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# Inhibitory Properties of a Latent Inhibitor After Preexposure in Compound With Novel Stimuli

Unai Liberal and Gabriel Rodríguez
University of Basque Country (UPV/EHU)

Geoffrey Hall
University of York and University of New South Wales

*Latent inhibition* refers to retardation of the development of a conditioned response when the conditioned stimulus (CS) is preexposed alone prior to its pairings with an unconditioned stimulus. Experiment 1 demonstrated this effect for rats trained in an appetitive conditioning procedure and confirmed that the effect is found when the target stimulus is presented in compound with another or with a range of other stimuli during preexposure. Previous work has shown that a latent inhibitor does not reliably reduce the level of conditioned responding supported by an excitatory CS when the 2 stimuli are presented in compound (in a summation test). In Experiments 2, 3, and 4 we demonstrate that preexposure in which the target stimulus is presented in compound with a novel event on every trial will render that stimulus effective in a summation test. This outcome is uniquely predicted by the account of latent inhibition proposed by Hall and Rodríguez (2010), which suggests that the latent inhibition effect is a consequence both of a reduction in the associability of the stimulus and of a process of inhibitory associative learning that opposes the initial expectation that a novel event will be followed by some consequence.

*Keywords:* latent inhibition, inhibitory learning, appetitive conditioning, rats



The term *latent inhibition* refers to the retardation of classical conditioning produced by preliminary nonreinforced exposure to the event to be used as the conditioned stimulus (CS; for reviews, see Lubow, 1989; Lubow & Weiner, 2010). The use of the term *inhibition* in this context has sometimes been thought to be a misnomer, to the extent that the inhibitory properties of such a stimulus do not fully match those shown by a Pavlovian conditioned inhibitor (e.g., Wagner & Rescorla, 1972). Accordingly, theories that attribute the latent inhibition effect to a change in the attention paid to the stimulus have come to dominate (see, e.g., Lubow, 1989). The general aim of the present study is to reassess the role of conditioned inhibition in the latent inhibition phenomenon, and, in particular, to test predictions of our account of latent inhibition (Hall & Rodríguez, 2010, 2011) in which inhibitory and attentional processes are seen, not as rivals, but as being jointly responsible for the phenomenon.

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Unai Liberal and Gabriel Rodríguez, Department of Psychology, University of Basque Country (UPV/EHU); Geoffrey Hall, Department of Psychology, University of York, and Department of Psychology, University of New South Wales.

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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.eus

In several formal theories of conditioning (e.g., Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981), it has been proposed that a "true" inhibitor is one that signals the absence of a given unconditioned stimulus (US). Thus, for example, an inhibitor in the Rescorla–Wagner formulation is a conditioned stimulus (CS) that has a negative value of their associative strength parameter; Pearce and Hall (1980; following Konorski, 1967) suggested that an inhibitor activates a no-US representation that inhibits activity in a particular US representation. In a seminal article, Rescorla (1969) made the widely accepted proposal that a double test is necessary to demonstrate that a given stimulus possesses such inhibitory properties. Specifically, it is necessary to perform both a retardation test and a summation test. In the retardation test, a stimulus with inhibitory properties should take longer to become associated with the US (and thus to evoke a conditioned response [CR], when it is paired with the US in an excitatory conditioning procedure). In the summation test, a genuine inhibitor should suppress activation of the US representation and, thus, when presented in compound with it, should reduce the effectiveness of any CS that has an excitatory connection with that US. Clearly, a latent inhibitor "passes" the retardation test; its effectiveness in the summation test procedure is more debatable.

Experiments using a summation test to assess the effects of nonreinforced preexposure have been reported by Kremer (1972), Reiss and Wagner (1972), Rescorla (1971), and Solomon, Lohr, and Moore (1974). These studies all tested, in slightly different experimental designs, the effect of adding a second stimulus (A) to a previously established excitatory CS (X). In the experimental condition, A had been trained as a latent inhibitor by prior presentation alone, in the absence of reinforcement. In the control conditions, A was either novel at the beginning of the test (Kremer, 1972; Rescorla, 1971) or had been preexposed much less often

than in the experimental condition (Reiss & Wagner, 1972; Solomon et al., 1974). If nonreinforced preexposure endows a latent inhibitor with genuine inhibitory properties, the ability of A to disrupt the CR evoked by X should be greater in the experimental than in the control conditions. Only the study by Kremer has produced results consistent with this prediction (Hall & Rodríguez, 2010 have argued that this result is likely to be a consequence, not of inhibition, but of habituation of the unconditioned response to the preexposed stimulus). Solomon et al. (1974) found no differences between the experimental and control conditions, and Rescorla (1971) and Reiss and Wagner (1972) found the opposite result: a reduced CR to AX in the control condition, indicating that prolonged nonreinforced preexposure had decreased the ability of the stimulus to disrupt the CR. We should acknowledge that the possible role of inhibition in these studies might have been obscured by other processes that could affect transfer of responding from X to AX (e.g., X could suffer generalization decrement with the addition of A, or a novel A might evoke unconditioned responses capable of being confounded with the CR; see Hall, 1991). But what this set of results clearly indicates is that, although it is relatively easy to demonstrate the apparently inhibitory properties of a preexposed stimulus with a retardation test, evidence of its ability to pass a summation test is much more problematic.

Although it is a problem for the inhibitory account, this pattern of results fits well with attempts to explain the phenomenon in attentional terms. If a latent inhibitor is a stimulus that fails to command attention, it will be difficult to establish that stimulus as a CS during subsequent CS–US pairings, and it will show a reduced, or null, ability to disrupt the CR evoked by another CS in a summation test. Acceptance of the attentional, rather than the inhibitory, account of latent inhibition was strengthened by the demonstration (e.g., Rescorla, 1971) that latent inhibition retards subsequent inhibitory learning just as it retards excitatory learning. If nonreinforced preexposure allows the acquisition of inhibitory properties, it should facilitate the subsequent acquisition of conditioned inhibition; but if preexposure reduces the power of the CS to command processing, then retarded acquisition, even of conditioned inhibition, is to be expected. On the basis of these considerations Wagner and Rescorla (1972) accepted that latent inhibition required an attentional explanation, and offered the specific suggestion that it reflected a reduction in the value of the learning-rate parameter (alpha) of their formal model, the parameter that determines the associability of the CS. The suggestion was taken up by others, and Pearce and Hall (1980) made the notion of changes in associability (in alpha) central to their account.

The central principle of the Pearce and Hall (1980) model was that the alpha-value of a CS declines when it is reliably followed by a given consequence. This was applied to a range of conditioning procedures but was not fully worked out for the case of latent inhibition itself, in which no event follows stimulus presentation. This omission was addressed in the development of the model proposed by Hall and Rodríguez (2010, 2011). In brief, Hall and Rodríguez suggested that the nonreinforced stimulus presentations of the latent inhibition procedure should be treated theoretically in the same way as those involved in an extinction procedure (see also Westbrook & Bouton, 2010). The theory interprets the latter as producing inhibitory learning, specifically the development of a CS–no US association, generated by the omission of the expected US. There is no equivalent US expectation in latent inhibition, but we supposed that a novel salient stimulus would (perhaps by generalization) evoke the expectation of a consequence of some sort. Inhibitory learning (equivalent to extinction) could then occur, establishing a stimulus-no event association that would, given enough training, negate the expectation of the occurrence of some event. As the stimulus comes to predict its consequence (i.e., no event) accurately, its alpha value will fall to zero. This loss of associability will be an important source of latent inhibition, and readily explains the retardation of both excitatory and inhibitory conditioning.

