Journal of Experimental Psychology: Animal Learning and Cognition
2014, Vol. 40, No. 4, 425–430

2329-8456/14/$12.00 http://dx.doi.org/10.1037/xan0000035

BRIEF REPORT

The Hall-Rodriguez Theory of Latent Inhibition: Further Assessment of Compound Stimulus Preexposure Effects

Gabriel Rodríguez, Raúl Márquez, Marta Gil, and Gumersinda Alonso
Universidad del País Vasco (UPV/EHU)

Geoffrey Hall
University of York and University of New South Wales

According to a recent theory (Hall & Rodriguez, 2010), the latent inhibition produced by nonreinforced exposure to a target stimulus (B) will be deepened by subsequent exposure of that stimulus in compound with another (AB). This effect of compound exposure is taken to depend on the addition of a novel A to the familiar B and is not predicted for equivalent preexposure on which AB trials precede the A trials. This prediction was tested in 2 experiments using rats. Experiment 1 used an aversive procedure with flavors as the stimuli; Experiment 2 used an appetitive procedure with visual and auditory stimuli. In both, we found that conditioning with B as the conditioned stimulus proceeded more slowly (i.e., latent inhibition was greater) in subjects given the B−AB sequence in preexposure than in subjects given the AB−B sequence.

Keywords: latent inhibition, associability, preexposure, compound stimulus, rats

When the event to be used as the conditioned stimulus (conditional stimulus [CS]) is repeatedly presented alone, subsequent CS−unconditioned stimulus (US) pairings are, at least initially, less effective in producing successful conditioning (Lubow & Moore, 1959). This CS preexposure effect, also known as latent inhibition, has been demonstrated to occur over a wide range of stimuli, species, and conditioning preparations (see Lubow & Weiner, 2010, for a recent review). Recently, Hall and Rodriguez (2010) developed a theory (an elaboration of that proposed by Pearce & Hall, 1980) of what is learned during the stimulus-alone presentations and how this learning impairs subsequent associative learning and performance. The experiments reported here test a unique prediction of this theory.

The formalization offered by Hall and Rodriguez (2010) starts with the assumption that any novel stimulus will evoke, via a stimulus-event association, the expectation that some event will follow. The ability of a novel stimulus to activate this expectation may be innate or it could be a consequence of generalization from similar stimuli that the animal has experienced in the past as being followed by some outcome. In the latter case, 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 will be that coding for any feature that all the outcomes that are associated with the stimuli have in common. We will refer to this simply as the representation of an event.

The expectation activated by a novel stimulus will be contradicted by nonreinforced preexposure, in which no event follows the stimulus. Hall and Rodriguez (2010) formalized this by introducing an inhibitory learning process, directly based on that proposed for simple extinction in the original Pearce and Hall (1980) model. Hall and Rodriguez proposed 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 by:

$$\Delta V_{\text{no event}} = S\,\alpha\,\lambda_{\text{no event}} \quad (1)$$

where S is a constant parameter that depends on the intensity of the stimulus, α is a variable that represents the associability of the stimulus (assumed to be high for a novel stimulus), and $\lambda_{\text{no event}}$ represents the magnitude of the (inhibitory) reinforcer. In line with the analysis of inhibition offered by the original model, when an event is expected but does not occur, an inhibitory reinforcer is generated, its magnitude depending on the degree to which the event was expected; that is:

$$\lambda_{\text{no event}} = \sum V_{\text{event}} - \sum V_{\text{no event}} \quad (2)$$

where ΣV refers to the summed associative strength of all the stimuli present on a trial. Also in line with the original model, the value of α will then change according to this equation:

This article was published Online First August 4, 2014.

Gabriel Rodríguez, Raúl Márquez, Marta Gil, and Gumersinda Alonso, Facultad de Psicología, Universidad del País Vasco (UPV/EHU); and Geoffrey Hall, Department of Psychology, University of York and Department of Psychology, University of New South Wales.

Supported by the Spanish Ministerio de Economía y Competitividad (Grant PSI2011-2431), Spanish Ministerio de Ciencia e Innovación (Grant PSI2008-00412-PSIC), Gobierno Vasco (Grant IT-694-13), and the United Kingdom Biotechnology and Biological Sciences Research Council. We thank to SGIker for personal and technical support.

