

## Superconditioning and Overshadowing

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Conditioned lick suppression by water-deprived rats was used to investigate the phenomenon of superconditioning. In Experiment 1, animals received explicitly unpaired conditioned inhibition training with CS A or CS B and a footshock US. Both groups then received pairings of a CS A-light compound with shock. In testing, animals for which CS A was inhibitory displayed enhanced responding to the light, relative to animals for which CS A was associatively neutral. Thus, superconditioning relative to the conventional control group was demonstrated. However, an additional control group that first received inhibitory training with CS A and then the light alone paired with shock exhibited as much suppression to the light as the superconditioning group. This control, which was omitted from previous demonstrations of superconditioning, suggests that superconditioning is merely attenuated overshadowing of the added element by CS A as a result of prior inhibitory training with CS A. In Experiment 2, superconditioning was not obtained when partial reinforcement of CS A occurred during negative contingency training, despite the fact that in prior research the identically trained CS A had passed both summation and retardation tests for inhibition. In Experiment 3, exposure to CS A alone (i.e., latent inhibition treatment) sufficed to produce a superconditioning-like effect with respect to the light when the CS A-light compound was later paired with shock. In Experiment 4, conditioned inhibition and latent inhibition treatments of CS A were found to yield equivalent superconditioning-like effects with respect to the added element. Collectively,

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these studies suggest that superconditioning arises from a reduction in overshadowing and a comparable effect can be obtained through any initial treatment that diminishes the associability of the overshadowing CS. © 1989 Academic Press, Inc.

A major success of the Rescorla–Wagner (1972) model of associative learning was its *a priori* prediction of the phenomenon of superconditioning. Superconditioning refers to the enhanced conditioned response evoked by a target stimulus after that stimulus has been reinforced in compound with a conditioned inhibitor, relative to when the target stimulus has been reinforced in compound with an associatively neutral stimulus element. This effect has been reported with a number of different preparations since the initial demonstration by Rescorla (1971b), in which enhanced conditioned suppression of barpressing was observed. For example, comparable results have been obtained by Blanchard and Honig (1976) using pigeons in an autoshaping preparation as well as by Taukalis and Revusky (1975) who examined taste aversion in rats.

The prevailing explanation of superconditioning invokes the Rescorla–Wagner (1972) model and speaks in terms of the conditioned inhibitor (A) creating a negative expectation of the US such that reinforcement of a novel stimulus in the presence of A results in the US being more surprising than if A had been neutral. In other words, this Rescorla–Wagner interpretation of superconditioning assumes that the phenomenon reflects enhanced reinforcement value of the US.

Alternatively, superconditioning may represent an attenuation of competition between stimulus elements for associative strength as a result of prior experience with one of the elements. Given that stimuli trained in compound are typically thought to compete for associative strength or at least response strength (e.g., Mackintosh, 1971), this possibility appears worthy of consideration. Only Wagner (1971) included a control group to test the possibility that superconditioning of a novel stimulus in a reinforced compound was not simply an attenuation of overshadowing (i.e., competition) resulting from previous inhibitory training with the alternate element of the compound. Specifically, the relevant control group in Wagner's study received inhibitory training with Stimulus A in Phase 1 and reinforcement of the target stimulus, X, alone in Phase 2. Wagner reported that responding to the target stimulus by the superconditioning group exceeded that of this X-alone (i.e., no-overshadowing) control group. However, his test for excitation was rather unusual in that it consisted of a savings measure based upon 64 reinforced trials with the target stimulus alone following compound training. Moreover, the only statistic reported in support of Wagner's conclusion was a one-tailed statistical test over all 64 trials that would not have achieved significance had a two-tailed test been used.

With the exception of the study by Wagner (1971), all demonstrations of the superconditioning phenomenon have employed a single control condition. This "standard" superconditioning control group receives inhibitory training with an irrelevant stimulus (B) followed by reinforced presentations of the same AX compound stimulus experienced by the superconditioning group. Such a control condition invites the possibility that the superior responding to Stimulus X seen in the superconditioning group arises not from enhanced reinforcement value of the US, but from an attenuation of the overshadowing which might be expected to occur in the control group where two novel stimuli are reinforced in compound. An attenuated overshadowing explanation of the phenomenon of superconditioning is as compatible with the Rescorla-Wagner (1972) model as is the enhanced surprise explanation. However, the former explanation casts the superconditioning effect as a specific instance of overshadowing, a phenomenon that many models of learning and performance can explain (e.g., Mackintosh, 1975; Miller & Schachtman, 1985; Pearce & Hall, 1980; Rescorla & Wagner, 1972). In contrast, the enhanced surprise view of overshadowing relies on a mechanism that is relatively unique to, and hence supportive of, the Rescorla-Wagner model.

### EXPERIMENT 1

Our first experiment assessed the degree to which superconditioning arises from enhanced reinforcement value of the US, as opposed to an attenuation of overshadowing resulting from prior inhibitory experience with the other element of the compound. In Phase 1, three groups of rats received explicitly unpaired conditioned inhibition training (+/A-). For two groups Stimulus A was a white Noise (Groups N-NL and N-L) and for one group Stimulus A was a Click train (Group C-NL). The US was footshock for all groups. The parameters used were previously found to produce conditioned inhibition as indexed on both retardation and summation tests (see Schachtman, Brown, Gordon, Catterson, & Miller, 1987). In Phase 2, Groups N-NL and C-NL received a white Noise-Light compound reinforced with footshock, whereas Group N-L received the Light alone reinforced with footshock. All animals were then tested for conditioned suppression of licking, first with the light and then with the noise.

Greater suppression in the presence of the light by Group N-NL than Group C-NL would demonstrate superconditioning of the light relative to the standard superconditioning control group. More suppression in the presence of the light by Group N-L than Group C-NL would indicate overshadowing of the light by the noise. Finally, if Group N-NL displayed more suppression in the presence of the light than Group N-L, we could conclude that the superconditioning was due to more than the prevention of overshadowing; whereas, equal or less suppression in the presence

of the light by Group N-NL than Group N-L would allow the possibility that superconditioning was no more than an attenuation of overshadowing as a result of prior inhibitory training with the noise.

The original study used a total of 48 animals and yielded a treatment effect significant at  $p < .055$ . Consequently, a replication was performed and the results were pooled with the original data.

