimulus. For example, a loud noise is assumed to be of greater biological significance than an otherwise equivalent noise of moderate intensity. In contrast, a CS, prior to being paired with a US, is ordinarily a relatively neutral stimulus that elicits little or no responding; hence, we view them as initially being of low biological significance. Typically, CSs are auditory or visual cues of moderate intensities that elicit no more than a weak orienting response. On the other hand, pairing a CS with a US may endow the CS with acquired biological significance. That is, a stimulus of inherently low biological significance can presumably be elevated to high biological significance by pairing it with a stimulus of inherently high biological significance, such as food, water, or painful stimuli. In this case, the cue comes to acquire biological significance through association.

The potential importance of a stimulus's biological significance in determining the nature of information processing is suggested by a recent study by R. R. Miller and Matute (1996; also see Denniston, Miller, & Matute, 1996). They were interested in the failure of previous research to observe backward blocking effects in Pavlovian conditioning with animals (R. R. Miller, Hallam, & Oramhe, 1990; Rescorla & Durlach, 1981; Schweitzer & Green, 1982) in comparison with the successful occurrence of backward blocking seen in causal judgment by humans (e.g., Chapman, 1991; Shanks, 1985). In the typical two-group forward blocking experiment, Group 1 receives Phase 1 training consisting of CS1→US pairings followed by Phase 2 training with compound CS1+CS2→US pairings, while Group 2 receives only the Phase 2 training. Blocking is then seen in deficient behavioral control by CS2 at test in Group 1 in relation to Group 2. The effect is assumed to arise from the CS1→US association "blocking" either the acquisition or expression of the CS2→US association. In the backward blocking procedure, Phase 1 and Phase 2 training are reversed (i.e., CS1+CS2→US compound training occurs prior to CS1→US training).

R. R. Miller and Matute (1996) explained the failure to observe backward blocking in animal conditioning by suggesting that in the backward blocking procedure, the target stimulus (i.e., CS2) acquires biological significance (through CS1+CS2→US training) prior to the CS1→US treatment, and because of this acquired biological significance, it cannot be subsequently blocked. However, in the forward blocking procedure, CS2 does not have the opportunity to acquire biological significance in Phase 1, and consequently it is vulnerable to being blocked in Phase 2. Miller and Matute provided evidence suggesting that stimuli of high biological significance are relatively protected against cue competition (specifically, blocking), which suggests that stimuli of high and low biological significance are differentially processed. This finding can be extrapolated to the CS- and US-preexposure effects. It suggests that in the two preexposure procedures, subjects are preexposed to stimuli that are processed in fundamentally different ways because of the difference in biological significance of CSs and USs. In the CS-preexposure effect the subject is preexposed to a cue of low biological significance, whereas in the US-preexposure effect the subject is preexposed to a cue of high biological significance. Consequently, the observed differential consequences of extinguishing contextual associations after the CS- and US-preexposure effects are correlated with the biological significance of the preexposed stimulus. Thus, stimulus preexposure might be expected to yield different consequences in the two cases. This expectation is congruent with the previously described differential effect of extinguishing contextual associations between stimulus preexposure and CS-US pairings.

Thus, the apparent differences in the processing of CSs and USs, as exemplified in the case of context extinction following stimulus preexposure, could be accounted for by differences in conventional temporal position of CSs and USs, by the inherent discrepancy between CSs and USs in terms of their biological significance, or by both. The present research asks which of these two factors (temporal order or biological significance) is responsible for the differential processing of CSs and USs, using as a methodological tool the differential consequences of extinguishing contextual associations on the CS- and US-preexposure effects. In order to avoid arguments concerning definitions of CSs and USs, we use the terms Event 1 (E1) and Event 2 (E2), with E1 being defined as the first stimulus and E2 being defined as the second stimulus presented on each of the subsequent E1→E2 training trials that follow stimulus preexposure treatment. This leaves us free to discuss E1 and E2 independently of their being of high or low biological significance. If the critical difference between CSs and USs is that of biological significance, then the differential effect of extinguishing contextual associations between preexposure and training phases should be reversed if the biological significance (i.e., low or high) of E1 and E2 was reversed (i.e., high or low) in relation to normal conditions. In the case of the CS-preexposure effect, E1 is normally of low biological significance, and extinction of contextual associations does not attenuate the preexposure effect. However, if E1 were biologically significant during the preexposure phase, we would expect extinction of contextual associations to attenuate the E1-preexposure effect. Conversely, in the case of the US-preexposure effect, E2 is normally of high biological significance, and extinction of contextual associations between phases attenuates the preexposure effect. However, if E2 were not biologically significant during the preexposure phase, we would expect extinction of contextual associations between phases to not attenuate subsequent retardation of behavioral control by E2. This is the typical effects of extinguishing contextual assoc...
tions on the E1- and E2-preexposure effects would be reversed.1

In Experiments 1–4, the intensity of the preexposed stimulus was manipulated systematically to determine whether the inherent biological significance of the preexposed stimulus would influence the level of postconditioning responding to the test stimulus observed following extinction of contextual associations between preexposure treatment and training. Experiment 1, in which E1 was of conventional low biological significance, replicated both the basic CS-preexposure (i.e., E1-preexposure) effect (Lubow & Moore, 1959) and the finding of Hall and Minor (1984) that subjects extensively exposed to the training context between preexposure and conditioning still exhibit attenuated responding to E1. Experiment 2 examined this preexposure effect with an E1 of high intensity (i.e., E1 had high biological significance at the time of preexposure) to determine whether pretraining extinction of contextual associations would now reduce the deficit in responding to E1 produced by preexposure to E1 (a result typically seen in the E2-preexposure effect). Experiment 3 (using a sensory preconditioning procedure and an E2 of high biological significance) replicated both the basic US-preexposure (i.e., E2-preexposure) effect (Randich & LoLordo, 1979) and the finding of Randich (1981) that extensive extinction of contextual associations between phases attenuates the E2-preexposure deficit. Experiment 4 (also using a sensory preconditioning procedure) examined the E2-preexposure effect using an E2 of low intensity (i.e., E2 had low biological significance at the time of preexposure) to determine whether pretraining extinction of contextual associations would now fail to attenuate the deficit in responding to E1 produced by preexposure to E2. In order to determine whether the effects observed in Experiments 1–4 depended on the manipulation of biological significance or merely on stimulus intensity, in Experiment 5, the acquired biological significance of the preexposed stimulus (E1) was manipulated by briefly pairing the to-be-preexposed stimulus with the US prior to the preexposure phase.