Correspondence concerning this article should be addressed to Gabriel Rodríguez, Facultad de Psicología, Universidad del País Vasco, UPV/EHU, Avenida de Tolosa 70, 20018 San Sebastián, Spain. E-mail: gabriel.rodriguez@ehu.es

$$\alpha^n = \left|\lambda_{event} - (\sum V_{event} - \sum V_{no\ event})\right|^{n-1} \quad (3)$$

That is, the associability of the stimulus on trial n, α^n, is determined by the absolute value of the discrepancy between λ_{event} (which will be 0 during the nonreinforced preexposure trials) and the strength of the expectation that some event will occur ($\Sigma V_{event} - \Sigma V_{no\ event}$) on the basis of all the stimuli present on trial $n - 1$.

Applying these equations to the case of nonreinforced exposure to a single stimulus, on the first trial, learning will occur because α will be high. The occurrence of some event will be expected in the presence of the CS (V_{event} will adopt a positive value) but no consequence will occur (λ_{event} is equal to 0). Under these conditions, Equation 2 implies that an inhibitory reinforcer will be present, and Equation 1 that the CS−no event association will be strengthened. As $V_{no\ event}$ grows over trials, V_{event} will be neutralized and learning will stop as both $\lambda_{no\ event}$ (Equation 2) and α (Equation 3) fall to 0. The consequence will be a latent inhibition effect, the magnitude of which will depend on the strength of the CS−no event association (i.e., $V_{no\ event}$) learned during preexposure. The stronger the CS−no event association, the lower the stimulus associability (i.e., α) will be; and, in addition, the reduced expectancy that some event will follow the preexposed stimulus is assumed (Hall & Rodriguez, 2010) to interfere proactively with the formation and/or expression of the association established by subsequent CS−US pairings, in which an event (the US) now reliably follows the CS.

This theory was developed (Rodriguez & Hall, 2008) and tested (Hall & Rodriguez, 2011) with respect to its predictions about the effects of preexposure in which the target stimulus is presented in compound with another. Specifically, the theory predicts that exposure to a compound stimulus (AB) will generate more latent inhibition to A than will equivalent exposure to A alone. This is because the two elements of the compound stimulus will activate a stronger aggregate expectation that some event is going to occur; according to Equation 2, the magnitude of the inhibitory reinforcer ($\lambda_{no\ event}$) will be higher when A is presented in compound with B ($\lambda_{no\ event} = V^A_{event} + V^B_{event}$) than when A is presented in isolation ($\lambda_{no\ event} = V^A_{event}$). The bigger inhibitory reinforcer present during compound exposure will ensure faster extinction of the original CS−event association (i.e., faster acquisition of $V_{no\ event}$ strength; Equation 1), and faster decline in the stimulus associability of A (Equation 3). This prediction was supported by Rodriguez and Hall (2008) in a study using flavor-aversion learning procedures, and confirmed by Leung, Killcross, and Westbrook (2011, 2013) in experiments using a fear conditioning procedure.

Further studies have looked at the effects of preexposure to the B element in this paradigm. The potentiation of latent inhibition produced by compound (AB) exposure is taken to depend on the contribution of the added element of the compound (B) in enhancing the expectation that some event is going to occur. Accordingly, nonreinforced preexposure to B (prior to the AB trials), which would extinguish that expectation, should attenuate or abolish the potentiation effect. This prediction has been confirmed by Hall and Rodriguez (2011) and by Leung et al. (2013). The experiments to be reported here address a further prediction of the theory about the effects of a preexposure schedule that involves exposure to a stimulus compound (e.g., AB) and to one element of that compound in isolation. In this case, however, we focus on the prediction regarding subsequent conditioning with stimulus B as the CS.

