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Representation-mediated Inhibitory Learning in the Conditioned-suppression Procedure

Sarah Leonard and Geoffrey Hall
University of York, York, U.K.

In three experiments, rats in group I (for intermixed) were given non-reinforced exposure to two compound stimuli, AX and BX, where A and B represent different auditory cues, and X represents a visual cue. AX and BX were presented in alternation. Group B (blocked) received similar exposure except that subjects experienced a block of AX trials and then a block of BX trials. Subsequent shock reinforcement of A was found to endow B with inhibitory strength in group I, as assessed by retardation (Experiments 1 and 2) and summation tests (Experiment 3). This outcome confirms and extends the results reported by Espinet, Iraola, Bennett, and Mackintosh (1995) and constitutes a further example of mediated learning in which the associative strength of a stimulus is found to be modified as a consequence of training given to some other event with which that stimulus is associated.

There has recently been an upsurge of interest in the proposition (first explored explicitly by Holland, 1981; but see also Konorski, 1967) that the associatively activated representation of a stimulus might be able to enter into associations with other events (for reviews see Hall, 1996; Holland, 1990). A particularly intriguing instance of such representation-mediated learning was demonstrated in series of experiments reported by Espinet, Iraola, Bennett, and Mackintosh (1995). In these experiments rats were given extensive exposure to a pair of compound flavour stimuli (designated AX and BX). The rats were allowed to consume two fluids, both of which were flavoured with saccharin (the common element X), but which differed in that one also contained salt and the other citric acid (the unique elements A and B). Presentations of AX and BX occurred on alternate days over the course of 12 days. The rats were then given aversion conditioning with lithium chloride (LiCl) as the unconditioned stimulus (US) and with Flavour A, presented on its own, as the conditioned stimulus (CS). This treatment appeared to endow the other unique element (Flavour B) with inhibitory properties, as assessed both by retardation and summation tests.

Requests for reprints should be sent to G. Hall, Department of Psychology, University of York, York, YO1 5DD, U.K. E-mail GH1@york.ac.uk

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Espinet et al.'s (1995) interpretation of their result was based partly on standard associative learning principles. According to such principles, pre-exposure to alternating presentations of AX and BX would be likely to establish both excitatory and inhibitory connections among the various elements of these compound stimuli. During the initial trials of pre-exposure it can be expected that excitatory links would form between the elements that co-occur (i.e. between A and X and between B and X). Once established, the presence of these excitatory connections would allow further stimulus presentations to generate inhibitory links between the unique elements (A and B) of the two compounds. Presentation of AX, for example, would activate the representation of B by way of the excitatory X–B link. According to standard theory (e.g. Wagner & Rescorla, 1972) this combination would result in inhibition forming between the stimulus that is present and the representation that is activated only associatively. As the excitatory X–B association will be maintained on BX trials, it can be predicted that inhibitory power will accrue chiefly to Stimulus A. Similarly, on BX trials, B will acquire an inhibitory connection with A. In short, mutually inhibitory links will be formed between the unique elements of each compound (i.e. between A and B), as A occurs only on trials when B does not, and vice versa (see McLaren, Kaye, & Mackintosh, 1989).

Further assumptions are required, however, to explain why the existence of these links should allow reinforcement of A to establish B as an inhibitor for the US. Espinet et al. (1995) suggested the following. Just as the presentation of a given stimulus may be assumed to produce a state of positive activation in its central representation, so the inhibition of a representation may be assumed to produce a state of negative activation (see, e.g. McClelland & Rumelhart, 1985). In these experiments, therefore, the inhibitory links established during the first phase of training will mean that stimulus A, when it is presented on the conditioning trial, will be able to produce negative activation of the B representation. Espinet et al. further proposed a new rule describing the conditions in which associations are formed. They suggested that the co-occurrence of positive and negative states of activation will cause inhibition to develop between the representations so activated. On the conditioning trial of their experiments the representation of B will be in a state of negative activation at a time when reinforcement of A occurs—that is, when the US representation is positively activated. Stimulus B should thus acquire the power to act as an inhibitor for that US.

