Previous blocking trials impede learning about the added CS during compound conditioning trials with an intensified US.

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Abstract

A conditioned suppression experiment was conducted with rats in an attempt to test the predictions offered by the Rescorla-Wagner (1972), Mackintosh (1975), and Pearce-Hall (1980) models regarding blocking effect. A four-stage within-subject design was employed in which rats received consistent pairings of a conditioned stimulus, CS_A, with a weak shock in Stage 1. A simultaneous compound formed by the pre-trained CS_A and an added stimulus CS_B was paired with the weak shock in Stage 2. Subsequently, the same AB compound was paired with a stronger shock in Stage 3. A high level of suppression to CS_A but no suppression to CS_B was found in the final test phase. This pattern of results suggests that CS_A blocked conditioning to stimulus CS_B in both Stage 2 and Stage 3. This finding supports the predictions of the Mackintosh model, but not those offered by the Rescorla-Wagner and Pearce and Hall models.

Key words: blocking, unblocking, Rescorla-Wagner model, Pearce-Hall model, Mackintosh model, conditioned suppression, rats

Resumen

Se presenta un experimento de supresión condicionada con ratas en el que se intentó poner a prueba las predicciones ofrecidas por los modelos de Rescorla y Wagner (1972), Mackintosh (1975) y Pearce y Hall (1980), acerca del fenómeno de bloqueo. Se empleó un diseño intrasujeto de 4 fases, en el que las ratas recibieron inicialmente emparejamientos de un estímulo condicionado, EC_A, con una descarga débil durante la Fase 1. Durante la Fase 2, se emparejó un compuesto formado por el estímulo entrenado previamente, EC_A, y un elemento añadido, EC_B, con la misma descarga débil empleada en la Fase 1. Posteriormente, ese mismo compuesto AB fue emparejado con una descarga más intensa durante la Fase 3. En la fase final de prueba se observó un alto nivel de supresión ante el EC_A pero ninguna supresión ante el EC_B. Este patrón de resultados sugiere que el EC_A bloqueó el condicionamiento del EC_B durante las Fases 2 y 3. Este resultado apoya las predicciones ofrecidas por el modelo de Mackintosh, pero no las ofrecidas por los modelos de Rescorla y Wagner y Pearce y Hall.

Palabras clave: bloqueo, desbloqueo, modelo Rescorla-Wagner, modelo Pearce-Hall, modelo Mackintosh, supresion condicionada, ratas.

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A substantial part of the empirical evidence that has had a profound impact on modern theorizing in Pavlovian conditioning over the last 30-40 years has been derived from experiments investigating the *blocking* effect (Kamin, 1969). In a typical blocking experiment, the experimental condition initially receives consistent pairings of a conditioned stimulus (CS_A) with an unconditioned stimulus (US). During a second stage, a compound formed by the pre-trained CS_A and an added CS_B is paired with the same US. The blocking effect becomes evident in the final test trials when CS_B is presented by itself, eliciting a lesser conditioned response (CR) in the experimental condition than in a control condition in which the initial training with CS_A was not received. That is, the pre-training with CS_A apparently *blocks*, or prevents, conditioning to CS_B that would otherwise occur on the reinforced AB compound trials.

Traditionally, two kinds of explanations have been proposed to account for the blocking effect: those that explain blocking in terms of changes in US effectiveness and those that explain blocking in terms of changes in CS effectiveness (see for a recent review, Le Pelley, 2004).