Experiment 1

The purpose of this experiment was to replicate, with an E1 of conventional low biological significance, both the E1-preexposure effect (Lubow & Moore, 1959) and the findings of Hall and Minor (1984) that extinction of contextual associations between E1 preexposure and conditioning would not serve to attenuate the E1-preexposure effect. The procedure for Experiment 1 is depicted in Table 1. All subjects were first given preexposure to either the target stimulus to be later conditioned (A) or a nontarget stimulus (C). Half of the subjects in the former condition (Group preexposure–context [P–C]) and in the latter condition (Group no preexposure–context [NP–C]) then received extinction of context associations, while the remaining subjects in each condition (Group preexposure–home cage [P–HC] and Group no preexposure–home cage [NP–HC], respectively) remained in the home cage. All subjects then received A→US training and were subsequently tested for suppression of drinking in the presence of A. If the results previously obtained by Lubow and Moore and by Hall and Minor are replicated here, we should observe less suppression of drinking in the presence of A by both Group P–C and Group P–HC than by Groups NP–C and NP–HC, respectively. Specifically, we expected to see a basic E1-preexposure effect (as seen in less suppression of drinking in the presence of A by Group P–HC than by Group NP–HC) and maintenance of this effect despite intervening extensive extinction of associations to the training context (as seen in less suppression of drinking in the presence of A by Group P–C than by Group NP–C). There was no effort to directly assess whether our extinction treatment actually

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Note. P-C = preexposure–context; NP-C = no preexposure–context; P-HC = preexposure–home cage; NP-HC = no preexposure–home cage. A represents the target E1 (Event 1); C represents the nontarget E1; US (unconditioned stimulus) represents footshock; minus sign represents nonreinforcement; HC represents home cage with equivalent handling. The only difference between Experiments 1 and 2 was that Stimuli A and C were auditory cues of moderate intensity in Experiment 1 and of high intensity in Experiment 2.

1 Although we emphasize Hall and Minor's (1984) finding that extensive extinction of associations to the training context between preexposure and conditioning ordinarily does not reduce the CS-preexposure effect, Baker and Mercier (1982) and Wagner (1979) have reported that extinction of contextual associations does reduce the response deficit constituting the CS-preexposure effect. On the one hand, Hall and Minor identified problems with the data of Baker and Mercier and of Wagner. On the other hand, if CSs sometimes do yield effects as do USs (e.g., reduction of the preexposure effect through extinction of contextual associations), this would suggest that there are further commonalities in the ways CSs and USs are processed. Such commonalities would be indirectly supportive of the biological significance view of the difference between CSs and USs rather than the temporal order view, because biological significance is surely a continuum on which CSs vary. Thus, the finding that the CS-preexposure effect might sometimes look like the US-preexposure effect can be accommodated by the biological significance view by assuming that the CS was biologically more significant in those CS-preexposure studies in which extinction of contextual associations reduced the usual response deficit than in those in which it did not. Unfortunately, because the procedures used were quite different among the aforementioned studies, across-experiment comparisons with respect to the relative biological significance of the stimuli used during treatment are difficult.
extinguished associations to the context. If we had found no effect of extinguishing contextual associations, such a measure would have been required to interpret our results. But as our extinction treatment did modulate the effect of stimulus preexposure prior to training (Experiments 2, 3, and 5), we saw little need to further document the effectiveness of our extinction treatment.

Method

Subjects

The subjects were 48 naive, adult male and female rats (Rattus norvegicus). They were bred in our colony from Holtzman Sprague-Dawley stock (Madison, WI). Body weight ranges were 200–350 g for males and 170–200 g for females. Each animal was randomly assigned to one of four groups (n = 12), counterbalanced for sex. Each animal was individually housed in standard, hanging, wire-mesh cages in a vivarium that was maintained on a 16-hr light, 8-hr dark cycle. Experimental manipulations occurred near the midpoint of the light portion of this cycle. Purina Laboratory Chow was freely available in the home cages. One week prior to the start of the study, all subjects were progressively deprived of water. By Day 1 of the experiment, access to water in the home cage was limited to 10 min/day, occurring 18–22 hr prior to any treatment scheduled for the following day. All subjects had been handled three times per week for 30 s, from the time of weaning until the initiation of the study.

Apparatus

Two types of enclosures were used. Enclosure R was a rectangular, clear Plexiglas chamber measuring 22.75 × 8.25 × 13 cm (length × width × height). The floor was constructed of stainless steel rods that were 0.48 cm in diameter and spaced 1.5 cm apart, center to center. The rods were connected by NE-2 neon bulbs, which allowed constant-current footshock to be delivered by means of a high-voltage ac circuit in series with a 1.0-MΩ resistor. Enclosure R was dimly illuminated by a 2-W (nominal at 120 VAC) incandescent house light driven at 56 VAC. The houselight bulb was mounted approximately 30 cm from the center of each enclosure. Six copies of Enclosure R were used, each encased in a separate environmental isolation chest.

Enclosure V was a 30-cm-long box in the shape of a vertical truncated V. The enclosure was 28 cm high and 21 cm wide at the top, narrowing to 5.25 cm wide at the bottom. The floor and 30-cm-long sides were constructed of sheet metal. The floor consisted of two 30-cm-long parallel metal plates, each 2 cm wide, with a 1.25 cm gap between them. A constant-current footshock could be delivered through the floor of the enclosure. The end walls of the enclosure were black Plexiglas, and the roof was clear Plexiglas. Enclosure V was dimly illuminated by a 1/2-W (nominal at 120 VAC) incandescent house light driven at 56 VAC. The houselight bulb was mounted approximately 30 cm from the center of each animal enclosure. Each of six copies of Enclosure V was encased in its own environmental isolation chest. Light entered the enclosure primarily by reflection from the roof of the environmental chest. Because of its opaque walls, Enclosure V was illuminated similarly to Enclosure R despite the presence of brighter light bulbs in Enclosure V.

Enclosures R and V could both be equipped with lick tubes that protruded 2.0 cm from the far end of a cylindrical drinking niche. Each niche was 5 cm in depth and 4.4 cm in diameter and was set into one of the narrow walls 6.5 cm above the chamber floor. The axis of the niche was perpendicular to the enclosure wall and was 2 cm from the wall. An infrared photobeam, 0.3 cm in front of the lick tube, was used to detect when the subjects had their heads inserted into the drinking niche.

Auditory CSs could be presented in Enclosures R and V through three speakers located on each of three interior walls of the environmental chests. The speakers could deliver a complex (3000 and 3200 Hz) tone 10 dB(C) above background and a train of six clicks per second 10 dB(C) above background, designated as Stimuli A and C, counterbalanced within groups. All CS presentations were 5 s in duration. The auditory background was a 74-dB(C) ambient sound level created largely by the ventilation fans of the environmental chests. All USs were 1.5-mA footshocks of 0.5 s duration, presented at the offset of the CS.

Procedure

Acclimation. On Day 1, all animals were acclimated to the conditioning chamber. Enclosures R or V, counterbalanced within groups, served as the experimental context for all phases of the study. During this 60-min session, lick tubes were available to the subjects.

EI preexposure. Prior to this phase of the experiment, all lick tubes were removed from the chambers. On each of Days 2–5, Groups P-C and P-HC received thirty 5-s nonreinforced presentations of A (the target stimulus), while Groups NP-C and NP-HC received, for control purposes, thirty 5-s nonreinforced presentations of C (a non-target stimulus, i.e., no preexposure to A). The mean intertrial interval was 2 min with a range of 1–3 min.

Extinction of contextual associations. On Days 6–10, Group P-C and Group NP-C received 120 min of exposure to the conditioning chamber. On these days, Groups P-HC and NP-HC remained in the home cage but received equivalent handling.

Conditioning. On Day 11, all subjects received four reinforced presentations of A. Footshock onset occurred at CS termination. These pairings occurred at 10, 20, 37, and 50 min into a 60-min session.

