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Counterconditioning of an Overshadowed Cue Attenuates Overshadowing

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In 3 Pavlovian conditioned lick-suppression experiments, rats received overshadowing treatment with a footshock unconditioned stimulus such that Conditioned Stimulus (CS) A overshadowed CS X. Subjects that subsequently received CS X paired with an established signal for saccharin (CS B) exhibited less overshadowing of the X–footshock association than subjects that did not receive the X–B pairings (Experiment 1). Experiment 2 replicated this effect and controlled for some additional alternative accounts of the phenomenon. In Experiment 3, this recovery from overshadowing produced by counterconditioning CS X was attenuated if CS B was massively extinguished prior to counterconditioning. These results are more compatible with models of cue competition that emphasize differences in the expression of associations than those that emphasize differences in associative acquisition.

An implicit (or explicit) assumption of most theoretical accounts of associative learning is that, in order to support conditioning, an unconditioned stimulus (US) or reinforcer must elicit an unconditioned response. That is, the US must be *biologically significant* to effectively support acquired behavior. However, despite the wide recognition of the importance of the biological significance of the outcome (US or reinforcer), little attention has been devoted to the influence of a conditioned stimulus' (CS) biological significance. In fact, the meaning of biological significance as applied to a CS has been inconsistent. For some researchers, biological significance (or some similar term) has been related to stimulus intensity (e.g., Kamin [1965] concluded that both rate of acquisition and asymptote of conditioned responding increased with the intensity of the CS [also see Hull, 1949]). Other researchers have used biological significance (or some analogous term) to refer to the acquired motivational value of an initially neutral cue (Eisenberger, 1992; Grice, 1948).

In this article, we apply the term biological significance to those stimuli that can produce a strong response, either inherently or through an associative history. Thus, our definition of biological significance combines both prior uses of the phrase, *inherent* and *acquired* biological significance. Stimuli that are of inherent biological significance unconditionally control responding, for example, food, sex, painful stimuli, and intense stimuli. Initially neutral stimuli can acquire biological significance through pairings with stimuli that are inherently biologically significant. For example, a CS that evokes conditioned responding has acquired biological significance through pairings with a US. Presumably, biological significance varies along a continuum from low (e.g., stimuli that elicit mild orienting responses that readily habituate) to high (e.g., stimuli that elicit vigorous responding that does not readily habituate, and indeed may sensitize). For purposes of discussion, we refer to stimuli having high biological significance as biologically significant stimuli and stimuli having low biological significance as biologically nonsignificant stimuli. This research is concerned with how the biological significance of a CS influences cue competition, specifically overshadowing. Overshadowing is a phenomenon, discovered by Pavlov (1927), in which an overshadowed stimulus elicits less vigorous conditioned responding after being paired with a US in compound with another (usually more salient) overshadowing stimulus, than after being paired with the US in the absence of the other stimulus.

A biologically significant stimulus not only controls strong responding (and supports responding to stimuli paired with it), but it also likely receives privileged activation of its representation. For example, subjects might be expected to differentially attend to biologically significant over biologically nonsignificant stimuli. (By attention, we mean a response variable and not its conventional use in learning theory as a variable of acquisition, such as associability [e.g., Mackintosh, 1975; Pearce & Hall, 1980]). This privileged activation of biologically significant stimuli might, in turn, provide protection from cue competition effects such as overshadowing.

Oberling, Bristol, Matute, and Miller (1999) tested the

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hypothesis that biologically significant stimuli are protected from cue competition effects, using stimuli of inherent or acquired biological significance. In one experiment, subjects received overshadowing treatment with overshadowed and overshadowing cues of either high intensity (i.e., inherently biologically significant) or low intensity (i.e., inherently biologically nonsignificant). Oberling et al. observed less overshadowing of cues that were inherently biologically significant; that is, high intensity cues appeared to be protected from overshadowing. Two further experiments by Oberling et al. revealed parallel failures to demonstrate the relative stimulus validity effect and the degraded contingency effect when cues of high, but not low, intensity were used. Moreover, Miller and Matute (1996) found that blocking is also attenuated when the to-be-blocked CS is inherently biologically significant. Thus, inherently biologically significant stimuli appear to be protected from cue competition. However, one could explain all of these results in terms of stimulus intensity alone, without resorting to the concept of biological significance. Therefore, Oberling et al. conducted an experiment that assessed overshadowing of biologically significant and biologically nonsignificant cues (both of low intensity) to address this possibility. Prior to overshadowing treatment with an aversive footshock as the US, the acquired biological significance groups received pairings of the target CS with a strong saccharin solution (foods that are intensely sweet can be used to control behaviors such as bar pressing; thus, by our definition, they are biologically significant). The biologically nonsignificant groups received saccharin pairings with an irrelevant stimulus, leaving the target CS without biological significance. At test, after overshadowing treatment with a footshock US, subjects that had received target CS–saccharin pairings prior to overshadowing training exhibited stronger conditioned suppression (behavior appropriate to a footshock US) than did subjects that had not received target CS–saccharin pairings. Thus, low-intensity cues that acquire biological significance through pairings with an inherently biologically significant stimulus prior to cue competition treatment appear to be at least partially protected from cue-competition effects just as are CSs of inherent biological significance.

The results presented above could potentially be interpreted in terms of an increase in associability of high intensity or saccharin-pretrained cues, which is consistent with some CS-modification acquisition accounts of cue competition effects (e.g., Mackintosh, 1975). Additionally, some US-modification accounts in which associability of a CS is a constant (e.g., Rescorla & Wagner, 1972) can explain the attenuation of overshadowing when the target CSs are intrinsically biologically significant because higher associability should result in more acquisition per trial, and these models do not predict cue competition on the first overshadowing trial. Therefore, the stronger conditioned response elicited by a high-intensity stimulus than a low-intensity stimulus could reflect greater acquisition on the first trial rather than any protection from overshadowing. However, these models cannot explain the attenuation of cue competition that is observed when the target CS has acquired biological significance. Nevertheless, the results described above are compatible with the general notion that stimuli of inherent (e.g., high intensity) or acquired (e.g., through pairings with a nontarget US) biological significance simply increase attention to the CS during training with the target US, thereby alleviating the detrimental effects of competing stimuli (e.g., those that are more salient or more valid predictors of the target US) on the acquisition of conditioned responding to the target CS.

In contrast to the acquisition-focused approaches presented above, an expression-focused approach to cue competition posits that the protective effects of biological significance arise at the time of testing, and not during training. In this framework, cue competition is explained by processes that occur during the test-trial retrieval and response generation stage of information processing. For example, the comparator hypothesis of Pavlovian responding (Miller & Matzel, 1988; Miller & Schachtman, 1985) posits that cue competition effects are failures to express information that was acquired during CS–US pairings. Responding to the CS is determined by the strength of the CS–US association relative to the associative strength of the other (i.e., comparator) stimuli that were present during CS training. At test, the US representation activated directly by the presentation of the target CS is compared with the US representation activated indirectly through the sequential action of the CS–comparator stimulus association and the comparator stimulus–US association. Excitatory responding to the CS is assumed to increase with the strength of the directly activated US representation and decrease with the strength of the indirectly activated US representation during testing. For example, in overshadowing in which a more salient CS (A) overshadows a less salient CS (X), the strong US representation activated indirectly through the X–A and A–US associations effectively competes with the US representation activated directly by X. Thus, little conditioned responding to X is observed at test. Evidence that an X–US association was acquired during overshadowing training comes from manipulations of posttraining deflation (i.e., extinction) of A that recover responding to X at test (e.g., Kaufman & Bolles, 1981; Matzel, Schachtman, & Miller, 1985). Additional reports have shown recovery from other forms of cue competition, such as blocking and the relative stimulus validity effect (e.g., Blaisdell, Gunther, & Miller, 1999; Cole, Barnet, & Miller, 1995; Dickinson & Charnock, 1985). Thus, biological significance may exert its protective effect through increased processing of the biologically significant CS at the time of testing rather than at the time of training.