Consider the case in which a series of presentations of B in isolation is followed by a series of presentations to the AB compound. During its preexposure in isolation, B will lose its associability to the extent that it loses its ability to evoke the expectation that some event will occur. Thus, during the subsequent AB trials, B will do little to enhance the expectation that an event is going to occur (abolishing the potentiation effect on stimulus A, as was demonstrated by Hall & Rodriguez, 2011). However, the novel stimulus A will evoke that expectation and this will allow further strengthening of the association of B with no event during the AB trials. With sufficient training, latent inhibition to B will be deepened.

Figure 1 presents a simulation of these effects (the condition labeled B−AB), using Equations 1–3. We simulated the effects of

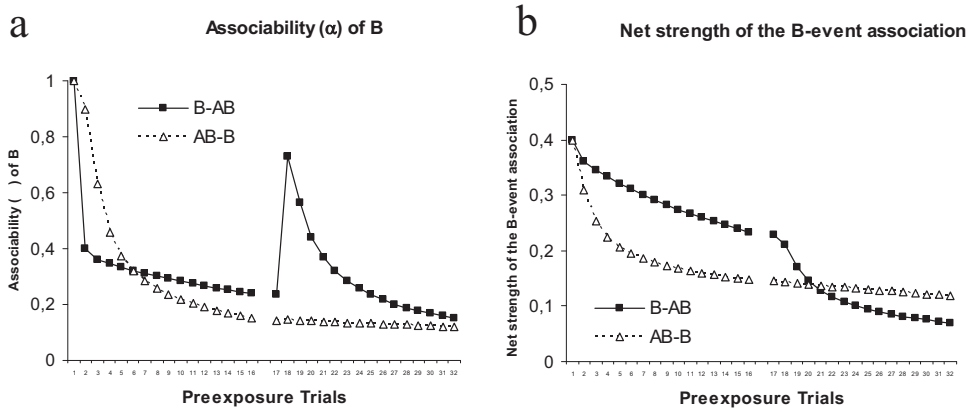

Figure 1. Simulation using the Hall and Rodriguez (2010) model. Stimulus A had a salience (S) with a value of 0.1, an initial associability (α) with a value of 1, and an initial V_{event} value of 0.4. Stimulus B had a salience (S) with a value of 0.2, an initial associability (α) with a value of 1, and an initial V_{event} value of 0.5. Increments in α (a). Increments in the net V_{event} for B (b). In the B−AB condition, 16 nonreinforced presentations to B were followed by 16 presentations of the AB compound; in the AB−B condition, the order of the presentations of B and AB was reversed.

giving exposure to two stimuli differing in salience: a more salient stimulus B and a less salient stimulus A. In our previous experimental studies (Hall & Rodriguez, 2011; Rodriguez & Hall, 2008), we made use of this stimulus arrangement (a more salient taste as B and a less salient odor as A) because, according to the theory, the enhancement in the latent inhibition acquired by the target stimulus in those experiments (stimulus A, the odor) will be more evident if its companion (stimulus B, the flavor) is more salient and thus activates a stronger expectancy that some event is going to occur. The compound latent inhibition effects predicted by the theory are not limited to those cases in which stimulus B is more salient than A, and we have conducted simulations with a range of starting values (including the case in which the stimuli are equal in salience) to confirm this. But in the example presented here, we chose a value for the salience of stimulus B that was bigger (0.2) than that of stimulus A (0.1); and, accordingly, the initial value chosen for V_{event} of stimulus B was also bigger (0.5) than that for stimulus A (0.4). The starting value of α for both stimuli was 1. We chose low numeric values for the salience of the stimuli in order to slow down the acquisition process (Equation 1), thus allowing a clear presentation of the effects with long learning curves across a relatively high number of simulated trials (see Figure 1).

The effect of exposure to AB after preexposure to B may be conveniently demonstrated by comparison with a condition in which subjects experience just the same events, but in a different order. If the AB trials precede rather than follow the B trials, the theory anticipates that during the initial AB presentations, the associability of B will decrease markedly (indeed, will achieve a lower level, than when presented in isolation, thanks to the potentiation produced by the presence of A, as demonstrated by Rodriguez & Hall, 2008). But subsequent presentations of B alone would have little further effect because B, on its own, it will be able to activate the expectation that some event is going to occur only very weakly. A simulation of the AB−B condition is also presented in Figure 1. With the parameters chosen, the final value of associability is much the same for the two conditions, but the net strength of the B-event association is lower after B−AB training than after AB−B training. In these circumstances, the difference between the conditions in subsequent conditioning will be determined principally by the strength of the associations established at the end of preexposure (see also Leung et al., 2011). When B is now reliably followed by an event (the US), development of the conditioned response will proceed less readily in the B−AB than in the AB−B condition.