### *Method*

*Subjects.* Ninety-six naive, adult, male and female, Sprague-Dawley-descended rats bred in our colony from Holtzman (Madison, WI) stock served as subjects in to balanced replications. Body weight ranges were 270–370 g for males and 180–300 g for females. Subjects were quasi-randomly assigned to treatment groups counterbalancing for sex, litter of origin, body weight, and baseline lick performance prior to differential treatment. All animals were individually housed in hanging wire-mesh cages in a vivarium that was maintained on a 16-h light/8-h dark daily cycle. Experimental manipulations occurred near the midpoint of the light portion of this cycle. Purina Laboratory Chow was freely available in the home cages. One week prior to the initiation of a study, all subjects were progressively deprived of water and by Day 1 of the study were limited to 10 min per day of home-cage access to water provided approximately 23 h prior to any treatment scheduled for the following day. All subjects were handled thrice weekly for 30 s from weaning until the initiation of the study.

*Apparatus.* Twelve identical chambers measuring  $24.1 \times 12.7 \times 20$  cm (l  $\times$  w  $\times$  h) were used. Each chamber was housed in a separate controlled-environment isolation shell. Three of the chamber sidewalls were opaque Plexiglas, whereas the ceiling and remaining sidewall were clear Plexiglas. Diffused illumination was provided in each chamber by a 1.12-W bulb (No. 1819) located behind one opaque wall. Chamber floors consisted of parallel stainless-steel rods interconnected by NE-2 neon bulbs. The output from a high voltage AC power source in series with a 1- $\Omega$  resistor could be passed through these bulbs to provide a constant current, 0.7-mA, 0.5-s footshock. Each chamber was equipped with a water-filled lick tube that extended 0.1 cm through a slot in one side wall, 2.5 cm above the floor. By licking the tube, the animal completed a circuit between the tube and the grid floor that allowed the number of licks to be monitored during sessions in which footshock was not administered. Nominal CSs consisted of a 2.8-W light (No. 1820) mounted on the chamber ceiling which flashed 0.5 s on/0.5 s off when activated and a white noise delivered by a 10-cm, 45- $\Omega$  overhead speaker. This auditory CS was 8 dB(C), re. SPL, above an 80-dB(C) background noise level produced by a ventilation fan mounted on the side of each envi-

ronmental chest. A 3-s click train could also be administered through the same speaker and at the same intensity as the white noise.

*Procedure.* On Days 1 and 2 animals were allowed 20 min to acclimate to their conditioning chambers and drink from the lick tubes. Latencies to complete the first and second 25 licks were recorded. Following acclimation, there were 4 days (Days 3–6) of Phase 1 conditioning. On each of these days, all subjects were placed in their chambers for a 60-min session and received 42 unsignaled footshocks. Shocks were distributed randomly with the constraint that no more than one was given per minute. During each session, Groups N-NL and N-L ( $n_s = 36$ ) also received eight nonreinforced 60-s presentations of the white noise, that is, no footshock occurred during or within 50 s of a noise presentation. Group C-NL ( $n = 24$ ) received the same treatment, but with clicks in place of the white noise.

Phase 2 treatment occurred during a single 60-min session in the conditioning chambers on Day 7. For Groups C-NL and N-NL, it consisted of eight 60-s presentations of a compound CS consisting of the simultaneous occurrence of the white noise and flashing light. This compound was consistently reinforced upon termination with footshock. Group N-L received the same treatment without the noise being present, that is, eight light-shock pairings. To reestablish baseline licking, on Days 8 and 9 subjects were placed into their chambers for 20 min and allowed to drink freely from lick tubes. Latencies to complete Licks 0–25 and 25–50 were recorded. No stimuli were presented on these days.

On Day 10, all subjects were returned to their conditioning chambers for 10 min and allowed to drink from lick tubes. At the conclusion of the first 25 licks, the flashing light was presented and remained on until 25 additional licks were completed. Latencies to complete Licks 0–25 and 25–50 were recorded. On Day 11, lick suppression to the white noise was tested in the same manner as with the light on the preceding day. Because the first 25 licks on Days 10 and 11 took no subject more than 30 s to complete, the 10-min session provided an effective ceiling latency for the second 25 licks of 9.5 min. In practice, no animal failed to complete the second 25 licks during any test session. Therefore, similar group means are not artifacts of ceiling effects. The same argument against the presence of ceiling effects also can be made for Experiments 2–4.

### *Results and Discussion*

As expected owing to the counterbalancing of subjects, no between-group differences were found in latencies to complete Licks 0–25 or Licks 25–50 on Days 2 and 9 and to complete Licks 0–25 on Days 10 and 11,  $p_s > .20$ . Analysis of variance (ANOVA) detected a difference among the treatment groups on the flashing light test,  $F(2, 90) = 5.60$ ,

$p = .005$  (see Fig. 1). Planned comparisons revealed differences between the superconditioning group (N-NL) and the standard superconditioning control group (C-NL),  $F(1, 90) = 4.39$ ,  $p < .05$ , but not between the superconditioning group (N-NL) and the no-overshadowing control group (N-L),  $F(1, 90) = 1.75$ ,  $p > .10$ .

ANOVA on suppression to the noise also proved significant,  $F(2, 90) = 69.94$ ,  $p < .001$ . Planned comparisons found Group C-NL to exhibit more suppression to the noise than Group N-NL,  $F(1, 90) = 12.00$ ,  $p < .001$ , indicative of a retardation effect which suggests that Phase 1 conditioned inhibition training of Group N-NL was successful. Moreover, Group N-NL exhibited more suppression than Group N-L,  $F(1, 90) = 10.96$ ,  $p < .01$ , which is consistent with the lack of Phase 2 reinforcement of the noise in Group N-L.

The observed difference in suppression to the flashing light between Group N-NL and Group C-NL demonstrates superconditioning with respect to the control group commonly used to assess superconditioning. Moreover, the failure of Group N-NL to exceed the suppression of Group N-L in response to the light suggests that superconditioning may be no more than a reduction in overshadowing of the light by the noise as a result

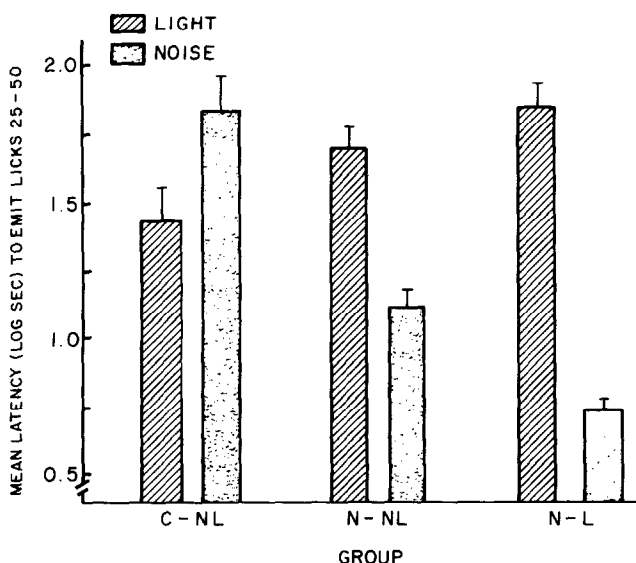


FIG. 1. Mean latencies in Experiment 1 to complete 25 licks in the presence of the flashing light and in the presence of the white noise. In Phase 1, Groups N-NL and N-L received explicitly unpaired negative contingency training with the Clicks. In Phase 2, Groups N-NL and C-NL experienced the Noise-Light compound paired with shock, while Group N-L received comparable Light-shock pairings in the absence of the noise. Brackets denote standard errors.

