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BRIEF REPORT

Blocking of Potentiation of Latent Inhibition

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We present a theory of latent inhibition based on the Pearce–Hall (Pearce & Hall, 1980) model for classical conditioning. Its central features are (1) that the associability of a stimulus declines as it comes to predict its consequences and (2) that nonreinforced exposure to a stimulus engages an associative learning process that makes the stimulus an accurate predictor of its consequences (in this case, the occurrence of no event). A formalization of this theory is shown to accommodate the finding that preexposure in compound with another cue can potentiate latent inhibition to the target cue. It further predicts that preexposure to the added cue will eliminate the potentiation effect. An experiment using rats and the flavor-aversion procedure confirmed this prediction.

Keywords: latent inhibition, blocking, associability, Pearce–Hall model, rats

Hall (1991) proposed a "hybrid" theory of latent inhibition in which the effect was attributed to two (related) learning processes. First, it was argued that nonreinforced exposure to a stimulus produces a reduction in the associability of the stimulus (in the α parameter of models such as that of Pearce & Hall, 1980). Second, it was suggested that stimulus exposure engages an associative learning process in which the subject learns the relationship between the stimulus and its consequence (in this case, the occurrence of no event). Loss of associability was taken to be a consequence of this associative change. On the basis of this informal account, Rodriguez and Hall (2008) derived the prediction that, in certain circumstances, the development of latent inhibition to a target stimulus would be potentiated by preexposure in which the target was presented in compound with another, and this effect was demonstrated in a series of experiments with rats using flavor-aversion and flavor-preference learning procedures. In the present article, we develop a formalization of the hybrid theory and confirm its ability to predict the potentiation effect. We then derive a further prediction, concerned with what may be regarded at a procedural level as "blocking" of latent inhibition, and we report a further flavor-aversion experiment designed to test it.

Theory

According to the Pearce–Hall model of conditioning (Pearce & Hall, 1980) the pairing of a conditioned stimulus (CS) and an unconditioned stimulus (US) will produce an increase in the associative strength of the CS as follows:

$$\Delta V = S \cdot \alpha \cdot \lambda \quad (1)$$

where ΔV represents the change in associative strength, S represents a parameter determined by the salience of the CS, λ represents a parameter related to the intensity of the US, and α represents the associability of the CS. The value of α is assumed to be high for a novel stimulus but can change as a result of experience. Specifically, it was suggested that the value of α on trials after the first was determined by events occurring on the preceding trial:

$$\alpha^n = |\lambda - \Sigma V|^{n-1}. \quad (2)$$

Here the value of α on trial n is set to equal the absolute value of the discrepancy between the value of λ and the summed associative strength (ΣV) of all CSs that were present on trial $n - 1$. This formulation implies that repeated pairings of a CS and a US will generate increases in associative strength (Equation 1) that will rise to an asymptote as the concurrent decline in α (Equation 2) occurs. At asymptote, the value of α will be zero. The introduction of ΣV into Equation 2 allows for cue competition effects, such as overshadowing. Thus, if two cues are conditioned as a compound, both will acquire strength (Equation 1). Although the associative strength acquired by each element of the compound will be less than λ, conditioning will stop when their summed strength reaches λ (Equation 2), at which point the α value of each will be zero.

Although developed for Pavlovian conditioning, these equations supply a possible account of latent inhibition. If a CS is presented alone, without a consequent US, the value of λ in Equation 2 will be zero and α will be set to zero for the next trial; acquisition of associative strength will not then be possible until the value of α has been restored, and conditioning will be impeded. This analysis supplies a useful starting point but is in some ways unsatisfactory. One problem is that it does not accommodate the gradual, incremental nature of the latent inhibition effect. A related, perhaps

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more fundamental, issue is that it does not capture the proposed parallel between the loss of associability suffered by a CS during conditioning and that suffered by a stimulus during nonreinforced preexposure. The logic underlying the Pearce–Hall model of conditioning was that an animal needs to attend to (be ready to learn about) a novel stimulus, the consequences of which are uncertain, but does not need to attend (in this sense) to a stimulus of which the consequences are known. Loss of associability during conditioning is thus a consequence of the acquisition of associative strength. This principle does not form a part of the account of latent inhibition just described.