We now present two experiments examining the prediction that a preexposure schedule consisting of presentations of B followed by presentations of AB will be more effective in producing latent inhibition to B than a schedule in which the order of B and AB presentations is reversed. In Experiment 1, we used the same stimuli and procedures as were used in the previous studies of Hall and Rodriguez (2011) and Rodriguez and Hall (2008). In Experiment 2, we used a similar design, but an appetitive procedure involving visual and auditory stimuli.

Experiment 1

Rodriguez and Hall (2008) noted that demonstration of the effects predicted by their theory required the use of appropriate stimuli (stimuli that interact at the sensory level could produce generalization decrement effects that would obscure the phenomena of interest) and an appropriate number of training trials. Their study established parameters suitable for demonstrating the potentiation of latent inhibition effect, with a less salient odor (almond) and a more salient taste (saline) as the stimuli, and these were adopted for the present study. (The difference in salience between these stimuli has been demonstrated by the results of previous experiments in which a higher rate of conditioning was observed to the taste than to the odor.) There were two groups of subjects. One (the B−AB group) was given an initial phase of preexposure to the saline alone and a second phase of exposure to a compound in which the saline was presented along with almond. The other group (the AB−B group) was given these two phases of preexposure in the reverse order. After preexposure, all rats were given aversion conditioning with saline as the CS and an injection of lithium chloride (LiCl) as the US. According to the predictions of the theory outlined above, conditioning to saline should occur more slowly (indicating more latent inhibition) in Group B−AB than in Group AB−B.

Method

Subjects were 16 experimentally naïve, male hooded Lister rats (mean ad libitum body weight: 323 g; range: 299–370 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; Supercook, Leeds, England), 0.16 M saline, and a compound of saline and almond mixed so 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 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. Rats were then randomly assigned to one of two equal-sized treatment groups for the 12-day preexposure phase. Those in the Group B−AB received access to 10 ml of saline during the morning session of the first 6 days of this phase. This group then received access to 10 ml of the saline−almond mixture on the second 6 days of the phase. Rats in the Group AB−B received the presentations of the compound on the first 6 days and the presentations of the saline alone on the second 6 days. 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 saline 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 saline solution for the 30-min trial. After a further recovery day, rats were given a nonreinforced test trial consisting of free access to the saline solution for 30 min in the morning session.

Data analysis. Data were analyzed with analysis of variance (ANOVA) or, where appropriate, *t* tests. Simple effects were examined using Duncan's multiple-range tests. A criterion of statistical significance of *p* less than .05 was adopted. Effect sizes for ANOVAs are reported as partial eta squared and those for pairwise comparisons are reported using Cohen's *d*. The 95% confidence intervals (CIs) around the effect sizes are also reported.

Results and Discussion

Rats drank almost all the fluid made available to them during the preexposure phase. The results for the conditioning trials and the test are shown in Figure 2a. On the first conditioning trial, all rats drank almost all of the 10 ml made available. A *t* test conducted on these scores revealed no significant differences between the two groups ($t < 1$). The effect of this first trial, evident on the next trial, was to suppress consumption in both groups (i.e., evidence of conditioning), but suppression was less marked in Group B−AB than in Group AB−B. The difference between the groups was maintained on the test trial. An ANOVA conducted on the data for the second conditioning and test trials revealed significant effects of group, $F(1, 14) = 10.68$, $\eta_p^2 = 0.43$, 95% CI [0.06, 0.66], and of trial, $F(1, 14) = 27.91$, $\eta_p^2 = 0.67$, 95% CI [0.28, 0.80]. The interaction between these two variables was not significant ($F < 1$). Although a condition given no preexposure to the CS was not included in this experiment, our previous work using this training procedure (e.g., Rodriguez & Hall, 2008) showed that, in such conditions, acquisition of the aversion occurs very rapidly, with consumption being totally suppressed after two trials. We concluded that latent inhibition occurred in both groups in the present experiment, but was more profound in the B−AB group than in the AB−A group.