of prior conditioned inhibition training with the noise. Of course superconditioning might result from both attenuated overshadowing and negative expectation of the US operating as two independent mechanisms. The present experiment (and subsequent ones in this series) gives credence to the attenuated overshadowing explanation without directly refuting the possibility that, under select conditions, negative expectation of the US may underlie superconditioning, an explanation that is unique to the Rescorla–Wagner (1972) model. However, parsimony would suggest that so long as the published data, including the present data, are consistent with the possibility that attenuated overshadowing is a fully adequate explanation of superconditioning, there is no reason to evoke a second explanatory mechanism.

The preceding remarks notwithstanding, there is an alternative to attenuated overshadowing as an explanation for the superconditioning group (N-NL) not surpassing the level of conditioned suppression to the light seen in the no-overshadowing control group (N-L). Specifically, for Group N-NL the noise may have lost its inhibitory qualities early in Phase 2 (owing to both reinforcement of the noise and an absence of unsignaled shock) and, after becoming excitatory, may have overshadowed the light on later trials relative to Group N-L. For this hypothesis to be viable in the framework of the Rescorla–Wagner (1972) model, the initially inhibitory noise must have become excitatory as a result of the repeated reinforcements during Phase 2. In other words, superconditioning of the light caused by a negative expectation of the US might have occurred early in Phase 2, but could have been masked by overshadowing of the light by the noise on subsequent trials. However, our results indicate that this did not take place because a test on the noise revealed that it never accrued appreciable excitatory strength in Group N-NL (see Fig. 1). In the Rescorla–Wagner framework, a critical comparison of attenuated overshadowing and negative expectation of the US as explanations of superconditioning would employ a single trial in Phase 2 because Rescorla–Wagner predict no overshadowing with a single compound trial, but negative expectation of the US should act on a single trial. However, published demonstrations of one-trial overshadowing (in contradiction to the Rescorla–Wagner model) make such differential predictions a poor means of distinguishing between these two explanations of the present results (e.g., Mackintosh & Reese, 1979).

Experiments 2–4 were designed to provide Phase 1 treatments with the noise that did not confound conditioned inhibition of Stimulus A with the loss of ability of Stimulus A to overshadow Stimulus X. Our selection of Phase 1 treatments to differentiate conditioned inhibition from attenuated overshadowing was directed by the Pearce and Hall (1980) model and supporting data which suggest that consistent outcomes following a stimulus reduce the stimulus' associability, that is, the potential

of the stimulus to change associative status. Thus, associative competition during Phase 2 compound conditioning might be attenuated when this conditioning is conducted subsequent to consistent nonreinforced exposure to one of the elements such as occurs during conventional inhibitory training. During Phase 1 of Experiment 2, the noise was made into a conditioned inhibitor without its having consistent outcomes, and in Phase 1 of Experiment 3, the noise had consistent outcomes without its being made into a conditioned inhibitor. Collectively, these studies tested the hypothesis that the critical consequence of Phase 1 treatment in Experiment 1 was that consistent experience with the noise by Group N-NL attenuated Phase 2 overshadowing of the light by the noise relative to overshadowing of the light in Group C-NL.

## EXPERIMENT 2

During Phase 1 of Experiment 2, the noise was again made into a conditioned inhibitor using negative contingency training. However, rather than use the explicitly unpaired procedure of Experiment 1 in which the noise was never reinforced, in Experiment 2 the noise was occasionally paired with footshock (i.e., partially reinforced). The parameters used were *identical* to those with which Schachtman *et al.* (1987) demonstrated that the noise so trained would pass both summation and retardation tests for conditioned inhibition.

According to the enhanced surprise explanation of superconditioning (Rescorla & Wagner, 1972), superconditioning should result whenever a stimulus is paired with a US in the presence of a conditioned inhibitor specific to that US. Of course, a stimulus made inhibitory with a partial reinforcement schedule would not be expected to be as strong an inhibitor as one produced with an explicitly unpaired procedure; hence it should support weaker superconditioning. Nevertheless, the enhanced surprise explanation of superconditioning predicts that some superconditioning would occur. Conversely, an attenuated overshadowing explanation employing Pearce and Hall's (1980) rules for modifying the associability of the noise as a function of experience predicts that the Phase 1 partial reinforcement schedule would allow the inhibitor (Stimulus A) to retain a high level of associability because of its inconsistent reinforcement history in Phase 1 (see Pearce, Kaye, & Hall, 1982, for supporting data). Consequently, following inhibitory training, the noise should retain its potential to overshadow the light in Phase 2. Thus, an overshadowing interpretation of superconditioning that is based on decreased associability of the noise predicts that *no* superconditioning should be observed when a partially reinforced inhibitor is used because such a stimulus would not have undergone a loss of associability.

A reduction in the contribution of overshadowing to an apparent superconditioning effect is also compatible with the Rescorla and Wagner



(1972) model. However, for Rescorla and Wagner the associability of Stimulus A is constant and the reduced overshadowing of Stimulus X arises from Stimulus A's being retarded in becoming an excitator in Phase 2 owing to its initially being an inhibitor. Because partial reinforcement of the white noise during inhibitory training in Phase 1 would make the noise a weaker inhibitor than complete nonreinforcement of the noise, the white noise should be somewhat less retarded in Phase 2, thereby reducing but not eliminating superconditioning of the light relative to Experiment 1. Thus, both potential mechanisms of superconditioning that are compatible with the Rescorla-Wagner model (enhanced surprise and reduced overshadowing due to less retardation of the noise) predict that some superconditioning of the light should occur, despite Phase 1 partial reinforcement of the noise, provided that the noise becomes at all inhibitory. In contrast, Pearce and Hall predict that partial reinforcement of the noise in Phase 1 will completely eliminate superconditioning.

### *Method*

*Subjects and apparatus.* Forty-eight naive rats of the same description as those used in Experiment 1 were used. The apparatus was the same as that of Experiment 1.