To deal (in part) with this issue, Hall (1991) proposed a hybrid theory in which it was suggested that nonreinforced preexposure engaged an associative learning process (characterized as the formation of a stimulus–no event association) and that the loss of associability produced by such preexposure was a consequence of this learning process. The theoretical complexities introduced by the notion of a no-event representation were acknowledged but not fully examined. What we offer now is an attempt to formalize one version of this suggestion based on a version of the Pearce–Hall (Pearce & Hall, 1980) model of conditioning. Our interpretation dispenses with the problematic notion of a no-event representation; rather, we propose that a novel stimulus activates a representation of "some event" and that its ability to do so is reduced during exposure by standard inhibitory learning processes. The background to this account is presented more fully by Hall and Rodriguez (2010); we present here a summary of the basic features of the formalization relevant to the experimental test to be reported later.

We start with the assumption that any novel stimulus will evoke the expectation that some event will follow, that there will be a stimulus–event association with some initial strength. This might be expected to arise as a consequence of generalization from similar stimuli that the animal has experienced in the past as being followed by some outcome. Each of the stimuli supporting generalization will tend to activate the particular outcome with which it has been associated, but the representation most effectively activated by the novel stimulus will be that coding for any feature that all of these outcomes have in common. We refer to this simply as the representation of an event. Given the variety of outcomes likely to be activated by generalization in this way, the event representation activated by a novel stimulus is likely to be motivationally neutral. We allow, however, that the current motivational state of the animal might promote preferential retrieval of outcomes that are relevant to that state, a notion supported by experimental results reported by Killcross and Balleine (1996). We assume that the expectation that an event will follow a given stimulus will be greater, the more salient that stimulus.

We now need to express that this expectation is negated by the fact that in nonreinforced preexposure, no event follows the stimulus; we thus introduce an inhibitory learning process directly based on that proposed for simple extinction in the original model. We suppose that nonreinforced exposure results in the development of a stimulus–no event association that acts to oppose the activation of (or the effects of) the existing stimulus–event association. Its growth over successive trials is given as follows:

$$\Delta V_{\text{no event}} = S\alpha\lambda_{\text{no event}}. \quad (3)$$

In line with the analysis of inhibition offered by the original model, the value of the inhibitory reinforcer, $\lambda_{\text{no event}}$, depends on the degree to which an event is expected; that is:

$$\lambda_{\text{no event}} = \Sigma V_{\text{event}} - \Sigma V_{\text{no event}}. \quad (4)$$

The value of α will then change in a way similar to that described by Equation 2:

$$\alpha^n = |\lambda_{\text{event}} - (\Sigma V_{\text{event}} - \Sigma V_{\text{no event}})|^{n-1}. \quad (5)$$

Applying these equations to nonreinforced exposure generates the following. On the first trial, α will be high and learning will occur. Because an event is expected (V_{event} has a positive value) but no consequence occurs, the inhibitory reinforcer will be present, its value given by Equation 4, and the CS–no event association will be strengthened (Equation 3). As this association grows, V_{event} will be neutralized and learning will stop as $\lambda_{\text{no event}}$ (Equation 4) and α (Equation 3) fall to zero. Subsequent conditioning, in which this stimulus is used as a CS, will be slow because α will be low and also because the expectation that an event will follow the stimulus (net V_{event}) has been lowered. (The role of V_{event} in determining behavior has yet to be fully specified; our working assumption is that a high value will facilitate performance of the conditioned response generated by the formation of an association between the CS and a particular US.)