This interpretation has at least two important implications. First, although the notion of negative activation and the learning rule espoused by Espinet et al. (1995) are widely accepted in some versions of connectionist theorizing, they are novelties as far as associative theories of animal learning are concerned. To adopt them in this context could have far-reaching implications for standard associative theory, in particular for our analysis of mechanisms of inhibitory conditioning. Second, the account offered by Espinet et al. (1995) requires us to accept that pre-exposure to intermixed presentations of AX and BX will produce inhibitory connections between A and B. This is of interest because the formation of such associations, previously postulated but not demonstrated for stimuli of this type, forms the basis of an account developed by McLaren et al. (1989) for certain instances of perceptual learning.

The perceptual learning effect of interest here is demonstrated by the observation that pre-exposure to flavours AX and BX, presented on alternate trials, reduces the extent to

which an aversion subsequently conditioned to AX will generalize to BX (e.g. Mackintosh, Kaye, & Bennett, 1991; Symonds & Hall, 1995). McLaren et al. (1989) point out that, for control subjects not given such pre-exposure, an excitatory association would be likely to form between the constituents of the trained flavour (i.e. X–A) during the conditioning phase of this procedure. On the generalization test, BX would be able to activate a representation of the conditioned flavour A, which would then contribute to evoking the conditioned response (CR). Pre-exposure that was effective in establishing inhibitory associations between A and B would eliminate this effect—activation of the representation of A would be inhibited by the presence of B, and this source of test responding would be absent.

Results consistent with this interpretation come from experiments by Symonds and Hall (1995; see also Honey & Bateson, 1996; Honey, Bateson, & Horn, 1994) showing that the effect of pre-exposure depends on the way in which stimulus presentations are scheduled—generalization from AX to BX is reduced only when the stimuli are experienced on alternate trials during pre-exposure (a procedure likely to generate inhibition between A and B); generalization remains substantial after pre-exposure consisting of a block of AX trials and a separate block of BX trials. Blocked pre-exposure is unlikely to result in the formation of inhibitory links between the unique features of the stimuli. According to standard associative theory (e.g. Wagner & Rescorla, 1972), the ability of an AX trial to generate such a link will depend on there being a pre-established excitatory X–B link; similarly, a BX trial will generate inhibition only when there is an effective excitatory X–A association. Thus there can be no inhibitory learning on the first block of (AX) trials as no X–B association will yet have been formed; and on the second block of (BX) trials the excitatory X–A association established during the first block can be expected to extinguish, so that the opportunity for inhibitory links to be formed will soon be minimal. But this interpretation of the differing effects of blocked and intermixed pre-exposure is not uncontested (see Symonds & Hall, 1995); hence the importance of the results reported by Espinet et al. (1995) in supplying a further line of evidence that alternating AX/BX pre-exposure does indeed result in the development of inhibition between A and B.

Given the theoretical importance of the effect demonstrated by Espinet et al. (1995) we undertook, in the experiments reported here, to attempt to replicate the basic phenomenon—to demonstrate the development of inhibitory power by B after reinforcement of A in animals given alternating presentations of AX and BX during pre-exposure. In order to extend the generality of the effect we employed the conditioned-suppression procedure rather than flavour-aversion learning and made use of a control procedure different from the procedures used by Espinet et al. Our control subjects received pre-exposure consisting of a block of AX trials followed by a separate block of BX trials. As has just been noted, this comparison (between the intermixed and blocked schedules of pre-exposure) generates a clear example of the perceptual learning effect in experiments using the flavour-aversion procedure. It should thus, according to the analysis presented by McLaren et al. (1989), be capable of generating inhibition between A and B in the intermixed condition, and if this is critical in generating the effect reported by Espinet et al., it should allow reinforcement of A to render B inhibitory.

EXPERIMENT 1

In Experiment 1, rats in the critical experimental condition (group I, i.e. intermixed) first received a phase of training consisting of intermixed, or alternating, presentations of the two compound stimuli AX and BX, where A and B designate auditory cues (click and noise), and X designates a visual cue (light offset). In a second phase, Stimulus A was presented alone, followed by a shock US. Finally, the acquisition of inhibitory properties by B was assessed in a retardation test comprising B–US pairings. Control subjects (group B, i.e. blocked) differed only in the way in which stimulus presentations were scheduled during the pre-exposure phase. They received a block of consecutive presentations of one of the stimulus compounds (AX) for half of the pre-exposure trials, followed by a block of presentations of the other compound (BX) for the rest of pre-exposure. In the experiment by Espinet et al. (1995), on which this experiment was modelled, the control subjects received presentations of A and B alone during pre-exposure. An advantage of the control procedure used here is that it equates the two groups in their experience of all elements of the stimuli (all animals receive presentations of both AX and BX) while still allowing the possibility that inhibitory links between A and B will form in one case but not in the other.

