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# Overshadowing and Latent Inhibition Counteract Each Other: Support for the Comparator Hypothesis

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In 4 conditioned lick suppression experiments with rats, the combined effects of latent inhibition treatment followed by overshadowing treatment were assessed as a test of the comparator hypothesis's (R. R. Miller & L. D. Matzel, 1988) explanations of overshadowing and latent inhibition. Experiments 1 and 2 confirmed the prediction of the comparator hypothesis that combined latent inhibition and overshadowing treatments attenuate the response deficit produced by either treatment alone. Furthermore, consistent with the comparator hypothesis, posttraining changes in the associative status of the putative comparator stimulus altered responding to the target conditioned stimulus (Experiment 3), and switching contexts between latent inhibition and overshadowing treatments (Experiment 4) eliminated the interaction between the latent inhibition and overshadowing treatments.

Over the past 3 decades, attempts to explain cue competition effects (i.e., diminished responding to one cue when a second cue is present during training) have been a driving force in the study of Pavlovian conditioning. One well-known example of cue competition is overshadowing, in which two novel cues (e.g., A and X, usually with A being more salient than X) are compounded and paired with an unconditioned stimulus (US). Overshadowing is evident in weaker subsequent responding to X than would be observed if X had been paired with the US in the absence of A. Although the overshadowing effect was first reported by Pavlov (1927), not until the late 1960s did overshadowing, along with other cue competition effects, for example, blocking (Kamin, 1968, 1969) and relative stimulus validity (Wagner, Logan, Haberlandt, & Price, 1968; Wasserman, 1974), receive theoretical attention.

The fruits of this research culminated in the development of several formal mathematical models of Pavlovian conditioning that provided mechanisms to account for cue competition effects (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972). For example, the Rescorla–Wagner model attributes overshadowing to a failure by the subject to associate the target conditioned stimulus (CS), X, with the US (i.e., an acquisition failure). In this model, associative acquisition is determined by the discrepancy between expectancy of the US (i.e., $V_{Total}$ in terms of the model) and the impact of the US that actually occurs on a given trial ($\lambda$ in terms of the model). During overshadowing treatment, the overshadowing CS (A), as well as the overshadowed CS (X), gains associative value on the first few AX–US trials, thereby reducing the discrepancy between the expectation of the US and the actual US that occurs on subsequent AX–US trials. Thus, X acquires less associative strength on these later trials than it would if A were not present. Attentional models of Pavlovian conditioning differ from the Rescorla–Wagner model in that they explain overshadowing in terms of decrements in the learning rate parameter for CS X (i.e., the associability of X) as a result of X being trained in the presence of A (Mackintosh, 1975; Pearce & Hall, 1980). Although the mechanisms posited by these attentional models differ from that of the Rescorla–Wagner model, all of these models, including the Rescorla–Wagner model, view overshadowing as an acquisition failure.

In contrast, expression deficit models assume that all cues (including background cues) that are present during training acquire associations with the US (e.g., Gibbon & Balsam, 1981). Cue competition effects are treated as failures to express information that was acquired. The strength of the association between each cue and the US is presumably determined by the degree of contiguity between the cue and the US. The comparator hypothesis is an expression deficit model that specifically addresses cue competition effects (see Figure 1; Miller & Matzel, 1988; see also Miller & Schachtman, 1985). It provides a qualitative response rule stating that responding to a CS is determined by the strength of the CS–US association relative to the associative strength of the other cues (i.e., comparator stimuli) that were present during CS training. In the framework of the comparator

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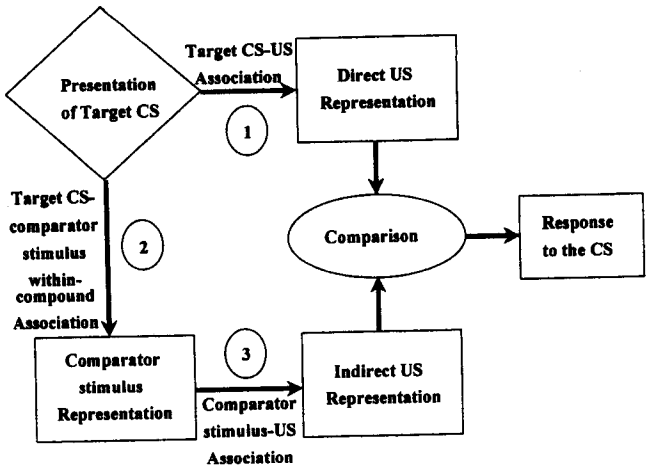


*Figure 1.* The comparator hypothesis. CS = conditioned stimulus; US = unconditioned stimulus.

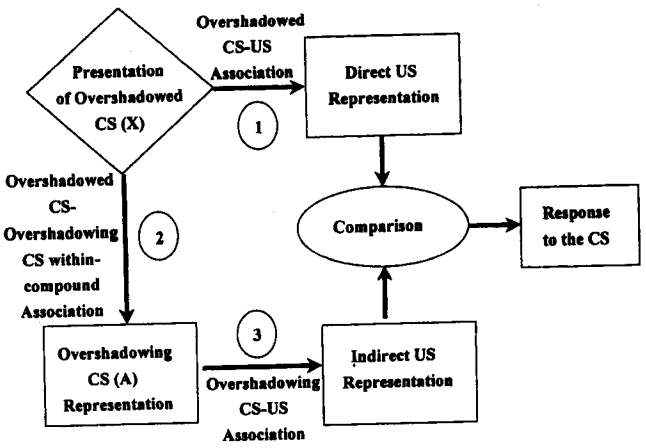


*Figure 2.* The comparator hypothesis applied to overshadowing. CS = conditioned stimulus; US = unconditioned stimulus; A = more salient cue; X = less salient cue.

hypothesis (as illustrated in Figure 1), the US representation directly activated by the target CS through the target CS–US association (Link 1) is compared at test with the US representation indirectly activated by the target CS through the conjoint action of the target CS–comparator stimulus association (Link 2) and the comparator stimulus–US association (Link 3). Excitatory responding to the CS increases with the strength of the direct US representation and decreases with the strength of the indirect US representation. Thus, although many cues acquire an association with the US, only the cues with associations that are strong relative to that of their primary comparator stimulus elicit a conditioned response.

For example, after overshadowing training (i.e., AX–US) with A being a more salient CS than X, A has greater associative strength than does X, although X's associative strength is presumably not reduced because of its being trained in compound with A. Presenting X at test activates a direct representation of the US (Link 1 of Figure 2), which is compared with the indirect US representation activated by X sequentially through the X–A association (Link 2 of Figure 2) and the A–US association (Link 3 of Figure 2); hence, the strength of the activated indirect US representation is proportional to the product of these two associations. Because of the strong X–A and A–US associations formed during the overshadowing treatment, the indirectly activated US representation is strong relative to the directly activated US representation, which results in weak conditioned responding to X. Supportive of this view, Kaufman and Bolles (1981); Matzel, Schachtman, and Miller (1985); and Matzel, Shuster, and Miller (1987) found that posttraining extinction of A restored responding to X. Presumably, this weakened Link 3 and possibly Link 2 of Figure 2, thereby increasing the magnitude of the directly activated US representation relative to the indirectly activated US representation.

Latent inhibition (Lubow, 1973) refers to a deficit in conditioned responding to a reinforced CS as a result of pretraining exposure to that CS alone, prior to reinforced training (i.e., CS–US). Most acquisition-focused models of Pavlovian responding view latent inhibition as a failure to acquire the CS–US association as a result of the nonreinforced preexposure decreasing attentional or memorial processing of the CS (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Wagner, 1981). However, posttraining manipulations such as reminder treatments (Kasprow, Catterson, Schachtman, & Miller, 1984) or extension of the training–testing retention interval (Kraemer, Randall, & Carbary, 1991) have recovered conditioned responding to a preexposed CS, thereby providing evidence for an expression deficit interpretation of latent inhibition. In the framework of the comparator hypothesis, nonreinforced CS preexposure (i.e., X-) produces a strong X–context association (Link 2 of Figure 3), thus strongly establishing the training context as the primary comparator stimulus for X. Reinforcing X in the training context during Phase 2 establishes a strong context–US

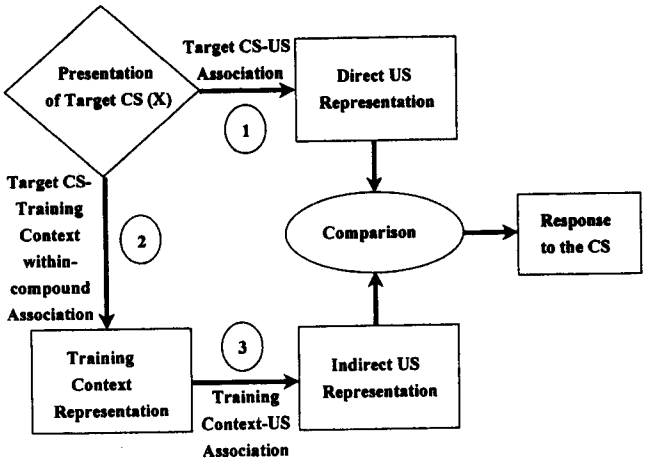


*Figure 3.* The comparator hypothesis applied to latent inhibition. CS = conditioned stimulus; US = unconditioned stimulus; X = less salient cue.

association (Link 3 of Figure 3). Then at test, the strong indirect US representation activated conjointly by the X-context and context–US associations successfully competes with the X–US association (Link 1 of Figure 3). Consistent with this view, Grahame, Barnet, Gunther, and Miller (1994) found that posttraining extinction of the experimental context attenuated the latent inhibition deficit in conditioned responding. Thus, the comparator hypothesis explains overshadowing and latent inhibition through the same comparator mechanism, but there is a difference in which stimulus (A or the training context, respectively) is serving as the comparator stimulus for X.