This analysis was applied (Hall and Rodriguez, 2010) to our 2008 study (Rodriguez & Hall, 2008) of the effects on latent inhibition of preexposing the target stimulus in compound with another. Using rats and an aversive flavor-conditioning paradigm, we (Rodriguez & Hall, 2008) demonstrated that the acquisition of an aversion to an odor was impeded in rats given previous exposure to the odor (i.e., they obtained the latent inhibition effect). The new finding was that rats given preexposure to the odor in compound with a taste (salt) showed an enhanced latent inhibition effect when subsequently conditioned with the odor alone as the CS. The same potentiation effect was demonstrated with an appetitive conditioning paradigm, confirming that this result is not confined to procedures using aversive conditioning techniques.

This outcome can be predicted by the extended version of the Pearce–Hall model outlined earlier. Figure 1 presents the results of a simulation (using Equations 3, 4, and 5) of changes in the associability and associative strength of the target stimulus (the odor) over six exposure trials, using a value of 0.4 for S, a starting value of 1 for α, and an initial value of V_{event} of 0.4. The element condition represents the case in which the odor is presented alone. The compound condition represents the case in which the odor is preexposed in compound with a more salient stimulus (such as the taste is assumed to be). For this, the taste stimulus was given a starting value of α of 1, the value of S was set at 0.6, and the initial value of V_{event} was set at 0.6. As the simulation shows, the presence of the added stimulus in the compound condition means that inhibitory learning to (both elements of) the compound occurs very rapidly and the net value of V_{event} quickly falls to zero, along with the value of α for the target stimulus. Presenting two stimuli together will generate a strong expectation that an event will follow; in the absence of an event, the magnitude of the inhibitory reinforcer (Equation 4) will be greater than when the element is presented alone, $V_{\text{no event}}$ will grow rapidly, and V_{event} will reach zero in fewer trials than are needed when the target stimulus is presented alone. Not only will associability be lower after compound

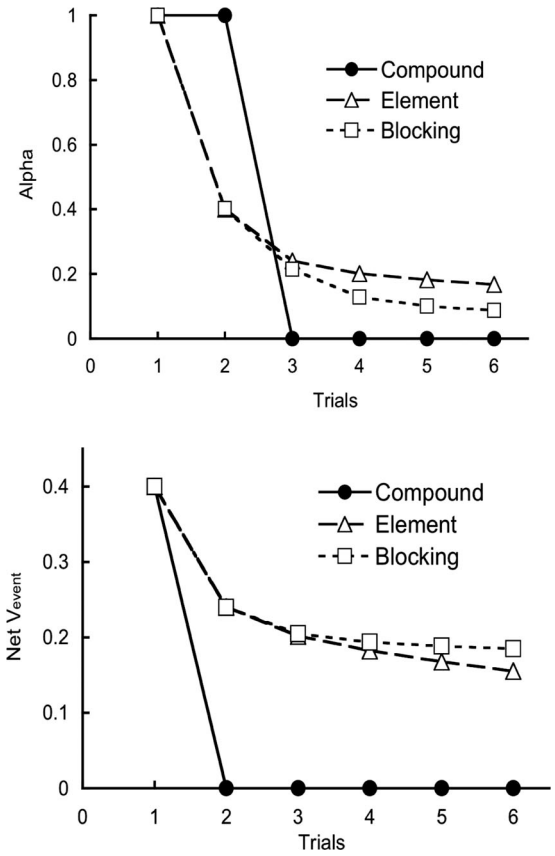

Figure 1. Simulation of the effects of six nonreinforced presentations of a stimulus with initial salience (S) of 0.4, an α value of 1, and an initial value of V_{event} of 0.4. In the element condition, the stimulus was presented alone. In the compound condition, the stimulus was presented in compound with another, with an S of 0.6. Rats in the blocking condition received compound exposure after six nonreinforced previous presentations of the nontarget stimulus.

training than after element training, but also the strength of the V_{event} association will be lower. What follows is that, after an appropriate number of trials, latent inhibition will be enhanced by this procedure. This was the result obtained by Rodriguez and Hall (2008).

Experiment

The value of this account of latent inhibition depends on its ability to make new (and accurate) predictions about the phenomenon. Although the outcome was a potentiation effect, the experiments by Rodriguez and Hall (2008) were concerned with what is procedurally an overshadowing design, in which the target stimulus was preexposed in compound with another. We consider now the blocking design, in which the added cue is presented several times alone, before the phase of compound preexposure. How will this influence the latent inhibition accruing to the target stimulus?