Reacclimation. Prior to reacclimation, lick tubes were returned to all chambers. On Days 12–14, all subjects were given 60 min of exposure to the conditioning chamber in order to restore a baseline level of drinking prior to testing.

Testing. On Day 15, all subjects were tested for suppression of drinking in the presence of A during a 16-min test session. Animals were placed in their chambers and were permitted to drink. Upon completion of 5 cumulative seconds of drinking after placement in the chamber, the test CS was presented and the time to complete an additional 5 cumulative seconds of drinking in the presence of A was recorded. Any animal that took longer than 60 s to complete the first 5 s of drinking (i.e., prior to CS onset) was to be eliminated from all statistical analyses in this and all subsequent studies. In Experiment 1, no animals met this criterion. Three subjects from each group were eliminated from all analyses because of experimenter error on the test day.

Lick suppression data ordinarily yield distributions with a strong positive skew. In order to better approximate a normal distribution and thereby justify the use of parametric statistics, a log (base 10) transformation was performed on each suppression score. An alpha level of .05 was adopted for all tests of statistical significance.

Results and Discussion

The present experiment demonstrated that with an EI of conventional low biological significance (a) the EI-
preexposure effect was obtained with our parameters and (b) extensive extinction of contextual associations between the E1-preexposure and conditioning phases resulted in no significant attenuation of the E1-preexposure effect (see Figure 1), thereby replicating the findings of Hall and Minor (1984). The following analyses support these observations.

A 2 × 2 analysis of variance (ANOVA)—Preexposed Stimulus (A or C) × Context Manipulation (context extinction or home cage)—was conducted on time to complete 5 cumulative seconds of licking in the presence of A on Day 15. A main effect of preexposed stimulus, F(1, 32) = 30.87, was observed, but neither the main effect of context manipulation nor the interaction proved significant. Two planned comparisons were then conducted in order to assess the basic E1-preexposure effect and the effects of posttraining extinction of associations to the context. Group P–HC suppressed licking in the presence of A less than did Group NP–HC, F(1, 32) = 12.61, providing evidence for the E1-preexposure effect with our parameters. Furthermore, Group P–C suppressed licking in the presence of A less than did Group NP–C, F(1, 32) = 18.84, providing evidence for the E1-preexposure effect even after associations to the experimental context had been extinguished.

An alternative interpretation of the present results is that the high suppression in the presence of A by Groups NP–C and NP–HC reflects unconditioned suppression to A and that the lack of suppression to A by Groups P–C and P–HC was a consequence of habituation of the unconditioned response to A that occurred during preexposure treatment. However, this interpretation is implausible because (a) A was an auditory stimulus of moderate intensity and (b) in unpublished research in our laboratory, A has not been observed to elicit more than a few seconds of unconditioned suppression of drinking when it is tested without prior pairings with the US. Moreover, an interpretation in terms of unconditioned responding is unable to explain the pattern of results observed in Experiment 2.

Analysis of the time required of each subject to complete 5 cumulative lick seconds prior to CS onset on Day 15 found no significant differences between groups. This suggests that following reacclimation there was no difference between groups in fear of the context (p > .10). Thus, the observed between-groups differences in responding to the test stimulus likely reflected differences in the CS–US association as opposed to differences in fear of the context. This lack of differential fear of the context was also observed in all of the subsequent studies in this series.

Experiment 2

Experiment 2 sought to determine whether attenuation of the E1-preexposure effect, as a result of extinction of contextual associations between the preexposure and conditioning phases, occurs when the target E1 has inherent biological significance at the time of E1 preexposure. E1 was presented at a more intense level than in Experiment 1 (30 dB rather than 10 dB above background) because, presumably, the greater the intensity of a stimulus, the higher its inherent biological significance will be. If biological significance is a critical component in determining whether a cue will be processed as a CS or US, then using an E1 of inherently high biological significance should result in a pattern of results typically seen with the US-preexposure effect (i.e., attenuated responding to the target E1 without extinction treatment, and unattenuated responding to the target E1 with extinction treatment between phases). Because the only difference between Experiments 1 and 2 was that E1 was more intense in Experiment 2, the group designations were the same (i.e., P–C, NP–C, P–HC, and NP–HC).

Method

Subjects and Apparatus

The subjects were 48 naive, adult male and female rats of Sprague-Dawley descent. Body weight ranges were 250–375 g for males and 190–230 g for females. Subjects were randomly assigned to one of four groups (n = 12), counterbalanced for sex.
Animal care and maintenance as well as the apparatus were the same as in Experiment 1.

Procedure

Experiment 2 was an exact replication of Experiment 1 (see Table 1) except that the intensity of El (i.e., A and C) was raised from 10 to 30 dB(C) above background. One subject from each of Groups P-HC and NP-C was eliminated from all subsequent analyses because of an equipment failure.

Results and Discussion

With an El of inherent biological significance (i.e., high intensity), the El-preexposure effect was again observed in subjects that did not receive extinction of contextual associations between preexposure and conditioning phases. However, subjects that received extensive extinction of contextual associations between the preexposure and conditioning phases did not exhibit the deficit in responding that ordinarily arises from El preexposure (see Figure 2). This latter finding was not observed in Experiment 1, in which El was not biologically significant during stimulus preexposure. These observations are consistent with the pattern of results obtained by Randich (1981) with the US-preexposure effect, in which the preexposed stimulus is normally biologically significant but serves as El rather than El. The following analyses support these conclusions.

A $2 \times 2$ ANOVA—Preexposed Stimulus (A or C) $\times$ Context Manipulation (context extinction or home cage)—was conducted on the lick suppression data from Day 15. A main effect of preexposed stimulus, $F(1, 42) = 15.41$, and a Preexposed Stimulus $\times$ Context Manipulation interaction, $F(1, 42) = 4.75$, were observed. Two planned comparisons were then conducted in order to assess the basic El-preexposure effect and the impact of extinction of contextual associations following El preexposure on this basic effect. Group P-HC suppressed licking in the presence of A less than did Group NP-HC, $F(1, 42) = 18.48$, providing evidence for the El-preexposure effect. Additionally, attenuation of the deficit in responding to A as a result of extinction of contextual associations was seen in the greater responding by Group P-C in relation to that of Group P-HC, $F(1, 42) = 5.02$. The El-preexposure effect was not observed when associations to the experimental context were extinguished between preexposure and conditioning, as was indicated by the similar suppression of drinking by A in Groups P-C and NP-C, $F(1, 42) = 1.52$.

Importantly, the low responding to A by Group P-HC indicates that the high responding observed in Group P-C was not an unconditioned response resulting from A’s high intensity. Although A was presumably of high biological significance by virtue of its high auditory intensity in this experiment (i.e., unpublished studies found that it elicited a strong startle response over repeated presentations in a startle chamber), preliminary research had determined that it did not suppress drinking after its first few presentations unless it had been paired with a US. (The data from Group P-HC in Experiment 3 further confirm this conclusion.) This apparent habituation of unconditioned suppression of drinking by the intense A stimulus is not problematic for the assertion that A is of high biological significance, because our view of the importance of the preexposed stimulus's biological significance with respect to the consequences of extinction of contextual associations between preexposure and conditioning phases focuses on the biological significance of the preexposed stimulus at the time of preexposure treatment.