All stimuli present during CS training can potentially serve as the comparator stimulus at test, but it has been demonstrated that these stimuli compete for comparator status (e.g., Miller, Esposito, & Grahame, 1992). The likelihood that a stimulus will serve as a CS's comparator stimulus is assumed to increase with increases in its association with the target CS (i.e., Link 2 of Figure 1) and with its saliency. However, given the existence of comparator competition, the comparator hypothesis would predict an interesting new phenomenon if overshadowing and latent inhibition treatments were combined. By presenting a target CS (X) in the training context prior to overshadowing treatment (AX–US), one would expect a strong X–context association to develop (i.e., Link 2 in Figure 3; Grahame et al., 1994). As previously stated, overshadowing training normally causes the formation of a within-compound X–A association (Link 2 in Figure 2), thereby establishing A as the comparator stimulus for X. But if CS-preexposure treatment precedes overshadowing treatment (i.e., X alone prior to AX–US), then a strong X–context association (Link 2b in Figure 4) established during CS-preexposure might interfere with effective establishment of A as the primary comparator stimulus for X (Link 2a in Figure 4) during the AX–US trials. Consequently at test, the representation of the context and not the overshadowing stimulus should be activated by X and thus serve as the comparator stimulus for X (provided that the number of AX–US trials is small relative to the number of preceding CS–context trials). This leads to the counterintuitive hypothesis that latent inhibition treatment of X should reduce its vulnerability to overshadowing by a punctate cue. Furthermore, the presence of the highly salient CS A during the reinforced trials (i.e., AX–US) should degrade the effective context-US association relative to subjects that are trained in the absence of A (i.e., X–US). As the comparator hypothesis attributes latent inhibition to a strong X–context association and a strong context-US association (Grahame et al., 1994), the presence of A should attenuate the latent inhibition effect that would otherwise be observed. Although the mechanism for competition between the context and A for comparator status is not specified here, we discuss some possibilities in the General Discussion section.

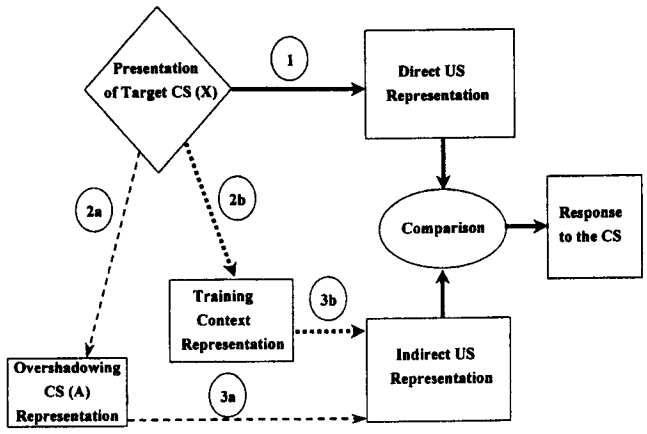


*Figure 4.* The comparator hypothesis applied to latent inhibition and overshadowing. CS = conditioned stimulus; US = unconditioned stimulus; A = more salient cue; X = less salient cue.

The purpose of the present research was (a) to explore the possibility that latent inhibition treatment might reduce overshadowing and that overshadowing treatment during training might reduce latent inhibition and then (b) to assess the extent to which comparator processes might play a role in producing this interaction. Experiment 1 directly tested the above-mentioned predictions of the comparator hypothesis regarding the interaction of latent inhibition and overshadowing treatments. Experiments 2 and 3 were designed to empirically determine which stimulus was acting as the primary comparator stimulus for X. We used posttraining associative inflation of candidate comparator stimuli. Finally, Experiment 4 was designed to investigate the same issue, but through a context switch between latent inhibition and overshadowing treatments. The overall goal was to provide a novel test of the comparator hypothesis's explanation of overshadowing and of the latent inhibition effect.

## Experiment 1

Experiment 1 was designed to initially examine interactions between latent inhibition treatment and overshadowing treatment. Phase 1 consisted of CS preexposure to the target CS (X) or an alternate CS (Y). During Phase 2, the groups that had been preexposed to either X or Y were subdivided and assigned to either overshadowing or overshadowing control conditions. Overshadowing subjects received overshadowing treatment (AX–US, with the US being a footshock), whereas control subjects received elemental excitatory conditioning only with the target CS (X–US). All subjects were then tested for conditioned suppression of ongoing drinking by the test stimuli. Our expectations on the basis of the comparator hypothesis were that (a) preexposure to X prior to pairings of X alone with the US would yield a latent inhibition deficit; (b) without preexposure to X, the presence of A (a more salient stimulus than X) during Phase 2 would overshadow X; and (c) preexposure to X followed by overshadowing treatment would yield more conditioned responding than either latent inhibition treatment alone or overshadowing treatment alone. Subjects receiving neither preexposure nor overshadowing training were expected to exhibit vigorous conditioned responding. Another control group, generalization decrement (GenDec), was included to determine the extent to which overshadowing observed in

our overshadowing condition arose from stimulus generalization decrement as a result of training with AX and of testing with X.

## Method

### Subjects

Thirty male (192–312 g) and 30 female (192–267 g) Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to one of five groups (*n*s = 12), counterbalanced for sex. Subjects were individually housed in wire-mesh cages in a vivarium maintained on a 16 hr light–8 hr dark cycle. Experimental manipulations occurred approximately midway through the light portion of the cycle. A progressive water deprivation schedule was imposed over the week prior to the beginning of the experiment until water availability was limited to 10 min per day. All subjects were handled for 30 s three times per week from weaning until the initiation of the study.

### Apparatus

Twelve clear Plexiglas chambers, 27 × 10 × 15 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 0.7-mA 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 house light driven at 60 VAC, mounted on the back wall 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 4 cm above the grid floor. Within each niche, there was a water-filled lick tube (opening = 0.3 cm in diameter) that extended 1 cm into the cylindrical niche. A horizontal photobeam 0.5 cm in front of the lick tube detected when a subject put its snout into the niche. 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 6 dB (C-scale) above the background sound level. A second 45-Ω speaker, mounted on the left interior side of each environmental chest, could deliver a white noise stimulus 6 dB (C-scale) above background. The tone and white noise, counterbalanced, served as X and Y. A 100-W incandescent bulb was mounted on the back wall of each environmental chest 30 cm from the floor of the conditioning chamber. This bulb could be flashed (0.25 s on–0.25 s off) to serve as CS A. The house light was turned off when the light stimulus was being flashed on and off. These stimulus parameters were selected on the basis of preliminary studies that indicated that the light (designated as A) would overshadow the tone or the white noise (which served as X, counterbalanced within groups). All CSs were 10 s in duration. The 0.7-mA footshock US was 1.0 s in duration. Ventilation fans in each environmental enclosure provided a constant 76-dB (C-scale) background noise.

### Procedure

The critical aspects of the experimental procedure are summarized in Table 1. All training and testing occurred in the same chamber for any given subject.

Table 1
*Design Summary: Experiment 1*

| Group | LI | OV | Test |
|---|---|---|---|
| LI + OV | X | AX → US | X |
| OV | Y | AX → US | X |
| LI | X | X → US | X |
| Acq | Y | X → US | X |
| GenDec | Y | X → US | AX |

*Note.* LI and OV refer to latent inhibition and overshadowing treatment, respectively. Acq and GenDec refer to control groups for assessment of acquisition and generalization decrement, respectively. X and Y denote two stimuli, counterbalanced within groups. A represents the overshadowing conditioned stimulus, and US represents the unconditioned stimulus.

*Acclimation.* On Day 1, each subject was acclimated to its experimental chamber with the lick tube in place. During this 40-min session, subjects were given access to water, and each received two nonreinforced 10-s presentations of the A, X, and Y stimuli with an average intertrial interval (ITI) of 6 min. This brief preexposure to each cue was intended to reduce unconditioned responding to any cue during testing. Without this preexposure procedure, group GenDec would have experienced A for the first time during testing, thereby making unconditioned suppression of drinking likely.

*Phase 1 (latent inhibition).* Prior to the initiation of Phase 1, the lick tube was removed from each chamber. On Days 2–5, subjects received 30 daily presentations of the X stimulus (Groups latent inhibition [LI] + overshadowing treatment [OV] and LI) or the Y stimulus (Groups OV, acquisition [Acq], and GenDec) with an average ITI of 2 min (range = 2–4 min), during these daily 60-min sessions. Group names are explained in the note to Table 1.

*Phase 2 (overshadowing).* During daily 60-min sessions on Days 6 and 7, subjects in Groups LI + OV and OV received five trials consisting of the AX compound followed immediately by footshock. Groups LI, Acq, and GenDec received five trials consisting of the X stimulus followed immediately by shock. Trials occurred at 8, 15, 28, 37, and 50 min into each daily session.

*Reacclimation.* On Days 8 and 9, the lick tubes were reinserted, and subjects were allowed to drink during each of the daily 40-min sessions. This treatment served to restabilize baseline levels of drinking. These sessions did not include any nominal stimulus presentations.

*Testing.* On Day 10, all subjects except those in Group GenDec were tested for responding (i.e., conditioned lick suppression) to X by presenting X immediately on completion of 5 cumulative s of licking (as measured by the total amount of time the infrared photobeam was disrupted). Thus, all subjects were drinking at the time of CS onset. Subjects in Group GenDec were similarly tested for conditioned lick suppression to the AX compound. Time to complete this initial 5 cumulative s of licking and time to complete an additional 5 cumulative s of licking after the onset of the test CS (or compound, Group GenDec) were recorded. Test sessions were 16 min in duration with a ceiling score of 15 min being imposed on the time to complete 5 cumulative s of drinking in the presence of the test CS.

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. Data from 2 subjects from each group were lost because of equipment failure. Additionally, following the convention of our laboratory, subjects that required more than 60 s to complete their first 5 cumulative s of licking (i.e., prior to CS onset), thereby exhibiting an unusual

reluctance to drink in the test context, were eliminated from all analyses. No rat met this criterion in Experiment 1.