According to the theory, the blocking cue will itself undergo latent inhibition during the first phase of training; that is, with sufficient training, the value of α and of net V_{event} for this stimulus will fall to zero. When it comes to the phase of compound preexposure, therefore, this cue will contribute nothing to the expectation that some event will follow the target stimulus; learning about the target stimulus will proceed in much the same way as when this stimulus is presented alone. Figure 1 includes the blocking condition, a simulation of changes occurring to the target stimulus over six trials of exposure in compound with another cue that has itself been preexposed alone for six trials before the start of this phase. Starting parameter values were the same as described previously. As Figure 1 shows, the decline in α and in $V_{no\ event}$ matches that shown by the condition in which the target element is presented alone; that is, the potentiation of latent inhibition produced by compound preexposure is predicted to be blocked.

To test this prediction, we conducted an experiment based on the procedure described by Rodriguez and Hall (2008). In this, Lister rats were given aversion conditioning with an odor as the CS. To demonstrate latent inhibition, one group of rats (the element group) was given previous exposure to the odor, whereas a second group (the control group) was not. A third group (the compound group) was given preexposure, in which the odor was presented in compound with a further cue (the taste of saline); these rats should show the potentiation effect from Rodriguez and Hall's (2008) study. The focus of interest was the performance shown by a fourth group (the blocking group) that was given an initial phase of preexposure to the saline alone before exposure to the compound. Will this blocking procedure abolish or attenuate the potentiation effect?

Method

The subjects were 32 male hooded Lister rats (mean ad lib body weight, 422 g; range = 330–480 g). They were singly housed with continuous access to food in a colony room that was lit from 9:00 a.m. to 8:00 p.m. each day. Access to water was restricted as detailed later. The solutions used as experimental stimuli were presented in the rats' home cages in 50-ml centrifuge tubes equipped with steel, ball-bearing-tipped, spouts. They were almond (2% vol/vol; almond flavoring from Supercook, Leeds, United Kingdom), 0.16 molar (M) saline, and a compound of saline and almond mixed so as to maintain these concentrations of the taste and the odor. Consumption was measured by weighing the tubes before and after trials. The US was an intraperitoneal injection of 0.15 M lithium chloride (LiCl) at 10 ml/kg of body weight.

A schedule of water deprivation was established in which access was restricted to two daily sessions of 30 min at 11:00 a.m. and 5:00 p.m. The rats were then randomly assigned to one of four equal-sized treatment groups for the 12-day preexposure phase. Those in the blocking group received access to 10 ml of saline during the morning session of the first 6 days of this phase. (As six preexposure trials are enough to generate latent inhibition with our stimuli, we judged that this amount of preexposure would be enough to produce the changes in the taste stimulus necessary for the blocking of potentiation effect.) The rats then received access to 10 ml of the saline–almond mixture on the second 6 days of the phase. Rats in the compound group received water in the morning sessions on the first 6 days and the almond–saline compound on the second 6 days. Rats in the element group received water on the first 6 days and access to 10 ml of the almond solution on the second 6 days. Finally, the nonpreexposed control group received water throughout this phase. All rats were given free access to

water for 30 min during the afternoon drinking sessions. On the day after completion of preexposure, all rats received a conditioning trial in which 10 ml of the almond solution was presented for 30 min in the morning session, followed immediately by an injection of LiCl. Free access to water was allowed during the afternoon session. The next day was a recovery day, with free access to water in both drinking sessions. The second conditioning trial, on the morning of the next day, was identical to the first except that the rats were given free access to the almond solution for the 30-min trial. After a further recovery day, the rats were given a nonreinforced test trial consisting of free access to the almond solution for 30 min in the morning session.