Although recovery from cue competition, particularly as a consequence of posttraining extinction of the competing stimulus (A), supports the comparator hypothesis, the effects of biological significance better fit some acquisition-focused models (e.g., Mackintosh, 1975). However, acquisition-focused interpretations of biological-significance effects suggest that protection should be observed only if the target cue has biological significance at the time of training. Here we test this prediction using an overshadowing preparation.

As Oberling et al. (1999) have shown, a CS that has either inherent or acquired biological significance at the time of training is protected from the overshadowing deficit. Although these results are compatible with both acquisition-focused (e.g., Mackintosh) and expression-focused (e.g., Miller & Matzel, 1988) approaches, these two families of models make some contrasting predictions regarding the effects of biological significance on the expression of cue competition. If cue competition effects are due to a failure to express an acquired CS–US association (e.g., the comparator hypothesis), then giving the target CS biological significance even after training should attenuate the response deficit that would normally result from cue competition training. For example, in overshadowing in which A overshadows X as a signal for a footshock US, making X biologically significant (e.g., through pairings with saccharin as we did in the present research) after AX–footshock pairings should alleviate the overshadowing deficit (i.e., produce strong responding to X, appropriate for a footshock US, at test) relative to a group for which X was not made biologically significant. However, if cue-competition effects result from a failure to acquire a CS–US association (e.g., Mackintosh), then posttraining increases in the target CS's biological significance through counterconditioning should not affect responding to the CS. That is, in the framework of the Mackintosh model, A prevents the encoding of an X–US association. Posttraining increases in X's biological significance (e.g., through pairings with saccharin) are expected to have no effect on responding to X due to the absence of the X–US association required for such responding.

The following experiments tested the prediction of the comparator hypothesis that inflation of a target CS's biological significance through counterconditioning following overshadowing treatment should decrease the overshadowing of that CS. Such pairings of a to-be-overshadowed CS with saccharin *prior* to training have previously been shown to provide protection against overshadowing (Oberling et al., 1999). Experiment 1 tested the validity of this prediction. Experiment 2 replicated this effect, controlling for some additional alternative accounts of the phenomenon. Experiment 3 explored whether acquired biological significance can be reduced through extinction treatment, as is known to be the case with a CS's potential to elicit conditioned responding; that is, does the protection from cue competition afforded by biological significance wane in direct proportion to the CS's response potential?

Experiment 1

The specific design used for Experiment 1 (see Table 1) consisted of interspersing B–saccharin pairings with C– trials in Phase 1; this was intended to make B biologically significant. Preliminary research found that such discrimination training decreased generalization from B to other stimuli. In Phase 2, half of the rats received overshadowing treatment (with mild footshock as the US) using parameters that were found in preliminary studies to allow a higher intensity cue (A) to overshadow a lower intensity cue (X; i.e., AX–US; Groups BS and OV), whereas the other half received simple acquisition training (i.e., X–US; Groups Counter and Acquisition [Acq]). Following Phase 2 training, half of the subjects received X–B pairings (Groups BS and Counter), whereas the remainder of the subjects received an equivalent number of X–C pairings (Groups OV and Acq). Thus, X was expected to become biologically significant through pairings with the signal for saccharin (B), but not through pairings with the irrelevant stimulus (C).[1] Subsequently, all subjects were tested for conditioned suppression to X. Strong suppression to X in Group BS was expected as a result of X becoming biologically significant during the X–B pairings of Phase 3. In comparison, Group OV was expected to exhibit little responding to X relative to Group Acq (i.e., overshadowing) because the X–C pairings of Phase 3 should have had little effect on X's biological significance. Group Counter was included to assess the possible counterconditioning effects of X–B pairings on the level of conditioned suppression supported by X as a result of the X–footshock pairings.

Table 1
Design Summary: Experiment 1

Group	Phase 1	Phase 2	Phase 3	Test
BS	B → sac/C–	AX → shock	X → B	X
OV	B → sac/C–	AX → shock	X → C	X
Counter	B → sac/C–	X → shock	X → B	X
Acq	B → sac/C–	X → shock	X → C	X

Note. OV and BS refer to overshadowing and biological significance enhancing treatments, respectively. Counter and Acq refer to control groups for assessment of counterconditioning and acquisition, respectively. B and C denote the tone and noise stimuli, counterbalanced within groups. A represents the overshadowing conditioned stimulus, X represents the overshadowed conditioned stimulus, sac denotes the saccharin reinforcer, shock represents the footshock unconditioned stimulus, and the slash (/) separates events that were interspersed.

[1] Our initial plan was that rats were to receive overshadowing treatment. Then, half of the subjects were to receive 48 X–saccharin pairings (i.e., counterconditioning). However, there was a concern that the counterconditioning might retroactively interfere with the expression of the X–footshock association, thus working against our observing a conditioned response to the test stimulus (X) consistent with the footshock US. The present experiments used conditioned lick suppression to assess stimulus control. Thus, the X–saccharin pairings of counterconditioning might have resulted in nose poking into the niche that was to be used at different times for both saccharin delivery during counterconditioning and presentation of the water lick tube used to assess conditioned suppression. Such saccharin-motivated behavior would, of course, be incompatible with conditioned suppression of nose poking in the niche. To reduce this possibility, we used a variation on the simple design described in the text. Specifically, the saccharin manipulation was embedded in a second-order conditioning procedure. In this design, following overshadowing treatment, the overshadowed cue (X) was paired with B (in six trials), which itself was a second-order reinforcer as a consequence of prior B–saccharin pairings. Six X–B pairings were not expected to create the strong response competition that would be expected from 48 X–saccharin pairings.

Method

Subjects

Twenty-four male (250–360 g) and 24 female (200–255 g) naive, Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Subjects were individually housed in wire-mesh cages in a vivarium maintained on a 16-hr light–8-hr dark cycle. Running was done approximately midway through the light portion of the cycle. A progressive water-deprivation schedule was imposed over the week prior to the beginning of the experiment, until water availability was limited to 10 min per day. All animals were handled three times a week for 30 s, from time of weaning to the initiation of the study. Subjects were randomly assigned to one of four groups (ns = 12) counterbalanced for sex.