Method

Subjects

The subjects were 16 experimentally naive male hooded Lister rats with a mean ad lib weight of 485 g (range: 455–535 g). The animals were maintained at 80% of their free-feeding weights by daily weighing and restricted feeding. They were housed in pairs in a colony room lit from 8:00 a.m. to 8:00 p.m.

Apparatus

Four identical Skinner boxes, supplied by Campden Instruments Ltd., were used. Each of the boxes contained a recessed food tray to which 45-mg food pellets could be delivered. Access to this food tray was by means of a rectangular aperture 6 cm high and 5 cm wide. A transparent plastic flap of the same dimensions was attached by a hinge to the top of the entrance to the food tray. Pushing this flap inward from its vertical resting position allowed subjects to gain entry to the food tray. This movement activated a microswitch, and each closing of this switch was recorded as a single response. The retractable levers with which the box was fitted were withdrawn throughout the course of the experiment. A loudspeaker mounted in the ceiling of the box was used to present the subjects with a white noise at an intensity of 70 dB, and a 30-Hz clicker at 80 dB. A ventilation fan provided a constant background noise of 65 dB. Background illumination was provided by a 3-W jewel light (rated for 24 V but operated at 16 V) mounted 10 cm above the food tray. This light was turned off to create the dark element of the compound stimuli. There were two jewel lights situated 5 cm below and 5 cm either side of this central light; these were not used in the present experiment (but see Experiment 3). The floor was constructed of stainless steel rods, which could be electrified by a Campden Instruments Ltd. shock generator (Model 521C) and shock scrambler (Model 521S). The boxes were housed in sound- and light-attenuating shells which contained a ventilation fan and were remotely controlled by a BBC microcomputer.

Procedure

Pretraining. On the first two days of the experiment the rats received 40-min sessions of magazine training in which 45-mg food pellets were delivered, on a 30-sec variable-time schedule on Day 1 and on a 60-sec variable-time schedule on Day 2. On Day 3 the rats were given a session of continuous reinforcement training, in which each response to the magazine flap was rewarded by the delivery of a food pellet. After 25 reinforcers had been earned the animal was removed from the box. All subsequent sessions were 40 min long. In the next session pushing the flap was reinforced on a variable-interval 30-sec (V1-30) schedule, and on the final two days of pretraining it was reinforced on a variable-interval 60-sec (V1-60) schedule. The V1-60 schedule remained in force throughout the rest of the experiment.

Pre-exposure. The subjects were randomly assigned to one of two equal-sized groups (group I or group B) for the eight daily sessions of this phase. In each session subjects in group I received four presentations of AX and four of BX, presented in an alternating sequence. Subjects in group B received eight presentations of AX in each of the first four sessions and eight presentations of BX in each of the last four sessions. For all subjects dark served as stimulus X. For half the animals in each group A was the noise and B was the clicker; for the remaining subjects this arrangement was reversed. These elements were presented as simultaneous compounds. Each stimulus presentation was 60 sec long and the interval between trials (ITI) was 220 sec.

Conditioning. On the next day all subjects received conditioning with Stimulus A. Following Espinet et al.'s (1995) procedure, just a single reinforced trial was given. Stimulus A was presented halfway through the 40-min session and was followed immediately by a 0.5-sec, 0.5-mA shock. This trial produced a loss of baseline responding in some animals. Accordingly, all subjects received a recovery session about 4 hr later during which no stimuli were programmed to occur, but responding continued to be reinforced on the V1-60 schedule. Similar recovery sessions were given after each of the conditioning sessions of the next phase.