Results and Discussion

The rats drank all the fluid made available to them during the preexposure phase. The results for the conditioning trials and the test are shown in Figure 2. On the first conditioning trial, all rats drank the full 10 ml. The effect of this trial, evident on the next trial, was to produce a suppression of consumption only in the group that was given no preexposure to almond; the others, therefore, showed evidence of latent inhibition. All groups showed evidence of conditioning on the test trial given after the second conditioning trial, but suppression of consumption was much less marked in the group given compound preexposure than it was in the other groups, which did not differ among themselves. An analysis of variance conducted on the data for the second conditioning and test trials revealed significant effects of group, $F(3, 28) = 12.03$; of trial, $F(1, 28) = 145.82$; and of the interaction between these variables, $F(3, 28) = 3.97$. (A significance level of $p < .05$ was adopted.) Analysis of simple effects showed that the groups differed both on the conditioning trial, $F(3, 56) = 7.78$; and on the test trial, $F(3, 56) = 10.69$. Pairwise comparisons using Tukey's honestly significant difference test showed that, on the second conditioning trial, the nonpreexposed group differed significantly from each of the other three groups and that these did not differ among themselves. On the test trial, the compound group differed significantly from each of the other groups, which again did not differ among themselves.

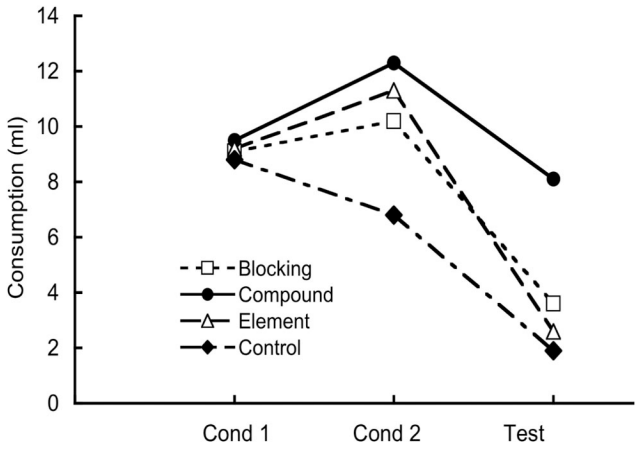

Figure 2. Group mean scores for consumption of almond during aversion conditioning (Cond) trials and a nonreinforced test. Rats in the element group had received previous exposure to almond alone, and the compound group received exposure to a compound of almond and saline. The blocking group received exposure to saline alone before exposure to the compound. The control group had preexposure to neither taste nor odor.

That the group given previous exposure to the odor that was used as the CS should learn less readily than the nonpreexposed group constitutes an example of the latent inhibition effect. The yet slower learning of the group that was given preexposure to the odor in compound with saline replicates the findings previously reported by Rodriguez and Hall (2008) and is an example of the effect that we have referred to as potentiation of latent inhibition. The blocking group, which was given exposure to saline alone before exposure to the compound, learned at the same rate as the group given exposure only to the odor; that is, they showed latent inhibition, but the potentiation effect, otherwise produced by compound preexposure, was abolished. This is the outcome predicted by the theoretical analysis outlined previously.

Conclusions

The account of latent inhibition advanced here makes the following predictions: Preexposure to a target stimulus (A) will result in impeded subsequent acquisition; preexposure in which the stimulus is compounded with another salient cue (AB preexposure) will potentiate this latent inhibition effect; previous exposure to this other cue (i.e., B/AB preexposure) will abolish the potentiation effect. The experiment reported here confirms these predictions. It may be worth adding that we have twice repeated the essential features of this experiment with minor procedural variations and have generated exactly the same pattern of results.