Experiment 3

Experiment 3 was designed to replicate the basic E2-preexposure effect (Randich & LoLordo, 1979) and the findings of Randich (1981) that the E2-preexposure effect can be attenuated with extinction of context associations between the preexposure and conditioning phases. In order to be able to manipulate the inherent biological significance of the preexposed E2 stimulus, the present experiment used a sensory preconditioning procedure. In a typical E2-preexposure experiment, a biologically significant US is
presented repeatedly prior to conditioning. The problem with this procedure for the present series is that it is difficult to make a conventional US (e.g., a footshock) biologically insignificant by reducing its intensity and still obtain observable behavior that could be used to assess the effects of preexposure. Therefore, the procedure we adopted involved using as E2 a high-intensity auditory stimulus (presumably of high inherent biological significance) in Experiment 3 or a moderate intensity auditory stimulus (presumably of low inherent biological significance) in Experiment 4. After the preexposure (to stimulus A) phase, the context extinction phase, and the sensory preconditioning (B→A) phase (which was our critical conditioning phase with A serving as E2), we used the second phase of a sensory preconditioning procedure to make A capable of suppressing drinking; this was done by pairing B with a footshock (see Table 2). Importantly, Cole, Barnet, and Miller (1995) previously demonstrated that B→A trials prior to B→US trials (i.e., backward sensory preconditioning) results in appreciable responding to A. Here, we used their finding as a tool for investigating the role of the inherent biological significance of E2 (surrogate US) on the effects of extinguishing contextual associations following B preexposure.

The intent of Experiments 3 and 4 was to investigate the E2-preexposure effect. As we merged the E2-preexposure procedure with a sensory preconditioning procedure, one might question whether we were truly looking at the conventional E2-preexposure effect. However, in anticipation of the outcome of Experiment 3, the fact that Experiment 3 successfully replicated both the E2-preexposure effect and the attenuation of that effect as a result of extinction of contextual associations between phases (both of which are ordinarily seen with a biologically significant E2) suggests that the introduction of the sensory preconditioning procedure did not alter the nature of the E2-preexposure effect provided E2 was of high biological significance (e.g., Experiment 3).

For control purposes, half of the subjects were preexposed to a nontarget control stimulus (C rather than A), and half of the subjects in each of these two preexposure conditions (A and C) did not have associations to the training context extinguished between the preexposure and conditioning phases (i.e., they remained in the home cage). The sensory preconditioning phase (i.e., B→A) served as the conditioning treatment that might be impaired by preexposure to A. In the last phase, all subjects received reinforced presentations of B (i.e., B→US). All animals were then tested for suppression of drinking in the presence of A. Thus, the study used the same basic 2 × 2 factorial design that had been used in Experiments 1 and 2, with the two variables being preexposure (P) versus no preexposure (NP) and context (C) extinction versus home cage (HC) between the preexposure and conditioning phases. Consequently, as in the prior experiments, the four group designations were P-C, P-HC, NP-C, and NP-HC.

As a result of our using a sensory preconditioning procedure in conjunction with an E2-preexposure procedure, we were able to manipulate the inherent biological significance of E2. In Experiment 3, both E2s (A and C) were auditory cues presented at a high intensity in order to make them biologically significant throughout the experiment, as would have been the case if conventional USs rather than auditory cues had been used as E2s. As a result, we expected to observe the basic E2-preexposure effect, as well as attenuation of that effect in subjects that were extensively exposed to the training context between the preexposure and conditioning phases.

**Method**

**Subjects and Apparatus**

The subjects were 48 naive, adult male and female rats of Sprague-Dawley descent. Body weight ranges were 350-425 g for males and 180-295 g for females. Subjects were randomly assigned to one of four groups (n = 12), counterbalanced for sex. Animal care and maintenance as well as the apparatus were the same as in Experiments 1 and 2. However, in addition to the click and tone stimuli used in Experiments 1 and 2, a white noise, delivered by a 45-watt speaker mounted on the interior wall of the chamber, was also used. The tone and the white noise, both presented at 30 dB(C) above background, served as Stimuli A and C, counterbalanced within groups. The click, presented at 10 dB(C) above background, served as Stimulus B.

**Table 2**

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<td>P-C</td>
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<td>Context extinction</td>
<td>B→A</td>
<td>B→US</td>
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<td>NP-C</td>
<td>C-</td>
<td>Context extinction</td>
<td>B→A</td>
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<tr>
<td>P-HC</td>
<td>A-</td>
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<td>B→A</td>
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<tr>
<td>NP-HC</td>
<td>C-</td>
<td>HC</td>
<td>B→A</td>
<td>B→US</td>
<td>A-</td>
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*Note.* P-C = preexposure-context; NP-C = no preexposure-context; P-HC = preexposure-home cage; NP-HC = no preexposure-home cage. A = target E2 (Event 2, surrogate unconditioned stimulus [US]) during sensory preconditioning (SPC); C represents the nontarget E2; B represents the conditioned stimulus used for sensory preconditioning training; US represents footshock; minus sign represents nonreinforcement; HC represents home cage with equivalent handling. The only difference between Experiments 3 and 4 was that Stimuli A and C were auditory cues of high intensity in Experiment 3 and of moderate intensity in Experiment 4. B was an auditory cue of modest intensity in both experiments.
Procedure

Acclimation. On Day 1, all animals were acclimated to the conditioning chamber. During this 60-min session, lick tubes were available to the subjects.

E2 preexposure. Prior to this phase, all lick tubes were removed from the chambers. Subjects were then divided into two conditions. On each of Days 2–5, subjects in Groups P-C and P-HC received thirty 5-s nonreinforced presentations of A (E2), while subjects in Groups NP-C and NP-HC received thirty 5-s nonreinforced presentations of C (i.e., no preexposure to A). The mean intertrial interval was 2 min with a range of 1–3 min.

Context extinction. On Days 6–10, half of the subjects from Condition P (i.e., Group P-C) and half of the subjects from Condition NP (i.e., Group NP-C) received 120 min of exposure to the conditioning chamber. On these days, the remaining subjects (i.e., Groups P-HC and NP-HC, respectively) remained in the home cage but received equivalent handling.

Sensory preconditioning. On each of Days 11–15, all subjects received four presentations of B followed immediately by A (i.e., B→A). These pairings constituted the critical conditioning that might be affected by stimulus preexposure. Session length was 60 min. Trials were initiated 10, 20, 37, and 50 min into the session.

Conditioning. On Day 16, all subjects received four reinforced presentations of B during a 60-min session (i.e., B→US). Trials were initiated 10, 20, 37, and 50 min into the session. These pairings were intended to imbue B with associations that would suppress drinking and that we expected would be transferred to A through a B→A sensory preconditioned association.

Reacclimation. Prior to this phase of the experiment, lick tubes were reininserted into the chambers. On Days 17 and 18, subjects were placed in the conditioning chamber for 60 min per day with no CSs or USs programmed to occur.

Testing. On Day 19, all subjects were tested for suppression of drinking in the presence of A during a 16-min test. The procedure for testing was the same as in Experiment 1. One subject from Group NP-HC was eliminated from the following analyses because of the subject's having failed to meet the aforementioned criterion of taking less than 60 s to complete 3 cumulative seconds of drinking prior to onset of A on Day 19.