Hall and Rodríguez (2010) were neutral about the possible contribution of the reduction in stimulus-event association to latent inhibition effects. They acknowledged the possibility that the strength of this association might influence further conditioning but could find no evidence to compel acceptance of this interpretation. But what is critical for our present concerns is the fact that, according to this account, the amount of acquired inhibition is not enough to turn the preexposed stimulus into a net inhibitor—rather, standard preexposure will simply reduce to some extent the preexisting excitatory strength that allows the animals to expect that the presence of a stimulus will be followed by some event. Hence, our account of latent inhibition, in common with other accounts adopting an attentional view, can explain the ability of a latent inhibitor to pass the retardation but not pass the summation test. It differs from other attentional theories, however, in that it does have a component of inhibitory associative learning. This allows us to predict that for certain forms of nonreinforced preexposure it will be possible to turn a stimulus into a net inhibitor of the occurrence of a subsequent event and thus produce a latently inhibited stimulus that will be effective in a summation test. Specifically, the account predicts that the target stimulus will acquire inhibitory properties when it is presented in compound with a novel event on each preexposure trial. The basis for this prediction is best presented in terms of the formal model of Hall and Rodríguez (2010).

In brief, the model assumes that any novel stimulus will evoke the expectation that some event will follow—that there is a stimulus-event association that has some initial strength. When this expectation is contradicted by the fact that, in nonreinforced preexposure, no event follows the stimulus, inhibitory learning occurs. Following the account of extinction of the original Pearce-Hall model, we suppose that nonreinforced exposure results in the development of a stimulus-no event association (of strength $V_{\text{no event}}$), 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 salience of the stimulus (and is mainly determined by its intensity); $\alpha$ is a variable that represents the amount of processing afforded by the stimulus (its associability); and $\lambda_{\text{no event}}$ represents the magnitude of the inhibitory reinforcer. The magnitude of the inhibitory reinforcer will depend on the degree to which the event was expected; that is,

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

Here, $\Sigma V_{\text{event}}$ represents the summed strength of any stimuli present predicting some event; $\Sigma V_{\text{no event}}$ is the summed strength

of stimuli predicting no event. In line with the original Pearce-Hall model, the value of α will then change according to the equation

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

where the associability of the stimulus on trial n, $\alpha^n$, is determined by the absolute value of the discrepancy between $\lambda_{\text{event}}$, which will be zero during the nonreinforced preexposure trials—and the total strength of the expectation that some event was going to occur ($\Sigma V_{\text{event}} - \Sigma V_{\text{no event}}$) on the basis of all the stimuli present on trial n-1. The basic principle of the Pearce and Hall (1980) model remains unchanged: the associability of a stimulus on the early trials of the nonreinforced preexposure will be high because there will be a discrepancy between what is expected (that some event is going to occur) and what actually happens (that no event happens). Repeated presentations of the stimulus will eliminate this discrepancy and associability will fall to zero.

The formalization just presented takes account of the summed strength of all stimuli present on a trial, and this allows novel predictions about the latent inhibition effects to be expected when compound stimuli are used. When a stimulus compound is presented each constituent element will activate an expectancy that some event will occur, and that the sum of these expectancies will generate the overall expectancy ($\Sigma V_{\text{event}}$). All the constituent elements of the compound will thus contribute to generating the inhibitory reinforcer ($\lambda_{\text{no event}}$) when the expected event does not occur during nonreinforced preexposure (Equation 2). Each element of the compound will undergo inhibitory learning (i.e., there will be increments in $V_{\text{no event}}$) as a function of its individual salience and associability and of the magnitude of the inhibitory reinforcer (which will be the same for all the elements present; Equation 1). Let us consider the case of the repeated presentation of a compound consisting of a target stimulus A and a nontarget stimulus B. Their concurrent presentation will generate a greater inhibitory reinforcer ($\lambda_{\text{no event}}$) than that generated by presenting A in isolation (in the compound condition both $V_{\text{event}}$ of A and $V_{\text{event}}$ of B will contribute to $\lambda_{\text{no event}}$). Inhibitory learning to the target stimulus A (and the corresponding reduction in associability) will thus occur faster when the target stimulus is presented in compound than when it is presented alone. Early studies of the effect of this compound exposure procedure (e.g., Honey & Hall, 1988, 1989; Mercier & Baker, 1985; Rudy, Krauter, & Gaffuri, 1976) failed to find such an effect (indeed some found a reduction in the magnitude of latent inhibition). But, as the authors of several of these reports themselves pointed out, their results could have been a consequence of generalization decrement effects. That is, the presence of the B stimulus might have modified the way in which stimulus A was perceived, so that learning about that stimulus during preexposure would fail to transfer to the A stimulus presented alone on test. More recently we (Hall & Rodríguez, 2011; Rodríguez & Hall, 2008; Rodríguez, Márquez, Gil, Alonso, & Hall, 2014; see also, Leung, Killcross, & Westbrook, 2013) have investigated the effects of this compound exposure procedure using stimuli selected to reduce or eliminate generalization decrement effects, and these experiments have uniformly confirmed the prediction that the presence of the nontarget B can potentiate the effect of exposure to A.

We now consider the case in which the target stimulus is repeatedly exposed in compound, not with the same partner on every trial, but with a different novel stimulus on each trial (i.e., exposure to $An_1$, $An_2$, $An_3$ . . .). The effects of this form of exposure will be rather different from those generated by preexposure schedules in which the stimulus, or stimuli, remain constant (e.g., when the preexposed event is always A in isolation, or A in simultaneous compound with B). On these "constant" preexposure schedules, the amount of inhibitory strength ($V_{\text{no event}}$) acquired by the target during the preexposure cannot exceed the amount of excitatory strength ($V_{\text{no event}}$) governed by the stimulus at the start of preexposure—as the preexposed event acquires more inhibitory strength ($V_{\text{no event}}$), the absence of the expected event grows less surprising, and the magnitude of the inhibitory reinforcer ($\lambda_{\text{no event}}$, Equation 2), and the associability of the event ($\alpha^n$, Equation 3) progressively decrease. That is, for such constant schedules, the model anticipates that nonreinforced preexposure will neutralize the preexisting excitatory value of the target but will not endow it with net inhibitory properties (i.e., produce a stimulus possessing more inhibitory strength, $V_{\text{no event}}$, than excitatory strength, $V_{\text{event}}$). However, net inhibitory status could be achieved by presenting the target in compound with a different novel stimulus on each trial ($An_1$, $An_2$, $An_3$ . . .). Under these conditions, the presence of a novel nontarget stimulus on each trial ($n_1$,$n_2$, $n_3$ . . .) will ensure a substantial activation of the expectancy that some event will occur, and accordingly $\lambda_{\text{no event}}$ will be maintained at a relatively high value throughout preexposure. As a consequence, even when the target stimulus A has acquired inhibitory strength ($V_{\text{no event}}$) equivalent to the amount of its preexisting excitatory strength ($V_{\text{no event}}$), it will still be able to acquire more inhibitory strength, thanks to the permanent contribution of the novel "n" stimulus maintaining the activation that some event is going to occur and thus in maintaining a relatively substantial inhibitory reinforcer, $\lambda_{\text{no event}}$. Under these conditions, the model anticipates that the target stimulus will suffer not only rapid extinction of the expectancy that some event is going to occur (Equation 1), but also predicts that A can become a net inhibitor of the expectancy that some event will occur.