### *Results and Discussion*

As can be seen in Figure 5, latent inhibition treatment and overshadowing treatment each produced the expected response deficits. However, both treatments jointly produced strong conditioned responding to the target CS. This confirms our prediction that latent inhibition and overshadowing treatments would counteract one another rather than summate. The following analyses support these conclusions.

A one-way analysis of variance (ANOVA) conducted on pre-CS times to complete 5 cumulative s of licking revealed no significant group differences in baseline drinking behavior in this or any of the following experiments (all $F$s $< 1.0$, $ps > .45$). A one-way ANOVA conducted on suppression scores during the test CS (or compound) revealed a treatment effect, $F(4, 45) = 9.57$. A 2 × 2 ANOVA with Phase 1 treatment (X or Y) and Phase 2 treatment (AX or X) as factors was conducted on suppression scores from the first four groups (Groups LI + OV, OV, LI, and Acq). This revealed an interaction, $F(1, 36) = 29.14$, but no significant main effects, $F$s$(1, 36) < 2.10$, $ps > .15$. Inspection of Figure 5 indicates that this interaction arose from both latent inhibition treatment and overshadowing treatment impairing conditioned responding, but the two treatments together counteracting each other rather than summating. We conducted planned comparisons by use of the overall error term from the one-way ANOVA conducted on CS suppression scores. Subjects in Groups OV and LI suppressed less to X than did subjects in Group Acq, thereby demonstrating overshadowing, $F(1, 45) = 18.37$, and latent inhibition, $F(1, 45) = 6.70$, respectively. Subjects that received the dual latent inhibition and overshadowing treatments (Group LI + OV) suppressed more than did subjects in either Group OV, $F(1, 45) = 20.00$, or Group LI, $F(1, 45) = 7.70$. This

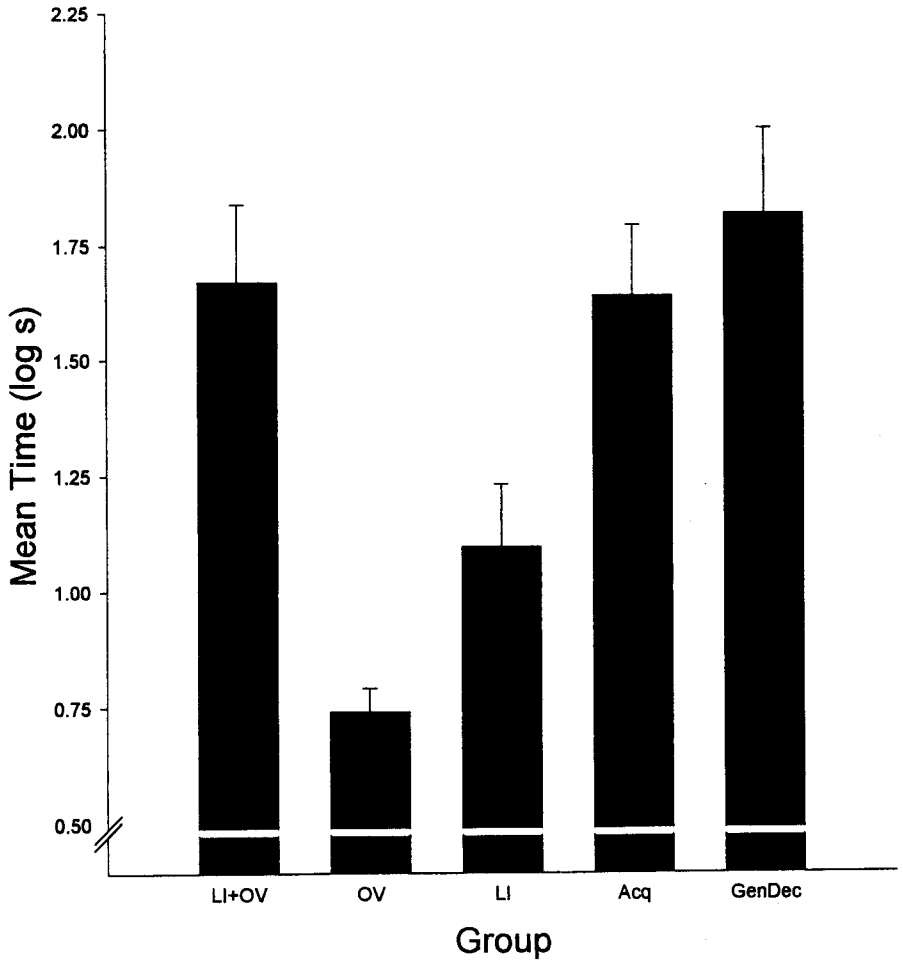


*Figure 5.* Experiment 1: Mean times (log s) to complete 5 cumulative s of licking in the presence of the target conditioned stimulus. All groups were tested on less salient cue, X, except Group generalization decrement (GenDec), which was tested on compound stimulus (AX). Error bars represent standard errors of means. LI = latent inhibition; OV = overshadowing treatment; Acq = acquisition.

indicates that the response-attenuating effect of CS-preexposure and overshadowing treatments together was less than that produced by either treatment alone.

A possible explanation of any overshadowing effect is stimulus generalization decrement as a result of the switch from training with AX to testing with X alone (e.g., Pearce, 1987). To assess generalization decrement as a source of the overshadowing deficit in responding observed in Group OV, we included Group GenDec, which was trained with X alone and tested with AX. Thus, both Groups OV and GenDec experienced transitions from AX or X alone to X alone or AX, respectively, between training and testing. Presumably, generalization decrement would be the same in these two groups (see Pearce, 1987, for a defense of this assumption). That Group GenDec suppressed more than Group OV, $F(1, 45) = 26.73$, suggests that generalization decrement was not the mechanism that produced overshadowing in this experiment. The nonsignificant tendency of Group GenDec to suppress more than Group Acq, $F < 1.0$, $p > .36$, may be a result of stimulus intensity dynamism (Hull, 1949), arising from Stimulus A (flashing light) being of high saliency and Group GenDec being tested on AX, whereas Group Acq was tested on X alone.

The stimulus novelty created by adding A during Phase 2 for Group LI + OV possibly served as a context change, which might explain the loss of latent inhibition (Hall, 1991). But if the effects of latent inhibition were attenuated because of a context shift that resulted from the addition of A, testing X alone should have restored the initial context of preexposure and consequently produced attenuated responding to X. Moreover, even if latent inhibition was decremented by a context shift because of the introduction of A, we should at least have observed overshadowing of X by A. Instead of overshadowing alone or overshadowing summated with latent inhibition, we observed stronger responding to X in Group LI + OV relative to Group OV. Furthermore, the hypothesis that adding A to X in Phase 2 acted as a context change for Group LI + OV cannot explain the results of Experiments 3 and 4 (see below). Therefore, we discount the possibility that this mechanism appreciably contributed to the preexposure–overshadowing effect.

## Experiment 2

The results of Experiment 1 support the prediction of the comparator hypothesis that overshadowing and latent inhibition should counteract each other. This outcome is counterintuitive in the framework of every other model of Pavlovian conditioning. Specifically, our prediction that preexposure to X caused the context rather than A to act as X's comparator at test is congruent with the outcome of Experiment 1. However, support for our interpretation of the present results would be strengthened by additional evidence that the cause of the observed effect was the manipulation of which nontarget stimulus (A or the context) acted as the primary comparator stimulus for X. If the preexposure-overshadowing effect did arise from the CS-preexposure treatment's causing the context to serve as the primary comparator stimulus for X, then posttraining manipulation of the associative status of the context should have an effect on responding to X, whereas posttraining manipulation of the associative status of A should have no effect on responding to X. Experiment 3 was designed to examine this prediction; Experiment 2 was performed to establish parameters for Experiment 3.

Posttraining deflation of the associative value of X's potential primary comparator stimuli would not provide a sensitive test because X was already eliciting a strong response in Group LI + OV of Experiment 1. (Posttraining associative deflation of a CS's primary comparator stimulus is known to enhance conditioned responding to the target CS.) Consequently, posttraining inflation of the associative value of X's potential comparator stimuli appeared to be the manipulation of choice. Posttraining inflation of the comparator stimulus would be expected to make the comparator stimulus more effective as a comparator stimulus for the target CS, thereby attenuating responding to the CS. However, posttraining inflation effects (e.g., decreased conditioned responding to the target CS) have proved difficult to obtain (e.g., Miller, Hallam, & Grahame, 1990; but see Miller & Matute, 1996). Recent evidence suggests that this difficulty reflects the conservative strategy of a strong resistance on the part of subjects to decrementing (but not to incrementing) the conditioned response potential of a stimulus after it has been acquired. Denniston, Miller, and Matute (1996) and Miller and Matute (1996) circumvented this problem in detecting effects of posttraining inflation of comparator stimuli by embedding both target CS training and posttraining inflation of the comparator stimulus within Phase 1 of a sensory preconditioning procedure (Rizley & Rescorla, 1972). Denniston et al. (1996) accomplished this with a backward blocking procedure (i.e., AX → US followed by A → US) embedded within a sensory preconditioning procedure. They paired the AX compound with a neutral Stimulus B (i.e., AX → B) in Phase 1 followed by further pairings of A with B (i.e., A → B) in Phase 2 of treatment. Thus, B served as a surrogate US. In Phase 3, B was paired with a conventional US, and subjects were subsequently tested for conditioned responding to X (the blocked CS). Thus, in Denniston et al.'s procedure, Phases 1 and 2 corresponded with Phase 1 of sensory preconditioning, and Phase 3 corresponded with Phase 2 of sensory preconditioning. This ploy allowed the assessment of posttraining inflation in a situation that did not at that time support conditioned responding (i.e., none of the stimuli during training or posttraining associative inflation was biologically significant).