Retardation Test. In each of the next four sessions all subjects received two presentations of B followed immediately by a 0.2-sec, 0.1-mA shock over the course of the 40-min session. (Espinet et al., 1995, similarly reduced the magnitude of the reinforcer for the retardation tests of their experiments.) B was presented for 60 sec, and the ITI was 780 sec. There followed two further sessions of testing in extinction, identical to the conditioning sessions except for the omission of the shock. Response rates were recorded separately during CS presentations and during the 60-sec stimulus-free (pre-CS) period that preceded each trial. Suppression during B was assessed by means of a ratio of the form $a/(a+b)$ where a is the number of responses made during the CS, and b is the number made during the 60-sec pre-CS period.

Results and Discussion

No formal data were collected during the initial phases of training, but the baseline response established during pretraining appeared to be well maintained until the occurrence of the shock on the reinforced trial with Stimulus A. At this point responding was markedly suppressed in several subjects. Responding was re-established during the recovery session, however, and the mean rates during the conditioning sessions with Stimulus B (computed by pooling the scores for all pre-CS periods during this phase of training) were 11.06 responses per min (rpm) for group I and 8.35 rpm for group B. These scores

did not differ significantly, $F(1, 14) = 1.38$. (A rejection criterion of $p < .05$ was adopted for this and all subsequent statistical tests.)

Acquisition of suppression to Stimulus B is shown separately for the two groups on the left of Figure 1. For each subject the responses emitted during both trials on each day were pooled before the suppression ratio for that day was calculated. It is evident that both groups acquired suppression over the course of conditioning and did so at much the same rate. An analysis of variance conducted on the data summarized in Figure 1, with trial block and group as the variables, revealed only a significant effect of block, $F(3, 42) = 14.68$; the effect of group and the interaction between the variables were both non-significant, (Fs < 1). By this measure there was thus no indication that the treatment given to group I had been effective in making B an inhibitor for the US. However, the results of the extinction test with B (shown on the right of Figure 1) tell a different story. Group B showed enhanced suppression on the first block of this phase (presumably as a result of the reinforced trials experienced on the previous block), but group I did not. Both groups showed some loss of suppression by the second block of extinction, but the difference between the groups was maintained. An analysis of variance was performed on the extinction test data with group and trial block as the variables. This analysis showed there to be a significant main effect of group, $F(1, 14) = 5.13$, and of block, $F(1, 14) = 8.51$, and no significant interaction between the variables, ($F < 1$). The groups did not differ in their baseline response rates during this test. The rates (computed by pooling all the pre-CS periods of this phase) were 9.35 rpm for group I and 8.95 rpm for group B, ($F < 1$).

The proposal that B would be rendered inhibitory by reinforcement of A in animals given intermixed presentations of AX and BX during pre-exposure gains limited support from these results. Excitatory conditioning to Stimulus B in group I showed no retardation during the reinforced trials of the test phase, but the lesser degree of suppression shown in the final extinction test seems to indicate that the amount of

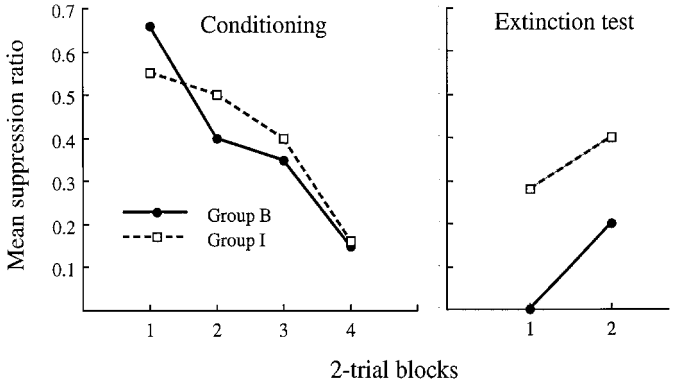

FIG. 1. Experiment 1: Group mean suppression ratios for conditioning trials in which Stimulus B was paired with shock and for the test in which Stimulus B was presented in extinction. Group I had received pre-exposure to alternating presentations of the stimulus compounds AX and BX; group B had received a block of AX trials followed by a block of BX trials during pre-exposure. Both groups received a reinforced trial with Stimulus A prior to conditioning with Stimulus B.

associative strength acquired in this group by the end of conditioning was rather less than that acquired by group B. To this extent, the effect demonstrated by Espinet et al. (1995) in flavour-aversion learning can be obtained in this very different training procedure. Encouraged by this partial success we conducted a further experiment, adding a necessary control condition and refining our procedure in the hope of generating a more robust effect.