Results and Discussion

A basic E2-preexposure effect (with a US surrogate substituting for a conventional US) was observed. Additionally, extensive extinction of contextual associations between the stimulus preexposure and (sensory pre-) conditioning phases was seen to attenuate that effect. These outcomes are the same as we observed in Experiment 2, in which the preexposed stimulus was also of high inherent biological significance, but there the preexposed stimulus was E1 rather than E2.

A 2 × 2 ANOVA—Preexposed Stimulus (A or C) × Context Manipulation (context extinction or home cage)—conducted on the lick suppression data from Day 19 verified these conclusions. Group means are illustrated in Figure 3. A main effect of preexposed stimulus, F(1, 43) = 13.40, a main effect of context manipulation, F(1, 43) = 9.23, and a Preexposed Stimulus × Context Manipulation interaction, F(1, 43) = 9.80, were observed. Planned comparisons were then conducted in order to assess the basic E2-preexposure effect and the consequences of extensive context preexposure between preexposure and (sensory pre-) conditioning phases on this effect. Group P-HC suppressed licking in the presence of A less than did Group NP-HC, F(1, 43) = 22.85, providing evidence for the E2-preexposure effect. Moreover, the E2-preexposure effect was not observed when associations to the experimental context were extinguished between preexposure and conditioning, as is indicated by the similar suppression of drinking in the presence of A by Groups P-C and NP-C, F(1, 43) = 0.13. Additionally, attenuation of the effect of stimulus preexposure was seen as a result of our extinguishing contextual associations; this is evident in the greater suppression of licking in the presence of A by Group P-C than by Group P-HC, F(1, 43) = 19.41. As in Experiment 2, the low responding to the test stimulus by Group P-HC indicates that the high responding observed in Group P-C was not an unconditioned response to the test stimulus resulting from its high intensity.
Experiment 4

Experiment 4 used the same procedure as Experiment 3 in order to determine whether the attenuation of the E2-preexposure effect seen in Experiment 3 as a result of extinction of contextual associations would be altered if E2 (i.e., A) were biologically neutral. In order to provide an E2 of low biological insignificance, A was presented at an appreciably less intense level than in Experiment 3. If biological significance is a critical factor in determining whether a stimulus is processed as a CS or US, preexposure to a biologically neutral E2 should result in a pattern of results typically seen with the conventional E1-preexposure effect (i.e., attenuated responding to the target CS with or without context extinction between phases). Because the only difference between Experiments 3 and 4 was that E2 was less intense in Experiment 4, the group designations were the same (i.e., P–C, NP–C, P–HC, and NP–HC).

Method

Subjects and Apparatus

The subjects were 48 naive, adult male and female rats of Sprague-Dawley descent. Body weight ranges were 350–420 g for males and 175–295 g for females. Subjects were randomly assigned to one of four groups (n = 12), counterbalanced for sex. Animal care and maintenance as well as the apparatus were the same as in the prior experiments.

Procedure

Experiment 4 was an exact replication of Experiment 3 (see Table 2) except that the intensities of A and C were lowered from 30 to 10 dB(C) above background.

Results and Discussion

With an E2 of low biological significance (i.e., moderate intensity), the basic US-preexposure effect was still obtained, but no attenuation of this effect as a result of extensive extinction of contextual associations between preexposure and (sensory pre-) conditioning phases was observed. These outcomes are the same as those of Experiment 1, in which the preexposed stimulus was also of low inherent biological significance, but there the preexposed stimulus was E1 rather than E2.

A 2 × 2 ANOVA—Preexposed Stimulus (A or C) × Context Manipulation (context extinction or home cage)—was conducted on the lick suppression data from Day 19. Group means are illustrated in Figure 4. Only a main effect of preexposed stimulus, F(1, 44) = 40.97, was observed. Planned comparisons were then conducted in order to assess the basic E2-preexposure effect and the consequences of extensive extinction of contextual associations between the preexposure and (sensory pre-) conditioning phases on this effect. Group P–HC suppressed licking in the presence of A less than did Group NP–HC, F(1, 44) = 14.12, providing evidence for the E2-preexposure effect. Moreover, Group P–C suppressed licking less than did Group NP–C, F(1, 44) = 28.01, providing evidence for the E2-preexposure effect even after associations to the training context had been extinguished. Additionally, no attenuation of this response deficit was seen to A as a result of extinction of contextual associations; responding to A was highly similar in Groups P–C and P–HC, F(1, 44) = 0.14.

Experiment 5

As the same variable (i.e., intensity) was manipulated throughout Experiments 1–4, it might be more parsimonious to interpret the results of Experiments 1–4 in terms of stimulus intensity rather than the broader concept of biological significance. In order to give added meaning to the concept of biological significance, we must demonstrate convergent results using distinctly different manipulations of biological significance. We previously defined high biological significance as being either inherent to the cue (e.g., food, water, pain, sex, or intense auditory cues) or acquired as a result of a neutral cue being paired with a stimulus of
inherent biological significance. As we identify biological significance with control of behavior, control may arise from unconditioned factors such as stimuli being of high physical intensity or it may be acquired. Although Dennis- 
ton et al. (in press) have already demonstrated that these two sources of biological significance have similar effects on cue competition, it remains to be determined whether they have similar effects on the consequences of context extinction for stimulus preexposure effects. Experiments 1–4 of the present series examined the effect of biological significance on the E1- and E2-preexposure effects by manip- 
utating only the inherent biological significance of the preexposed event through the physical intensity of that event. Consequently, in Experiment 5 we sought to manipulate the acquired biological significance of a stimulus prior to preexposure treatment. Experiment 5 sought to extend the concept of biological significance beyond physical intensity by manipulating the acquired biological significance of E1 in an E1-preexposure procedure (see Table 3).

In Experiment 5, the acquired biological significance of E1 was manipulated by giving reinforced presentations of E1 prior to the preexposure phase. Thus, groups high-preexposure–context (H-P–C), high-preexposure–home cage (H-P–HC), and high-no preexposure–context (H-NP–C) received reinforced presentations of A (Event 1) in Phase 1, which made A into a biologically significant stimulus prior to preexposure to A. During this phase, Groups low-preexposure–context (L-P–C) and low-no preexposure–context (L-NP–C) received reinforced pre- 