Experiment 2

To establish the generality of the effect demonstrated in Experiment 1, we made use, in Experiment 2, of an appetitive conditioning procedure using a noise as stimulus A and a light as stimulus B (in a previous pilot experiment, we found that conditioning proceeded more readily to the light than to the noise, indicating that the light was more salient than the noise). The same two groups (B−AB and AB−B) were studied, but because this was the first time that we used the present procedure and parameters in assessing predictions on the magnitude of the latent inhibition effect, we also included a control (CTRL) group given no preexposure to the stimuli.

Method

Subjects were 24 experimentally naïve male Wistar rats with a mean ad libitum body weight of 367 g (range: 302–421 g) at the start of the experiment. They were housed in pairs in a colony room on a 12-hr light–dark cycle (lit from 7 a.m. to 7 p.m.) with training taking place during the light part of the cycle. They had free access to water, but were food deprived prior to the beginning of the experiment, and maintained at 85% of their ad libitum body weights throughout the experiment.

Four identical conditioning chambers (30.5 × 24.1 × 21.0 cm) from Med Associates (St. Albans, VT) were used. Each chamber was housed in a sound and light attenuating shell with background noise of 65 dB produced by ventilation fans. The floor of each chamber consisted of 19 steel rods 4.8 mm in diameter and 11.2 mm apart. These bars were perpendicular to the wall where the food tray was located. This wall and the wall opposite it were made of aluminum. The remaining walls and the ceiling of the chamber were of transparent plastic. The food tray was connected to a magazine pellet dispenser (Model ENV-203M; Med Associates) that delivered 45-mg Noyes pellets (Improved Formula A; P. J. Noyes, Lancaster, NH). A head entry into the food tray was recorded by interruption of a LED photocell. Two different stimuli