In the present research, we applied the same ploy to the preexposure-overshadowing effect. If CS-preexposure, overshadowing, and inflation of the potential comparator stimuli are sequentially embedded in Phase 1 of sensory preconditioning (i.e., before a biologically significant US is presented), on the basis of the Denniston et al. (1996) and Miller and Matute (1996) findings, posttraining inflation of X's comparator stimulus should be effective in decrementing subsequent conditioned responding to X. Specifically, inflating the associative value of the experimental context (Figure 4, Link 3b) should produce a decrement in respond-

ing to X. Moreover, inflating A's associative strength (Figure 4, Link 3a) should have no effect on responding to X because A is presumably not the primary comparator stimulus for X because of a weak within-compound X–A association relative to the X–context association. Demonstration of these predicted outcomes would provide strong support to the comparator hypothesis's view that the preexposure-overshadowing effect arises from CS-preexposure altering the within-compound association between X and its potential comparator stimulus during the subsequent overshadowing treatment. The purpose of Experiment 2 was only to replicate the results of Experiment 1 by use of a sensory preconditioning procedure (i.e., there was no posttraining inflation manipulation), thereby facilitating our examination of the effects of posttraining inflation of A or the context in Experiment 3. If our hypothesis that the context, and not A, served as the primary comparator stimulus for X in Group LI + OV of Experiment 1, then in Experiment 3, inflation of the context, but not of A, should decrement responding to X in an otherwise comparably treated group.

The critical aspects of Experiment 2 are depicted in Table 2. Phase 1 consisted of preexposure to the target CS or an alternate stimulus (X and Y). During Phase 2, Groups LI + OV and OV received overshadowing treatment in which the AX compound was followed by B, which served as the surrogate US (i.e., AX → B), whereas all other groups received noncompound pairings of X with B (i.e., X → B). In Phase 3, all subjects received 4 days of handling (to equate retention intervals and handling between training and testing for Experiments 2 and 3). In Phase 4, subjects received trials in Context 2 in which B was paired with the footshock US (i.e., B–US) in order to give B aversive attributes, thereby motivating behavior on the subsequent test trial. This was administered in a different context in order to avoid inflating the associative value of the training context at the same time B was given motivational value. Subsequently, all subjects were tested in the context used for the B–US pairings. Each group was tested for suppression of drinking by the target stimulus (X), except for Group GenDec, which was tested on the AX compound. This last group served as a control for a generalization decrement interpretation of overshadowing in our sensory-preconditioning preparation.

Table 2
*Design Summary: Experiment 2*

| Group | $[LI]_1$ | $[OV]_1$ | Interval | $[B–US]_2$ | $[Test]_2$ |
|---|---|---|---|---|---|
| LI + OV | X | AX → B | handle | B → US | X |
| OV | Y | AX → B | handle | B → US | X |
| LI | X | X → B | handle | B → US | X |
| Acq | Y | X → B | handle | B → US | X |
| GenDec | Y | X → B | handle | B → US | AX |

*Note.* LI and OV refer to latent inhibition and overshadowing treatment, respectively. Acq and GenDec refer to control groups for assessment of acquisition and generalization decrement, respectively. The subscript numbers to the right of the brackets denote the context in which the treatment occurred. X and Y denote two stimuli, counterbalanced within groups. A represents the overshadowing conditioned stimulus. B represents the surrogate unconditioned stimulus (US) used for sensory preconditioning.

## Method

### Subjects and Apparatus

Thirty male (340–440 g) and 30 female (230–310 g) Sprague–Dawley rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Subjects were randomly assigned to one of five groups (*ns* = 12), counterbalanced for sex. The animals were housed and maintained as in Experiment 1. The apparatus and stimuli were identical to those used in Experiment 1 except where otherwise noted.

There were two sets of contexts. Context 1 was the same as that used in Experiment 1 with the addition of a Plexiglas plate covering the grid floor. Context 2 consisted of a different instance of Context 1, and additionally an odor cue (2 drops of methyl salicylate on a wooden block) was present, and the floorplate was removed. Additionally, a third 45-Ω speaker in both contexts was able to deliver a 6-s click train stimulus 8 dB(C) above the background sound level. This 10-s click train served as the surrogate US (B) required for sensory preconditioning. Previous experiments performed in our laboratory have determined that these parameters served to make the clicks an effective surrogate US in a sensory preconditioning design (Denniston et al., 1996; Miller & Matute, 1996).

### Procedure

The critical aspects of the training procedure are summarized, and group names are explained in Table 2. All aspects of the procedure were the same as in Experiment 1, except where otherwise noted.

*Acclimation.* On Day 1, each subject was acclimated to Context 1 as in Experiment 1, except that the lick tube was absent. On Day 2, each subject was acclimated to Context 2 in the same manner as with Context 1 on the preceding day, except that the water-filled lick tube was present. This latter session served to establish licking behavior in what would subsequently serve as the test context.

*Phase 1 (preexposure).* On Days 3–6, subjects received exposure to either X or Y (see Table 2) in Context 1 as in Experiment 1.

*Phase 2 (overshadowing).* On Days 7 and 8, subjects received training in Context 1 as in Experiment 1, except that all CSs were followed by Stimulus B (i.e., clicks) rather than the footshock US.

*Phase 3 (handling).* On Days 9–12, all subjects were placed in a transport cart, taken to the experimental room, handled there for 30 s, and immediately returned to the colony. This procedure served to match retention intervals and handling between Experiments 2 and 3.

*Phase 4 (making Stimulus B biologically significant).* On Days 13 and 14, all subjects received four trials per day of the clicks (B) followed immediately by shock in Context 2. This treatment served to make B biologically significant and facilitate subsequent responding to X.

*Reacclimation.* On Days 15 and 16, subjects received exposure to Context 2 as in Experiment 1.

*Testing.* On Day 17, subjects were tested for conditioned lick suppression to X (or AX for Group GenDec) in Context 2 by use of the same procedure as in Experiment 1. No animal took longer than 60 s to complete their first 5 cumulative s of licking (i.e., prior to CS onset).

### *Results and Discussion*

Experiment 2 successfully replicated the pattern of results obtained in Experiment 1 within a sensory preconditioning preparation. The central observations from Experiment 2 were that the preexposure-overshadowing treatment of latent inhibition, followed by overshadowing, produced strong conditioned responding to the target CS, whereas either treatment alone produced attenuated conditioned responding. These outcomes are illustrated in Figure 6. The following analyses support these conclusions.

A one-way ANOVA conducted on suppression scores to complete 5 cumulative s of licking in the presence of the target CS (or compound) revealed a main effect of training, $F(4, 55) = 17.83$. A $2 \times 2$ ANOVA with the first factor being Phase 1 treatment (X or Y) and the second factor being Phase 2 treatment (AX or X) conducted on the data from the first four groups (Groups LI + OV, OV, LI, and Acq) found an interaction, $F(1, 44) = 46.78$, whereas no other effects were significant, $Fs(1, 44) < 1.0$, $ps > .37$. As is evident in Figure 6, this interaction arose from latent inhibition and overshadowing treatments counteracting each other, rather than their response attenuating effects summating. Planned comparisons were conducted by use of the overall error term from the one-way ANOVA. Subjects in Groups OV and LI suppressed less to X than did subjects in Group Acq, thereby demonstrating overshadowing, $F(1, 55) = 23.10$, and latent inhibition, $F(1, 55) = 28.13$, respectively. Subjects that received both latent inhibition and overshadowing (Group LI + OV) suppressed more than did subjects in either Group OV, $F(1, 55) = 16.19$, or Group LI, $F(1, 55) = 20.45$. The difference in performance between Groups Acq and GenDec was not significant, $F(1, 55) < 1, p > .17$; thus, overshadowing in this instance cannot readily be viewed as resulting from generalization decrement. The results of Experiment 2,

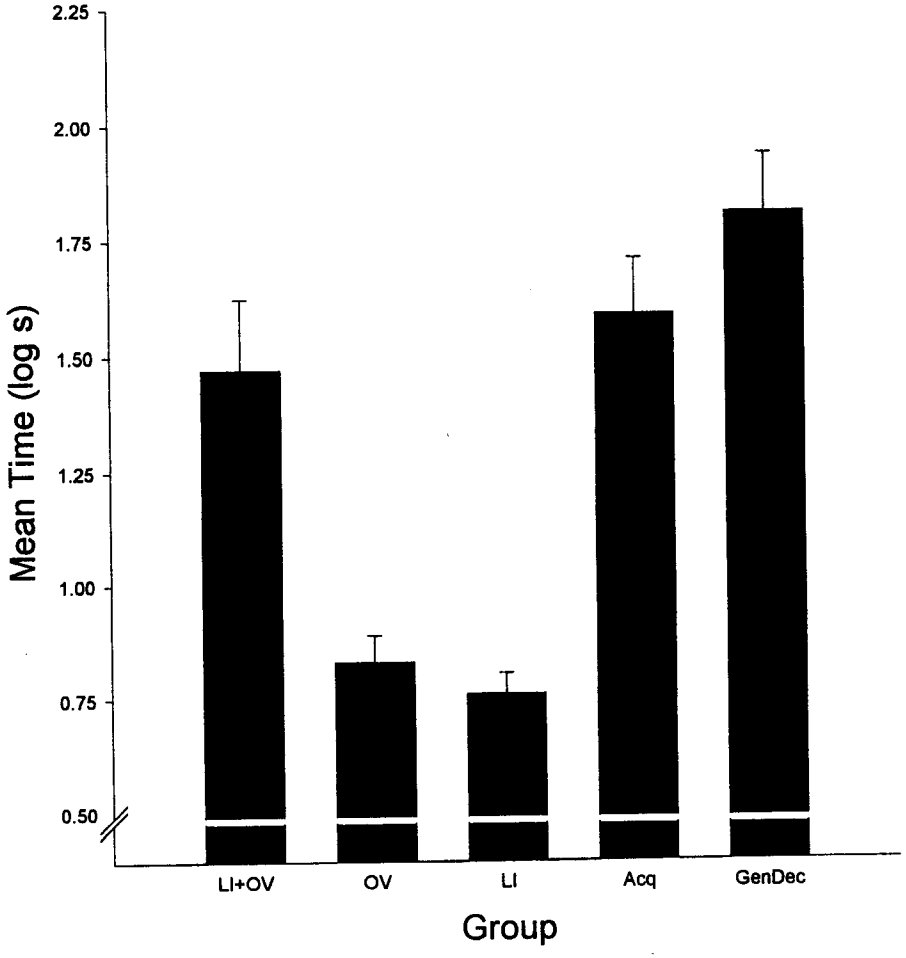


*Figure 6.* Experiment 2: Mean times (log s) to complete 5 cumulative s of licking in the presence of the target conditioned stimulus. All groups were tested on less salient cue, X, except Group generalization decrement (GenDec), which was tested on compound stimulus (AX). Error bars represent standard errors of means. LI = latent inhibition; OV = overshadowing treatment; Acq = acquisition.

which included a sensory preconditioning procedure, replicated the findings of Experiment 1. This outcome indicates the robustness of the preexposure-overshadowing effect.