Figure 1 shows the results of a simulation, using Equations 1 through 3, for two exposure conditions, one in which the target stimulus (A) is exposed alone (A condition) and other in which the target is exposed in compound with a different novel stimulus ($n_1$, $n_2$, $n_3$ . . .) on each exposure trial (the AN condition). We used starting values of 0.4 for S (salience), α (associability), and the net $V_{\text{event}}$ parameter for all the stimuli. As the figure shows, in the A condition, the process of extinction of the expectancy that some event will occur (the growth of $V_{\text{no event}}$) neutralizes the associative value of A (net $V_{\text{event}}$ reaches a value close to zero). With it, the value of alpha declines steadily. In the AN condition, the presence of a novel stimulus on each trial generates larger values for alpha from the second trial of preexposure. This is because each "n" stimulus ($n_1$, $n_2$, $n_3$ . . .) will contribute with its $V_{\text{event}}$ to produce a marked discrepancy between what actually happens and the expectation that some event was going to occur (Equation 3). This will produce an initial superiority of the values of alpha in the AN condition with respect to the A alone condition, although even in this condition alpha will decline across trials as inhibitory learning about the continuously presented Stimulus A acts to reduces the error term and therefore associability (Equation 3). More important, for our present purposes, in the AN condition, the value of $V_{\text{no event}}$ for Stimulus A will continue to grow, coming to

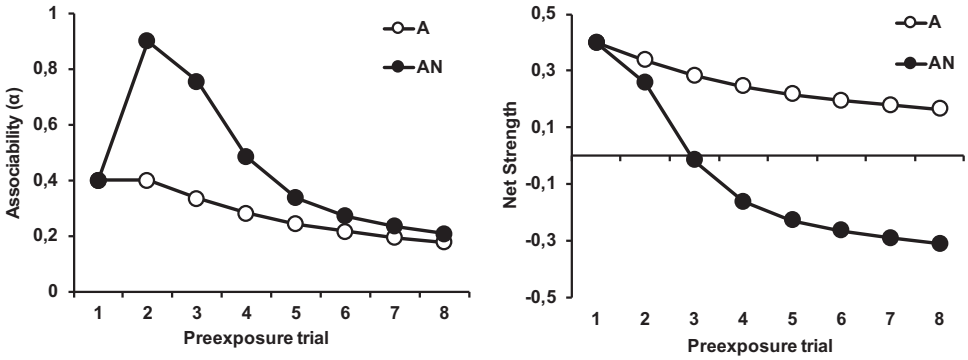


*Figure 1.* Simulation using the Hall and Rodríguez (2010) model of changes in the properties of Stimulus A. Stimulus A had a salience (S) with a value of 0.4, an initial associability (α) with a value of 0.4, and an initial $V_{event}$ value of 0.4. Each of the "N" stimuli had a salience (S) with a value of 0.5, an initial associability (α) with a value of 0.5, and an initial $V_{event}$ value of 0.5. The left panel shows the associability values of A for the two conditions, over the course of 8 preexposure trials. The right panel shows the net $V_{event}$ values during that training. In the A condition, there were eight nonreinforced presentations to A alone; in the AN condition, there were eight nonreinforced presentations to A in compound with a different novel stimulus on each trial ($An_1$, $An_2$, $An_3$, . . . , $An_8$).



exceed the starting value for $V_{event}$ for that stimulus and establishing A as a net inhibitor of the expectancy of an event.

The principle aim of the experiments be reported here is to test the implications of the conclusion that AN training will make A a net inhibitor of this sort. We adopt the assumption that such a stimulus (one that suppresses the expectation of some event) will reduce the excitatory power of another cue that has previously signaled the occurrence of some specific event (such as, e.g., the presentation of food), in other words, we assume that this form of latent inhibition training will allow the stimulus to pass a summation test. Experiments 2, 3, and 4 test this proposal. The implications of compound (AN) preexposure for a retardation test are less clear. It might be thought that a net inhibitor would be learned about only slowly, but it will be noted from Figure 1 that our model anticipates that the alpha value for stimulus A might be higher after the AN preexposure than after the A-alone preexposure. Any difference between these preexposure schedules in their effects on acquisition to A will depend on the balance of these factors (i.e., the associative value and associability of A). But despite our inability to make a firm prediction in this case, we though it worthwhile to investigate the effects of these preexposure arrangements on conditioning to A as an excitatory CS to confirm that both (the AN and A-alone procedures) are capable of producing the basic latent inhibition effect (i.e., a retardation of conditioning) with the stimuli and training procedures to be used in the later experiments investigating summation effects.

## Experiment 1

In this experiment, and those that follow, the subjects were rats trained in an appetitive classical conditioning procedure, with food as the US, the CR being the tendency of the rat to approach the site of food delivery in the presence of the CS. In this first experiment, we aimed to demonstrate that the stimulus exposure conditions, to be used later in studies using summation tests, were able to establish the target preexposed Stimulus, A, as a latent inhibitor (i.e., as a stimulus that acquires the ability to control a CR only slowly in a retardation test).

The experimental design is shown in Table 1. Three groups of rats received appetitive conditioning in which CS A (a light) was used to signal the occurrence of the US (food). The groups differed in the treatment that they received during the initial preexposure phase prior to conditioning. There were two conditions that received exposure to the target stimulus, A: Group AN and Group A. Subjects in Group A received presentations of A alone (i.e., the standard latent inhibition preexposure procedure). Subjects in Group AN received nonreinforced presentations of A in compound with a novel auditory stimulus on each trial (An1, An2, An3 . . . , An32). (This choice of stimuli, with a visual cue as the target and auditory cues as the N stimuli, was dictated by the fact that only with the auditory input could we generate the large number of N stimuli needed). We also included the standard control condition (Group NP; no preexposure); these subjects received exposure to the apparatus but no preexposure to A. Although we cannot predict whether there will be a difference between Groups AN and A, we expected to confirm that acquisition of conditioning would be retarded in both these groups relative to Group NP.

### Method

**Subjects.** The subjects were 24 naïve male adult Sprague–Dawley rats. The experiment was carried out in two identical replications. The mean ad lib weight of the 12 subjects of the first replication was 366 g (range = 295–444 g), and that for the 12 subjects of the second replication was 298 g (range = 272–311 g).