Apparatus

Twelve identical chambers, each measuring 30 × 25 × 32 cm (length × width × height), individually housed in environmental isolation chests, were used. The floor of each chamber was constructed of 0.5-cm diameter stainless steel rods, 1.5 cm center to center, connected by NE-2 neon bulbs that allowed a 1.0-mA, 0.5-s constant-current footshock to be delivered by means of a high-voltage AC circuit in series with a 1.0-MΩ resistor. Each enclosure was dimly illuminated by a 2-W (nominal at 120 VAC) incandescent houselight driven at 60 VAC, mounted on the ceiling of the environmental isolation chest. Each chamber was equipped with a cylindrical niche 4.5 cm in diameter mounted with its axis perpendicular to the wall of the chamber on which the niche was affixed. The niche was left–right centered at one end of the chamber with its bottom 4 cm above the grid floor. Within each niche, there was a water-filled tube (opening = 0.3 cm in diameter) that extended 1 cm into the cylindrical niche. In addition to the lick tube, the drinking recess of each chamber could be equipped with an 80201 Liquid Dispenser System (Lafayette Instruments, Lafayette, IN), which included a metallic spout located 1.75 cm into the niche and 4.5 cm from the niche base. This dispensing system was capable of delivering variable amounts of liquid at specified intervals. A horizontal photobeam was projected 0.5 cm in front of the lick tube. To drink from the tube, subjects had to insert their heads into the niche, thereby breaking the horizontal infrared photobeam. Thus, the amount of time the photobeam was disrupted could be monitored; this served as our dependent measure for both B–saccharin conditioning in Phase 1 (increased time in the photobeam during presentation of B) and for the X–shock association at test (longer pause outside the photobeam during presentation of X). A 45-Ω speaker mounted on the interior back side of each environmental chest could deliver a high-frequency complex (3000 and 3200 Hz) tone 8 dB(C-scale) SPL above the background sound level. A second 45-Ω speaker, mounted on the ceiling of each experimental chamber, could deliver a click train (6/s) 6 dB(C-scale) above background. A third 45-Ω speaker, mounted on the side wall of each environmental chest, could deliver a white-noise stimulus 8 dB(C-scale) above background. A 75-W (nominal at 120 VAC) incandescent bulb driven at 100 VAC, mounted on the back wall of each environmental chest 30 cm from the floor of the conditioning chamber, could be flashed (0.25 s on–0.25 s off). The tone and white noise, counterbalanced within groups, served as Stimuli B and C. The flashing light always served as Stimulus A (the overshadowing stimulus). The houselight was turned off when the light stimulus was being flashed on and off. The clicks always served as Stimulus X (the overshadowed stimulus). All CSs were 10 s in duration. A saccharin–water solution (0.05 ml at 0.04 M) could be delivered by the liquid-dispenser system into a cup at the bottom of the same niche in which the lick tubes could be placed. Ventilation fans in each enclosure provided a constant 76-dB(C-scale) background noise.

Procedure

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

Acclimation. On Day 1, all subjects had access to the water-filled lick tubes during a 60-min session. No nominal stimuli were presented. This session allowed for the acquisition of licking behavior.

Phase 1 (biological significance treatment). Prior to the initiation of Phase 1, the lick tubes were removed from each chamber and replaced with the liquid dispensers. On Days 2–7, all subjects received eight daily exposures to Stimulus B followed immediately by the saccharin solution pseudorandomly interspersed with eight nonreinforced presentations of Stimulus C, with a mean intertrial interval of 3.75 min (range ± 1.25 min), during each 60-min daily session. To determine whether the animals had learned the B–saccharin association, duration of nose poking into the reinforcement niche was measured during each cue presentation. A discrimination ratio (R) was then calculated (R = [B − C]/[B + C]), with B and C being the sum for any given day of the nose-poke durations recorded during the presentation of cues B and C, respectively. A quotient of 0 was indicative of a total lack of discrimination between B and C, and a quotient of 1 was indicative of a perfect discrimination. This testing procedure allowed assessment of the animal's acquisition of the B–saccharin solution contingency and, hence, the acquired biological significance of the first-order stimulus (B).

Phase 2 (overshadowing treatment). On Days 8 and 9, subjects in Groups BS and OV received three AX–shock pairings (with common onset and termination of A and X followed immediately with shock presentation) during each daily 60-min session, for a total of six presentations. Subjects in Groups Counter and Acq received three X–shock pairings per day, for a total of six presentations. These trials occurred 15, 34, and 50 min into the session. Neither water nor saccharin was available during these sessions.

Phase 3 (biological-significance-of-X treatment). On Day 10, subjects in Groups BS and Counter received six X–B trials in a single 60-min session. Subjects in Groups OV and Acq received six X–C trials. These trials (with X terminating coincidentally with B or C onset) occurred 8, 15, 20, 30, 44, and 52 min into the session. Neither water nor saccharin was available during these sessions.

Reacclimation. Prior to the initiation of reacclimation, water-filled lick tubes were reinstalled in each chamber. On Days 11 and 12, subjects were allowed to drink during each daily 60-min session to restabilize baseline levels of drinking. There were no nominal stimulus presentations during these sessions.

Testing. On Day 13, all subjects were tested for conditioned lick suppression to X by presenting X immediately on completion of 5 cumulative seconds of licking for water (as measured by the total amount of time the infrared photobeam was disrupted). Thus, all subjects were drinking at the time of X onset. Time to complete this initial 5 cumulative seconds of licking in the absence of X and time to complete an additional 5 cumulative seconds of licking in the presence of X were recorded. Test sessions were 16 min in duration, with a ceiling of 15 min being imposed on the time to complete the 5 cumulative seconds of drinking in the presence of X.

Suppression data were transformed to log (base 10) scores to

facilitate the use of parametric statistics. An alpha level of .05 was adopted for all tests of statistical significance.

Results and Discussion

The central observation from Experiment 1 was that there was less responding to X, indicative of greater overshadowing of X by A, in Group OV, in which X was paired in Phase 3 with a stimulus (C) that was not biologically significant, than in Group BS, in which X was paired in Phase 3 with a stimulus (B) that was biologically significant (see Figure 1).

On the last day of Phase 1 training, all groups exhibited greater nose-poke responding to the stimulus paired with saccharin (B) than to the stimulus explicitly unpaired with saccharin (C), indicating that the subjects learned to discriminate between the two stimuli (R ± *SEM* = .31 ± .20; .68 ± .10; .32 ± .18; .31 ± .18 for Groups BS, OV, Counter, and Acq, respectively). Thus, within our definition of biological significance, despite considerable between-subjects variability, Stimulus B was made biologically significant for each group during Phase 1. Notably, a post hoc test (Newman–Keuls) revealed no significant differences in between-groups variability, $p > .60$.

A 2 × 2 analysis of variance (ANOVA) with Phase 2 treatment (AX–shock or X–shock) and Phase 3 treatment (X–B or X–C) as factors conducted on the pre-CS times to complete 5 cumulative seconds of licking revealed no main effects or interaction in baseline drinking behavior, Fs < 1.0. Moreover, no subjects took over 60 s to complete their first 5 cumulative seconds of licking (i.e., prior to CS onset). A similar 2 × 2 ANOVA conducted on suppression to X scores revealed a main effect of Phase 2 treatment, $F(1, 44) = 13.86$, $p < .001$, and an interaction, $F(1, 44) = 7.76$, $p < .01$, but no main effect of Phase 3 treatment, $F < 1.0$.

Planned comparisons were conducted on the suppression to X scores using the overall error term from the latter 2 × 2 ANOVA. Subjects in Group OV suppressed less to X than did subjects in Group Acq, demonstrating overshadowing (and therefore a failure of Phase 3 X–C trials to eliminate overshadowing by serving as a reminder treatment), $F(1, 44) = 21.18$, $p < .001$. Group BS suppressed more to X than did Group OV, demonstrating recovery from overshadowing, $F(1, 44) = 6.73$, $p < .02$. Groups Counter and Acq did not differ, suggesting little effect of counterconditioning due to pairing X with a stimulus that signaled saccharin following the X–shock pairings (i.e., B), $F(1, 44) = 1.81$, $p > .10$.