## Experiment 3

On the basis of the finding of a preexposure-overshadowing effect in Experiment 2, in Experiment 3 we used a sensory preconditioning procedure for the purpose of inflating either A or the context to assess which of these two stimuli served as the comparator stimulus for X in each of the critical groups of Experiment 2. The specific purpose of Experiment 3 was to determine through a posttraining inflation manipulation the identity of the primary comparator stimulus for the focal experimental group (Group LI + OV). This was done by posttraining inflation of either the training context alone or stimulus A within the training context. If preexposure to X established the context (i.e., Link 2b of Figure 4) and not A (i.e., Link 2a of Figure 4) as X's primary comparator stimulus, then we expected inflation of the context (i.e., of Link 3b of Figure 4) to produce a decrement in conditioned responding to X, whereas inflation of A (i.e., Link 3a of Figure 4) should have no such effect.

The critical aspects of this experiment are depicted in Table 3. A 2 × 2 × 2 design, with an extra control group, LI + OV (N), was used in which Phase 1 consisted of CS preexposure to either the target or an alternate stimulus (X or Y, respectively); Phase 2 consisted of either overshadowing training of an association to B (AX → B) or simple training of an association to B (X → B); and Phase 3 consisted of either inflation of the A–B association in the training context (A → B) or inflation of the training context alone (B). Subsequently, in Phase 4 subjects received B–US pairings in Context 2 to make B biologically significant. Finally, all subjects were tested in Context 2 for suppression to the target stimulus (X). Group LI + OV (N) received preexposure to X in Phase 1, followed by AX → B overshadowing training in Phase 2, followed by handling alone in Phase 3.

Table 3
*Design Summary: Experiment 3*

| Group | $[LI]_1$ | $[OV]_1$ | $[Inflation]_1$ | $[B–US]_2$ | $[Test]_2$ |
|---|---|---|---|---|---|
| LI + OV (N) | X | AX → B | None | B → US | X |
| LI + OV (A) | X | AX → B | A → B | B → US | X |
| OV (A) | Y | AX → B | A → B | B → US | X |
| LI (A) | X | X → B | A → B | B → US | X |
| Acq (A) | Y | X → B | A → B | B → US | X |
| LI + OV (Con) | X | AX → B | B | B → US | X |
| OV (Con) | Y | AX → B | B | B → US | X |
| LI (Con) | X | X → B | B | B → US | X |
| Acq (Con) | Y | X → B | B | B → US | X |

*Note.* LI and OV refer to latent inhibition and overshadowing treatment, respectively. N, A, and Con refer to no inflation, inflation of the A–B association, and inflation of the Context 1–B association, respectively. The subscript numbers to the right of the brackets denote the context in which the treatment occurred. X and Y denote two stimuli, counterbalanced within groups. A represents the overshadowing conditioned stimulus. B represents the surrogate unconditioned stimulus (US) used for sensory preconditioning. Acq represents acquisition.

### Method

#### Subjects and Apparatus

Sixty male (300–545 g) and 57 female (220–340 g) Sprague–Dawley rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to one of nine groups (*ns* = 13), counterbalanced for sex. The animals were housed and maintained as in Experiments 1 and 2. The apparatus and stimuli were the same as those used in Experiments 1 and 2 except where otherwise noted. Because of the large number of subjects, the number of experimental chambers was increased to 24, with the 12 added chambers being identical to those used in Experiments 1 and 2.

#### Procedure

The critical aspects of the training procedure are summarized and group names are explained in Table 3.

On Days 1 and 2, all subjects received acclimation to the experimental chambers as in Experiment 2. On Days 3–6, subjects received exposure to either X or Y as in Experiments 1 and 2. On Days 7 and 8, subjects received overshadowing treatment as in Experiment 2. On Days 9–12, subjects in Group LI + OV (N) received handling treatment as in Experiment 2. Subjects in Groups LI + OV (Con), OV (Con), LI (Con), and Acq (Con) received 5 nonreinforced B trials alone per day in Context 1 for a total of 20 trials (i.e., inflation of the training context-B association). Subjects in Groups LI + OV (A), OV (A), LI (A), and Acq (A) received 5 A → B trials per day for a total of 20 trials in Context 1 (i.e., inflation of the A–B association). Presumably, A at least partially overshadowed the context on these trials, minimizing the accompanying inflation of the context-B association. All trials occurred 8, 15, 28, 37, and 55 min into the daily 60-min sessions. On Days 13 and 14, all subjects received B–US pairings as in Experiment 2. On Days 15 and 16, subjects were reacclimated to Context 2 as in Experiment 2. On Day 17, subjects were tested for conditioned lick suppression to X as in Experiments 1 and 2. Data from one animal in Group Acq(Con) were deleted due to illness. No animals took longer than 60 s to complete their first 5 cumulative s of licking (i.e., prior to CS onset).

#### Results and Discussion

Experiment 3 successfully replicated the results of Experiment 2 in that Group LI + OV (N), which received preexposure-overshadowing training followed by no posttraining inflation treatment, displayed strong responding (i.e., the preexposure-overshadowing effect). The central observations from Experiment 3 were that inflation of A had no attenuating effect on conditioned responding in the preexposure-overshadowing condition (i.e., LI + OV). In contrast, inflation of the context produced a strong decrement in conditioned responding in the preexposure-overshadowing condition. The outcomes of these manipulations provide direct evidence supporting the comparator hypothesis's explanation of the preexposure-overshadowing effect. These outcomes are illustrated in Figure 7.

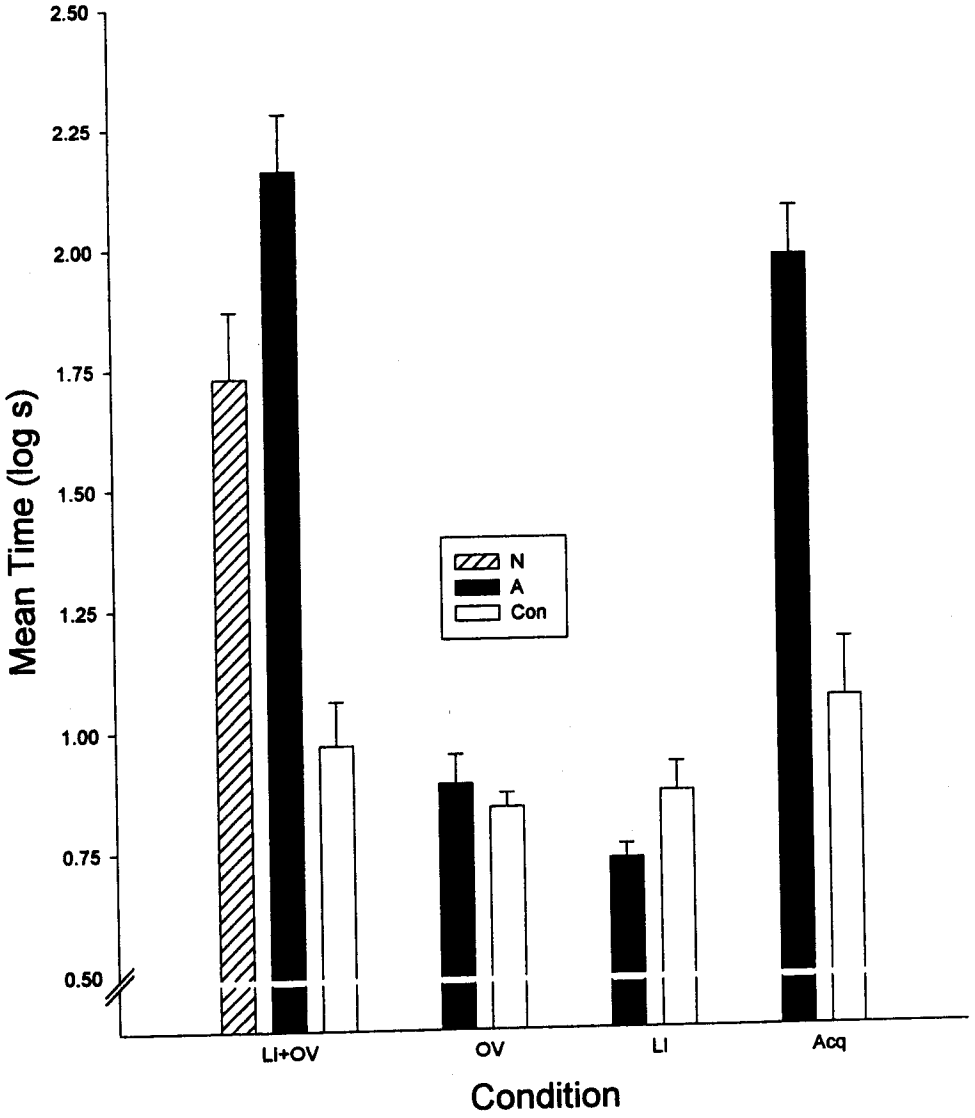


*Figure 7.* Experiment 3: Mean times (log s) to complete 5 cumulative s of licking in the presence of the target conditioned stimulus. All groups were tested on less salient cue, X. Error bars represent standard errors of means. LI = latent inhibition; OV = overshadowing treatment; Acq = acquisition; N = no inflation; A = inflation of the A–B association; Con = inflation of the context 1–B association.