All procedures relating to the maintenance and use of animals were in accordance with the European Law of Animal Welfare and were approved by the Animal Welfare Committee of the University of the Basque Country (UPV/EHU). Animals were housed in pairs and were provided free access to water throughout the study. At the beginning of the experiment, a gradual food deprivation schedule was initiated in order to maintain the rats at 85% of their

Table 1
*Experimental Designs*

| Group | Preexposure | Conditioning | Test |
|---|---|---|---|
| | Experiment 1: Retardation | | |
| AN | 32 × AN ($An_1$, $An_2$, $An_3$ . . . . . $An_{31}$, $An_{32}$) | A→US | |
| A | 32 × A (A, A, A . . . . . A, A) | A→US | |
| NP | | A→US | |
| | Experiment 2: Summation | | |
| AN | 32 × AN ($An_1$, $An_2$, $An_3$ . . . . . $An_{31}$, $An_{32}$) | X→US | X, AX |
| BN | 32 × BN ($Bn_1$, $Bn_2$, $Bn_3$ . . . . . $Bn_{31}$, $Bn_{32}$) | X→US | X, AX |
| A | 32 × A (A, A, A . . . . . A, A) | X→US | X, AX |
| NP | | X→US | X, AX |
| | Experiment 3: Summation | | |
| AN | 32 × AN ($An_1$, $An_2$, $An_3$ . . . . . $An_{31}$, $An_{32}$) | X→US | X, AX |
| AB | 32 × AB (AB, AB, AB . . . . . AB, AB) | X→US | X, AX |
| | Experiment 4: Summation | | |
| | 16 × (AN/B) ($An_1$, B, B, $An_2$, $An_3$, B . . . . . B, B, $An_{32}$) | X→US | AX, BX |

*Note*. Each letter represents a cue (auditory or visual). The unconditioned stimulus (US) was two food pellets.



ad lib body weight. The animals were handled, weighed and fed a restricted amount of food at the end of each session to keep them at this weight for the course of the experiment. The colony room was artificially lit from 8 a.m. to 8 p.m. each day; the experimental procedures occurred during the light cycle.

**Apparatus and stimuli.** The apparatus consisted of four conditioning boxes (28 cm × 28 cm × 24 cm) made of clear acrylic plastic, each installed inside a sound-attenuating chamber. The front walls and lids were transparent; the exterior of all other sides was covered with opaque black paperboard. The floor consisted of bars, 3 mm in diameter, mounted parallel to the side walls and spaced 1.8 cm apart. The floor bars were staggered so that the odd-numbered bars were mounted 6 mm above the even-numbered bars. On the right wall of each box, a 6 cm × 6 cm square opening positioned 3 cm above the floor and 1.5 cm from the back wall, gave access to a recessed stainless-steel food cup. To record the food cup entries, an infrared photocell was mounted 3 mm behind the opening and 2.5 cm from the bottom. When the apparatus was functioning, there was a constant background noise (60 dB) measured at in the floor of the boxes.

There was no background illumination in the boxes, but three different lights were available for use as experimental stimuli, all of them positioned on the right wall. A small 28-V keylight (referred to as the left-light) was mounted on the left side of the wall, 18 cm above the floor, 2 cm from the top edge of the box and 8.5 cm from the front wall. Another light, referred as the right-light, was mounted in the equivalent position on the right side of the wall. The third light, the flashing-light, was mounted centrally, 12 cm above the floor. Only the left-light and the flashing-light were used in this experiment. Auditory stimulation was presented through a speaker mounted on the ceiling of the sound isolating chambers, 30 cm above the floor of each box. Fragments of different audio files were used as auditory stimuli. The 32 fragments used are listed in Table 2. Presentations of these sounds were controlled by a computer (linked to the main equipment that controlled the other functions of the apparatus). This additional computer had an audio device that equated the sound intensity of all the stimuli (at 80 dB) and synchronized their appearance in compound with visual stimuli. All the stimuli employed were 30 s in duration.

**Procedure.** Experimental sessions were given daily and lasted 60 min. Rats were assigned to one of three equal-sized groups (Groups AN, A, and NP; $n = 4$, in each of the two replications) before the start of preexposure phase. For half of the animals in each group, the Stimulus A was the left-light; for the remaining, A was the flashing-light. The N stimuli consisted of the sounds listed in Table 2.

Preexposure started when all the rats reached the required weight, and was carried out on 4 consecutive daily sessions of 60 min. In each of these sessions, animals in Group AN received 8 exposure trials, each of which consisted of the simultaneous presentation of the target light, A, and one of the novel sounds (An1, An2, . . . An8). A different set of eight sounds was used in each session. The intertrial interval (ITI) was variable, with a mean of 353 s, and the first trial of each session occurred 511 s after the beginning of the session. Animals in Group A received eight presentations of A alone in each session, with the same ITI as was used for the AN group. Subjects in Group NP were placed in the apparatus without any stimulus presentations.

On the day following the last preexposure session, all the animals received a single 30-min session of magazine training. In this session, rats were placed in the boxes, and four food pellets were delivered immediately. The animals were then allowed to explore the box for 15 min with no other events occurring. During the last 15 min of the session, a total of 50 food pellets (25 presentations of two pellets) were delivered at random intervals, thus allowing to the rats to learn to approach to the food cup at the sound of the feeder's activation.

On each of the three next days, rats received 12 presentations of the target light, A, each of which was immediately followed by the presentation of two food pellets. The mean of the variable ITI between the onset of the successive trials was 240 s, and the first

Table 2
*Auditory Stimuli*

| N stimulus | Description |
|---|---|
| n1 | Synthesizer arpeggio |
| n2 | Crickets chirping |
| n3 | Church bells pealing |
| n4 | Bird song |
| n5 | Old telephone ringing |
| n6 | Cow bellowing |
| n7 | School bell ringing |
| n8 | Electronic snare drum |
| n9 | Sea waves |
| n10 | Tambourine played rhythmically |
| n11 | Triangle played rhythmically |
| n12 | Women and children singing |
| n13 | Old mechanical typewriter |
| n14 | Random sounds made by a 8-bit sound machine |
| n15 | Piano ballad |
| n16 | Crowd clapping |
| n17 | Audio introduction for TV news program |
| n18 | Car horns |
| n19 | Male speech on radio |
| n20 | Morse code |
| n21 | Tuning radio sound |
| n22 | Electric guitar solo |
| n23 | Brazilian batucada (percussive samba) |
| n24 | Fireworks |
| n25 | Water fountain |
| n26 | Videogame sound |
| n27 | Chimes |
| n28 | Lawnmower |
| n29 | Laser gun shots |
| n30 | R2D2 robot sounds |
| n31 | Trumpet |
| n32 | Star Wars theme |

*Note*. Stimulus N in the AN compound varied from trial to trial. There were 32 versions of the auditory (N) stimulus, comprising 30-s fragments of different audio files.

trial of each session occurred 314 s after the beginning of the session.

**Data analysis.** In the conditioning phase, magazine entries were recorded during each 30-s CS and during the 30-s preCS period. Subtracting the number of responses made during the preCS period from the number of responses made during the CS period gave an elevation score for each trial. Data were analyzed with analysis of variance (ANOVA), and, where appropriate, *t* tests or Tukey's test. A criterion of statistical significance of $p < .05$ was adopted. Effect sizes for ANOVAs are reported as partial eta squared ($\eta_p^2$) and those for pairwise comparisons are reported using Cohen's *d*. The 95% confidence intervals (CIs) around the effect sizes are also reported in parentheses following the effect size.