The recovery from overshadowing observed in Group BS as a consequence of a postacquisition change in biological significance of X is congruent with expression-failure views of overshadowing, although no current expression-failure model speaks explicitly to the mechanism by which a posttraining increase in the biological significance of an overshadowed CS acts to reverse overshadowing. An alternative interpretation of overshadowing is provided by the possibility that stimulus generalization decrement attenuated suppression to the overshadowed element alone after training with a compound of the overshadowed and overshadow-

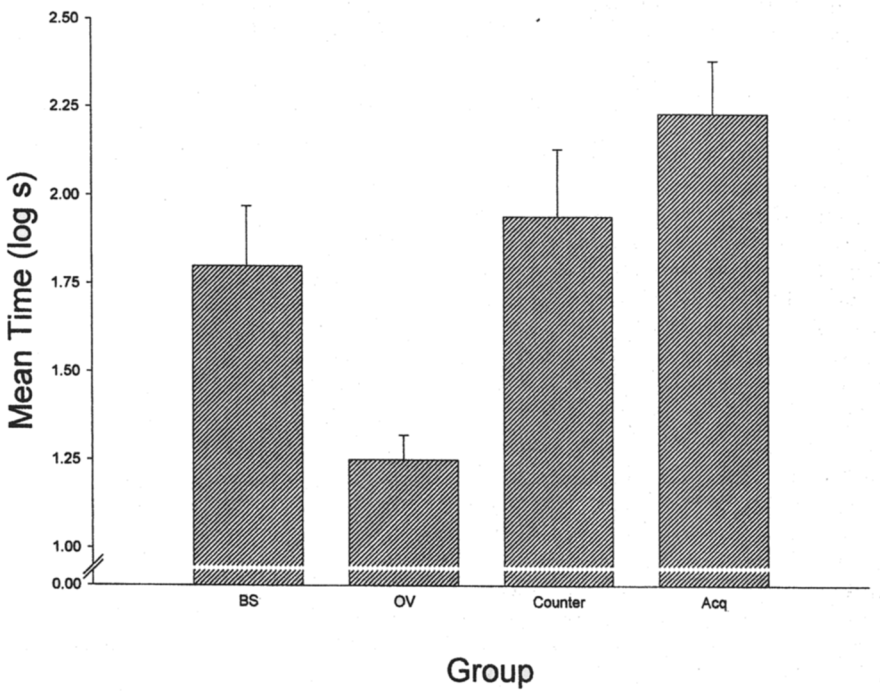

Figure 1. Experiment 1: Mean times (log s) to complete 5 cumulative seconds of licking in the presence of the target conditioned stimulus (CS). All groups were tested on CS X. Error bars represent standard errors of means. OV and BS refer to overshadowing and biological significance enhancing treatments, respectively. Counter and Acq refer to control groups for assessment of counterconditioning and acquisition, respectively.

ing stimuli. Although we did not include an explicit control for such an interpretation of the present overshadowing effect, Group BS provides an implicit control. A generalization decrement account of overshadowing does not predict X–B pairings in Phase 3 would facilitate generalization of suppression from the AX compound to the X element preferentially in Group BS compared to Group OV, which received X–C pairings in Phase 3. Thus, a stimulus generalization decrement account of the present overshadowing effect would anticipate an equal level of overshadowing in both groups, which clearly is contrary to what was observed.

Experiment 1 demonstrated that CSs that acquired biological significance following overshadowing treatment are subject to an attenuation of the overshadowing deficit. This finding is congruent with the prior finding of Oberling et al. (1999) that CSs acquiring biological significance prior to overshadowing treatment were protected against overshadowing. Although the observations of Oberling et al. could, in principle, be explained by either acquisition-focused or expression-focused models of Pavlovian responding, the present results appear to preclude an explanation in terms of acquisition-focused models. If the Phase 3 X–B pairings given to Group BS resulted in any associative acquisition, then they should have made X a second-order CS for nose poking, thus enhancing rather than suppressing nose poking in the presence of X.

Experiment 2

The purpose of Experiment 2 was to address two alternative accounts of the central finding of Experiment 1; that is, greater suppression to X in Group BS than in Group OV. The first alternative is that the X–B and X–C postovershadowing pairings may have modulated extinction of X instead of influencing overshadowing. That is, we may have failed to produce overshadowing (note that we included no overshadowing-treatment-only group in Experiment 1), but instead may have had strong responding to X after the overshadowing treatment that underwent a differential amount of extinction, depending on whether X was subsequently paired with B or C during Phase 3. As B was an appetitive excitor and C was possibly an appetitive inhibitor, we might have expected them to function as aversive inhibitors and excitors, respectively (e.g., Dickinson & Pearce, 1977; but see Lovibond & Dickinson, 1982). If B acted as an aversive inhibitor, it could have protected the X–shock association from extinction during the X–B pairings in Group BS, relative to the X–C pairings of Group OV (a mechanism analogous to the account of superconditioning that is provided by the Rescorla–Wagner model, Rescorla, 1971). In the same framework, the negative contingency between C and saccharin in Phase 1 may have established C as an appetitive inhibitor which, like an aversive excitor, might augment extinction of the X–shock association during X–C pairings. These possibilities are discouraged by the observation that the difference in performance between Groups Counter and Acq of Experiment 1 (for which X was demonstrably excitatory) was in the opposite direction (although nonsignificant) of that between OV and BS. However, the high level of aversive conditioning in Groups Counter and Acq may have precluded much extinction to X. Therefore, to provide a stronger test of this alternative account, we compared conditioned suppression to X in two groups (Group OV.B and OV.C below) that received post-overshadowing pairings of X with B or C, respectively, with suppression to X in subjects that received no further treatment with X after overshadowing training (Group OV.None; see Table 2). If B is acting as an aversive inhibitor, then it should protect the X–shock association from extinction, and we should observe equal responding in Groups OV.B and OV.None (i.e., we should fail to demonstrate overshadowing with this procedure). Moreover, these two groups should both suppress more than Group OV.C in which C is acting as an aversive excitor, thereby facilitating extinction of the X–shock association. This prediction is based on the assumption that we are observing facilitation of extinction in Group OV.C instead of protection from overshadowing in Group OV.B. However, on the basis of the putative role of biological significance, we expected to observe high suppression in Group OV.B and low suppression (i.e., overshadowing) in Groups OV.C and OV.None, allowing us to conclude that the X–B pairings, but not X–C pairings, attenuated overshadowing of X.

A second alternative account of the greater suppression to X observed in Group BS relative to Group OV of Experiment 1 is that the Phase 3 X–B trials might conceivably have made X a cue for frustration because X potentially was associated with the nonreinforcement of an appetitive CS (B). To evaluate this alternative account, we compared the effects of X–B pairings on responding to X in subjects that did (Group OV.B) or did not (Group NoOV.B) receive prior overshadowing treatment. In contrast with the frustration-effect explanation raised above, we expected suppression in Group OV.B to be stronger than in Group NoOV.B, demonstrating the necessity of the Phase 2 X–shock pairings in producing strong conditioned suppression to X. Furthermore, we expected no difference in suppression to X by rats that received X–B (Group NoOV.B) or X–C (Group NoOV.C)

Table 2
Design Summary: Experiment 2

Group	*n*	Phase 1	Phase 2	Phase 3	Test
OV.B	12	B → sac/C−	AX → shock	X → B	X
OV.C	12	B → sac/C−	AX → shock	X → C	X
OV.None	12	B → sac/C−	AX → shock	—	X
NoOV.B	6	B → sac/C−	A → shock	X → B	X
NoOV.C	6	B → sac/C−	A → shock	X → C	X

Note. OV and NoOV refer to overshadowing and no overshadowing treatments in Phase 2, respectively. B, C, and None refer to pairings of X with B, X with C, or context exposure in Phase 3, respectively. B and C denote the tone and noise stimuli, counterbalanced within groups. A represents the overshadowing conditioned stimulus, X represents the overshadowed conditioned stimulus, sac denotes the saccharin reinforcer, shock represents the footshock unconditioned stimulus, and the slash (/) separates events that were interspersed. *n* refers to the number of subjects in each group. Dash represents equivalent context exposure without nominal stimulus presentations.

pairings in the absence of overshadowing treatment. That is, if Phase 3 X–B pairings induce frustration, then rats that received this treatment should evidence greater suppression to X, than rats that received X–C pairings.

Method

Subjects and Apparatus

Twenty-four male (250–365 g) and 24 female (190–235 g) naive, Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to one of five groups (Group OV.B, OV.C, and OV.None, *n*s = 12; and Groups NoOV.B and NoOV.C, *n*s = 6). The animals were housed and maintained as in Experiment 1. The apparatus and stimuli were identical to those used in Experiment 1.