Changes in US effectiveness

This view, advanced by Kamin (1968), argues that over the course of consistent CS-US pairings the effectiveness of a US decreases as its occurrence becomes less surprising due to the fact that it is signaled by the CS. Thus, in a blocking experiment CS_A blocks CS_B because the US that follows AB compound trials in Stage 2 is reduced in effectiveness by being signaled by CS_A. This explanation was taken up and formally developed by Rescorla and Wagner (1972) in their Pavlovian conditioning model. In short, the basic assumptions of this model are as follows. First, the strength of a CR is directly related to the *associative strength* of the CS, which is the strength of the connection between internal representations of the CS and US. Second, the increment in associative strength on a given CS-US pairing is given by the following equation:

$$\Delta V_A = \alpha_A \beta (\lambda - V_T) \qquad (1)$$

Where the change in associative strength of the CS_A, (ΔV_A), is directly related to the discrepancy between an asymptotic value set by the magnitude of the US (λ) and the sum of the associative strengths of all the stimuli present on that trial (V_T). This *error term* (λ- V_T) represents the effectiveness of the US. If the occurrence of the US is not well predicted by the presence of all the stimuli (that is, if the value of V_T is low) the US will be very effective (the error term will be large), and hence the conditioning trial will be successful (there will be a large increase in V_A). As we can see, the extent of the change in V_A is also modulated by two learning-rate parameters: a_A, which reflects the associability or conditionability of the CS_A and is a function of the intensity or salience of the CS_A; and b, which is a function of the intensity of the US.

When applied to the blocking effect, Rescorla-Wagner model predicts that sufficient initial CS_A-US pairings allow V_A to reach λ. According to Equation 1, during subsequent AB-US trials the value of the discrepancy (λ - V_T) in the experimental condition will

be near to zero due to the contribution of V_A to the value of V_T. As a result, increments in V_B on every trial of Stage 2 will be smaller than those in a control condition which did not receive the initial training with CS_A.

Changes in CS effectiveness

An alternative approach to the blocking effect has instead emphasized variations in the CS effectiveness as responsible for modulating the effectiveness of a CS-US pairing (e.g., Mackintosh, 1975; Pearce & Hall, 1980). According to this view the effectiveness of a CS, and hence the readiness to learn about it, changes as a result of the prior training undergone by that CS. Below we will consider briefly two of the most influential models of associative learning that have incorporated this notion: the Mackintosh (1975) and Pearce-Hall (1980) models. In the Rescorla-Wagner model outlined above the associability of the CS (a) was a fixed parameter depending on its intensity or salience. In Mackintosh and Pearce-Hall models, however, a is a variable able to change as a result of experience. Interestingly, although these two models share some common assumptions they both hold opposing views of the way in which a changes as a result of the experience with the CS, therefore taking different views of the process underlying blocking.

The Mackintosh (1975) model

Mackintosh (1975) stated that the change in associative strength of the CS_A on a given CS-US pairing is given by:

$$\Delta V_A = \alpha_A (\lambda - V_A) \qquad (2)$$

Although this equation is clearly similar to that of Rescorla-Wagner outlined above, it includes two important differences. First, the error term $(\lambda - V_A)$ is given by the value of the discrepancy between l and the individual strength of the CS_A (V_A), not by the sum of the strengths of all the stimuli present in that trial. And second, the influence of the error term is not only modulated by constant parameters. In Equation 2, a is a variable reflecting changes in the associability of (or in the attention paid to) the CS. According to Mackintosh, a is initially determined by the physical properties of the CS and the sensorial system of the organism. As of the first trial, α of a CS will increase if that CS predicts the US more accurately than other stimuli present in that situation but will decrease if it predicts the US less accurately. This notion is formally expressed in the following equations:

$$\Delta a_A \text{ is positive if } |\lambda - V_A| < |\lambda - V_X| \qquad (3)$$
$$\Delta a_A \text{ is negative if } |\lambda - V_A| \geq |\lambda - V_X| \qquad (4)$$

Where $\Delta\alpha_A$ is the change in associability of CS_A; λ is the magnitude of the US; V_A is the associative strength of CS_A; and V_X is the total associative strength of all

stimuli other than CS_A present. If the value of $|\lambda - V_A|$ is less than $|\lambda - V_X|$, a_A will increase as CS_A is a better predictor of the US than the other stimuli ($V_A > V_X$). On the other hand, if the value of $|\lambda - V_A|$ is greater than or equal to $|\lambda - V_X|$, a_A will decrease as CS_A is a worse or equal predictor respectively, of the US than the other stimuli ($V_A \leq V_X$).