A one-way ANOVA conducted on time to complete 5 cumulative s of licking in the presence of the target CS revealed an overall effect of treatment, $F(8, 107) = 33.92$. A 2 × 2 × 2 ANOVA (Phase 1, Phase 2, and inflation treatments as factors) conducted on the data with Group LI + OV (N) omitted revealed the following: (a) a main effect of inflation treatment (A vs. context), $F(1, 95) = 67.18$; (b) a two-way interaction between Phase 1 (latent inhibition) and Phase 2 (overshadowing) treatments, $F(1, 95) = 132.92$; and (c) a three-way interaction between all factors, $F(1, 95) = 79.69$. No other effects were significant, $Fs(1, 95) < 3.45$, $ps > .065$. Planned comparisons, which used the overall error term from the one-way ANOVA, were then used to determine the sources of these differences.

The first planned comparison found that Group LI + OV (Con) suppressed less than did Group LI + OV (N), $F(1, 107) = 32.02$. Moreover, Group LI + OV (A) did not suppress less than did Group LI + OV (N); in fact, the difference was in the opposite direction (see later discussion and Figure 7). Thus, posttraining inflation of the training context in Group LI + OV (Con), but not posttraining inflation of A in Group LI + OV (A), succeeded in attenuating responding to X relative to Group LI + OV (N).

This indicates that the training context (Context 1) and not A served as the comparator stimulus for X in the preexposure-overshadowing condition (i.e., LI + OV).

Examination of the remaining data (see Figure 7) found no effect of inflating either A or the context in Conditions OV and LI. That is, Groups OV (A) and LI (A) suppressed less to X than did Group Acq (A), $Fs(1, 107) = 66.61$ and 87.42, respectively, and Group LI + OV (A), $Fs(1, 107) = 91.13$ and 114.59, respectively). Presumably, this occurred because responding to X was already low without inflation, because of overshadowing in Condition OV and latent inhibition in Condition LI. Therefore, a floor effect likely operated here, masking any potential effects of inflation of comparator stimuli. However, prior studies that used post-training deflation (rather than inflation) treatment identified the context as the comparator stimulus for the CS in latent inhibition studies (Grahame et al., 1994) and the overshadowing cue (A) as the comparator stimulus in overshadowing studies (e.g., Kaufman & Bolles, 1981; Matzel et al., 1985).

In Condition Acq, inflation of the training context did decrease responding relative to inflation of A, $F(1, 107) = 45.70$. A was not presented during training in this condition, so Group Acq (A) can be viewed as a control group that indicates full expression of the X–US association acquired during training. The decrease in responding to X produced by posttraining inflation of the context identifies the training context as the comparator stimulus for X when X is trained alone, as is predicted by the comparator hypothesis.

A post hoc comparison that used the overall error term from the one-way ANOVA revealed that Group LI + OV (N) suppressed less than Group LI + OV (A), $p < .0045$ (Newman–Keuls). There were no a priori expectations that these two groups would differ in this direction. Rather, in the framework of the comparator hypothesis, inflation of A was expected to decrease responding to X relative to Group LI + OV (N) if it were to have any effect at all. This increase in responding suggests second-order conditioning of X mediated by A. The comparator hypothesis posits the existence of both X–A and A–US associations but anticipates a resultant impairment in responding to X on the basis of the X–US association as a result of the X–A (and A–US) association. Thus, the comparator hypothesis is unable to explain second-order conditioning. Seemingly, an explanation of second-order conditioning must be left to a theory of learning, whereas the comparator hypothesis is a theory of associative expression.

## Experiment 4

The differential effects of inflating potential comparator stimuli observed in Experiment 3 provide supportive evidence for the comparator hypothesis's explanation of the preexposure-overshadowing effect. Experiment 4 was designed to provide another means of testing this explanation. Latent inhibition, which normally retards the acquisition of conditioned responding, has been shown to be relatively context specific (e.g., Bouton, 1993; Channell & Hall, 1983). When a subject receives CS preexposure in one context followed by excitatory training in a different context, the response attenuation constituting the latent inhibition effect is observed only when testing occurs in the preexposure context and not when testing occurs either in the second context or in a novel context (but see Lovibond, Preston, & Mackintosh, 1984). The comparator hypothesis explains CS preexposure as arising from strong CS–context associations that are established during the CS-preexposure treatment (see Link 2 in Figure 2; Grahame et al., 1994). In the framework of the comparator hypothesis, the context specificity of latent inhibition arises from the CS-alone presentations in the preexposure context establishing the preexposure context as the comparator stimulus for the CS. Subsequent CS–US pairings in a second context condition both the CS and the second context, but not the preexposure context, which continues to act as the comparator stimulus for the CS. Thus, the comparator hypothesis predicts that a context shift between preexposure treatment and overshadowing treatment should eliminate the preexposure-overshadowing effect as well as prevent latent inhibition, thereby leaving only the overshadowing deficit. To test this prediction, we gave latent inhibition training in Context 1 and overshadowing training in either the same or a different context (i.e., Contexts 1 or 2, respectively). Subsequently, we tested all animals in a neutral context (Context 3). We expected to observe attenuated responding in the preexposure-overshadowing group that received a context switch between treatment phases relative to the strong responding in the preexposure-overshadowing group lacking a context switch. Consistent with the context specificity of latent inhibition, we anticipated strong responding in a group that received latent inhibition treatment in Context 1 and excitatory training in Context 2. We also included groups to assess the context specificity of overshadowing. The comparator hypothesis predicts that overshadowing will readily transfer from a training context to most any other test context. The two purposes of these manipulations were to further isolate the effects of each treatment (latent inhibition and overshadowing) and to more adequately account for the context's role as the primary comparator stimulus in the preexposure-overshadowing effect.

### *Method*

#### *Subjects and Apparatus*

Forty-eight male (355–550 g) and 48 female (255–370 g) naive, Sprague–Dawley rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to one of eight groups ($ns = 12$). The animals were housed and maintained as in the prior experiments. The apparatus and stimuli were identical to those used in Experiment 3 except where otherwise noted.

Contexts 1 and 2 were the two contexts that were used in Experiment 3, physically counterbalanced within groups as to which served as Context 1 and which served as Context 2. Context 3 consisted of using a different instance of the same rectangular chambers (Context 1 for half the subjects in each group and Context 2 for the other half), but with Plexiglas plates covering the

grid floor, a banana-scented odor cue (distinctly different from methyl salicylate) being present, and the house light turned off.

## Procedure

The design of Experiment 4 is depicted and group names are explained in Table 4.

*Acclimation.* On Day 1, subjects received acclimation to the context condition that consisted of the experimental chamber with no odor cue (Chamber 1 or 2, counterbalanced). One nonreinforced 10-s presentation each of the A, X, and Y (light, noise, and tone) stimuli, with an average ITI of 10 min, was given in this 40-min session. Lick tubes were absent. On Day 2, subjects received acclimation to the experimental context with the methyl salicylate odor (Chamber 2 or 1). All other manipulations were identical to Day 1 treatment. On Day 3, subjects were acclimated to Context 3 (which would later serve as the test context). Day 3 treatment was the same as treatment on Days 1 and 2, except for context, the absence of any nominal stimulus presentations, and the presence of lick tubes.

*Phase 1 (preexposure).* On Days 4–7, subjects received exposure to either X or Y in Context 1 and equal exposure to Context 2 without X or Y being present, as in the prior experiments.

*Phase 2 (overshadowing).* On Days 8 and 9, subjects received training (X–US or AX–US) as in Experiment 1, except that half of the subjects in each group received this training in the same context as preexposure (i.e., Context 1), and the other half received it in Context 2. Additional exposure to Contexts 2 and 1, respectively, equated subjects' total exposure to these two contexts.

*Reacclimation.* On Days 10 and 11, subjects received exposure to Context 3 as in the prior experiments.

*Testing.* On Day 12, subjects were tested for conditioned lick suppression to X in Context 3 as in the prior studies.

One subject in Group LI (switch) was eliminated prior to testing due to illness. Nine animals (1 each from Groups LI [same], OV [same], and OV [switch], and 2 each from Groups LI [switch], Acq [same], and Acq [switch]) failed to complete their first 5 cumulative s of licking in 60 s; thus, their data were eliminated from the study.

Table 4
*Design Summary: Experiment 4*

| Group | Latent inhibition treatment | Overshadowing treatment | Test |
|---|---|---|---|
| LI + OV (same) | $[X]_1[\ ]_2$ | $[AX \rightarrow US]_1[\ ]_2$ | $[X]_3$ |
| OV (same) | $[Y]_1[\ ]_2$ | $[AX \rightarrow US]_1[\ ]_2$ | $[X]_3$ |
| LI (same) | $[X]_1[\ ]_2$ | $[X \rightarrow US]_1[\ ]_2$ | $[X]_3$ |
| Acq (same) | $[Y]_1[\ ]_2$ | $[X \rightarrow US]_1[\ ]_2$ | $[X]_3$ |
| LI + OV (switch) | $[X]_1[\ ]_2$ | $[\ ]_1[AX \rightarrow US]_2$ | $[X]_3$ |
| OV (switch) | $[Y]_1[\ ]_2$ | $[\ ]_1[AX \rightarrow US]_2$ | $[X]_3$ |
| LI (switch) | $[X]_1[\ ]_2$ | $[\ ]_1[X \rightarrow US]_2$ | $[X]_3$ |
| Acq (switch) | $[Y]_1[\ ]_2$ | $[\ ]_1[X \rightarrow US]_2$ | $[X]_3$ |

*Note.* LI and OV refer to latent inhibition and overshadowing treatments, respectively. The subscript numbers to the right of the brackets denote the context in which the treatment occurred. The word at the end of each group name denotes whether overshadowing treatment occurred in the same or a switched context relative to latent inhibition treatment. X and Y denote two stimuli, counterbalanced within groups. A represents the overshadowing conditioned stimulus. Empty brackets $[\ ]_1$ and $[\ ]_2$ refer to time spent in Contexts 1 and 2, respectively, without the presentation of any nominal stimulus. US represents unconditioned stimulus, and Acq denotes acquisition.