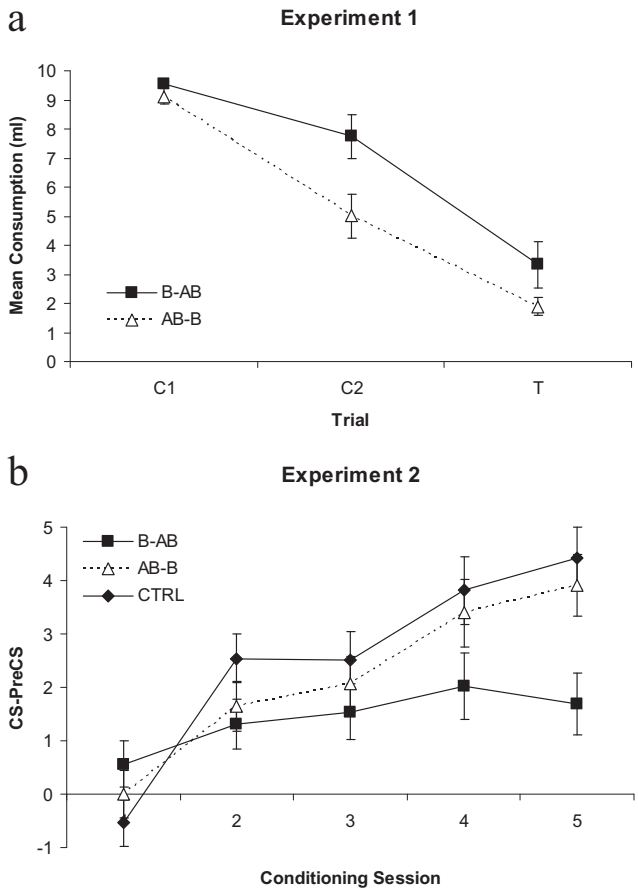

Figure 2. For Experiment 1, group mean consumption during two conditioning trials and the test trial (a). For all subjects, the conditioned stimulus (CS) on these trials was saline (stimulus B). Before the conditioning, subjects in Group B−AB received six presentations of saline (B) followed by six presentations of the almond plus saline (AB) compound; subjects in Group AB−B received the B and AB presentations in the reverse order. Vertical bars represent the standard errors of the mean. For Experiment 2, mean responding to the CS (differences between the number of magazine entries during the CS and during the pre-CS) across the five conditioning sessions (b). For all subjects, the CS was light (stimulus B). Before conditioning, subjects in Group B−AB had received 12 presentations of light (B) followed by 12 presentations of the noise plus light (AB) compound; subjects in Group AB−B received the B and AB presentations in the reverse order; subjects in the control (CTRL) group did not receive stimulus presentations before conditioning. Vertical bars represent the standard errors of the mean.

were used. Stimulus A was a white noise of 85 dB, generated by a Campden Instruments (Loughborough, England) noise generator, and delivered by a speaker located at the ceiling of the chamber (or on the inside front wall of the shell). The second (stimulus B) was the illumination supplied by the shielded houselight (operated at 20 V) located on the wall opposite the food tray. A Pentium III 800 MHz computer running MED-PC for Windows (Version 4.0) controlled experimental events with 10-ms resolution.

All experimental sessions were conducted in darkness. In the first two 20-min sessions, the animals were trained to retrieve pellets from the food tray; pellets were delivered according to a variable time 60-s schedule during these sessions. Rats were then randomly assigned to one of three equal-sized groups (Group B−AB, Group AB−B, and Group CTRL) before starting the preexposure phase.

There were four preexposure sessions, each lasting 40 min. Each of these sessions contained six stimulus presentations with an average interstimulus interval of 315 s. All stimulus presentations were 15-s long. Animals in Group B−AB received presentations of light (stimulus B) on the first two sessions of preexposure, and presentations of noise plus light simultaneous compound (the AB compound) on the second two sessions; animals in Group AB−B received presentations of the compound on the first two sessions and presentations of light on the second two sessions. Finally, animals in Group CTRL did not receive any stimulus presentations during the four preexposure sessions.

Five sessions of conditioning followed. For all animals, each of these sessions comprised six presentations of the light followed by two pellets of food. The number of times the animal inserted its head into the food tray was recorded during the stimulus presentation (the CS period) and during the equivalent period of time that preceded each stimulus presentation (the pre-CS period). The principal response measure was the difference score, calculated as CS responses minus pre-CS responses.

Results and Discussion

No data were recorded during the preexposure phase. Performance during the conditioning phase with light as the CS is shown in Figure 2b, where CS and pre-CS scores are averaged across trials to produce a single difference score for each session. This shows that responding during the presentation of light increased progressively across sessions, but at different rates, for the three groups. It is evident that Group B−AB showed a slower increase in responding than Group AB−B, which showed a conditioning rate only marginally less than that shown by Group CTRL. ANOVA with group and session as the variables confirmed the statistical reliability of these differences in responding. There were significant effects of session, $F(4, 84) = 20.77$, $\eta_p^2 = 0.50$, 95% CI [0.32, 0.59], and of group, $F(2, 21) = 4.17$, $\eta_p^2 = 0.28$, 95% CI [0.00, 0.50]. The Group × Session interaction was also significant, $F(8, 84) = 2.28$, $\eta_p^2 = 0.18$, 95% CI [0.00, 0.25]. Subsequent analyses performed to reveal the source of this interaction showed that the effect of group was not significant from Session 1 to Session 4, Fs$(2, 21) < 2.23$, ps $> .13$, but was significant on Session 5, $F(2, 21) = 6.41$, $\eta_p^2 = 0.38$, 95% CI [0.04, 0.57]. Post hoc comparisons with the Duncan test showed that on this session scores of Group B−AB differed from those of Group AB−B, $d = 1.26$, 95% CI [0.17, 2.33], and Group CTRL, $d = 1.67$, 95% CI [0.49, 2.80].