tsentations of B, which was irrelevant to the study but controlled for nonassociative effects. As a result, the target event (A) was not biologically significant for the subjects in the low condition. During Phase 2 (the E1-preexposure phase), the three high condition groups (H-P–C, H-P–HC, H-NP–C) and Group L-P–C received a nonreinforced presentation of A, while Group L-NP–C received nonreinforced presentations of C. For the preexposed groups (H-P–C, H-P–HC, and L-P–C), the preexposed stimulus was the stimulus that would later be conditioned, whereas for the no preexposure groups (H-NP–C and L-NP–C), the preexposed stimulus was a stimulus different from that which would later be conditioned. In Phase 3 (the extinction phase), all groups except Group H-P–HC received extinction of the training context. Finally, in Phase 4 (the conditioning phase), Groups H-P–C, H-P–HC, L-P–C, and L-NP–C received reinforced presentations of A, while Group H-NP–C received reinforced presentations of C. Then, all subjects were tested for suppression to A. Thus, Group H-P–C was our critical experimental group, having received a biologically significant E1 prior to preexposure and conditioning. If acquired biological significance has properties similar to those of inherent biological significance, then we would expect to see vigorous responding to A in this group, replicating the results of Experiment 2, which found that context extinction attenuated the E1-preexposure deficit, provided E1 was biologically significant during preexposure. Group L-P–C received identical treatment as Group H-P–C, except that E1 was of low biological significance for Group L-P–C. This group should not show attenuation of the E1-preexposure deficit as a result of context extinction (i.e., this group constitutes a replication of the conventional E1-preexposure effect and of Group P–C from Experiment 1). Additionally, Group H-P–HC did not receive extinction of the training context and therefore should not show any attenuation from this deficit. Group H-NP–C was included to demonstrate that any drink-suppressing potential of A that resulted from Phase 1 A→US treatment had been extinguished as a consequence of the preexposure phase of the study. Presumably, this extinction of any drink- 

 suppression potential acquired during Phase 1 did not fully attenuate the acquired biological significance of A gained during this Phase 1. Finally, Group L-NP–C was preex- 

posed to a control stimulus (B) and received simple conditioning of E1 (A), thereby serving as a basis of comparison for the E1-preexposure conditions.

Method

Subjects and Apparatus

The subjects were 120 naive, adult male and female rats of Sprague-Dawley descent. Body weight ranges were 315–460 g for males and 185–300 g for females. Subjects were randomly assigned to one of five groups (n = 24), counterbalanced for sex. Animal care and maintenance as well as the apparatus were the same as in Experiments 1–4. However, the click, tone, and white

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Experimental Design for Experiment 5 (E1 Preexposure)</th>
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<tr>
<td>Group</td>
<td>Acquisition of biological significance</td>
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<td>L-P–C</td>
<td>B–US</td>
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Note. H-P–C = high-preexposure–context; H-P–HC = high-preexposure–home cage; H-NP–C = high-no preexposure–context; L-P–C = low-preexposure–context; L-NP–C = low-no preexposure–context. A represents the target E1 (Event 1); B and C represent control stimuli; US (unconditioned stimulus) represents footshock; minus sign represents nonreinforcement; HC represents home cage with equivalent handling. All stimuli were of moderate intensity.
noise stimuli described previously served as Stimuli A, B, and C, fully counterbalanced within groups. All stimuli were presented at 10 dB(C) above background throughout the experiment.

Procedure

**Acclimation.** On Day 1, all animals were acclimated to the conditioning chamber. During this 60-min session, lick tubes were available to the subjects.

**Acquisition of biological significance.** Prior to this phase, lick tubes were removed from all chambers. On Day 2, Groups H-P-C, H-P-HC, and H-NP-C received four reinforced presentations of A, while Groups L-P-C and L-NP-C received four reinforced presentations of B. Session duration was 60 min, and stimulus presentations occurred at 10, 20, 37, and 50 min into the session.

**EI preexposure.** On Days 3-10, Groups H-P-C, H-P-HC, H-NP-C, and L-P-C received thirty 5-s nonreinforced presentations of A, whereas Group L-NP-C received thirty 5-s nonreinforced presentations of C. Session duration was 60 min, and the intertrial interval averaged 2 min, ranging from 1 to 3 min. Notably, this treatment now served two purposes. First, it was the critical preexposure treatment that was expected to produce subsequent retardation of conditioned responding to EI when EI was later paired with the US; second, it was intended to extinguish the response eliciting potential of A established during the first phase of treatment. Control groups were included to ensure that this preexposure treatment did in fact achieve these ends.

**Context extinction.** On Days 11-15, Groups H-P-C, H-NP-C, L-P-C, and L-NP-C received extinction of the training context during daily 2-hr sessions. Group H-P-HC remained in the home cage during this phase but received equivalent handling.

**Conditioning.** On Day 16, subjects in Groups H-P-C, H-P-HC, L-P-C, and L-NP-C received four reinforced presentations of A, while Group H-NP-C received four reinforced presentations of C. Session duration was 60 min, and stimulus presentations occurred at 10, 20, 37, and 50 min into the session.

**Reacclimation.** Prior to this phase, lick tubes were reintroduced into the conditioning chambers. On Days 17-18, subjects were reacclimated to the chambers during 60-min sessions each day with no CSs or USs programmed to occur.

**Testing.** On Day 19, all subjects were tested for suppression of drinking in the presence of A during a 16-min test. The procedure for testing was the same as in all previous experiments. One subject from Group L-P-C and one subject from Group L-NP-C were excluded from the following analysis because of illness detected prior to testing.

Results and Discussion

A basic EI-preexposure effect was observed. In addition, this effect was alleviated when context extinction was interposed between preexposure and conditioning phases, but only if EI had been made biologically significant prior to preexposure. These results are consistent with those of Experiments 1 and 2, except that in the current experiment, biological significance was acquired rather than inherent.

A one-way ANOVA across all groups was conducted on the lick suppression data from the test day, which revealed a main effect of group, F(4, 113) = 10.53. Group means are illustrated in Figure 5. Planned comparisons were then conducted in order to assess the basic EI-preexposure effect and the consequence of context extinction and biological significance on this effect. Group L-P-C suppressed less to A than did Group L-NP-C, F(1, 113) = 23.85, thereby demonstrating in Group L-P-C the basic EI-preexposure deficit with an EI of low biological significance despite extensive exposure to the context between preexposure and the subsequent conditioning trials. No groups lacking context extinction for which A had low biological significance at the time of preexposure were included in this study, as such groups would have been identical to Groups P-HC and NP-HC of Experiment 1. Note that Groups L-P-C and L-NP-C replicated Groups P-C and NP-C of Experiment 1 in procedure and yielded highly similar behavior. In the present study, the EI-preexposure deficit was eliminated as a result of context exposure between preexposure and the subsequent conditioning trials when A was made biologically significant prior to preexposure (i.e., Group H-P-C) but not when A was of low biological significance (i.e., Group L-P-C), F(1, 113) = 11.15. However, the greater responding observed in Group H-P-C might reflect the associative strength of A acquired directly during Phase 1 (acquisition of biological relevance) rather than during the
critical conditioning trials that immediately precede testing (see Table 3). But Group H-FP-C suppressed less to A than did Group H-FP-C, F(1, 113) = 15.12, indicating that the increased responding to A in Group H-FP-C was due to the critical A-US pairings immediately prior to testing rather than to greater conditioned suppression to A as a result of Group H-FP-C receiving A-US trials prior to nonreinforced preexposure to A. In other words, A’s control over lick suppression was greatly attenuated as a result of the extinction of A during the preexposure phase. Additional evidence of the impact of biological significance can be found through comparisons of groups that received preexposure to an E1 that had previously acquired biological significance. The potential of context extinction to attenuate the E1-preexposure effect can be seen in the greater responding to A by Group H-FP-C than by Group H-FP-HC, F(1, 113) = 4.53.