The Mackintosh model is applied to the blocking as follows. Initial CS_A-US pairings will establish CS_A as a good predictor of the US (V_A reaching a value near to λ). During subsequent AB-US trials the added CS_B will therefore be a poorer predictor of the US than CS_A. According to Equations 3 and 4, this will result in an increase of α_A (since $|\lambda - V_A|$ will be less than $|\lambda - V_B|$, as V_A is greater than V_B), and a decline of α_B (since $|\lambda - V_B|$ will be greater than $|\lambda - V|$, as V_B is less than V_A). Consequently, increments in V_B on every Stage 2 trial but the first (since CS_B is a new stimulus on this trial and α_B assumes its initial value) will be smaller than those in a control condition that did not receive the initial training with CS_A.

Pearce and Hall (1980) model

Similarly to the Mackintosh model, the Pearce-Hall (1980) model asserts that the associability of (or the attention paid to) the CS is initially determined by its physical properties but changes later according to its predictive accuracy. However, Pearce and Hall's view of the associability process is in some sense opposite to that taken by Mackintosh. Pearce and Hall suggest that a CS will only be attended to insofar as there is uncertainty about its consequences. The better predictor a given CS is of its consequences (the US on a conditioning trial), the less attention is paid to it. Specifically, Pearce and Hall expressed this notion formally in the following equation:

$$\alpha_A^{\,n} = |\lambda - V_T|^{\,n-1} \qquad (5)$$

where the associability of CS_A in a given trial *n* ($\alpha_A^{\,n}$) is determined by the absolute value of the discrepancy between the intensity of the US (λ) and the sum of the associative strengths of all the stimuli present, including CS_A, (V_T) on the previous trial in which CS_A was presented, *n*-1. The use of this summed error term to determine associability of a CS implies that for compound conditioning the associability of each element will be inversely related to the aggregate predictive accuracy of the compound.

The contribution of the associability of the CS to the increment in its associative strength on a given CS-US pairing is described by Equation 6:

$$\Delta V_A = S_A\, a_A\, \lambda \qquad (6)$$

where S_A and l are constant parameters that depend on CS_A intensity and US intensity respectively; and a_A represents the associability of A which is updated according to the Equation 5. An examination of Equation 6 merits two important comments. First, the influence of the intensity of the CS_A (S_A) on the acquisition of associative strength is explicitly separated from that of the changes in its associability (α_A). And second, the

error term in this equation is included substituting according to Equation 5 α_A for $|\lambda - V_T|$. The inclusion of this summed error term allows this model to account for blocking as follows.

Initial CS_A-US pairings in Stage 1 will establish CS_A as a good predictor of the US and hence α_A will decline (since according to Equation 5, as V_A increases $|\lambda - V_T|$ decreases). During the first AB-US trial in Stage 2 the novelty of added CS_B enables it to acquire a certain amount of associative strength as α_B assumes its initial value. According to Equation 5 the summed error term $|\lambda - V_T|$ determines associability of all the elements of a compound, and therefore as of the first trial α_B will decrease and be equal to a_A (as the value of $|\lambda - V_T|$ is close to zero due to the contribution of V_A to the value of V_T) [1]. As a result, increments in V_B on every trial of Stage 2 but the first (CS_B is a new stimulus on this trial and a_B assumes its initial value) will be smaller than those in a control condition where pre-training with CS_A is not given.

Learning after blocking

So far we have discussed the ability of the three models described above to account for the blocking effect. All three anticipate the smaller increments in V_B during AB-US trials as a result of the previous CS_A-US training. The models do, however, propose different underlying processes for this effect, and these differences become apparent when examining some variations in the blocking procedure. One such instance where these models can be distinguished is when they are applied to learning about the AB compound subsequent to blocking. Consider the training conditions described in Table 1. The question of interest here is what effect the prior blocking training (Stage 1 and Stage 2) has on the subsequent learning about CS_A and CS_B during Stage 3 in which AB compound is paired with an intensified US.