*Procedure.* As in Experiment 1, subjects were allowed 20 min of free access to water in their conditioning chambers on Days 1 and 2. Again, three different groups were formed: N-NL ( $n = 18$ ), C-NL ( $n = 12$ ), and N-L ( $n = 18$ ). In Phase 1 (Days 3–6), all subjects received 42 unsignaled footshocks in each of four 60-min sessions. During each session, Groups N-NL and N-L also received eight white noise exposures as in Experiment 1; only this time a randomly selected two of these noise presentations were terminated with footshock, that is,  $P(US/CS) = .25$ . Group C-NL received the same treatment, but with clicks in place of the white noise.

Phase 2 treatment, baseline reestablishment, and testing were identical to Experiment 1. In Phase 2, Groups C-NL and N-NL received eight compound stimuli presentations (white noise plus flashing light), each reinforced with footshock. Group N-L received the same treatment with the flashing light and shock but not the white noise. On Days 8 and 9, all subjects were placed in their conditioning chambers where they were allowed access to water-filled lick tubes for 20 min in order to reestablish baseline licking. On Days 10 and 11, subjects were tested for lick suppression in response to the light and noise, respectively.

### *Results and Discussion*

No between-group differences were detected in the latencies to complete 25 licks other than in the presence of the CSs on the test days. ANOVA revealed a difference on Day 10 among the treatment groups in the

presence of the flashing light,  $F(2, 45) = 3.21$ ,  $p = .05$  (see Fig. 2). Planned comparisons found that Groups C-NL and N-NL did not differ,  $p > .50$ , that is, no superconditioning was seen with respect to the standard superconditioning control group. Moreover, Group N-L suppressed more to the light than did Group C-NL,  $F(1, 45) = 6.55$ ,  $p < .02$ , and marginally more than Group N-NL,  $F(1, 45) = 3.76$ ,  $.05 < p < .10$ , indicative of overshadowing of the light by the noise in the latter two groups.

Analysis of suppression to the white noise on Day 11 detected differences between groups,  $F(2, 45) = 9.54$ ,  $p < .001$ . Planned comparisons found that Group C-NL suppressed more than Group N-NL,  $F(1, 45) = 5.11$ ,  $p < .05$ . This retardation of responding to the noise in Group N-NL testifies to the Phase 1 negative contingency treatment making the noise an effective conditioned inhibitor and is consistent with the retardation and summation test data presented by Schachtman *et al.* (1987) indicating that training of the noise with parameters identical to the present ones served to make the noise a functional conditioned inhibitor.

The absence of superconditioning to the light in the present experiment

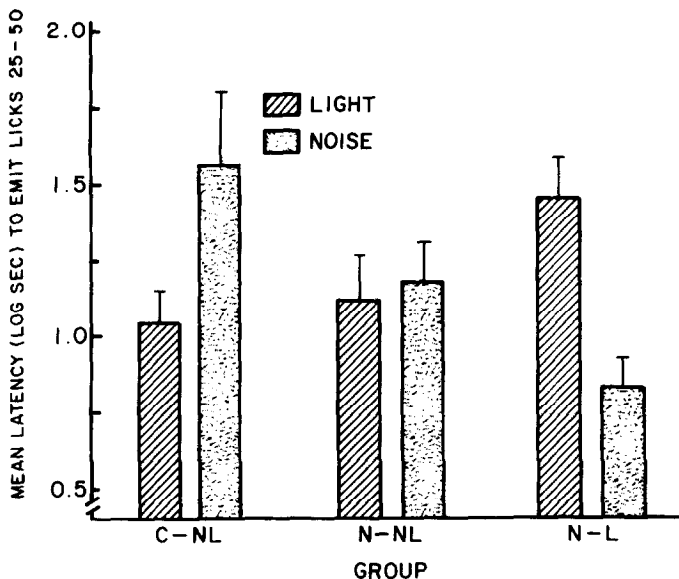


FIG. 2. Mean latencies in Experiment 2 to complete 25 licks in the presence of the flashing light and in the presence of the white noise. In Phase 1, Groups N-NL and N-L received partially reinforced negative contingency training with the white Noise and footshock, while Group C-NL received the same treatment with the Clicks. In Phase 2, Groups N-NL and C-NL experienced the Noise-Light compound paired with shock, while Group N-L received comparable Light-shock pairings in the absence of the noise. Brackets denote standard errors.

(Groups N-NL vs C-NL) contrasts sharply with the results of Experiment 1, which differed with respect to treatment only in that Phase 1 negative contingency training in Experiment 1 used an explicitly unpaired procedure in contrast to the occasional reinforcements of the noise during Phase 1 of Experiment 2. This lack of superconditioning could have arisen from the noise for Group N-NL being a weaker conditioned inhibitor in Experiment 2 than in Experiment 1. However, Schachtman *et al.* (1987) used exactly the same parameters as were used in Phase 1 of Experiment 2 and they found that the noise subsequently passed both summation and retardation tests for conditioned inhibition. Thus, although the noise was likely a weaker conditioned inhibitor in Experiment 2 than in Experiment 1, it was a conditioned inhibitor as defined by the commonly accepted empirical definition. Based on the presumed negative associative strength of any conditioned inhibitor, the Rescorla-Wagner (1972) model predicts that superconditioning should have occurred in Experiment 2 by virtue of enhanced surprise and/or attenuated overshadowing in Group N-NL, although the superconditioning would be expected to be somewhat smaller than in Experiment 1 owing to the noise in Experiment 2 being a weaker conditioned inhibitor than in Experiment 1. Thus, our rejection of the Rescorla-Wagner interpretation of superconditioning, at least on the basis of Experiment 2, must be tempered by the fact that Experiment 2 provided a null result and might have been insufficiently sensitive to detect superconditioning. Therefore, Experiments 3 and 4 were designed to test the altered associability interpretation of superconditioning by modifying associability through means other than varying the consistency with which CS A was nonreinforced during Phase 1 inhibitory training.

Experiment 2 used fewer animals than Experiment 1, so we entertained the possibility that the lack of apparent superconditioning in Experiment 2 was due to this decrease in group size. Consequently, we performed a power test which determined that even an order of magnitude increase in the number of subjects in Experiment 2 would not have produced a significant superconditioning effect. Essentially, Experiment 2 yielded not even a hint of superconditioning (see Fig. 2). In contrast with the unsupported prediction of the Rescorla-Wagner (1972) model, the lack of superconditioning in Experiment 2 but not Experiment 1 is consistent with the view that the superconditioning phenomenon reflects a decrease in overshadowing that arises from Phase 1 inhibitory training decreasing the associability of Stimulus A (i.e., the noise in Experiments 1 and 2). In Experiment 2, this decrease in the associability of Stimulus A presumably was prevented by the inconsistent reinforcement history of Stimulus A in Phase 1 (see Pearce & Hall, 1980).