## Results and Discussion

Experiment 4 successfully replicated the preexposure-overshadowing effects in that Group LI + OV (same), which received latent inhibition and overshadowing treatment in the same context, responded as strongly as did the excitatory control group (Group Acq [same]). The central observation from Experiment 4 was that the context switch between latent inhibition treatment and overshadowing treatment produced a strong decrement in conditioned responding relative to subjects that received both phases of training in the same context. This outcome is illustrated in Figure 8. Thus, the preexposure-overshadowing effect was attenuated by the context shift. These observations directly support the comparator hypothesis's interpretation of the preexposure-overshadowing effect.

A 2 (preexposure: X vs. Y) × 2 (overshadowing treatment: AX vs. X) × 2 (context: same vs. switch) ANOVA conducted on time to complete 5 cumulative s of licking in the presence of X revealed a main effect of Phase 1 preexposure treatment, $F(1, 78) = 4.76$, and Phase 2 overshadowing treatment, $F(1, 78) = 9.18$, as well as interactions between preexposure and overshadowing treatments, $F(1, 78) = 11.45$, and overshadowing treatment and context, $F(1, 78) = 11.82$. All other factors including the three-way interaction were not significant, $F$s$(1, 78) < 0.53$, $ps > .40$.

A series of planned comparisons were conducted by use of the overall error term from the ANOVA. Groups OV (same) and OV (switch) suppressed less than did Groups Acq (same) and Acq (switch), $F$s$(1, 78) = 4.23$ and 19.20, respectively, demonstrating overshadowing with and without a context shift. Group LI (same) suppressed less than did Group Acq (same), $F(1, 78) = 8.53$, demonstrating latent inhibition without a context shift. Moreover, Group LI (switch) suppressed more than did Group LI (same), indicating that the context shift did attenuate the latent inhibition effect, $F(1, 78) = 4.49$. However, there was still an effect of CS preexposure in Group LI (switch) relative to Group Acq (switch), $F(1, 78) = 6.21$, demonstrating that the latent inhibition effect was not completely degraded by the context switch manipulation. Perhaps this arose from some limited similarity, and hence stimulus generalization, between our contexts. Group LI + OV (same) suppressed more than Group LI (same), $F(1, 78) = 6.14$, which evidenced attenuation of a latent inhibition effect in Group LI + OV (same) as a result of overshadowing treatment. Moreover, Groups LI + OV (same) and Acq (same) both exhibited strong and equivalent responding, $F(1, 78) < 1.0$, $p > .55$. Thus, consistent with the prior experiments, the preexposure-overshadowing treatment of latent inhibition and overshadowing appear to have nearly fully counteracted each other, although it should be noted that the difference between Group LI + OV (same) and Group OV (same) did not quite reach statistical significance, $F(1, 78) = 2.17$, $p > .10$. Group LI + OV (switch) suppressed less than did Group Acq (switch), $F(1, 78) = 22.16$, suggesting that the prevention of a latent inhibition effect as a result of changing contexts between Phases 1 and 2 allowed the occurrence of

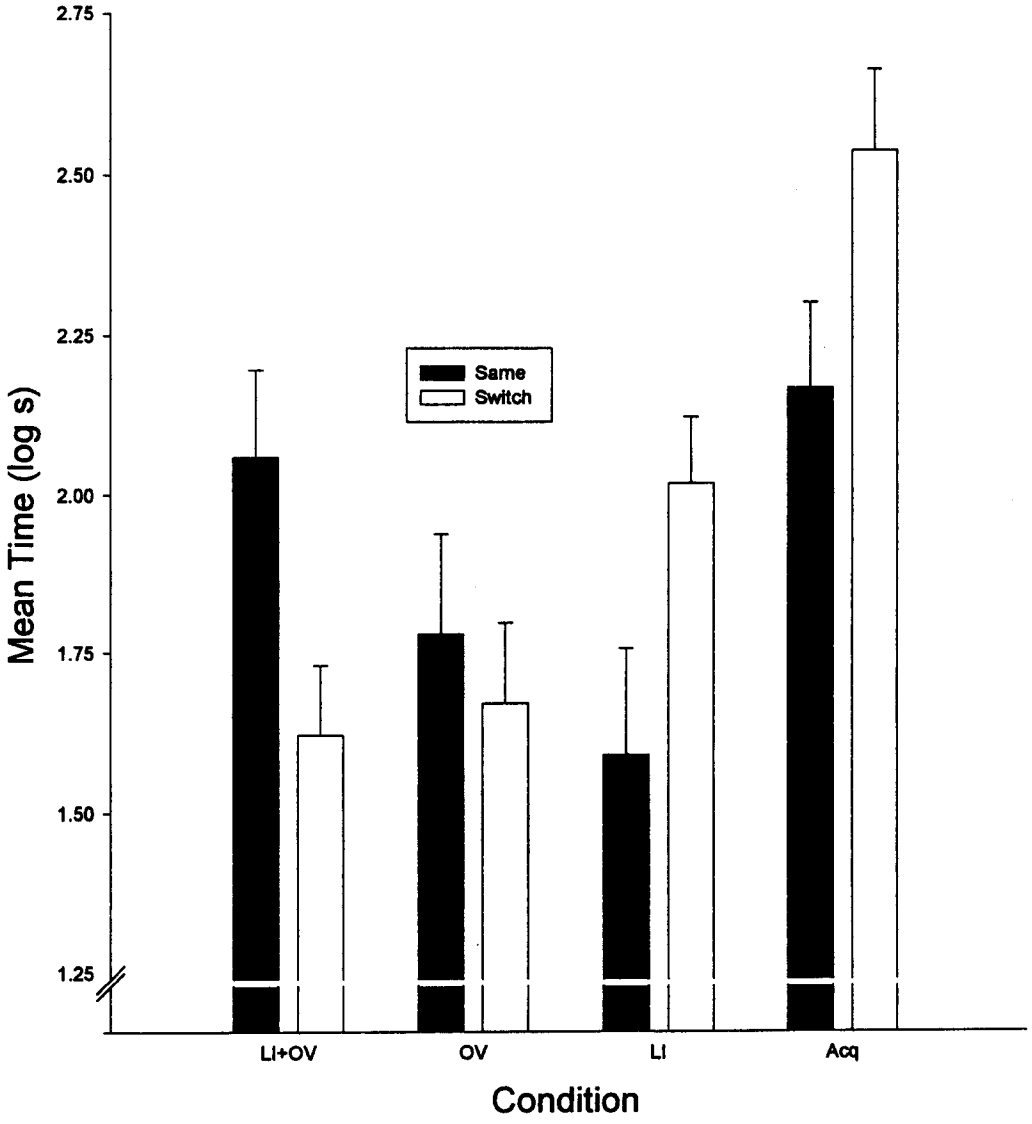


*Figure 8.* Experiment 4: Mean times (log s) to complete 5 cumulative s of licking in the presence of the target conditioned stimulus. All groups were tested on less salient cue, X. Error bars represent standard errors of means. LI = latent inhibition; OV = overshadowing treatment; Acq = acquisition.

overshadowing in Group LI + OV (switch). The reduction in the magnitude of the latent inhibition effect that was due to the context shift appears to have allowed the occurrence of overshadowing in Group LI + OV (switch) relative to the somewhat unimpaired excitatory responding observed in Group LI (switch), $F(1, 78) = 3.99$. Last, Groups Acq (same) and Acq (switch) did not differ, $F(1, 78) = 3.29$, $p = .07$, indicating that the apparent recovery from latent inhibition in Group LI + OV (switch) was not due to increased responding in Group Acq (switch).

## General Discussion

Experiment 1 showed that latent inhibition treatment followed by overshadowing treatment attenuated the response-diminishing effects of either treatment alone. We call the strong responding observed as a result of combining these two treatments the preexposure-overshadowing effect. The preexposure-overshadowing effect is anticipated by the comparator hypothesis and challenges all other contemporary models of Pavlovian conditioning, including both US-focused (e.g., Rescorla & Wagner, 1972) and CS-focused (e.g., Mackintosh, 1975; Pearce & Hall, 1980) models. Experiment 2 replicated the preexposure-overshadowing effect with a sensory preconditioning procedure, and Experiment 3 used a posttraining inflation of the comparator stimulus procedure to provide additional evidence, supporting the view that the preexposure-overshadowing effect was the product of altering the stimulus that served as the

primary comparator stimulus for the target CS. The switching of contexts between latent inhibition and overshadowing treatments in Experiment 4 provided further evidence that the preexposure-overshadowing effect depends on the training context, and not A, serving as the primary comparator stimulus for X. Collectively, these experiments confirm predictions that are based on the comparator hypothesis concerning the interaction of latent inhibition and overshadowing. Thus, these data support the existence of a comparator mechanism such as the one described by the comparator hypothesis (illustrated in Figure 1), and the version of it depicted in Figure 4 is able to explain the preexposure-overshadowing effect.