## Results and Discussion

No data were collected during the preexposure phase. Group mean elevation scores over the course of the six 6-trial blocks of conditioning with A as the CS, are summarized in Figure 2. Elevation scores increased progressively in all the groups, but at different rates. Groups for which A had been preexposed in the absence of reinforcement showed a lower rate of acquisition than that shown by the nonpreexposed control group (NP). Retardation of acquisition was somewhat more enduring in Group A than in Group AN.

This description of the results was confirmed by statistical analysis. A Group × Blocks of Trials × Replication ANOVA on the data summarized in the figure revealed significant main effects of group, $F(2, 18) = 9.33$, $p = .002$, $\eta_p^2 = 0.51$ (0.11–0.67), and block, $F(5, 90) = 31.39$, $p < .0001$, $\eta_p^2 = 0.63$ (0.49–0.70). The main effect of replication was not significant, $F(1, 18) = 3.22$, $p = .089$, nor were any of the interactions ($F$s $< 1$) apart from that of Group × Block, $F(10, 90) = 2.16$, $p = .027$, $\eta_p^2 = 0.19$ (0.00–0.25). Further analyses were performed in order to explore the source of the Group × Block interaction. There was an effect of block in each of the groups, reflecting the fact that the scores for all groups increased during conditioning: $F(5, 35) = 13.96$, $p < .0001$, $\eta_p^2 = 0.66$ (0.39–0.74) for Group AN; $F(5, 35) = 8.64$, $p < .0001$, $\eta_p^2 = 0.55$ (0.24–0.65), for Group A; and $F(5, 35) = 14.17$, $p < .0001$, $\eta_p^2 = 0.67$ (0.40–0.74), for Group NP. There were no differences among the groups on the first block, $F(2, 21) = .98$, $p = .393$, but, differences emerged over the course of training. There were significant differences among the groups in blocks 2, 3, 4, 5, and 6, $F$s$(2, 21) > 4.21$, $p$s $< .029$. Tukey post hoc comparisons showed that on blocks 2 and 3, Groups AN and A both differed from Group NP; that is, both showed the latent inhibition effect. The difference was sustained only in Group A; on Blocks 4, 5, and 6 only Group A differed from Group NP.

These differences in the elevation scores were not a consequence of differences in baseline response rates among the groups. Group mean pre-CS scores pooled over all conditioning trials were 2.63 (*SEM* = 0.32), 2.86 (*SEM* = 0.43), and 3.48 (*SEM* = 0.58) responses per trial, for groups Groups AN, A, and NP, respectively ($F < 1$).

## Experiment 2

In the previous experiment, we demonstrated that the preexposure conditions experienced by Groups AN and A successfully established the target stimulus A as a latent inhibitor, as assessed by the retardation of subsequent conditioning. We now test the ability of these preexposure conditions to allow the target stimulus to pass a summation test. According to previous literature, we

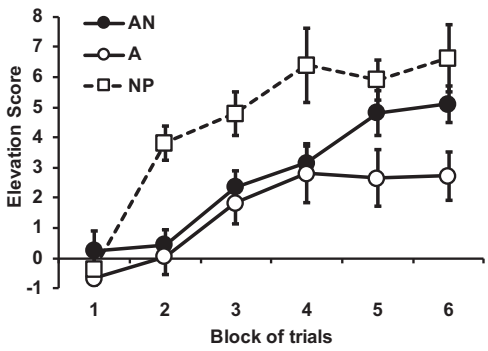


*Figure 2.* Experiment 1: Group mean elevation scores (±*SEM*) in the presence of A during conditioning. Group AN received prior exposure to A in compound with a novel stimulus on each trial during preexposure; Group A received preexposure to A alone; and the control group, NP, received exposure just to the experimental context.

should not find clear evidence in this regard in Group A, but according to the predictions of the Hall and Rodríguez (2010) account presented in the preceding text, evidence for an effect on a summation test can be expected in Group AN. The aim of the present experiment was to test this prediction.

The design of the experiment is shown in Table 1. After the preexposure phase, all subjects received appetitive conditioning in which a novel stimulus (X, a light) was used as a CS signaling the occurrence of the US (food). After this conditioning stage, the ability of the target stimulus A (a second different light) to interfere with the CR evoked by X was assessed in nonreinforced test trials with X presented either alone, or in compound with A. There were four groups that differed in the treatment that they received during the initial preexposure phase. Two groups received the same conditions of exposure to the target stimulus, A, as were tested in Experiment 1: Groups AN and A. There were two control conditions that received no exposure to A during the preexposure phase: Group NP (which, as in Experiment 1, was simply preexposed to the apparatus) and Group BN. This last group was added in order to confirm that the effects of exposure to the set of novel N stimuli in Group AN, were dependent on their occurring along with A, Thus, during preexposure, Group BN received nonreinforced presentations of a nontarget stimulus (B; a second nontarget light) in compound with the same set of novel stimuli that were presented to Group AN (i.e., Bn1, Bn2, Bn3, . . . , Bn32). According to the predictions of the Hall-Rodríguez account outlined above, the ability of stimulus A to interfere with the CR evoked by X should be greater in Group AN than in the other groups.

## Method

The subjects were 64 naïve male adult Sprague–Dawley rats. The experiment was carried out in two identical replications. The mean ad lib weight of the 32 subjects of the first replication was 384 *g* (range = 338–437 g), and that for the 32 subjects of the second replication was 377 *g* (range = 304–474 g). Animals were housed and maintained in the same conditions as those described in Experiment 1, in this and all the following experiments.

Stimuli n1-n32 were the same 32 fragments of different audio files used in Experiment 1 (see Table 2). The left-light and the flashing-light served as Stimuli A and B, counterbalanced between subjects. For all the animals, the right-light served as the CS X. As in Experiment 1, all the stimuli used were 30 s in duration.

Rats were assigned to one of four equal-sized groups (Groups AN, BN, A, and NP; $n = 8$, in each of the two replications) before the start of preexposure phase. The procedure in this phase for Groups AN, A, and NP was the same as that described in Experiment 1. Group BN received treatment identical to that received by Group AN, but the exposed compounds included the nontarget light, B, rather than the target A. On the day following the last preexposure session, all the animals received a single session of magazine training. On each of the three next sessions, all received 12 presentations of the nontarget light, X, each of which was immediately followed by food.

On the day following the last conditioning session, all subjects received a test session with a presentation of X alone and a presentation of AX. The first stimulus presentation occurred 511 s after the beginning of the session, and the second 330 s later. The order of presentation of the test stimuli was counterbalanced. For half the subjects in each Group X was presented first and AX second; for the rest of the animals the order was reversed. We gave only a single test trial with each stimulus as pilot work had shown that effects were most evident on the first trial of a series of tests. Details of the procedure not specified here were the same as those described for Experiment 1.