The results of Experiment 2 also allow us to reject another possible explanation of the superconditioning effect seen in Experiment 1. Specifically, Groups C-NL and N-NL in Experiment 1 were both trained

with the noise–light compound in Phase 2 and subsequently tested on the light alone. Thus, both groups would be expected to exhibit a generalization decrement. However, the superconditioning group (N-NL) experienced the noise without the light in Phase 1 which might have reduced configuring between the light and the noise in Phase 2, thereby attenuating generalization decrement in that group between Phase 2 and the light test. Group N-NL in Experiment 2 also experienced the noise without the light in Phase 1. Thus, a generalization decrement explanation of the observed superconditioning seen in Experiment 1 would also predict superconditioning in Experiment 2. This did not occur.

### EXPERIMENT 3

We have suggested that the superconditioning observed in Experiment 1 occurred because consistent experience with the noise in Phase 1 reduced the associability of the noise, thereby attenuating overshadowing of the light by the noise during the subsequent reinforced compound presentations. If this analysis is correct, then any other Phase 1 treatment that reduces the associability of the noise should also yield a superconditioning-like enhancement in responding to the light as a result of Phase 2 training. (Because “superconditioning” is defined as enhanced responding to the stimulus element added in Phase 2 excitatory training following *inhibitory* training with the initial element in Phase 1, we shall refer to any enhanced responding to the added element observed after latent inhibition treatment in Phase 1 as a “superconditioning-like effect.”)

One manipulation believed to reduce CS associability is CS preexposure (i.e., latent inhibition) (Lubow & Moore, 1959). Both Rescorla (1971a) and Reiss and Wagner (1972) have demonstrated that exposure to a stimulus alone results in subsequent retarded acquisition of response strength for that stimulus when it is paired with a US, but the stimulus does not pass an inhibitory summation test, that is, it does not reduce conditioned responding to a known excitor when compounded with the excitor. Hence, the stimulus is not a conditioned inhibitor, and Rescorla and Wagner (1972) would not predict a superconditioning-like effect with respect to a second stimulus that is subsequently reinforced in the presence of the preexposed stimulus. This absence of a superconditioning-like effect is predicted because there should be no enhanced surprise on the Phase 2 AX+ trials and no reduction of overshadowing of X as overshadowing is understood within the Rescorla–Wagner framework (i.e., fixed associabilities). In contrast, the attenuated overshadowing interpretation of superconditioning based on decreased associability of Stimulus A predicts that the loss of associability resulting from latent inhibition treatment should allow Stimulus X to condition with less competition for associative strength.

Two existing reports led us to expect that prior nonreinforced exposures

to a stimulus would attenuate subsequent overshadowing by that stimulus, although neither experiment was performed with the implications for superconditioning in mind. Using a conditioned taste aversion preparation, Revusky and Garcia (1970) determined that serial overshadowing of one flavor by a second flavor could be reduced by prior nonreinforced exposure to the overshadowing flavor. Although the generalization from serial to simultaneous overshadowing is problematic, their results are encouraging. Carr (1974), using a barpress suppression preparation, found a decrease in simultaneous overshadowing as a result of prior nonreinforced exposure to the overshadowing stimulus alone. However, we regard Carr's observations as being only suggestive owing both to extremely weak overshadowing seen in his subjects that lacked latent inhibition treatment and to the novel statistical test he employed. Consequently, Experiment 3 was designed to investigate the effect of conditioning a stimulus in compound with a second element as a function of whether the subject had been reexposed to the second element.

### *Method*

*Subject and apparatus.* Forty-eight naive rats of the same description as in Experiment 1 were used. The apparatus was the same as in Experiment 1.

*Procedure.* On Days 1 and 2, all subjects were acclimated to their conditioning chambers for 20 min during which time they had free access to water. On Days 3–6 (Phase 1), subjects in Groups N-NL ( $n = 18$ ) and N-L ( $n = 18$ ) were given eight daily white noise presentations. Group C-NL ( $n = 12$ ) received equivalent treatment, but with clicks in place of the noise. No footshocks were given in Phase 1. Each session lasted for 60 min.

On Day 7, Phase 2 training was conducted during a 60-min session. For Groups C-NL and N-NL, training consisted of three compound presentations (flashing light and white noise) each reinforced at offset with footshock. For Group N-L, three flashing light presentations were reinforced with footshock. On Day 8, all subjects were placed in their conditioning contexts for 20 min in order to reestablish baseline licking. On Days 9 and 10, all subjects were tested for lick suppression to the flashing light and white noise, respectively, as in Experiments 1 and 2.

Thus, this study differed from Experiments 1 and 2 in that no footshock was given in Phase 1 and three rather than eight CS-US pairings were given in Phase 2. This reduction from the eight Phase 2 compound trials used in Experiments 1 and 2 was based on unpublished demonstrations in our laboratory that latent inhibition treatment produces less retardation than does conditioned inhibition treatment. Thus, a smaller number of Phase 2 trials was used in order to avoid both a ceiling effect that would mask differences between groups and the possibility of the noise becoming

excitatory on early Phase 2 trials and blocking the light on subsequent Phase 2 trials. Examination of the data confirmed that this procedure was effective in preventing the noise from becoming an appreciable excitator for Group N-NL.

### Results and Discussion

No between-group differences were found in any of the latencies to complete 25 licks other than in the presence of the CSs on the test days. ANOVA detected differences in suppression to the flashing light,  $F(2, 45) = 9.80, p < .001$  (see Fig. 3). Planned comparisons found Group N-NL suppressed more to the light than Group C-NL,  $F(1, 45) = 6.05, p < .025$ , indicating that a superconditioning-like effect had occurred. Although Group N-NL suppressed more to the light than Group C-NL, it suppressed less than Group N-L,  $F(1, 45) = 4.08, p < .05$ . Thus, preexposure to the noise during Phase 1 was effective in enhancing conditioned responding to the light with which it was compounded in Phase 2, but the performance of this superconditioning-like group did not exceed that of the no-overshadowing control group. The observation that latent inhibition treatment reduces the potential of a stimulus to subsequently