These differences were not a consequence of differences in baseline (pre-CS) responding. In all three groups, pre-CS rates were low and tended to decline slightly over sessions (mean response per minute for the five sessions of conditioning were: 0.97, 0.85, 0.70, 0.33, and 0.91 for Group B−AB; 1.22, 0.81, 0.68, 0.64, and 0.45 for Group AB−B; and 1.27, 0.87, 1.02, .52, and 0.45 for Group CTRL). ANOVA with group and session as the variables showed a borderline effect of session, $F(4, 84) = 2.19$, $p < .1$; neither the effect of group nor the interaction was significant (Fs < 1). The results of this study thus confirm those of Experiment 1, demonstrating that learning about the target stimulus B is more retarded after preexposure to the sequence B−AB, than after the sequence AB−B. A control group confirmed that conditioning proceeds readily when no preexposure was given.

Although it extends the generality of the previous finding, the procedure used in this experiment introduces a potential complication. As is customary with this procedure, the rats received pretraining in which food was delivered; food was then not available during preexposure. The start of stimulus preexposure thus coincided with the omission of food that might have been expected on the basis of the contextual cues. In these circumstances, the stimulus presented at the start of the preexposure phase could have come to act as a signal for the absence of food, in which case retarded subsequent learning could be a consequence (at least in part) of conditioned inhibition (rather than latent inhibition). Because Group B−AB received presentations of B alone at the start of preexposure, any such effect might be expected to be greater in this group than in the AB−B group. Although speculative, there is nothing in our results that allows us to reject this possible explanation; but an interpretation on the basis of modulation of latent inhibition might be preferred on the grounds of parsimony, given the parallel between these results and those of Experiment 1, for which an explanation in terms of conditioned inhibition does not apply.

General Discussion

The present study assessed a novel prediction made by the theory of latent inhibition proposed by Hall and Rodriguez (2010): that a stimulus (e.g., B) will acquire more latent inhibition when it is first presented in isolation and then in compound with a novel stimulus (e.g., B, B . . . AB, AB . . .), than when first presented in the compound and then in isolation (e.g., AB, AB . . . B, B . . .). The two experiments reported here confirmed this prediction both for flavor-aversion learning and in an appetitive conditioning procedure.

This prediction derives from a central tenet of the theory: that the development of latent inhibition will depend on the extent to which the preexposed stimulus predicts that some event will occur, and that the prediction error (generated by the fact that no event in fact follows the stimulus) will be greater when the target stimulus is presented in compound with another. Summation of the expectancies evoked by the two stimuli will enhance the magnitude of the prediction error. This account predicts the phenomenon referred to as "deepened" latent inhibition, in which the latent inhibition acquired after initial presentations of a target stimulus in isolation is enhanced by further presentations of that stimulus in

compound with another. This effect has been demonstrated by Leung et al. (2011, 2013) and was operating for stimulus B in the B−AB groups of our experiments.

In the experiments reported by Leung et al. (2011, 2013), the basic comparison made was between (in the present notation scheme) subjects given B−AB preexposure and subjects given equivalent exposure just to B (i.e., B−B). As they noted, this procedure would allow the formation, during preexposure, of a within-compound (B−A) association in the former condition and, with it, the possibility that the effect seen in the test stage might arise, in some way, from the unexpected omission of A in the B−AB condition. This possibility seems unlikely to be of importance in the present procedure in which comparison was made between groups that both received presentations of the AB compound at some stage during preexposure. And in both cases, the strength of any within-compound association is likely to be weak—exposure to B prior to AB might be expected to retard formation of the association; exposure to B after AB might allow extinction of the association.

We do not know, however, whether such within-compound associations as may be present at the start of conditioning will be equivalent in the two groups; and if it is hypothesized that the association is weaker in the AB−B condition than in the B−AB condition (i.e., that the extinction allowed by B presentations in the former group is particularly effective), then it may be possible to derive an explanation for our findings from the account of latent inhibition offered by Reed (1995). Reed suggested that after compound (AB) preexposure, the degree of latent inhibition shown to B will depend not only on the ability of B itself to activate a no-event representation but also on the ability of A to do so, A being activated associatively via the within-compound association. The assumption that this association is stronger after B−AB exposure than after AB−B exposure could thus explain our findings.