Procedure

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

Acclimation. On Day 1, all subjects were acclimated to the experimental context as in Experiment 1.

Phase 1 (biological significance treatment). On Days 2–7, all subjects received appetitive discrimination training (B–saccharin/C–) as in Experiment 1.

Phase 2 (overshadowing treatment). On Days 8 and 9, subjects received training identical to Phase 2 of Experiment 1, that is, AX–shock or X–shock pairings as indicated in Table 2, except for subjects in Groups NoOV.B and NoOV.C, which received equivalent A–shock pairings but not treatment with CS X.

Phase 3 (biological-significance-of-X treatment). On Day 10, subjects received training identical to Phase 3 of Experiment 1. That is, X was paired with either B or C, except for subjects in Group OV.None, which received equivalent context exposure without any nominal stimulus presentations.

Reacclimation. On Days 11 and 12, subjects were reacclimated to drinking in the apparatus as in Experiment 1.

Testing. On Day 13, all subjects were tested for conditioned lick suppression to X as in Experiment 1. Data from 1 subject (Group OV.B) were lost due to an equipment malfunction.

Results and Discussion

Experiment 2 replicated the recovery from overshadowing of X through X–B pairings (Group OV.B vs. Group OV.C), which was observed in Experiment 1 (see Figure 2). Furthermore, this effect was not due to X–B (as opposed to X–C) pairings protecting X from extinction (Group OV.B vs. OV.None and OV.C vs. OV.None, respectively). Rather, pairings with B, but not C, in Phase 3 appeared to have attenuated observed overshadowing of X at test. Finally, pairing X with B in Phase 3 did not appear to induce frustration to X (NoOV.B vs. NoOV.C).

On the last day of Phase 1 training, all groups exhibited greater nose-poke responding to the stimulus paired with saccharin (B) than to the stimulus explicitly unpaired with saccharin (C), indicating that the subjects learned to discriminate between the two stimuli (R ± *SEM* = .45 ± .10; .70 ± .08; .57 ± .12; .40 ± .11; .43 ± .09; for Groups OV.B, OV.C, OV.None, NoOV.B, and NoOV.C, respectively). Thus, within

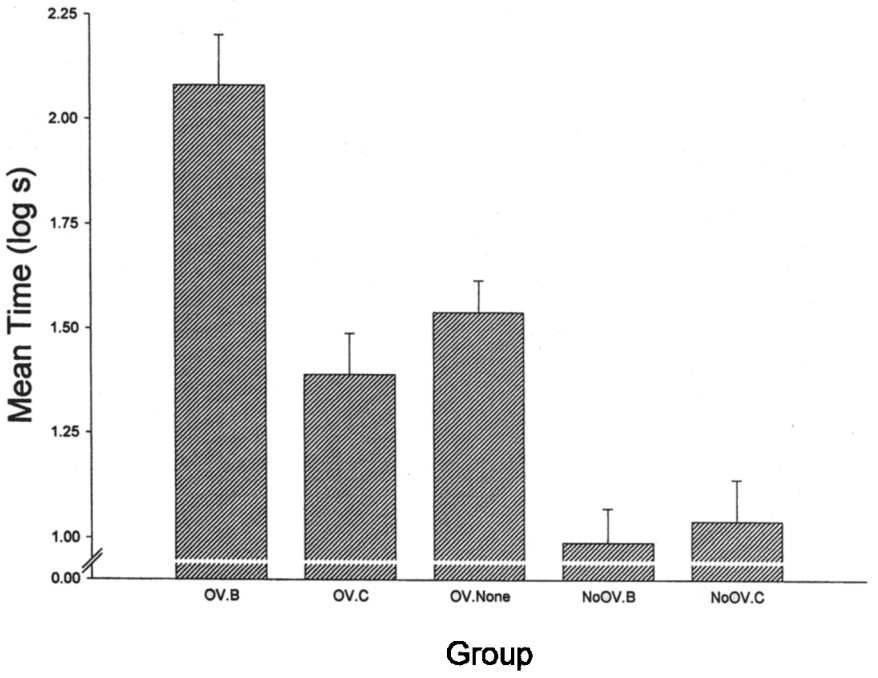

Figure 2. Experiment 2: Mean times (log s) to complete 5 cumulative seconds of licking in the presence of the target conditioned stimulus (CS). All groups were tested on CS X. Error bars represent standard errors of means.

our definition of biological significance, Stimulus B was made biologically significant for each group during Phase 1. Although there was considerable variability between groups, a post hoc test (Newman–Keuls) revealed no significant difference in this measure, $p > .24$.

A one-way ANOVA conducted on the pre-CS (all groups) times to complete 5 cumulative seconds of licking revealed no significant group differences in baseline drinking behavior, $F < 1.0$. Moreover, no subject took longer than 60 s to complete an initial 5 cumulative seconds of licking (i.e., prior to CS onset). A one-way ANOVA conducted on the suppression scores (Groups OV.B, OV.C, and OV.None) during the presentation of X revealed a treatment effect, $F(2, 32) = 13.10$, $p < .0001$. Planned comparisons were conducted, using the overall error term from the one-way ANOVA, to determine the source of this effect. Group OV.B demonstrated much greater suppression than did Group OV.C, $F(1, 32) = 24.13$, $p < .0001$, replicating the differential effect of postovershadowing pairings of X with B and C, respectively, that was observed in Experiment 1. Group OV.B also suppressed more to X than did Group OV.None $F(1, 32) = 14.37$, $p < .001$, indicating that pairing X with B after overshadowing treatment resulted in attenuation of the overshadowing effect. Postovershadowing pairings of X with C did not affect responding to X relative to Group OV.None, $F(1, 32) = 1.31$, $p > .25$. These two comparisons argue against an interpretation of postovershadowing X–B pairings protecting the X–shock association from extinction, whereas X–C pairings allowed or facilitated such extinction.

A 2 × 2 ANOVA conducted on suppression scores from Groups OV.B, OV.C, NoOV.B, and NoOV.C, with Phase 2 treatment (AX–shock or A–shock treatment) and Phase 3 (X–C or X–B pairings) as factors revealed a main effect of Phase 2 treatment, $F(1, 26) = 22.83$, $p < .0001$; a marginally significant main effect of Phase 3 treatment, $F(1, 26) = 3.79$, $p = .063$; and an interaction between Phases 2 and 3 treatment, $F(1, 26) = 6.64$, $p < .02$. Planned comparisons were conducted to isolate the source of the interaction. Group OV.B evidenced greater suppression to X than did Group NoOV.B, $F(1, 26) = 24.15$, $p < .0001$, indicating that X–B pairings alone (Group NoOV.B) failed to elicit strong conditioned suppression to X (i.e., no frustration effect was observed). Rather, X–B treatment increased conditioned suppression to X only if X had received overshadowing treatment in Phase 2 (Group OV.B). In the absence of prior overshadowing treatment, X–B pairings did not increase suppression to X relative to X–C pairings, Groups NoOV.B versus NoOV.C, $F(1, 26) < 1.0$, indicating that X–B pairings alone did not inflate suppression to X. These results argue against an account based on learned frustration.

Experiment 3

The purpose of Experiment 3 was to investigate whether and under what conditions the biological significance that a cue acquires through pairings with an inherently biologically significant stimulus can be extinguished through posttraining exposure. If a stimulus loses the potential to elicit vigorous responding, then it should no longer be protected from cue competition, nor should a stimulus that has lost biological significance through extinction be able to confer biological significance on another cue paired with it. It is this latter prediction that we tested in Experiment 3. We suggested above that biologically significant stimuli are protected from cue competition effects because they receive privileged postperceptual processing. Perhaps this greater amount of postperceptual processing also provides some protection of biologically significant stimuli from the effects of extinction treatment. If so, then as with the reversal of cue competition effects, extinction of a biologically significant CS should only be effective with massive posttraining extinction.