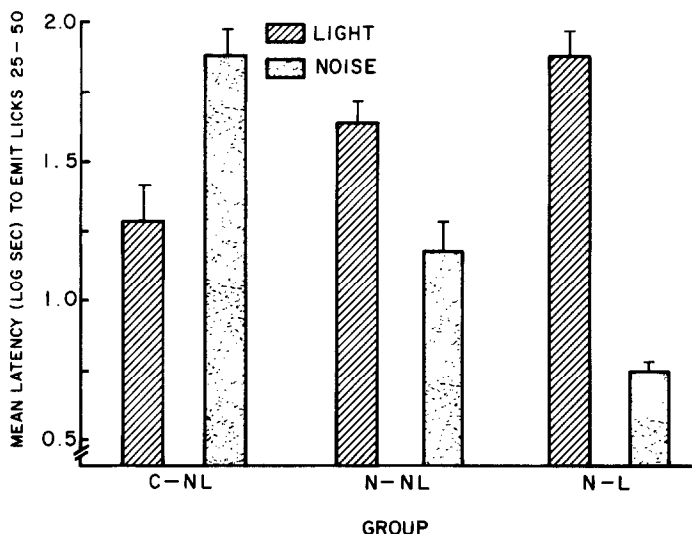


FIG. 3. Mean latencies in Experiment 3 to complete 25 licks in the presence of the flashing light and in the presence of the white noise. In Phase 1, Groups N-NL and N-L received exposure only to the white Noise (latent inhibition treatment), while Group C-NL received the same treatment with the Clicks. In Phase 2, Groups N-NL and C-NL experienced the Noise-Light compound paired with shock, while Group N-L received comparable Light-shock pairings in the absence of the noise. Brackets denote standard errors.

overshadow a second stimulus is consistent with prior reports by Carr (1974) and Revusky and Garcia (1970).

ANOVA of test trial suppression to the white noise detected differences between groups,  $F(2, 45) = 46.25$ ,  $p < .001$ . Planned comparisons found Groups C-NL to suppress more than Group N-NL,  $F(1, 45) = 34.41$ ,  $p < .001$ , indicative of Group N-NL's Phase 1 preexposure to the noise retarding conditioned responding to the noise, that is, the latent inhibition treatment was effective.

Experiment 3 demonstrates that a superconditioning-like effect can be obtained when a target CS is trained in the presence of a latent inhibitor. As latent inhibitors, by virtue of their not passing negative summation tests (Rescorla, 1971a; Reiss & Wagner, 1972), do not meet the established criterion for conditioned inhibitors, Experiment 3 indicates that conditioned inhibition is not necessary to obtain a superconditioning-like effect. Experiment 2 demonstrated that conditioned inhibition is not sufficient to obtain superconditioning. Thus, collectively these two studies suggest that conditioned inhibition is neither necessary nor sufficient to obtain a superconditioning-like effect. Conversely, the view that superconditioning is due to attenuation of the associability of the Phase 1 stimulus which in turn reduces overshadowing of the target stimulus in Phase 2 is consistent with each of the present experiments as well as prior studies of superconditioning.

#### EXPERIMENT 4

If the phenomenon of superconditioning is a consequence of attenuated associability of Stimulus A rather than Stimulus A being an inhibitor, Phase 1 latent inhibition treatment with Stimulus A ought to result in a superconditioning-like enhancement in responding to Stimulus X comparable in magnitude to that resulting from Phase 1 conditioned inhibition training with Stimulus A. Despite such a relationship being implied in our preceding discussion, the evidence for this rests upon an implicit comparison between Experiments 1 and 3. Not only is such a comparison tenuous because critical unidentified variables could have changed during the interval between the two experiments, but the number of Phase 2 reinforced trials (AX+) was not the same in Experiments 1 and 3. Although this difference in the number of Phase 2 trials was intentionally imposed to maximize the sensitivity of each study, it makes any comparison of the two studies somewhat problematic. A third difference between Experiments 1 and 3 is that the unsignaled shocks of negative contingency training in Experiment 1 made the contextual cues highly excitatory. Consequently Experiment 4 was conducted to permit a direct, unfounded comparison of the relative effectiveness of latent inhibition treatment and conditioned inhibition training in producing a subsequent superconditioning-like effect.

Experiment 4 brought together in a single study some of the groups from Experiments 1 and 3 under conditions of an equal (intermediate) number of Phase 2 pairings and minimal conditioning of the context. This last goal was achieved by using Pavlovian conditioned inhibition training (B+ /BA-) in Phase 1 rather than the negative contingency conditioned inhibition training (+ /A-) that was used in Experiment 1. To avoid a large number of treatment groups, the standard control group for superconditioning was omitted from this study. In order to obtain Pavlovian conditioned inhibition, we used stimuli and parameters similar to those that we have previously found result in Stimulus A becoming a robust conditioned inhibitor as seen on both retardation and negative summation tests (Hallam, Matzel, Sloat, & Miller, 1989). This necessitated a change in stimuli and parameters from those employed in Experiments 1 and 3; therefore, an no-overshadowing control group was included in the present study to make it fully self-contained.

### Method

*Subjects and apparatus.* Seventy-two naive rats of the same description as in Experiment 1 were used. Twelve subjects were assigned to each of six groups on the same quasi-random basis as in the preceding studies. The apparatus was the same as in Experiment 1 except that a 15-W, 120-V AC, white light capable of being flashed (0.5 s on-0.5 s off) was installed on the ceiling of each chamber. This light was normally off.

*Procedure.* The central features of the procedure are summarized in Table 1. On Days 1 and 2, all subjects were allowed free access to water in their conditioning chambers for 20 min. On Day 3, a flashing light

TABLE 1  
Design of Experiment 4

Group	Treatment <sup>a</sup>		
	Phase 1	Phase 2	Test
ACI-AX	16L + /32LA -	6AX +	X, A
ACI-X	16L + /32LA -	6X +	X, A
BCI-AX	16L + /32LB -	6AX +	X, A
BCI-X	16L + /32LB -	6X +	X, A
ALI-AX	16L + /32L - /32A - or 32 A -	6AX +	X, A
ALI-X	16L + /32L - /32A - or 32 A -	6X +	X, A

<sup>a</sup> Stimulus X was a tone, Stimulus L was a flashing light, and + was a footshock for all animals. For half the animals in each group, Stimulus A was a click train and Stimulus B was a white noise. For the remaining animals, these last two stimuli were reversed. Groups ALI-AX and ALI-X were divided in half with one-half of each group receiving one of the two listed Phase 1 treatments.



was made a conditioned excitator for most of the animals in order to facilitate subsequent inhibitory training. On this day, each subject spent 60 min in its conditioning context. During this session, half of the subjects in Groups ALI-AX and ALI-X received no treatment. For all other animals, the 15-W, flashing light was presented for 30 s at six pseudorandomly selected times, each followed immediately by 0.7-mA, 0.5-s footshock.