The results of Experiment 5 are consistent with those of Experiments 1 and 2, despite the different methods used for manipulating biological significance. Thus, the results obtained in Experiments 1 and 2 are not simply reliant on the physical intensity of the events but rather on the biological significance of E1 at the time of preexposure treatment, regardless of whether the biological significance was inherent or acquired through pairings with a stimulus of inherent biological significance. Furthermore, the results of Experiment 5 argue against any concern that the results of Experiments 1–4 arose from nonassociative factors such as habituation and sensitization that differed as a function of stimulus intensity.

General Discussion

The present series of experiments sought to examine the factors that determine whether a stimulus will be processed like a conventional CS or a conventional US. The CS- and US-preexposure procedures were chosen as tools to explore this question because of the differential outcomes obtained when subjects are extensively exposed to the training context between the preexposure and conditioning phases. Typically, context exposure (presumably, extinguishing the association between the context and the preexposed stimulus) between these phases results in attenuation of the response deficit constituting the US-preexposure effect (Randich, 1981) but not the response deficit constituting the CS-preexposure effect (Hall & Minor, 1984). The data presented here demonstrate that by manipulating the biological significance (operationalized in this research by stimulus intensity in Experiments 1–4 and by prior associations in Experiment 5) of the preexposed stimulus, these patterns of results can be obtained independently of whether the preexposed stimulus is the first or second event in the subsequent E1→E2 pairings. We first (Experiment 1) used an E1 of low biological significance and replicated the pattern of results obtained by Hall and Minor (1984) with the CS-preexposure effect. However, when we used an E1 of high biological significance (Experiment 2), we saw attenuation of the resultant response deficit when extensive extinction of contextual associations occurred between phases, a pattern of results that typically is seen with the US-preexposure procedure. Similarly, using an E2 of high biological significance (Experiment 3), we replicated the pattern of results observed by Randich (1981) with the US-preexposure effect. However, when we used an E2 of low biological significance (Experiment 4), we did not see any attenuation of the deficit in responding to the target stimulus when extensive extinction of contextual associations occurred between phases, a pattern of results that typically is seen with the CS-preexposure procedure. Experiment 5 expanded the concept of biological significance beyond stimulus intensity. This experiment replicated the pattern of results obtained in Experiments 1 and 2 with the E1-preexposure effect but manipulated the acquired (rather than inherent) biological significance of E1. Thus, the impact of extensive extinction of contextual associations appears to depend on the biological significance (either inherent or acquired) of the preexposed stimulus and not on whether it is the signaling or the signaled stimulus (i.e., E1 or E2). Apparently, stimuli of high biological significance yield a preexposure effect that is more readily attenuated by pretraining extinction of context associations than is the case with stimuli of low biological significance.

Consistent with our finding that the inherent biological significance of a stimulus is a critical determinant of information processing, Schur and Lubow (1976) reported that the magnitude of the CS-preexposure effect is a direct function of the intensity of the preexposed stimulus. Their results suggest that as the preexposed CS is made biologically more significant (i.e., more intense), the CS-preexposure effect is enhanced. Other studies (Randich, 1978; Renslow, 1974) have looked at the effects of US intensity on the US-preexposure effect. However, a problem exists in these studies in that they administered different US intensities across subjects in the preexposure phase and then used a single intensity for all subjects in the conditioning phase. This invites explanations based on differences in generalization decrement between phases. A better strategy would be to vary the intensities during both phases, but this might result in differences in asymptotic responding between groups. The present study circumvented this problem by using a sensory preconditioning procedure and making the ultimate US (i.e., footshock) identical for all subjects. However, the intensity of E2 (the surrogate for the US) was varied, thereby allowing us to compare the effects of the intensity of E2 on the E2-preexposure effect.

Although this series of experiments was designed explicitly to determine the basis of differential information processing of E1 and E2, the present results also have implications for our understanding of the E1- and E2-preexposure effects. Theoretical explanations for the US-preexposure effect have suggested both nonassociative and associative mechanisms. Nonassociative explanations assume that preexposure to the US reduces the subject’s responsiveness to subsequent presentations of that US (e.g., habituation). On the other hand, associative views posit that the deficit is due to a subliminal blocking of the US association by a context-US association previously formed.
during US preexposure. The associative view is more widely accepted because either a context shift or extinction of contextual associations (Baker, Mercier, Gabel, & Baker, 1981; Randich, 1981) between preexposure and conditioning ordinarily attenuates the US-preexposure effect. Because blocking is widely viewed as an acquisition deficit (e.g., Rescovia & Wagner, 1972), the US-preexposure effect is commonly regarded as reflecting a failure to acquire the CS-US association. However, Matzel, Brown, and Miller (1987) have reported that extensive extinction of associations to the training context following the CS-US pairings restores responding to the CS. Additionally, J. S. Miller, Jagielo, and Spear (1993) observed spontaneous recovery from the US-preexposure deficit. Thus, there is now reason to doubt the acquisition-failure account of the US-preexposure effect.

Similar controversy has surrounded explanations of the CS-preexposure effect (i.e., latent inhibition). Several theories have proposed (albeit through different mechanisms) that latent inhibition, like the US-preexposure effect, results from the failure of subjects to acquire the CS-US association (see, e.g., Lubow, Schuur, & Riklin, 1976; Mackintosh, 1975; Pearson & Hall, 1980). Each of these models assumes that repeated presentations of a CS prior to CS-US pairings decreases the ability to associate the CS with the US is impaired. However, studies that have demonstrated an increase in responding to the target CS as a result of various posttraining manipulations (other than further CS-US pairings) provide evidence that latent inhibition is at least in part the result of a failure to express acquired information rather than a failure to actually acquire that information. Examples of manipulations that have attenuated the latent inhibition effect include reminder treatments (e.g., presenting the US alone outside of the training context; Kasprow, Catterson, Schachtman, & Miller, 1984), spontaneous recovery (i.e., delayed testing; Aguado, Symonds, & Hall, 1994; Kraemer, Randall, & Carbary, 1991), and extinction of contextual associations following the CS-US pairings (Graham, Barnett, Gunther, & Miller, 1994). Any sort of posttraining attenuation of latent inhibition without further training is problematic for attentional explanations of latent inhibition that assume (a) that the CS-preexposure effect arises from a loss of attention and (b) that attention is necessary for acquisition.

However, if attention to the CS during training is assumed to facilitate later expression of learning rather than facilitate acquisition per se, these observations can still be reconciled with an attentional view (Kasprow et al., 1984; Townsend, 1974). This variant of attentional theory views attention during training as having at least part of its consequences on subsequent retrieval and response-generation processes. Extinction of associations to the context between preexposure and conditioning phases should have little effect, which is what is observed after preexposure to stimuli of low biological significance. Thus, this postacquisition attentional view is compatible with the results of Experiments 1 and 4, in which the preexposed stimulus was of low biological significance, but not with the results of Experiments 2, 3, and 5, in which the preexposed stimulus was of high biological significance. Perhaps when the preexposed stimulus is of high biological significance (Experiments 2, 3, and 5), attention to the preexposed cue is less apt to wane with mere exposure, and instead a context-stimulus or stimulus-context association may be formed that in turn modulates the acquisition (Wagner, 1981) or expression (Graham et al., 1994) of the CS-US association.