### *Competition Between Comparator Stimuli*

The comparator hypothesis interpretation of the preexposure-overshadowing effect rests on the context and not on A, serving the preexposure-overshadowing group as the comparator stimulus for X. As mentioned in the introduction, on the basis of application of the comparator hypothesis and previously published data (Grahame, Barnet, & Miller, 1992; Miller et al., 1992), we expected the establishment of the context as the comparator stimulus for X to block A from becoming the primary comparator stimulus for X. Thus, for both the latent inhibition and preexposure-overshadowing groups, the context is acting as the comparator stimulus for X. This explains why overshadowing of X by A was not observed in the preexposure-overshadowing group. But one may ask why latent inhibition of X did not occur in the preexposure-overshadowing group. Toward answering this question, we previously spoke of the presence of A on the AX → US trials attenuating the effective value of context-US association (Link 3), but we have said little regarding the nature of the processes that produce stimulus competition between A and the context as comparator stimuli. A full explanation of the current data requires a detailed explanation of the mechanism underlying this competition. In this article we propose two different mechanisms to explain competition between comparator stimuli. The first is an extension of the comparator hypothesis, and the second borrows a mechanism from another (but not contrary) theoretical framework.

### *Higher-Order Comparator Processes*

Within the framework of the comparator hypothesis, the strength of the direct US representation is determined by the absolute strength of the target CS–US association (i.e., Link 1 of Figures 1 and 4), but Links 2 and 3 (the target CS–comparator stimulus and comparator stimulus-US associations, respectively) are effective associations. That is, the comparator values of Links 2 and 3 are modulated by their own comparator stimuli. These second-order comparator processes determine the effectiveness of the context and of A to attenuate conditioned responding to X.

Specifically, for both Group LI + OV and Group LI of Experiment 1, preexposure to X in the treatment context established the context as the primary comparator stimulus for X. However, the effectiveness of this context in modulating responding to X differed between these two groups because of differential Phase 2 treatments. For Group LI + OV, the presence of A during Phase 2 training presumably allowed A to degrade the effectiveness of the context-US association that was presumably formed during Phase 2 treatment (see Table 1). Alternatively stated, the treatment context-US association that would attenuate responding to X if A had not been present is itself attenuated in its comparator role by the A–US association (i.e., the effectiveness of a comparator stimulus is itself modulated by its own comparator stimulus). (We tentatively assume that a target CS [X] cannot itself modulate the comparator value of its own comparator stimuli.) Thus, the effective strength of the context-US association was inversely related to the product of the context-A association and the A–US association. Because of the high saliency of A and the good contiguity between the context and A and between A and the US, this product should have been of large value, thereby weakening the effective context-US association. As a consequence, despite the strong effective X–context association (Link 2b in Figure 4), the weak effective context-US association (Link 3b in Figure 4) caused the comparator process (with the context as the comparator stimulus for X) to have little impact on expression of the X–US association. In this scenario, A, as the comparator stimulus for the context-US association, degraded the context as an effective comparator stimulus for X, thereby attenuating the latent inhibition effect. That is, although the context was a comparator stimulus for X, the effective context-US association was weak, reducing the potential of the context to modulate responding to X. The primary difference between Group LI + OV and Group LI was the presence of a strong A–US association in Group LI + OV, which reduced the effectiveness of the context as a predictor of the US and hence as a modulator of conditioned responding to X.

### *Interference Theory*

A second mechanism that can explain competition between potential comparator stimuli is interference between retrieval of different associations with common associates (Bouton, 1993; Osgood, 1949; Underwood, 1957). In Group LI + OV of Experiment 1, the strong X–context association created by preexposure treatment may have interfered at test with the activation of the relatively weak X–A association. Thus, A may have been unable to function as a comparator stimulus for X.

Now consider the context's role as a comparator stimulus in Group LI + OV. The context's effectiveness in attenuating responding to X was presumably directly related to the product of the activated X–context and activated context-US associations at test. In this scenario, activation of Comparator Links 2 and 3 can be reduced by interference. The activation of the X–context association was likely strong in Group LI + OV because of latent inhibition treatment, making the X–context association strong relative to the X–A association. But activation of the context-US association was subject to interference by the strong A–US association

(see Matute & Pineño, 1998, for evidence of interference in activating two associations with a common subsequent associate). Thus, although the context likely acted as the primary comparator stimulus for X, the effective context–US association was relatively weak because of interference from the strong A–US association. By contrast, in this interference framework, responding was weak in the latent inhibition group because there was no A–US association to interfere with the context–US association, which serves as Link 3 when the context is the comparator stimulus.

Both of these accounts (i.e., higher-order comparator processes and comparator interference) explain why combining latent inhibition treatment and overshadowing treatment should produce strong conditioned responding, rather than a response deficit that is due to latent inhibition alone, overshadowing alone, or a summation of these two effects. Within the framework of higher-order comparator processes, each comparator stimulus has its own set of comparator stimuli that modulate the effectiveness of that stimulus in serving as a comparator stimulus for other stimuli and modulate directly elicited conditioned responding to that stimulus.[1] According to a comparator interference account, comparator stimuli compete for activation, thereby reducing the effectiveness of the primary comparator stimulus in modulating responding to the target stimulus. Superimposed on the basic comparator hypothesis (see Figure 1), either of these accounts can explain the strong conditioned responding to X that we observed in Group LI + OV.

Furthermore, both mechanisms predict that posttraining associative inflation of the context (i.e., context → US) should make the treatment context a more potent comparator stimulus, thereby attenuating responding to X, as was observed in Experiment 3. Moreover, both mechanisms in conjunction with the comparator hypothesis lead to the prediction of recovery of latent inhibition in the preexposure-overshadowing group if Stimulus A was extinguished. The comparator mechanism anticipates this predicted recovery of latent inhibition as a consequence of A losing its potential to modulate downward the context-US association (i.e., Link 3b of Figure 4). In contrast, the interference model anticipates this predicted recovery of latent inhibition as a consequence of the A–US association losing its potential to interfere with the activation of context-US association in the comparator process (again, Link 3b) because of the superior retrievability of an A–no US association.

### *A Potentiation Explanation*

A possible alternative to either of the above competition explanations of the preexposure-overshadowing effect is that preexposure to X may have allowed A to potentiate X in entering into an X–US association during the subsequent overshadowing treatment. The argument has been made for the potentiation of odors by tastes (Bouton, Jones, McPhillips, & Swartzentruber, 1986; Palmerino, Rusiniak, & Garcia, 1980) and tastes by tastes (Bouton, Dunlap, & Swartzentruber, 1987) that potentiation results from the to-be-potentiated odor or taste being weakly conditionable at the time of compound stimulus pairings with the US (drug- or radiation-induced illness in taste aversion preparations). The weakly conditionable cue enters into a within-compound association with the stronger, more conditionable cue, which mediates strong responding to the weaker stimulus at test. Stimulus preexposure is one method that has been shown to reliably make a stimulus weakly conditionable (i.e., the latent inhibition effect). Thus, as an explanation of the preexposure-overshadowing effect, preexposure to X may have caused X to become weakly conditionable, thus allowing A to potentiate, rather than overshadow, X. However, there are several reasons why we must reject this alternative interpretation.

One reason for rejecting a potentiation explanation of the preexposure-overshadowing effect is that the outcome of Experiment 3 obviates such an interpretation. If strong responding to X in the preexposure-overshadowing group was due uniquely to the formation of a strong X–A within-compound association thereby allowing A to potentiate responding to X, then inflation of the training context should have had little effect on responding to X unless potentiation and comparator mechanisms, which modulated the effective A–US association, were both assumed to be involved, which is a decidedly unparsimonious position. However, inflation of the training context was seen to abolish the otherwise strong responding observed to X. Thus, it appears that a comparator mechanism was involved, which makes potentiation a superfluous process because a comparator process is both necessary in any case and is capable of explaining all of the present observations. Note that inflation of A did produce a small but significant increase in responding to X, providing some evidence that A may have a small potentiation effect on X. But that inflation of the training context produced a much larger effect on responding to X than did inflating A (see Figure 7), provides evidence favoring a comparator explanation over a potentiation explanation of the preexposure-overshadowing effect.

A second reason for rejecting a potentiation explanation of the preexposure-overshadowing effect is evident in Bouton et al.'s (1986) finding that, although preexposure to an otherwise conditionable odor produced latent inhibition of that odor (i.e., the odor alone was retarded in accruing control of behavior), preexposure to the odor in their study did not allow a taste to potentiate the odor during subsequent compound odor–taste aversion treatment. Thus, a potentiation explanation of the preexposure-overshadowing effect appears relatively implausible. However, more research is needed before a final judgment of the explanation is possible.

## *Conclusions*

The comparator hypothesis (Miller & Matzel, 1988; Miller & Schachtman, 1985) provides a qualitative response

[1] A third-order application of the comparator process is of course a possibility. But we assume that it would be of little or no consequence in determining the effectiveness of Links 2 and 3 of the second-order comparator process because of a damping effect that occurs with each higher order progression of the comparator process.

rule that was thought to uniquely predict the effect of posttraining inflation and deflation of comparator stimuli (i.e., retrospective revaluation) that have been reported in recent years. However, there are several recent revisions of the Rescorla–Wagner (1972) model (e.g., Van Hamme & Wasserman, 1994) that succeeded in explaining most of the reported instances of retrospective revaluation in terms of new values for existing associations, in contrast to the comparator hypothesis explaining these phenomena in the framework of changes in the expression of an unchanging association between the target CS and the US. The present data provide a new phenomenon that at this time is predicted uniquely by the comparator hypothesis. The other models that successfully explain retrograde revaluation effects cannot explain the preexposure-overshadowing effect, whereas our elaboration of the comparator hypothesis can do so. Thus, pretraining manipulation may serve as a powerful method of contrasting the predictions of models that emphasize processes occurring during testing (e.g., the comparator hypothesis) with models that emphasize processes that occur during acquisition.

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