According to the Rescorla-Wagner model, the increase in the intensity of the US in Stage 3 trials will result in an increase in the value of parameter λ. This increase will in turn augment the value of the discrepancy $(\lambda - V_T)$, thereby reflecting the surprise factor of the change in the US and a recovery of its effectiveness. According to Equation 1, the new value of the discrepancy $(\lambda - V_T)$ will enable CS_A and CS_B to acquire the same amount of associative strength during $AB\text{-}US_{strong}$ trials (providing the intensities of CS_A and CS_B are the same, or these stimuli are properly counterbalanced, and hence $\alpha_A = \alpha_B$).

The Mackintosh model offers different predictions. As we saw earlier, on Stage 2 trials α_B will decrease to a near-zero value while α_A will remain high. According to Equation 2, this result in CS_A acquiring a substantial amount of associative strength on

Table 1

Stage 1	Stage 2	Stage 3	Test
A _ US	AB _ US	AB _ US_{strong}	A, B

Note: US= 0.4 mA, 0.2 sec foot-shock; USstrong= 0.8 mA, 0.5 sec foot-shock; A and B= light and tone counterbalanced.

the first Stage 3 AB-US_{strong} trial, but CS_B will be hardly able to acquire any associative strength at all. This will result in CS_A becoming a better predictor of US_{strong} than CS_B. According to Equations 3 and 4, this means that α_A will continue to increase and α_B will continue to decrease (its value dropping closer and closer to zero) over the subsequent trials. Consequently, CS_A will acquire almost all the associative strength generated by US_{strong}, while CS_B will acquire almost none.

We saw above that according to the Pearce-Hall model (Equation 5), the associability of both CS_A and CS_B is determined by the summed error term. Thus, unlike the Mackintosh model, Pearce-Hall anticipates that α_A and α_B will remain equal following Stage 2. The surprising increase in the intensity of the US on the first Stage 3 trial will increase the value of $|\lambda - V_T|$ (since λ is now greater than V_T, which was acquired over the course of prior conditioning with the non-intensified US). According to Equation 3, this will lead to an initial increase in a_A and a_B on the second Stage 3 trial, followed by a decrease during subsequent trials as V_A and V_B rise and then V_T approach λ. According to Equation 6 [2], CS_A and CS_B will then acquire the same amount of associative strength in Stage 3 due to the equal values of α_A and α_B.

To summarize, the Rescorla-Wagner and Pearce-Hall models predict that CS_A and CS_B will acquire the same amount of associative strength in Stage 3. On the other hand, the Mackintosh model predicts that in this stage CS_A will acquire almost all the associative strength while CS_B will acquire almost none. In other words, while the Mackintosh model predicts that previous blocking trials (in Stage 2) will result in learning about pre-training CS_A but will impede learning about the added CS_B in Stage 3, the Rescorla-Wagner and Pearce-Hall models predict similar learning about pre-trained CS_A and added CS_B in this stage.

The study presented here aimed to test these predictions. To this end, a conditioned suppression experiment was conducted employing a within-subjects design according to the treatment described in Table 1. Initially, CS_A was paired with a weak US in Stage 1. A compound formed by the pre-trained CS_A and an added CS_B was paired with the same weak US in Stage 2. Subsequently, the AB compound was paired with a stronger US in Stage 3. Finally, levels of suppression to CS_A and CS_B were tested.

At this point, a comment about the different magnitudes of the US employed is relevant. A 0.4 mA foot-shock for a duration of 0.2 sec, and a 0.8 mA foot-shock for 0.5 sec were employed as the weak US and the strong US, respectively. A very weak US was employed in Stages 1 and 2 in order to make the most precise assessment possible of the predictions offered by the different models regarding the acquisition of associative strength by CS_A and CS_B in Stage 3. According to the Mackintosh model, CS_B will be only able to acquire associative strength on the first compound conditioning trial with the non-intensified US in Stage 2. The amount of associative strength that the CS_B will be able to acquire from its pairing with a very weak US will be very low, and will therefore hardly manifest itself in suppression to the CS_B on the test. Thus, according to the Mackintosh model we should expect to observe a high level of suppression to CS_A and scarce or non existent suppression to CS_B on the test. As with the Mackintosh model, the Rescorla-Wagner and Pearce-Hall models predict that responding to CS_A will be greater than to CS_B on the test. According to these two models, the final