In the absence of direct evidence of the strength of the within-compound associations in the two groups of our experiment, this interpretation will remain speculative. But some features of the details of the performance observed in Experiment 2 may argue against it. In this experiment, all subjects received 36 conditioning trials with the B stimulus, which may be presumed to be enough to allow extinction of the B−A association in both groups (the hypothesis under consideration required that the 12 B-alone trials given during preexposure were enough to substantially weaken the association for the AB−B group). Accordingly, any difference between the groups in their test performance that was produced by a difference in the strength of the within-compound association might be expected to be evident only at the start of conditioning. This was not what we observed, because the difference between the groups developed over trials and was most clearly seen in the final block of trials, when the contribution from within-compound associations would be minimal.

Our analysis of these training procedures was based on the assumption that the elements of the compound stimulus are perceived and treated as such during compound stimulus preexposure. But we must consider the possibility that the AB compound may be treated as a separate configural stimulus, different from A and B. In its most extreme form (assuming no generalization between the configure and its constituents), this analysis would imply that the AB trials for animals given the AB−B sequence would make no contribution to the latent inhibition accruing to B. The situation could be different, however, for subjects given the B−AB sequence. If initial presentations of B alone interfere with the formation of the configure and allow B to be perceived during the compound trials, latent inhibition to B could be acquired during both phases of preexposure. The result would be slower learning about B after B−AB than after AB−B preexposure—the outcome obtained.

There is nothing in the present data that allows us to reject this possible interpretation, but the results of a previous experiment render it untenable. Using stimuli and procedures identical to those of Experiment 1, Rodriguez and Hall (2008) made a direct comparison of the effects of exposure just to an element or to the compound and showed that latent inhibition was enhanced in the latter case. This could not have occurred had the compound been perceived as a configure different from its elements. This enhancement of latent inhibition is, however, a central prediction of the theory proposed by Hall and Rodriguez (2010).

References

Hall, G., & Rodriguez, G. (2010). Associative and nonassociative processes in latent inhibition: An elaboration of the Pearce-Hall model. In R. E. Lubow & I. Weiner (Eds.), *Latent inhibition: Data, theories, and applications to schizophrenia* (pp. 114–136). Cambridge, England: Cambridge University Press. doi:10.1017/CBO9780511730184.007

Hall, G., & Rodriguez, G. (2011). Blocking of potentiation of latent inhibition. *Journal of Experimental Psychology: Animal Behavior Processes, 37,* 127–131. doi:10.1037/a0020716

Leung, H. T., Killcross, A. S., & Westbrook, R. F. (2011). Additional exposures to a compound of two preexposed stimuli deepens latent inhibition. *Journal of Experimental Psychology: Animal Behavior Processes, 37,* 394–406. doi:10.1037/a0023714

Leung, H. T., Killcross, A. S., & Westbrook, R. F. (2013). A further assessment of the Hall–Rodriguez theory of latent inhibition. *Journal of Experimental Psychology: Animal Behavior Processes, 39,* 117–125. doi:10.1037/a0031724

Lubow, R. E., & Moore, A. U. (1959). Latent inhibition: The effect of non-reinforced exposure to the conditioned stimulus. *Journal of Comparative and Physiological Psychology, 52,* 415–419. doi:10.1037/h0046700

Lubow, R., & Weiner, I. (Eds.). (2010). *Latent inhibition: Cognition, neuroscience and applications to schizophrenia*. Cambridge, England: Cambridge University Press. doi:10.1017/CBO9780511730184

Pearce, J. M., & Hall, G. (1980). A model for Pavlovian conditioning: Variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review, 87,* 532–552. doi:10.1037/0033-295X.87.6.532

Reed, P. (1995). Enhanced latent inhibition following compound preexposure. *The Quarterly Journal of Experimental Psychology, Section B: Comparative and Physiological Psychology, 48,* 32–45.

Rodriguez, G., & Hall, G. (2008). Potentiation of latent inhibition. *Journal of Experimental Psychology: Animal Behavior Processes, 34,* 352–360. doi:10.1037/0097-7403.34.3.352

Received January 31, 2014
Revision received April 30, 2014
Accepted May 27, 2014 ■