## Results and Discussion

No data were recorded during preexposure. A preliminary analysis of preCS rates during the conditioning phase revealed no differences among the groups in their baseline levels of responding. In summary, the group mean scores pooled over all conditioning trials were 2.85 (*SEM* = 0.23), 3.01 (*SEM* = 0.28), 2.61 (*SEM* = 0.23), and 2.71 (*SEM* = 0.32) responses per trial, for Groups AN, BN, A and NP, respectively ($F < 1$). The conditioning trials with X as the CS established an elevated rate of magazine responding in the presence of X in all groups. The mean elevation scores during the six 6-trial blocks of conditioning are shown in the left panel of the Figure 3. Elevation scores increased progressively (indicating the acquisition of conditioning), and at a similar rate, in all the groups. A Group × Blocks of Trials × Replication ANOVA conducted on these data revealed significant main effects of block, $F(5, 280) = 59.71$, $p < .0001$, $\eta_p^2 = 0.51$ (0.43–0.57), and of replication, $F(1, 56) = 6.06$, $p = .017$, $\eta_p^2 = 0.09$ (0.002–0.25). Tukey's test revealed smaller elevation scores in Replication 1 than in Replication 2. The main effect of group was not significant, $F(3, 56) = 1.35$, $p = .268$, nor were any of the interactions, largest, $F(15, 280) = 1.21$, $p = .261$.

PreCS responding during the test phase was similar for all groups and for each of the stimuli. Mean scores were 1.62 (*SEM* = 0.37), 2.06 (*SEM* = 0.54), 1.25 (*SEM* = 0.37), and 2.18 (*SEM* = 0.49) for Groups AN, BN, A, and NP (respectively) for preCS periods prior to X, and 2.12 (*SEM* = 0.37), 2.18 (*SEM* = 0.51), 2.62 (*SEM* = 0.86), and 2.43 (*SEM* = 0.81) prior to AX. A Group × Stimulus × Replication ANOVA performed with these data revealed no significant effect: largest $F(3, 56) = 2.35$, $p = .082$, for the Stimulus × Group × Replication interaction.

The critical results of the test phase, group mean elevation scores for the trials with X and AX are presented in Figure 3. The elevation scores supported by the excitor on the X-alone trials were similar in all the groups, but there were marked differences in the ability of the added cue A to reduce responding to the excitor. Adding the (novel) A to the X produced almost no change in responding (between X and AX) in groups BN and NP. However, exposure to A (in Groups A and AN) seems to have endowed this stimulus with the ability to reduce responding to X, although the size of this reduction was substantial only in Group AN. Statistical analysis of the data summarized in the figure confirmed this description. A Group × Stimulus × Replication ANOVA revealed a significant effect of stimulus, $F(1, 56) = 9.84$, $p = .003$, $\eta_p^2 = 0.14$ (0.01–0.31), but no significant effects of either group, $F(3, 56) = 1.78$, $p = .162$, or replication, $F(1, 56) = .44$, $p = .506$. Critically, however, the interaction of Group x Stimulus was significant, $F(3, 56) = 3.26$, $p = .028$, $\eta_p^2 = 0.14$ (0.00–0.28). None of the remaining interactions were be significant, the largest being Group × Stimulus × Replication, $F(3, 56) = 1.37$, $p = .262$.

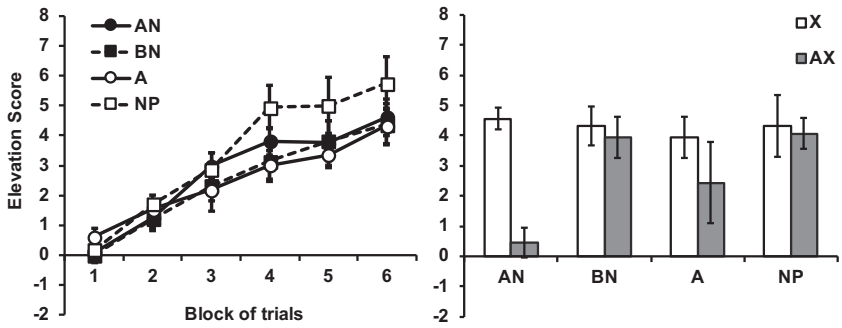


*Figure 3.* Experiment 2: Group mean elevation scores (±*SEM*s) to Stimulus X during conditioning (left panel) and to X and AX on the subsequent summation test (right panel). The AN group received prior exposure to the target Stimulus A in compound with a novel stimulus on each trial; the BN group received prior exposure to a nontarget Stimulus B in compound with a novel stimulus on each trial; the A group received exposure to A alone; and the control group, NP, received exposure just to the experimental context.



Further analyses were performed in order to explore the source of the Group × Stimulus interaction. There were no significant differences among groups in responding to X, $F(3, 60) = .13$, $p = .944$, but the groups differed in their responding to AX, $F(3, 60) = 4.13$, $p = .01$, $\eta_p^2 = 0.17$ (0.01–0.31). Tukey's HSD post hoc comparisons revealed that Group AN responded less to AX than did Groups BN and NP, consistent with the proposal that A had acquired inhibitory properties in the AN condition. Direct comparison of the response to the test stimuli for each group revealed a significant stimulus effect (less responding to AX than to X) in Group AN, $t(15) = 6.46$, $p < .001$, $d = 1.61$ (0.85–2.35); none of the other groups yielded a significant effect: the largest effect, for Group A, was $t(15) = 1.11$, $p > .285$. Thus, although responding to AX was somewhat less than to X in the group given standard latent inhibition training (the A group), there is no strong support for the idea that A had become inhibitory. Group AN, on the other hand, showed a clear and reliable suppression of responding when A was added to X. The results of the BN group, which like the AN group, received exposure to a range of novel stimuli, show that the effect of this treatment is dependent on these stimuli being presented in compound with the target stimulus A, as would be expected on the basis of the Hall and Rodríguez (2010) account.

## Experiment 3

The results of Experiment 2 support the proposal that preexposure in which the target stimulus (A) is presented repeatedly along with a novel stimulus (the conditions employed in Group AN) will endow Stimulus A with the ability to inhibit the CR controlled by an excitor. In the present experiment, we sought further evidence for this effect, in this case contrasting the performance of Group AN with that of a different control group. Table 1 shows the two conditions used in this experiment. Group AN received just the same treatment as the equivalent group in Experiment 2; the second group was added to control a possible confounding of variables in the previous experiment. In Experiment 2, Group AN was the only condition in which the target was presented in compound. That is, for Group BN there were compound presentations during preexposure, but without the presence of the target A; and in Group A, the target was presented alone. In this experiment we included a control condition in which A was preexposed in compound but always with the same stimulus, Group AB. If, as anticipated by the account of Hall and Rodríguez (2010), the inhibitory properties of A in Group AN are due to the presentation of the target A in compound with a series of novel stimuli, and not simply due to its presentation in compound during the preexposure, we can expect to replicate the summation test effect in Group AN but not in Group AB.

### Method

The subjects were 16 naïve male adult Sprague–Dawley rats, with a mean ad lib weight of 317 g (range = 287–355 g) at the start of the experiment. Stimuli n1 - n32 (see Table 2) were the same 32 fragments of different audio files used in the previous experiments. The left-light served as Stimulus A and a tone (of 3,000 Hz at 80 dB) as Stimulus B. For all the animals, the right-light served as the CS X. The rats were assigned to one of two equal-sized groups (Groups AN and AB; $n = 8$) before the start of preexposure phase. One subject in the Group AN became ill during the conditioning stage and was removed from the experiment.