On Days 4–7 (Phase 1), subjects in Groups ACI-AX and ACI-X received training designed to make Stimulus A a conditioned inhibitor. Specifically, during daily 60-min sessions these subjects received four flashing light–shock pairings ( $L+$ ) pseudorandomly intermingled with eight nonreinforced presentations of the flashing light simultaneously compounded with Stimulus A ( $LA-$ ). All CS presentations were 30 s in duration. Groups BCI-AX and BCI-X received identical conditioned inhibition training except that Stimulus B was substituted for Stimulus A ( $L+/LB-$ ). Groups ALI-AX and ALI-X received latent inhibition treatment with Stimulus A that consisted of eight 30-s exposures to Stimulus A alone ( $A-$ ). For half of the subjects in these last two groups, the Stimulus A presentations were pseudorandomly intermingled with four flashing light–shock pairings and eight presentations of the flashing light alone ( $L+/L-/A-$ ). The light alone and light–shock trials were intended to provide nonassociative experience (particularly shock experience) comparable to that of the four conditioned inhibition groups. These trials were not given to all the subjects in the two latent inhibition groups for fear that the light–shock/Stimulus A–no shock treatment would effectively provide differential inhibitory training. By giving the light–shock pairings to only half of the subjects in each latent inhibition group, the two subgroups within each of these treatment conditions could be compared to determine if the presence or absence of the light–shock pairings made any difference with respect to any subsequently observed superconditioning-like effect. For half the subjects in each of the six groups, Stimulus A was a three per second click train 10 dB(C) above background and Stimulus B was a white noise 12 dB(C) above background. The difference in intensity of these two stimuli was intended to approximately equate the clicks and white noise in terms of rapidity with which they would accrue control over behavior when paired alone with shock (i.e., equivalent saliences) on the basis of unpublished research in our laboratory. For the other half of the subjects, Stimuli A and B were reversed with respect to the clicks and the white noise.

On Day 8, Phase 2 training was conducted during a single 60-min session. Groups ACI-AX, BCI-AX, and ALI-AX received six 30-s presentations of a 3000-Hz, 8-dB(C) above background tone (Stimulus X) in compound with Stimulus A, each immediately followed by a 0.5-s, 0.7-mA footshock. Groups ACI-X, BCI-X, and ALI-X received identical

treatment except that Stimulus A was omitted; that is, these subjects experienced six tone (Stimulus X)–shock pairings.

On Day 9, all subjects were placed in their conditioning contexts for 20 min in order to reestablish baseline licking. On Days 10 and 11, each animal was tested for lick suppression to the tone (Stimulus X) and Stimulus A, respectively. The test procedure was identical to that used in the previous experiments.

In summary, this study used a  $3 \times 2$ , between-subject, factorial design in which subjects in Phase 1 received either inhibitory training with Stimulus A, inhibitory training with Stimulus B, or equivalent exposure to Stimulus A alone (i.e., latent inhibition treatment), and in Phase 2 received shock-reinforced trials with Stimulus X either alone or in compound with Stimulus A. To keep this experiment manageable, the standard control group for superconditioning (i.e., Phase 1 treatment matched for nonassociative effects) was excluded for the latent inhibition treatment. But the behavior of this group in Experiment 3 (Group C-NL) suggests that nonreinforced exposure to an irrelevant stimulus in Phase 1 has the same null effect on Phase 2 information processing as Phase 1 conditioned inhibition treatment with an irrelevant stimulus (Experiment 1 and this experiment).

### *Results and Discussion*

Conditioned lick suppression in response to Stimulus X (tone) was found not to differ between subjects in Group ALI-AX that received partially reinforced, flashing light trials in Phase 1 and those that experienced neither the light stimulus nor footshock in Phase 1; nor was any difference detected as a function of this factor within Group ALI-X, both  $ps > .50$ . The corresponding comparisons for suppression in response to Stimulus A as a function of the presence or absence of partially reinforced light presentations in these two groups also proved nonsignificant, both  $ps > .50$ . This latter observation in Group ALI-AX suggests that the parameters of Phase 1 treatment were not conducive to the development of differential conditioned inhibition as a result of the intermingling of light–shock pairings with nonreinforced presentations of Stimulus A. Consequently, the data from these two conditions were pooled within each of the two latent inhibition groups.

No between-group differences were found in any of the latencies to complete 25 licks other than in the presence of CSs on the test days. Group mean suppression in response to Stimulus X and Stimulus A are depicted in Fig. 4. ANOVA of suppression to the tone (Stimulus X) detected a interaction of Phase 1 treatment and Phase 2 treatment,  $F(2, 66) = 3.21$ ,  $p < .05$ , but no main effect of Phase 1 or Phase 2 treatment,  $ps > .10$ . Planned comparisons found that Group BCI-AX suppressed

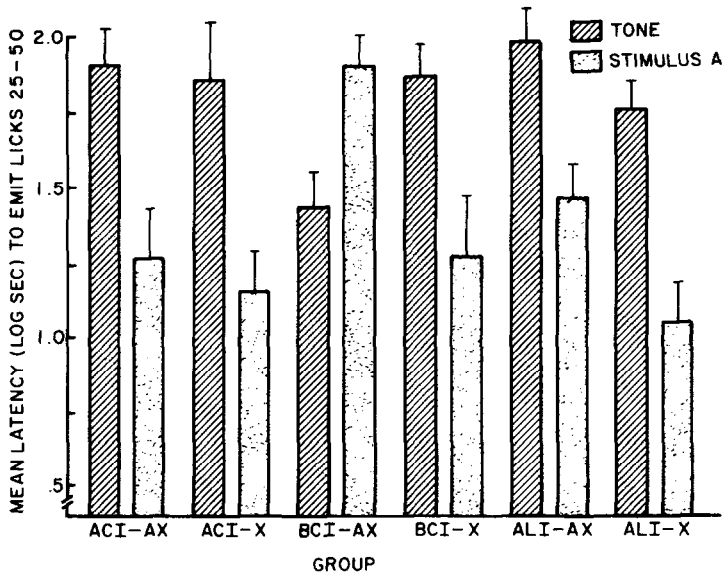


FIG 4. Mean latencies to complete 25 licks in the presence of the Stimulus X, which was a tone for all animals, and in the presence of Stimulus A, which was a click train for half the animals in each group and a white noise for the remaining animals. In Phase 1, Groups ACI-AX and ACI-X received Pavlovian inhibitory training that made Stimulus A into a Conditioned Inhibitor, Groups BCI-AX and BCI-X received Pavlovian inhibitory training that made Stimulus B (which was a white noise for half the animals and a click train for the remaining animals) into a Conditioned Inhibitor, and Groups ALI-AX and ALI-X received exposure only to Stimulus A (latent inhibition treatment). In Phase 2, Groups ACI-AX, BCI-AX, and ALI-AX experienced the tone-Stimulus A compound paired with shock, while Groups ACI-X, BCI-X, and ALI-X received comparable tone-shock pairings in the absence of Stimulus A. Brackets denote standard errors.

less to the tone than any of the other groups, all  $F_s(1, 66) \geq 4.24$ ,  $p_s < .05$ . None of the other groups differed from one another,  $p_s > .50$ .