associative strength of CS_A will be greater than that of CS_B, as a result of the contribution of the associative strength that CS_A acquired during Stage 1 and Stage 2. However, the Rescorla-Wagner and Pearce-Hall models predict that CS_B will acquire the same amount of associative strength as CS_A in Stage 3 using the intensified US. Given the relatively high US magnitude employed in this stage, these models predict a substantial increase in the associative strength of CS_B during the trials with-in this stage, and therefore a moderate level of suppression to CS_B on the test trial (which is, critically, the opposite of that predicted by the Mackintosh model).

METHOD

Subjects

The subjects were 20 male Wistar rats with a mean ad lib weight of 420 g (range: 348-452 g). They had served previously in an experiment of flavor aversion conditioning, receiving stimuli and treatment with no apparent relation to those used here. The animals were housed in pairs in cages located in an air-conditioned room maintained at a constant temperature (23º C) and humidity (50%) on a 12 h light/dark cycle with light on at 08:00 AM. The experimental procedures were conducted in a room away from the home room during the light portion of the cycle. Prior to the start of training, the weight of the subjects was gradually reduced to 80% of their ad lib weights. This deprivation schedule was maintained throughout the experiment, with subjects being given a reduced food ration daily at the end of each experiment session. The subjects had free access to water throughout the experiment.

Apparatus

Five Skinner boxes made by Coulbourn Instruments were used. The ceiling and front and rear walls of each box were made from aluminum, whereas the two side walls were made from transparent plastic. The floor of the box was composed of stainless steel rods 6 mm in diameter and spaced 1.5 cm apart center-to-center. The floor could be electrified by an AC shock generator. Each box was equipped with a response lever located on the front wall, 6 cm above the floor. The food tray was 2 cm from the floor in the center of the front wall, situated to the right of the lever, and was connected via a plastic tube to an external 45-mg pellet dispenser. Each box was housed in a sound-attenuated cubicle equipped with a fan that supplied a background noise of 40 dB. Two different stimuli were used as CSs. The first was the illumination supplied by the simultaneous lighting up of three bulbs (28 volt and 0.04 amp), aligned horizontally 11 cm over the response lever. The second CS was a continuous tone of 4.5 kHz and 85 dB, generated by a loud speaker located 6 cm over the bulbs. Both stimuli had a duration of 90 sec. These stimuli were counterbalanced in their role of A and B. For half of the subjects, stimulus CS_A was the tone and stimulus CS_B the light, whereas for the other half, the opposite was true.

Procedure

The design of the experiment is presented in Table 1. All experimental sessions were conducted in darkness and lasted 60 min, except for the three sessions of Stage 3, which lasted 40 min.

Initially, the animals received magazine training sessions. In each, food pellets were delivered on a variable-time (VT) 60-sec schedule while lever press responses were continuously reinforced. Each rat finished magazine training when it made 100 lever press responses. Subjects then received 12 sessions of lever press response training (baseline). The lever press response was reinforced with one food pellet on a variable interval (VI) 30-sec schedule during the first session. In the remaining sessions, reinforcement was delivered according to a VI 60-sec schedule. The following experimental sessions were conducted on the baseline of the lever press response.

Stage 1 consisted of 11 conditioning sessions to CS_A. Each session included 6 trials. In each trial CS_A was followed immediately by an electric shock of 0.4 mA and 0.2 sec. The inter-trial interval (ITI) was variable around a mean duration of 360 sec. Two conditioning sessions to the simultaneous AB compound were conducted during Stage 2. The number of trials, the ITI and the intensity and duration of the electric shock used in this stage were identical to those used in Stage 1.Three further compound conditioning sessions were conducted during Stage 3. In each of these sessions, rats received 2 trials in which the compound AB was followed by an electric shock of 0.8 mA and 0.5 sec. The first trial began 360 sec and the second trial 1560 sec after the start of the session. A non-reinforced trial for each of the stimuli, CS_A and CS_B, were conducted in a final test session. The first trial began 360 sec and the second trial 1560 sec after the start of the session. For half of the animals CS_A was presented on the first trial and CS_B on the second one**,** and the reverse was true for the remaining subjects.