The preexposure procedure for Group AN was the same as that described in Experiments 1 and 2. Group AB received identical treatment to that received by Group AN, except that on all the preexposure trials the compound consisted of the simultaneous presentation of the target light, A, and the tone, B.

On the day following the last preexposure session, all the animals received a single session of magazine. Conditioning to X followed. On each of the next 3 days, the rats received 12 presentations of the nontarget light, X, which were immediately followed by food. On the day following the last conditioning session, the rats received a test session in which they experienced one presentation of X alone and one presentation of AX. Details of the procedure not specified here were the same as those described for previous experiments.

### Results and Discussion

The mean elevation scores during the six blocks of trials of conditioning, with X as the CS, are summarized in the left panel of

the Figure 4. Elevation scores increased progressively during conditioning, at a similar rate in both groups. A Group × Blocks of trials ANOVA on these data revealed only a significant main effect of block, $F(5, 65) = 29.97$, $p < .0001$, $\eta_p^2 = 0.72$ (0.56–0.78); neither the main effect of group, $F(1, 13) = .14$, $p = .712$, nor the interaction between the two variables were significant, $F(5, 65) = 1.18$, $p = .328$. The groups did not differ in their responding during the pre-CS periods. The mean scores over all trials were 2.66 (*SEM* = 0.37), and 3.09 (*SEM* = 0.32) responses per trial for Groups AN and AB respectively: these means did not differ reliably, $t(13) = .87$, $p = .40$.

The results of the test phase are presented in the right panel of Figure 4. As in Experiment 2, Group AN showed an elevation score to AX that was much lower than that shown to the excitor, X, alone. Group AB, however, showed similar elevation scores to AX and to X alone. Statistical analyses were consistent with this description. A Group × Stimulus ANOVA showed nonsignificant main effects of group, $F(1, 13) = 1.86$, $p = .196$, and of stimulus, $F(1, 13) = 1.23$, $p = .288$, but there was a significant Group × Stimulus interaction, $F(1, 13) = 6.44$, $p = .025$, $\eta_p^2 = 0.33$ (0.003–0.59). Further analyses were performed in order to explore the source of this interaction. The groups did not differ in responding to X, $t(13) = .33$, $p = .748$, but they differed in their responding to AX, $t(13) = 3.33$, $p < .005$, $d = 1.72$ (0.49–2.91). In addition, there was a significant difference in responding to AX and to X in Group AN, $t(6) = 3.44$, $p = .014$, $d = 1.3$ (0.24–2.31), but not in Group AB, $t(7) = .88$, $p = .407$. Responding during the pre-CS periods had mean values of 1.28 (*SEM* = 0.68) and 2.75 (*SEM* = 0.72) for Groups AN and AB (respectively) on the test trial with X and 1.4 (*SEM* = 0.61) and 2.87 (*SEM* = 1.21), during the test trial with AX. These means did not differ reliably on either trial, $t$s(13) < 1.45, $p$s > .168.

These results indicate that the effect of giving preexposure to a target stimulus in compound with some other depends critically on the novelty of the added stimulus. In previous work (Hall & Rodríguez, 2011; Rodríguez & Hall, 2008; Rodríguez et al., 2014) we have investigated the effects of compound preexposure in which the added stimulus is constant. This has been shown to potentiate the development of latent inhibition as assessed by a retardation test, a result predicted by the Hall and Rodríguez (2010) theory, which supposes that the loss of associability will be rapid in this case. The theory does not predict, however, that the target stimulus would become a net inhibitor of the expectation of an event after this form of preexposure. Such an outcome can be expected, however, when the added stimulus is changed from trial to trial, generating the result obtained in this experiment in Group AN.

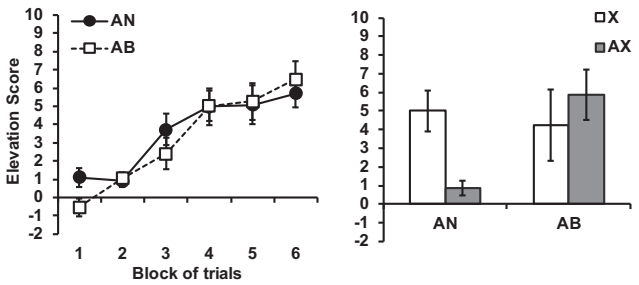


*Figure 4.* Experiment 3: Group mean elevation scores (±*SEM*) to X during its conditioning (left panel) and to X and AX on the subsequent summation test (right panel). Group AN received prior exposure to the target Stimulus A in compound with a novel stimulus on each trial; Group AB received prior exposure to a compound consisting of the concurrent presentation of the target stimulus A and a nontarget stimulus B.

## Experiment 4

In our previous experiments, we demonstrated the summation test result in Group AN, making comparison with a range of different control procedures. It remains the case, however, that we have not made comparison between this group and a control that has had equivalent experience of target Stimulus A and of a cue presented in compound with a range of novel stimuli. To achieve this, the present experiment made use of a within-subjects design in which all the rats received presentations of the target stimulus A in compound with the range of novel stimuli the AN procedure; these were intermixed with presentations of a nontarget light, B. After conditioning to a nontarget Stimulus X, we tested the ability of A and B to interfere with the response elicited by X in a summation test. According to the mechanisms proposed by Hall and Rodríguez (2010), we expect to find that A would have the ability to pass the summation test. There are no grounds for supposing that a similar effect would be obtained for Stimulus B; that is, we would expect less responding to AX than to BX.

### Method

The subjects were 16 experimentally naïve male rats with a mean ad lib body weight of 396 g (range = 368–437 g) at the start of the experiment.

The left-light and the flashing-light served as Stimuli A and B, counterbalanced between subjects. For all the animals, right-light served as the CS X. The auditory stimuli used for the N stimulus were drawn from those listed in Table 2.

In each of the four sessions of the preexposure phase, all the animals received four nonreinforced presentations of a light-sound (AN) compound in which the sounds were novel in each trial; there were also four nonreinforced presentations of the nontarget light (B) alone. The order of presentation was counterbalanced. Half of the animals received the sequence (AN, B, B, AN, B, AN, AN, B) on the odd days and the sequence (B, AN, AN, B, AN, B, B, AN) on the even days. The remaining subjects received the sequences in the opposite order.

After magazine training, on each of the following 3 consecutive days, the rats received pairings of a new light (X) with food. Each session of 66 min consisted of 12 reinforced presentations of the light. The stimulus presentation schedule was identical to the conditioning phase of the previous experiments.

The test session followed the last day of conditioning. All rats received a nonreinforced presentation of the compound consisting of the target light and the conditioned light (AX) and a nonreinforced presentation of the compound consisting of the nontarget light and the conditioned light (BX). The order of presentation of these trials was counterbalanced as in previous experiments. In details not specified here, the procedure was the same as described for the previous experiments.

## Results and Discussion

The left panel of Figure 5 shows the gradual increase in mean elevation scores during the conditioning phase with X as the CS. An ANOVA with block as the only variable, revealed a significant effect, $F(5, 75) = 22.44$, $p < .001$, $\eta_p^2 = 0.59$ (0.42–0.67). Responding during the pre-CS periods had mean values of 2.86 (*SEM* = 0.25) in the first block of trials and 2.91 (*SEM* = 0.28) during the final block.