To test this prediction, after giving rats discrimination training with B and C as in Experiment 1 (i.e., 48 B–saccharin trials interspersed with 48 C− trials), we manipulated the number of B–extinction trials administered following the B–saccharin pairings. Specifically, we gave rats either 80 B– trials, 1080 B– trials, 80 C– trials, or 1080 C– trials. Subsequently, we gave overshadowing training (AX–US pairings, as in Phase 2 of Experiment 1) and then paired target CS X with either B or C (as in Phase 3 of Experiment 1). At test, we expected to observe, as in Experiment 1, overshadowing of X in the groups that received overshadowing treatment followed by X–C pairings (regardless of the amount of extinction of C) and less overshadowing in the groups that received overshadowing treatment followed by X–B pairings provided B had not been extinguished. However, we anticipated a difference in suppression between the groups that received overshadowing followed by X–B pairings in which B was extinguished with either few (80) or many (1,080) trials. We expected to see more suppression (i.e., less overshadowing) in the group which received only 80 B–extinction trials than in the group that received 1,080 B– trials.

Method

Subjects and Apparatus

Thirty-six male (190–310 g) and 36 female (182–245 g) naive, Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to 1 of 10 groups (Group BS.Ext.Many and BS.Ext.Few, *ns* = 12; and Groups BS.NoExt.Many, BS.NoExt.Few, OV.Many, OV.Few, Counter.Many, Counter.Few, Acq.Many, and Acq.Few, *ns* = 6). The animals were housed and maintained as in Experiments 1 and 2. The apparatus and stimuli were identical to those used in Experiments 1 and 2.

Procedure

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

Acclimation. On Day 1, all subjects were acclimated to the experimental context as in Experiments 1 and 2.

Phase 1 (biological significance treatment). On Days 2–7, all subjects received appetitive discrimination training (B–saccharin/C–) as in Experiments 1 and 2.

Table 3
Design Summary: Experiment 3

Group	*n*	Phase 1	Phase 2	Phase 3	Phase 4	Test
BS.NoExt.Many	6	B → sac/C−	C− (many)	AX → shock	X → B	X
BS.Ext.Many	12	B → sac/C−	B− (many)	AX → shock	X → B	X
OV.Many	6	B → sac/C−	C− (many)	AX → shock	X → C	X
Counter.Many	6	B → sac/C−	C− (many)	X → shock	X → B	X
Acq.Many	6	B → sac/C−	C− (many)	X → shock	X → C	X
BS.NoExt.Few	6	B → sac/C−	C− (few)	AX → shock	X → B	X
BS.Ext.Few	12	B → sac/C−	B− (few)	AX → shock	X → B	X
OV.Few	6	B → sac/C−	C− (few)	AX → shock	X → C	X
Counter.Few	6	B → sac/C−	C− (few)	X → shock	X → B	X
Acq.Few	6	B → sac/C−	C− (few)	X → shock	X → C	X

Note. OV and BS refer to overshadowing and biological significance enhancing treatments, respectively. Counter and Acq refer to control groups for assessment of counterconditioning and acquisition, respectively. Ext and NoExt refer to extinction and no extinction of B, respectively. Many and Few refer to the number of extinction of B trials, 1,080 or 80, respectively. B and C denote the tone and noise stimuli, counterbalanced within groups. A represents the overshadowing conditioned stimulus, X represents the overshadowed conditioned stimulus, sac denotes the saccharin reinforcer, shock represents the footshock unconditioned stimulus, and the slash (/) separates events that were interspersed. *n* refers to the number of subjects in each group.

Phase 2 (extinction of biological significance). On Days 8–15, subjects in Group BS.Ext.Few received 10 B– extinction trials per daily session, for a total of 80 trials. Groups BS.NoExt.Few, OV.Few, Counter.Few, and Acq.Few received an equivalent number of C– trials. Subjects in Group BS.Ext.Many received 135 B– extinction trials per daily session, whereas subjects in Groups BS.NoExt.Many, OV.Many, Counter.Many, and Acq.Many received 135 C– trials per daily session, for a total of 1,080 trials. We monitored extinction by comparing daily duration of nose poking during B in Phase 2 relative to duration of nose poking during C on the last day of Phase 1. On the basis of these data, extinction of nose poking during presentation of B was evidenced by a performance index of R.

Phase 3 (overshadowing treatment). On Days 16 and 17, subjects received training identical to Phase 2 of Experiments 1 and 2, that is, AX–shock or X–shock as indicated in Table 3.

Phase 4 (biological-significance-of-X treatment). On Day 18, subjects received training identical to Phase 3 of Experiments 1 and 2; that is, X was paired with either B or C.

Reacclimation. On Days 19 and 20, subjects were reacclimated to drinking in the apparatus as in Experiments 1 and 2.

Testing. On Day 21, all subjects were tested for conditioned lick suppression to X as in Experiments 1 and 2.

Results and Discussion

Experiment 3 replicated overshadowing of X by A (Groups OV.Many and OV.Few) and recovery from overshadowing as a result of posttraining pairings of B (the signal for saccharin) with X (Groups BS.NoExt.Many and BS.NoExt.Few), which were demonstrated in Experiment 1. Furthermore, many (but not few) extinction of B trials abolished B's effectiveness in recovering responding to X (Group BS.Ext.Many).

On the last day of Phase 1 training, all groups exhibited greater nose-poke responding to the stimulus paired with saccharin (B) than to the stimulus explicitly unpaired with saccharin (C), indicating that the subjects learned to discriminate between the two stimuli (R ± *SEM* = .44 ± .17; .61 ± .14; .32 ± .22; .32 ± .17; .59 ± .15; .45 ± .15; .42 ± .20; .28 ± .28; .66 ± .17; .73 ± .11 for Groups BS.NoExt.Many, BS.Ext.Many, OV.Many, Counter.Many, Acq.Many, BS.NoExt.Few, BS.Ext.Few, OV.Few, Counter.Few, and Acq.Few, respectively). Thus, within our definition of biological significance, despite high variability, Stimulus B was made biologically significant for each group during Phase 1. Although there was considerable variability between groups, a post hoc test (Newman–Keuls) revealed no significant differences in this measure, $p > .50$. Figure 3 shows that on the last day of Phase 2 training, nose poking to B had extinguished in Groups BS.Ext.Many and BS.Ext.Few (R ± *SEM* = −.16 ± .23 and −.15 ± .21, respectively).[2]

A one-way ANOVA conducted on the pre-CS times to complete 5 cumulative seconds of licking revealed no significant group differences in baseline drinking behavior, $F < 1.0$. Moreover, no subject took longer than 60 s to complete an initial 5 cumulative seconds of licking (i.e., prior to CS onset). A one-way ANOVA conducted on the suppression scores during the presentation of X revealed a treatment effect, $F(9, 62) = 5.56$, $p < .0001$. Planned comparisons were conducted, using the overall error term from the one-way ANOVA, to determine the source of this effect. Groups BS.NoExt.Many and BS.NoExt.Few did not differ significantly, Groups OV.Many and OV.Few did not differ significantly, Groups Counter.Many and Counter.Few did not differ significantly, and Groups Acq.Many and Acq.Few did not differ significantly, all $Fs < 1.30$, $ps > .25$. Thus, the scores from the first two of these groups were pooled to create Group BS.NoExt.Pooled ($n = 12$), the

[2] It is interesting to note that the rate of extinction in both groups, as assessed by the total amount of nose poking during B per extinction day, was nearly equivalent despite the vast difference in the number of extinction trials per day in each group, 135 and 10 in Groups BS.Ext.Many and BS.Ext.Few, respectively (see Figure 3). This suggests that distributed extinction trials are at least as effective in decreasing responding to a CS as are massed extinction trials, which is contrary to the prevailing view (e.g., Rohrer, 1947).