Lever press responses were recorded and the suppression ratios to the CS were calculated, in accordance with the X/(X+Y) formula (Annau and Kamin, 1961). X was

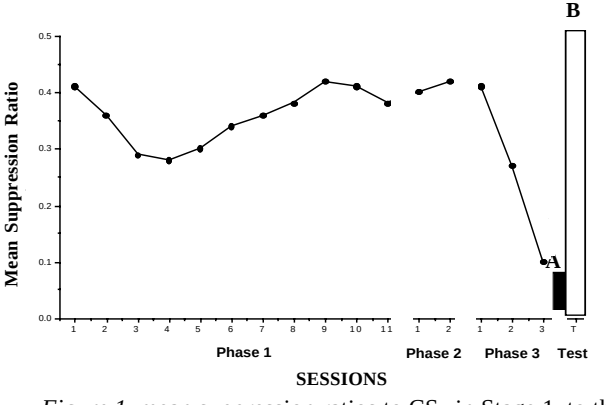

Figure 1. mean suppression ratios to CS_A in Stage 1, to the AB compound in Stage 2 and Stage 3, and to CS_A and CS_B during the final test trials.

the number of lever press responses during CS, and Y the number of lever press responses during a period of equal duration immediately prior to the onset of the CS.

Results

Subjects learned to press the lever systematically. The mean response rate was 15.28 responses per minute during the last session of the baseline training.

Figure 1 shows the mean suppression ratios to CS_A in Stage 1, to the AB compound in Stage 2 and Stage 3, and to CS_A and CS_B during the final test trials. Subjects exhibited weak suppression to CS_A during Stage 1. The moderately weak magnitude of the US employed in this Stage may explain the low level of suppression observed. An analysis of variance (ANOVA) examining the effect of session during this stage showed subjects reliably increased suppression, $F(10, 190) = 5.03$, $p < 0.001$. This result suggests that the weak US produced demonstrable conditioning to CS_A.

Apparently, the suppression level decreased slightly when the AB compound was paired with the same US during Stage 2. However, a repeated measures test comparing the suppression ratios to CS_A during the last session of Stage1 and the suppression ratios to the AB compound during the first session of Stage 2 revealed subjects did not decrease suppression $t(19) = 0.88$, $p = 0.388$. The effect of session in Stage 2 did not have a significant effect, $F(1, 19) = 2.44$, $p = 0.134$.

It seems that the intensification of the shock administered during Stage 3 trials considerably increased the suppression level. An ANOVA examining the effect of session during this stage showed subjects reliably increased suppression to the AB compound, $F(2, 38) = 83.02$, $p < 0.001$.

Finally, subjects showed higher suppression to CS_A than to CS_B, $t(19) = 8.33$, $p < 0.001$. Critically, scarce suppression to CS_B was observed during the final test trials. Although the mean suppression ratio to CS_B was lower than 0.5 (.47), no differences were found between this suppression level and a hypothetical level of 0.5, which seems to indicate an absence of suppression, $t(19) = 0.81$, $p = 0.4253$.

Discussion

Training in which the AB compound was paired with the same US with which CS_A had been previously paired resulted in a high level of suppression to CS_A but prevented the conditioning to the added CS_B during later training in which the AB compound was paired with an intensified US (see for similar results, Mackintosh & Turner, 1971 and Kruschke & Blair, 2000). This finding is in accordance with expectations of the Mackintosh (1975) model but not with those of the Rescorla-Wagner (1972) or Pearce-Hall (1980) models.