Right panel of Figure 5 shows the mean elevation scores to AX and BX on test. The elevation score to BX was much the same that shown to X alone in the last block of conditioning. However, the presence of A in the test with AX drastically reduced this elevation score. A paired samples *t* test with Stimulus (AX vs. BX) as the only factor, conducted on these data, revealed a significant effect, $t(15) = 3.66$, $p = .002$, $d = 0.91$ (0.31–1.49). Responding during the pre-CS periods had mean values of 2.68 (*SEM* = 0.64) during test with BX and 2.43 (*SEM* = 0.63) during test with AX. These means did not differ significantly, $t(15) = .34$, $p = .74$.

Preexposure training in this experiment consisted of presentations of a target stimulus (A) in compound with a variety of novel stimuli (N) intermixed with presentations to a nontarget stimulus B in isolation. After conditioning to X, subjects showed a considerable reduction in the CR when X was presented in compound with A but not when it was presented with B. These results thus confirm that the ability of a preexposed stimulus to reduce the CR critically depends its being preexposed in compound with a range of novel stimuli.

## General Discussion

The present series of experiments has identified preexposure conditions that will endow a latent inhibitor with the ability to pass a summation test. These conditions consist of preexposing the target stimulus in compound with a different (nontarget) novel stimulus on each preexposure trial. Subjects given this form of preexposure also showed the standard latent inhibition effect; that is, a retardation of subsequent conditioning with the target stimulus as the CS. Subjects exposed to the target stimulus on its own showed an effect on the retardation test but not on the summation test. These results accord with our theoretical account which proposes that the standard latent inhibition procedure will result in a loss of stimulus associability, whereas the compound-preexposure procedure used in these experiments will endow the target stimulus with inhibitory properties. It is appropriate, however, to consider alternative interpretations of our results.

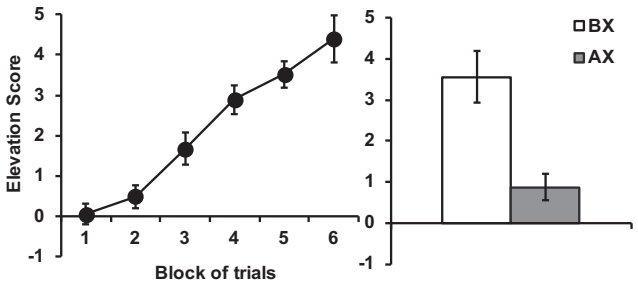


*Figure 5.* Experiment 4: Group mean elevation scores (±*SEM*s) to X during its conditioning (left panel) and to AX and BX on the subsequent summation test (right panel). The subjects had received prior training consisting of presentations of a target stimulus A in compound with different novel stimuli (An1, An2, An3. . .) intermixed with presentations of a nontarget Stimulus B, presented in isolation.

Previous work on latent inhibition and the summation test has compared the effects of a preexposed stimulus with those of a novel (or minimally preexposed) stimulus. As we have noted, those experiments making this comparison that have generated results consistent with the notion that the preexposed stimulus has acquired inhibitory properties are open to other interpretations. Specifically, a demonstration that the target stimulus (A) is effective in interfering with the CR evoked by a separately trained CS (X) on test might be explained in perceptual or attentional terms; that is, A might have its effect by modifying the perception of the CS (i.e., impeding recognition of X, generating a strong generalization decrement effect) or by distracting attention from it. It is difficult to see how an analysis of this sort could be devised for the results obtained in our experiments in which the critical comparison is between subjects preexposed to A and subjects preexposed to A in compound with novel stimuli (the AN condition). The results of Experiment 2 show a summation effect in the AN condition but no sign of a summation effect in the group given no preexposure. To explain this in terms of generalization decrement, it would be necessary to assume that AN preexposure has rendered the preexposed A even more effective than a novel A. But if this were true, conditioning of A, when it was used as the CS in Experiment 1 (i.e., in the retardation test) should have been facilitated in this preexposure condition with respect to the nonpreexposed control condition. In fact, in that experiment, the target A stimulus passed the retardation test in the critical preexposure conditions (i.e., A conditioned more slowly in group AN than in group NP).

Another possible alternative explanation of the performance of group AN on the summation test can be devised if we assume that pairing A with the wide variety of novel auditory stimuli ($n_1$, $n_2$, $n_3$ . . .) might have conditioned some sort of response generated by the presence of these latter stimuli (e.g., a mildly aversive state, some general level of "excitement" or, more simply, a startle response evoked by each of the novel stimuli). As a consequence, A could have acquired the tendency to evoke a response that would interfere with (and thus diminish) the target CR (magazine approach) governed by Cue X on the AX test trials. A reason for doubting this account comes, again, from the retardation test results of Experiment 1. It might be expected that the postulated interfering responses would be especially evident on this test making acquisition of the magazine approach CR in group AN much poorer that in Group A. No such difference was obtained, with rats in the AN condition learning slightly more readily than those in the A condition.

We should acknowledge that the force of these arguments, based on the results of the retardation test of Experiment 1, is undermined to some extent by the fact that, by our own account, these results will be determined by differences between conditions in the associability of the target cue (see Figure 1) which could act to obscure the effect produced by competing responses. Although this response–interference account cannot be

fully discarded (and should be object of future research), we now return to the implications of the view that the ability of A in the AN group to reduce the CR evoked by a separately trained excitor is to be explained by the proposal that A has acquired true inhibitory properties so that it is able to reduce the ability of the excitor to activate its associate.

The proposal that AN training will render A inhibitory is derived from, and thus supplies support for, the account of latent inhibition offered by Hall and Rodríguez (2010, 2011). According to this, latent inhibition is, at least in part, mediated by the reduction of an association between the target stimulus and the occurrence of a possible consequence. This involves inhibitory learning—the initial ability of any novel stimulus to evoke the expectancy that some event will follow will be disconfirmed during the nonreinforced exposure. When preexposure is to a stimulus in isolation, the amount of inhibition that the target will acquire will not be enough to neutralize its initial excitatory properties, and it will not acquire net inhibitory properties. However, when the target stimulus is presented in compound with a novel stimulus on each trial, the activation of the expectancy of a consequent event (ensured by the novel component) will occur even when the target has already neutralized its initial excitatory properties. In this case the target stimulus will to become a net inhibitor.

In conclusion, the significance of the results reported here is not so much that they have identified a set of preexposure conditions that allow a preexposed stimulus to pass both the retardation and the summation tests; rather it is that they lend support to an explanation of the latent inhibition phenomenon that finds a place for a contribution from inhibitory learning. Finally, we note that our theoretical position expects that the effects reported here would be found in other situations, beyond the magazine approach procedure, and the set of stimuli, employed here. Future research will be directed to determining the extent to which these effects are also found in procedures using aversive techniques (e.g., flavor aversion learning with rats) or with the neutral stimuli often used in work on human learning. Furthermore, the net inhibitory properties that the target A acquires during the compound preexposure schedule under consideration ($An_1$, $An_2$, $An_3$ . . .) should be evident in other sort of tests. For example, such a stimulus should be facilitated when it comes to further inhibitory conditioning; and it should be capable of producing protection from extinction, when it is presented in compound on nonreinforced trials with a previously trained CS.

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