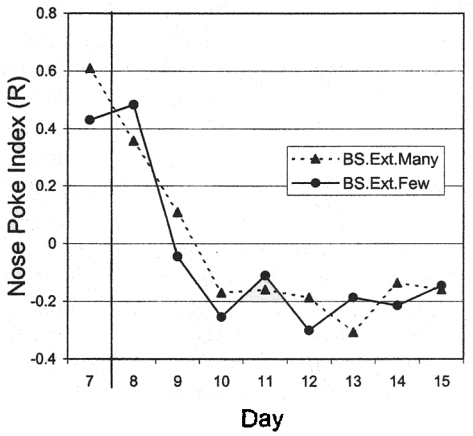

Figure 3. Experiment 3: Index (R) of nose poking during Stimulus B during Phase 2 extinction (Groups BS.Ext.Many and BS.Ext.Few). The last day of Phase 1 (Day 7) is included to illustrate baseline levels of nose poking prior to extinction.

scores from the third and fourth groups were pooled to create Group OV.Pooled (n = 12), the scores from the fifth and sixth groups were pooled to create Group Counter.Pooled (n = 12), and scores from the seventh and eighth groups were pooled to create Group Acq.Pooled (n = 12), for the purpose of further analysis.

A one-way ANOVA conducted on suppression scores of Groups BS.Ext.Many, BS.Ext.Few, BS.NoExt, OV, Counter, Acq revealed an effect of treatment, $F(5, 66) = 9.75$, $p < .001$ (see Figure 4). Additional planned comparisons using the error term from the latter one-way ANOVA revealed that Group OV suppressed less to X than did Group Acq, $F(1, 66) = 11.70$, $p < .01$, demonstrating overshadowing with our preparation. Group BS.NoExt suppressed more to X than did Group OV, $F(1, 66) = 13.49$, $p < .001$, demonstrating recovery from overshadowing as a function of X being paired with B (an established signal for saccharin). Additionally, Groups BS.NoExt and Counter did not differ significantly, $F(1, 66) < 1.0$, suggesting that recovery from overshadowing was largely complete. Group Counter did not differ from Group Acq, $F(1, 66) = 1.30$, $p > .25$, suggesting no appreciable counterconditioning effect from posttreatment pairings with B. Most important, Group BS.Ext.Few did not differ from Group BS.NoExt, $F(1, 66) = 1.27$, $p > .25$, whereas Group BS.Ext.Many suppressed less than either Group BS.NoExt $F(1, 66) = 14.13$, $p < .01$, or Group BS.Ext.Few, $F(1, 66) = 23.87$, $p < .001$, indicating that many, but not few, extinction-of-B trials sufficed to disrupt the recovery from overshadowing that X–B pairings conferred on X, despite equivalent losses of direct behavioral control by B.

General Discussion

Experiment 1 demonstrated recovery from overshadowing of an X–footshock association when the overshadowed CS (X) was made biologically significant through pairings with a signal for saccharin, even though these pairings took place after overshadowing treatment. Experiment 2 repli-

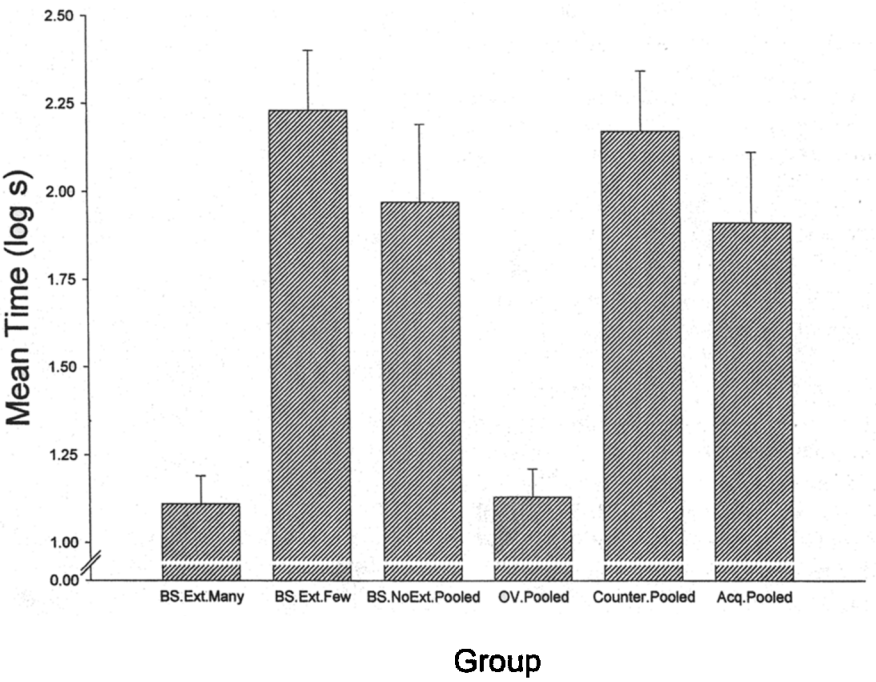

Figure 4. Experiment 3: Mean times (log s) to complete 5 cumulative seconds of licking in the presence of the target conditioned stimulus (CS). All groups were tested on CS X. Error bars represent standard errors of means.

cated this effect and ruled out a number of alternative accounts. Of critical interest in Experiment 3 was the effect of few versus many extinction-of-B (a signal for saccharin) trials on the potential of X–B pairings to restore behavioral control to X. Few extinction trials (i.e., 80) sufficed to extinguish nose-poke responding to B during Phase 2, yet were insufficient to prevent X–B pairings from restoring responding to the overshadowed CS X. Many extinction trials (i.e., 1,080) extinguished nose-poke responding to B during Phase 2 and prevented X–B pairings from restoring responding to X.

In the introductory section, we referred to biological significance as being indicated by the magnitude of the response controlled by a stimulus (as well as the stimulus' protection against cue competition). Clearly, the results of Experiment 3 indicate that this empirical definition requires modification. Seemingly, a stimulus that does or ever did support conditioned responding can provide protection against cue competition, but with sufficient extinction this protective quality can be attenuated. For example, in Experiment 3, 80 extinction-of-B trials were sufficient to reduce conditioned nose poking in response to B, but not sufficient to reduce B's potential to reverse overshadowing of X (which is indicative of B retaining biological significance). That is, X was protected from overshadowing (of conditioned lick suppression) through its pairings with a stimulus (B) that did not itself elicit a strong nose-poke response after 80 extinction trials. Apparently, the associative value of B fell below the threshold for response elicitation (i.e., as a signal for saccharin delivery) before it fell below the threshold for providing protection against cue competition (i.e., lost its biological significance). Thus, the degree to which responding is elicited by a stimulus is not a completely valid index of the biological significance of that stimulus.