As we saw before, the Mackintosh model predicted that CS_A would acquire almost all the available associative strength while CS_B would acquire almost none throughout the training. The high level of suppression to CS_A and the absence of

conditioned suppression to CS_B observed support these predictions. Recall, however, the consideration outlined above regarding the prediction of this model concerning an increase in the associative strength of CS_B on the first AB-US trial in Stage 2 (since CS_B is a new stimulus in this trial and a_B assumes its initial value). The non-existent level of suppression to CS_B observed on the test could seem to cast doubt on this assumption. Bearing in mind the low magnitude of the US used during this stage, it is reasonable, however, to assume that the amount of associative strength acquired by CS_B on this first trial (if any), was negligible. It could therefore be argued that such a low amount of associative strength would not be sufficient to become apparent in a suppression of lever press response on the test trial.

The results of the present experiment are, however, not so easily accommodated by the Rescorla-Wagner and Pearce-Hal models. It was observed that the increase in the magnitude of the shock in Stage 3 caused the final conditioned suppression to the AB compound to be practically total. If, as in the models that we are considering, a monotonic relationship is assumed between the value of the associative strength acquired and the magnitude of the CR, it seems logical to think that if CS_B had really acquired such an amount of associative strength during the course of the trials, this would have manifested itself in at least a moderate level of suppression to the CS_B on the test. Contrary to this prediction the blocking effect observed was apparently total, as suppression to CS_A was high but suppression to CS_B was practically non-existent[3].

A point of particular interest regarding the present findings is the similarity between our experimental situation and experiments in which the Hall-Pearce effect has been observed (Hall & Pearce, 1979; see also, e.g., Ayres, Moore & Vigorito, 1984; Kasprow, Schachtman & Miller, 1985; Schachtman, Channell & Hall, 1987; Swartzentruber & Bouton, 1986). Such experiments consist of two stages. During the first stage the critical experimental group receives pairings of a CS with a moderately weak electric shock. During the second stage, when the same CS is paired with a stronger shock than that administered in Stage 1, the experimental group acquires conditioned suppression more slowly than the control group whose subjects have not previously experience the CS. This result has been interpreted as evidence supporting the idea that consistent CS-US pairings result in a decrease of the associability of the CS. This effect, which to a great extent inspired the Pearce-Hall model, is incompatible with the Mackintosh model which predicts that consistent CS-US pairings will result in an increase of the associability of the CS. On the other hand, the results of the experiment reported here support Mackintosh's view of associability: consistent CS-US pairings in Stage 1 resulted in an increase of the associability of CS_A that allow it to acquire all the associative strength in Stage 3, in spite of CS_B. The existence of evidence supporting each of these opposing views suggests the possibility that the associability of a CS is not determined solely by one factor (see Le Pelley, 2004; Rodríguez, 2003; Rodríguez, Lombas & Alonso, 2002, for some attempts to combine Mackintosh's and Pearce-Hall's views of associability).

Notes

1. Throughout the paper we will discuss the predictions made by the original version of the Pearce-Hall model (1980), in which it is assumed that the value of a is determined only by the result of the conditioning trial immediately preceding the current one. According to this version of the model, a_B will be equal to a_A with its value dropping close to zero during the second trial in Stage 2. Subsequently, a modification was made to this model (Pearce, Kaye and Hall, 1982), in which the introduction of a weighting value – g - enabled the value of a also to be determined by the result of earlier trials.
2. Given that CS_A and CS_B were counterbalanced in this present experiment, S_A and S_B adopt the same constant value which is why this parameter is not taken into consideration in the development of this model's predictions.
3. The modified version of the Pearce-Hall model proposed by Pearce, Kaye and Hall (1982) can assume that a_B drops close to zero and equals a_A more slowly than in the original version of the model. This modified version therefore predicts that CS_B will also acquire a certain amount of associative strength in trials following the first trial of Stage 2 since a_B still has a value greater than zero. Bearing in mind the absence of suppression to CS_B observed during the final test trial of our experiment, this modification does nothing to help the predictions offered by the original model account for our results.

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