Before accepting the conclusions prompted by these findings, it is necessary to consider the results of several other experiments that have investigated the same basic (blocking) design in latent inhibition, with rather different results. Two studies (Rudy, Krauter, & Gaffuri, 1976, Experiment 1; Honey & Hall, 1988, Experiment 2) included the groups that we may summarize as preexposure to A alone (latent inhibition), to an AB compound (the potentiation design), and to B/AB (the blocking design). In neither case was potentiation observed (in the Honey & Hall study, latent inhibition was significantly attenuated in the AB group), and in both, the B/AB treatment resulted in substantially more rapid acquisition (i.e., less latent inhibition) than was shown by the subjects given preexposure to A alone. Honey and Hall (1988) interpreted their results as reflecting interactions between the stimuli and sensory/perceptual level. They suggested that preexposure to AB might attenuate latent inhibition because of generalization decrement from preexposure to the test—that the A stimulus used as the CS might be perceived somewhat differently from the A stimulus preexposed in compound with B, limiting the extent to which latent inhibition would transfer between the two stages. Previous exposure to B (in the B/AB procedure) might attenuate, but would not wholly eliminate, this effect. Honey and Hall used two tastes as their stimuli, and the possibility of sensory or perceptual interaction with this procedure is evident. Our choice of a taste and an odor in the present study was motivated by a desire to avoid generalization decrement effects (see Rodriguez & Hall, 2008) that otherwise might act to obscure evidence of the learning processes that we want to investigate.

The blocking design (B/AB) has also been investigated in a series of experiments by Reed and his collaborators, using appet-

itive conditioning procedures with rats as the subjects and two differently located lights as the stimuli (Reed, 1995a, 1995b; Reed, Petrochilos, Upsal, & Baum, 1997; although Reed, Anderson, & Foster, 1999, used a light and a tone). In these, it is reported that the initial phase of exposure to B (at least when it was extensive, Reed, 1995a) *enhanced* the latent inhibition that accrued to A as a consequence of AB exposure (i.e., the reverse of our finding). In some cases, this effect may not be of theoretical significance. In Reed (1995a, 1995b) comparison was made with a group given just the second phase of preexposure (i.e., preexposure just to AB). Given the similarity of the two stimuli and the possibility of generalization between them, the enhanced latent inhibition shown by the B/AB group may simply indicate that these rats received much more exposure to the critical features of the test stimulus.

In several studies, however, (Reed 1995a, 1995b; Reed et al., 1997; Reed et al., 1999) comparison has been between the blocking group and a control group given equivalent exposure to the B stimulus (specifically given B/A training, rather than the B/AB training given to the blocking group). Acquisition to A was again found to be slower in the blocking group than in the control group. This effect seems to depend on within-compound associations, formed between A and B in the second phase of preexposure. Reed et al. (1997) showed that extra presentations of A (i.e., B/AB/A training)—a treatment that might be expected to bring about extinction of such associations—abolished the effect so that learning with A as the CS now proceeded readily.

These are intriguing results that challenge our analysis and require explanation, although at this stage we can offer only speculation. There is nothing in our account to suggest that the learning mechanisms we postulate will apply only to aversion learning with tastes and odors and not to appetitive conditioning with tones and lights; we must suppose, therefore, that some other processes are operating in the procedure used by Reed and his colleagues to generate the results they obtained. One possibility is that the effects observed by Reed are the product not of latent inhibition but of a standard inhibitory learning mechanism. The appetitive conditioning procedure used in these experiments involved giving the rats extensive experience of food in the apparatus (magazine training) before stimulus exposure. Thus the introduction of stimulus B in the first phase of preexposure was coincident with the removal of food, a treatment that could endow B with conditioned inhibitory properties. The blocking procedure, in which B was subsequently presented in compound with A would then, by allowing the formation of an AB association, endow A with (second-order) inhibitory properties. The difference in test performance between the B/AB and B/A groups would then reflect a difference in conditioned rather than in latent inhibition. Further experimental work is needed to assess the validity of this account.

Although the basic procedure is very simple, there are many factors that can influence the outcome of an experiment on latent inhibition, generalization decrement, and associative inhibitory effects among them. With our training procedures (which were chosen to minimize the influence of such factors), we have demonstrated that preexposure to a compound potentiates the latent inhibition effect and that this potentiation is abolished by the blocking arrangement in which the added stimulus itself receives preexposure. This pattern of results appears to be uniquely predicted by the extension of the Pearce–Hall (Pearce & Hall, 1980) theory that we described earlier.

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