There are two models that assume that the CS-preexposure and US-preexposure effects arise from associations formed between the preexposed stimulus and the context. One is Wagner's (1981) SOP model and the other is R. R. Miller and Matzel's (1988a; also see R. R. Miller & Schachtman, 1985) comparator hypothesis, which was applied to the CS-preexposure effect by Graham et al. (1994). Both models predict a decrease in responding to a target stimulus as a result of associations between the preexposed stimulus and the context that are formed during preexposure treatment. In the framework of SOP, during the conditioning session the context primes the representation of a preexposed element into a state that is not conducive to the formation of the CS-US association. One problem with this interpretation is that the CS- and US-preexposure effects is that it cannot explain attenuation of these deficits through the various postconditioning manipulations that we have previously described. A second problem arises from SOP's assumption that CSs and USs are processed equivalently, except for differences that arise because of their temporal order, that is, CSs ordinarily precede USs. However, the present data suggest that conventional CSs and USs are differentially processed as a function of their biological significance rather than as a function of their temporal order.

The comparator hypothesis (R. R. Miller & Matzel, 1988) assumes that responding to a CS is a direct function of the strength of the US-US association and an inverse function of the product of the CS-context and context-US associative strengths. That is, rather than merely responding to the absolute predictive value of the CS, subjects respond to a CS to the degree that it predicts a change in US likelihood. Preexposure to a CS presumably strengthens the CS-context associations, and preexposure to the US presumably strengthens the context-US association, thereby attenuating responding to the CS after either CS or US preexposure, respectively. Thus, the comparator hypothesis is equipped to explain both basic preexposure effects. Additionally, it views these effects as deficits in the expression of associations rather than acquisition deficits, which is consistent with the observed attenuation of the two preexposure effects by the previously described postconditioning manipulations. Moreover, extensive context exposure should extinguish the association between the preexposed stimulus and the context that is formed during preexposure, thereby attenuating the preexposure effect (which is what we observed with events of high biological significance, but not those of low biological significance). Extinction of contextual associations either before or after conditioning should be effective in attenuating the preexposure effect. Graham et al. (1994) have indeed demonstrated that extensive ex-
tinction of contextual associations following the CS-US pairings attenuates the CS-preexposure effect, and Baker and Mercier (1982) and Wagner (1979) have reported that extinction of contextual associations between CS preexposure and training also produces attenuation of the CS-preexposure effect (notably, this is a more difficult to obtain than is the analogous effect with preexposure to biologically significant events; see Hall & Minor, 1984, and Experiment 1 in this article). Thus, there appear to be two mechanisms responsible for retardation of behavioral control because of prior stimulus exposure: attention loss and event-context associations. Most observed preexposure effects likely have components due to each of these two mechanisms, with the contribution from each depending on the biological significance of the stimulus at the time of preexposure. The comparator hypothesis can explain the component of preexposure effects that arises from contextual associations (more prevalent following preexposure to stimuli of high biological significance) but is unable to explain the component of preexposure effects that arises from losses in attention (more prevalent following preexposure to stimuli of low biological significance).

In contrast with Baker and Mercier (1982), Grahame et al. (1994), and Wagner (1979), the present data (Experiment 1) and Hall and Minor (1984) failed to detect any attenuation of the CS-preexposure effect (preexposure to an E1 of low inherent biological significance) as a result of extinction of contextual associations between CS preexposure and the CS-US pairings. Biological significance can provide an account for this seeming conflict at least with respect to the Grahame et al. study. In their experiment, extinction of contextual associations occurred after the CS-US pairings, at which point the CS (and perhaps the context) had already been made biologically significant through pairings with the US. However, in our Experiment 1 (in which extinction of contextual associations did not result in recovery from CS preexposure), extinction of contextual associations occurred after CS preexposure but prior to the CS-US pairings. At this time the CS had not yet been made biologically significant through pairings with the US. Consequently, it is possible that, in order for there to be attenuation of the deficit in responding to the CS, extended extinction of associations to the context must occur after the CS has been made biologically significant to the subject. (This explanation does not address the outcomes of Baker and Mercier and of Wagner, but see Footnote 1.) In contrast, the US-preexposure effect is not ordinarily influenced by this constraint, because USs are ordinarily of inherent biological significance. Thus, the stimulus associated with the context during US preexposure is typically of high biological significance both as well as after the CS-US pairings. Experiment 4 stands as an exception to the ordinary state of the US because E2 in that experiment was of low inherent biological significance. Consistent with this hypothesis, extinction of contextual associations between E2 (the surrogate US) preexposure and conditioning failed to attenuate the deficit in responding to the target stimulus in Experiment 4. Evidence supportive of this explanation can also be seen by looking at the results from Experiments 2 and 5. In these experiments, extinction of contextual associations between preexposure and conditioning likely resulted in attenuation of the E1-preexposure effect because E1 was biologically significant at the time of preexposure and, consequently, was biologically significant at the time of extinction of contextual associations.

There are several alternatives to attentional mechanisms versus contextual associations as possible explanations of why context extinction between preexposure and conditioning has more of an effect with events of high biological significance than with events of low biological significance. For example, extinction of an association between two stimuli proceeds more rapidly when the representation of the absent stimulus is active. Perhaps context exposure activates through association a representation of the preexposed stimulus, but that representation does not stay active for long unless it includes the attribute of high biological significance. If this is correct, the more prolonged activation of biologically significant representations should lead to more rapid extinction of contextual associations to these representations. A testable prediction of this position is that with sufficient (i.e., many) context extinction exposures between preexposure and conditioning, the deleterious effect of stimulus preexposure should be attenuated even when the preexposed stimulus is of relatively low biological significance.

Conclusion

The present data suggest that the information processing of signaling stimuli and of signaled stimuli is quite similar, provided that the biological significance of the stimuli are equivalent. Preexposing either type of stimulus results in a deficit in responding to the target stimulus at test. In addition, extinction of contextual associations between the preexposure and conditioning phases results in similar attenuation of the deficit, provided that the preexposed stimuli are of high biological significance at the time of preexposure. The fact that CSs ordinarily precede USs during the conditioning phase does not appear critical to this attenuation; that is, preexposure effects appear similar regardless of whether the preexposed stimulus is E1 or E2, provided the biological significance of E1 and E2 has been equated. More extensive research needs to be conducted to look closely at the ways in which organisms process different types of stimuli, but in light of the present data, biological significance appears to be an important variable in differentiating conventional CSs and USs.

In the framework of traditional theories of conditioning (e.g., Pavlov, 1927), this conclusion would not seem surprising. In recent years, however, great emphasis has been placed on the importance of a CS as a predictor of a US (e.g., Rescorla & Wagner, 1972). The implication of this is that for a CS to be important to a subject, it must precede the US, thereby focusing attention on the role of temporal order. Surely, within the framework of an ultimate analysis of behavior (i.e., functionally serving survival and reproduction), preparing for future events makes good sense. How-
ever, the apparent logic of this ultimate goal may have caused researchers to confuse proximate processes and ultimate functions of behavior. In our view, prediction of USs (as they enhance survival and reproduction) is the ultimate function of acquiring CS-US associations, but it may not be directly reflected in the proximate learning process. Rather, the learning mechanism may depend on biological significance and contiguity, with these variables controlling a proximate learning mechanism that functionally serves the ultimate goals of survival and reproductive success.

References


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