The present results do not support an attentional view of the effects of biological significance on cue competition as might be formulated based on the model of Mackintosh (1975). According to this view, changes in biological significance reflect changes in perceptual processing of the target stimulus. The increased perceptual or attentional processing of X that presumably comes with X's increased biological significance is assumed to prevent the overshadowing stimulus (A) from interfering with the establishment of an X–footshock association. But, if the overshadowed CS (X) is made biologically significant after overshadowing treatment, then any resulting increase in attention or perceptual processing of X should have no effect on subsequent associative responding to X because of the lack of an existing X–footshock association. That is, overshadowing should have progressed unimpeded, resulting in the failure to acquire an overshadowed CS–US association. However, biological significance, as used here and previously (e.g., Denniston, Miller, & Matute, 1996; Miller & Matute, 1996), refers to postperceptual processes that protect a CS from response-attenuating comparator processes. Biological significance appears to be a property of a stimulus that accords it special attention (hence facilitates retrieval of its representation) and is indirectly measurable in terms of the responding it elicits. Biological significance also appears to be subject to extinction (or habituation), but does not wane over extinction trials as rapidly as does the stimulus' potential to elicit responding, at least using simple conditioned responding as a measure. This conception of biological significance is compatible with a notion of attention as a determinate of performance (e.g., Sutherland & Mackintosh, 1971), which can attenuate cue competition at the time of testing, but not as a determinant of the acquisition of response-elicitation value as proposed by Mackintosh and by Pearce and Hall (1980). Thus, possibly the X–B manipulation enhanced attention to X during subsequent testing, thereby increasing responding to X despite overshadowing itself being caused by the acquisition of a relatively weak X–shock association. However, other posttraining manipulations that also successfully reverse overshadowing argue against differences in associative strength as a source of the overshadowing effect (e.g., Blaisdell, Denniston, & Miller, 1999; Kaufman & Bolles, 1981; Matzel et al., 1985).

The present results also rule out an alternative interpretation of the protective effects from cue competition of acquired biological significance. This alternative explanation relates to Holland's (1980) demonstration that the presence of the US during second-order conditioning (i.e., S1→US in Phase 1 followed by S2→S1→US in Phase 2) disrupts conditioning of S2 relative to standard second-order conditioning in which the US is absent during Phase 2 training (i.e., S1→US in Phase 1 followed by S2→S1 in Phase 2). Perhaps with Oberling et al.'s (1999) demonstration of acquired biological significance, pretraining the overshadowed CS X with saccharin allowed X to interfere with establishing an association between the overshadowing CS A and the US, thereby preventing overshadowing (A. Dickinson, personal communication, November 4, 1998). That is, $X–US_1$ pairings followed by $AX–US_2$ pairings resulted in a weaker $A–US_2$ association than groups that had not received $X–US_1$ pairings (US_1 = saccharin, US_2 = footshock). Because the overshadowed CS was not made biologically significant until after overshadowing training, such an explanation cannot apply to the current experiments.

One possible relationship between a stimulus' potential to elicit conditioned responding and its potential protection from cue competition derived from the concept of biological significance is that both reflect associative value, but the threshold for conditioned responding is higher than that for protection against cue competition. In this framework, 80 extinction trials may have brought the associative value below the threshold for elicitation of conditioned responding, but not below the threshold for protection against cue competition. However, 1,080 extinction trials seemingly reduced the associative value of both thresholds.

This associative threshold view of response elicitation value and protection from cue competition raises another issue. Mentioned above, after overshadowing training, extinguishing the overshadowing stimulus can recover responding to the overshadowed stimulus (Kaufman & Bolles, 1981; Matzel et al., 1985). How can we explain the apparent increase in the biological significance of the overshadowed CS (X) as a consequence of extinction of the overshadowing

stimulus (A)? If X is biologically nonsignificant after overshadowing training as indicated by weak responding to X, but biologically significant after extinction of A as evidenced by the strong conditioned responding to X, then when and how does X gain biological significance? We suggest that the comparator process masks a CS's associative value that would otherwise be seen in both the CS's potential to elicit conditioned responding and its potential to provide protection against cue competition. But when this mask is removed (e.g., by posttraining extinction of the CS's comparator stimulus), responding to the CS is restored. The increase in conditioned responding to the CS indicates that the CS now activates the motivational system that controls responding. Possibly access to the US-appropriate (e.g., shock) motivational system increases postperceptual processing of the CS, ergo its biological significance.

Although some attributes of a CS must be acquired during the learning event (e.g., acquisition of associative value is dependent on close temporal and spatial proximity between the two events), other attributes can be retrospectively revalued (e.g., biological significance). It is when these retrospective processes interact with the initial learning of associative value that a conditioned response or other learned behavior is expressed. Thus, when the overshadowed CS comes to control a conditioned response (e.g., through extinction of the overshadowing stimulus) it also becomes biologically significant, and it presumably would be able to interfere with the ability of other stimuli to control conditioned responding (e.g., blocking; Denniston, Savastano, Blaisdell, & Miller, 1999).

Finally, it is worth noting that this effect (recovery from overshadowing of an aversive association through postovershadowing pairings of the overshadowed CS with a signal for saccharin, an appetitive stimulus) resembles the opposite of the usual outcome of counterconditioning treatment. Conventional counterconditioning, such as when a signal for an aversive reinforcer subsequently receives posttraining pairings with an appetitive reinforcer, results in a loss in the response (such as suppression) associated with the first learning treatment rather than an increase in the first response (as demonstrated in the current experiments). One difference between the present procedure and conventional studies of counterconditioning is that the counterconditioning in these experiments consisted of second-order conditioning rather than the conventional first-order conditioning. Because the overshadowed CS (X) had not been directly paired with saccharin, X did not acquire an appreciable propensity to elicit a nose-poke response. Moreover, the test context was changed from that of counterconditioning in that at test the water lick tube rather than the saccharin dispenser was present.

In this and previous work (e.g., Denniston, Miller, & Matute, 1996; Gunther, Miller, & Matute, 1997; Miller & Matute, 1996; Oberling et al., 1999), we have attempted to provide the beginnings of a principled account of biological significance by systematically exploring the various ways this variable interacts with other better known variables (e.g., associative value) to determine responding. Until now, biological significance has played a vague and inconsistent role in many theories of Pavlovian and instrumental learning (e.g., Hull, 1943; Pavlov, 1927; Thorndike, 1911). Our investigations of the role of biological significance in Pavlovian conditioning suggest that biological significance is a useful intervening variable that is potentially as important to understanding conditioned behavior as are other better established constructs, such as response threshold, temporal and spatial information, and associability.

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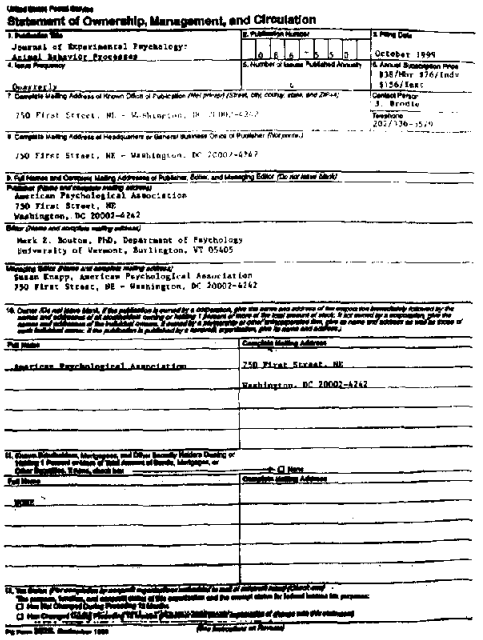

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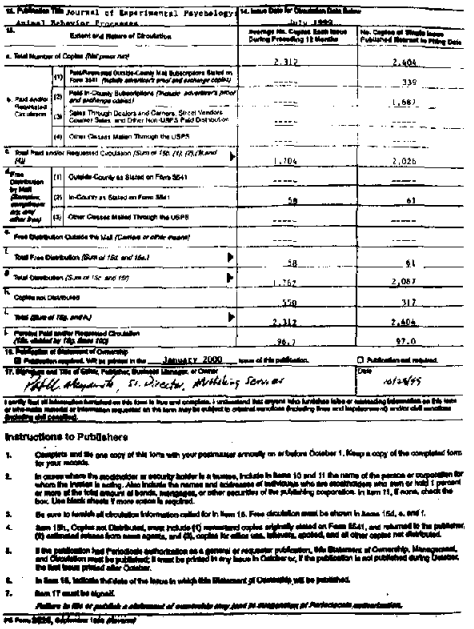

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