ANOVA of suppression to Stimulus A (click or white noise) found a main effect of Phase 1 treatment (ACI, BCI, or ALI),  $F(2, 66) = 3.67$ ,  $p < .05$ , and a main effect of Phase 2 treatment (AX+ or X+),  $F(1, 66) = 8.70$ ,  $p < .005$ , but no interaction of these two factors,  $p > .20$ . Planned comparisons found that Group BCI-AX suppressed more to Stimulus A than any of the other groups, all  $F_s(1, 66) \geq 4.16$ ,  $p_s < .05$ . None of the other groups differed from one another,  $p_s > .05$ .

In summary, a reciprocal relationship was seen between suppression to the tone and suppression to Stimulus A, with Group BCI-AX suppressing to Stimulus A but not the tone and all other groups suppressing to the tone but not Stimulus A. This outcome is entirely expected in the three groups that received reinforcement of the tone alone in Phase 2. When the compound of Stimulus A and the tone was reinforced in Phase 2,

Stimulus A overshadowed the tone provided that no relevant treatment had occurred in Phase 1 (Group BCI-AX vs Group BCI-X). However, when Stimulus A had been made into a conditioned inhibitor (Group ACI-AX vs Group ACI-X) or had been presented alone (Group ALI-AX vs Group ALI-X), Stimulus A failed to overshadow the tone.

The greater suppression to the tone by Group ACI-AX than Group BCI-AX indicates that superconditioning occurred relative to the standard superconditioning control group, but the lack of difference between Group ACI-AX and Group ACI-X, consistent with Experiment 1, demonstrates that the benefit of superconditioning was no greater than the absence of overshadowing. Moreover, the great similarity of Group ACI-AX and Group ALI-AX in suppression to the tone under comparable conditions in a single experiment supports the conclusion that the occurrence of a superconditioning-like effect is not dependent upon excitatory conditioning in the presence of a conditioned inhibitor, but rather depends upon conditioning in the presence of a second stimulus that does not overshadow the target stimulus as much as in control subjects. Of course the observed superconditioning-like effect in these two cases in principle could arise from two different mechanisms, but parsimony would argue against such a complex interpretation unless further evidence necessitates it.

## GENERAL DISCUSSION

Based upon the Rescorla–Wagner (1972) model, superconditioning has generally been believed to be a consequence of the target stimulus benefitting from the US in Phase 2 being more surprising than it would have been in the absence of the conditioned inhibitor (e.g., Rescorla, 1971b). However, the early studies advocating this position did not control for the possibility that reduced overshadowing relative to the standard superconditioning control group might have been responsible for the observed differences. Supportive of this view, conditioned responding to the tone by the superconditioning groups (N-NL in Experiment 1 and ACI-AX in Experiment 4) did not exceed that of the no-overshadowing control groups (N-L in Experiment 1 and ACI-X in Experiment 4). Although the present data do not prove that all previous demonstrations of superconditioning were merely the result of a reduction in overshadowing, the available evidence is entirely consistent with this viewpoint. Because there are many demonstrations of overshadowing beyond those embedded in studies of superconditioning, parsimony suggests that superconditioning not be regarded as an independent phenomenon until it can be shown to be of greater magnitude than any accompanying overshadowing effect.

There are at least two not mutually exclusive mechanisms by which overshadowing of Stimulus X by Stimulus A in Phase 2 might have been reduced. First, attenuated overshadowing could have arisen from the Phase 1 CS being retarded in accruing control of excitatory responding

owing to its starting Phase 2 with negative associative strength. This mechanism is consistent with the Rescorla–Wagner (1972) model. Second, attenuated overshadowing in Phase 2 could have resulted from a reduction in the associability of Phase 1 CS as a consequence of consistent non-reinforcement during the inhibitory training of Phase 1. This latter mechanism is congruent with Pearce and Hall's (1980) theory, which posits variable associabilities, but is inconsistent with the Rescorla–Wagner model, which views associabilities of specific stimuli as fixed. Although both of these potential sources of attenuated overshadowing could have contributed to the superconditioning that was observed in Experiment 1, the results of Experiments 2 and 3 are understandable in terms of altered associability of Stimulus A, but not in terms of retardation of Stimulus A arising from Stimulus A starting Phase 2 with a negative associative strength. If one subscribes to the concept of a conditioned inhibitor having negative associative strength, in Experiment 2 at least some tendency toward superconditioning should have been evident. Moreover, latent inhibition treatment in Experiments 3 and 4 should not have endowed Stimulus A with negative associative strength; hence, no superconditioning-like effect should have occurred following this treatment. As no superconditioning of Stimulus X was observed in Experiment 2 and a superconditioning-like effect on Stimulus X was seen following latent inhibition treatment with Stimulus A in Experiments 3 and 4, the present research suggests that at least with the present parameters superconditioning originates in attenuated overshadowing due to Phase 1 treatment reducing the associability of Stimulus A.

The current data are in accord with the Pearce and Hall (1980) model, which postulates that following a history of consistent outcomes, a stimulus' ability to form future associations is reduced. Applied to Experiment 1, explicitly unpaired inhibitory training in Phase 1 reduced the associability of the noise. Therefore, the group that received inhibitory training with the noise in Phase 1 (N-NL) was less ready to learn new associations to the noise when it was reinforced in compound with the light during Phase 2 than was the standard superconditioning control group (C-NL); this presumably attenuated overshadowing of the light by the noise in the former group. When the same mechanism is applied to Experiment 3, repeated nonreinforced exposures to the noise in Phase 1 should have reduced the associability of the noise because the noise was a consistent antecedent of nonreinforcement. Thus, animals that received latent inhibition training (N-NL) were also less ready to learn new associations about the noise when it was reinforced in compound with the light during Phase 2, once again attenuating overshadowing of the light by the noise.

The Pearce and Hall (1980) model is also applicable to the data from Experiment 2. With respect to Experiment 2, the model predicts that superconditioning should not have occurred because the noise in Phase

1 was not a consistent antecedent of either reinforcement or nonreinforcement, owing to the 25% partial reinforcement schedule that was used during negative contingency training. Therefore, the lack of superconditioning seen in Experiment 2 is consistent with Pearce and Hall. In contrast, the Rescorla-Wagner (1972) model successfully predicted superconditioning (although not necessarily its underlying mechanism) in Experiment 1, but incorrectly predicts superconditioning in Experiment 2 and the absence of a superconditioning-like effect in Experiment 3.

In summary, the present research suggests that superconditioning is a consequence of attenuated overshadowing rather than heightened surprise value of the US. Moreover, the observed pattern of superconditioning-like behavior in the present research is in accord with Pearce and Hall's (1980) prediction that consistent consequences should decrease the associability of a CS.

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