EXPERIMENT 2

Two of the groups in this experiment (groups I–A+ and B–A+) matched those of the previous experiment—that is, after intermixed or blocked pre-exposure to AX and BX they received conditioning with Stimulus A(A+) followed by a retardation test with B. They differed only in that pre-exposure was extended from 8 to 12 sessions. If the difference between the groups observed in the test phase of Experiment 1 depends on the establishment of inhibitory links between A and B during pre-exposure, then increasing the opportunity for such links to be formed might be expected to enhance the magnitude of the effect. However, to obtain a difference between these two groups in their performance to Stimulus B would not establish that the effect depends on a learning process engaged during conditioning with A (as the interpretation proposed by Espinet et al., 1995, supposes). It could be that intermixed pre-exposure to AX and BX is, for some reason, in itself sufficient to retard subsequent excitatory conditioning to B. Accordingly, a third group of subjects (group I–A/+) was included. These animals received intermixed pre-exposure to AX and BX as did group I–A+, but this was followed by a session in which A and the shock were presented unpaired (A/+). Thus this group had the opportunity to form A–B inhibitory links but did not receive excitatory conditioning with A prior to the test with B. According to the hypothesis of Espinet et al., B will not be established as an inhibitor for shock in these circumstances.

This control condition addresses a further issue. The results from Experiment 1 indicated that excitatory conditioning to B occurred less readily in group I than in group B. This outcome is consistent with the suggestion that B had acquired inhibitory properties in group I, but there are other possibilities. In particular, there might be generalization to B of excitation acquired by A as a consequence of the reinforced trial with that stimulus. If this generalization occurs less readily in subjects given intermixed pre-exposure than in subjects given blocked pre-exposure, then conditioning to B would appear to be retarded in the former group. The treatment given to group I–A+, on the other hand, provides a suitable test. These subjects received intermixed pre-exposure but no excitatory conditioning to A. There is thus no possibility of generalization of excitation from A to B; if the effect seen in Experiment 1 reflects a lack of generalization in group I then the acquisition of suppression to B in subjects in group I–A/+ should be comparable to that shown by subjects in group I–A+. On the other hand, if the treatment given to group I–A+ renders B genuinely inhibitory, then this group might be expected to show slower acquisition to B than to either of the other groups.

Method

The subjects were 24 male hooded Lister rats with a free-feeding weight of 415 g (range: 370–460 g). They had previously been used in a conditioned flavour-aversion experiment, but they were naive to the procedure and stimuli used in the present experiment. The apparatus was that used in Experiment 1.

After the baseline response had been established during pretraining, the subjects were randomly assigned to one of three groups. Two of these groups (groups I–A+ and I–A/ +) were given intermixed pre-exposure to AX and BX, the procedures used being the same as those described for Experiment 1, except that 12 rather than 8 pre-exposure sessions were given. Group B–A+ received six sessions of exposure to AX followed by six sessions of exposure to BX. On the next session groups I–A+ and B–A+ received a single shock-reinforced trial with stimulus A as the CS. Animals in Group I–A/ + received a presentation of A after 19 min of the 40-min session and an unsignalled presentation of the shock 12 min after the termination of A. On the next day all animals received a baseline recovery session in which responding was reinforced according to the VI-60 schedule. Similar recovery sessions followed each of the shock-reinforced sessions of the retardation test phase. The test phase consisted of four conditioning sessions, each containing two reinforced B trials followed by two further sessions in which B was presented in the absence of shock. In all other respects the procedure was identical to that described for Experiment 1.

Results and Discussion

No data were collected during the pre-exposure and conditioning phases of the experiment. The results from the reinforced trials of the retardation test with Stimulus B are shown in the left-hand panel of Figure 2. All three groups acquired suppression during these trials but, as in Experiment 1, there were no obvious differences among the groups in the rate at which this occurred. An analysis of variance conducted on the data summarized in Figure 2 revealed only a significant effect of trial block, $F(3, 63) = 55.43$; the main effect of group and the interaction between group and block were both

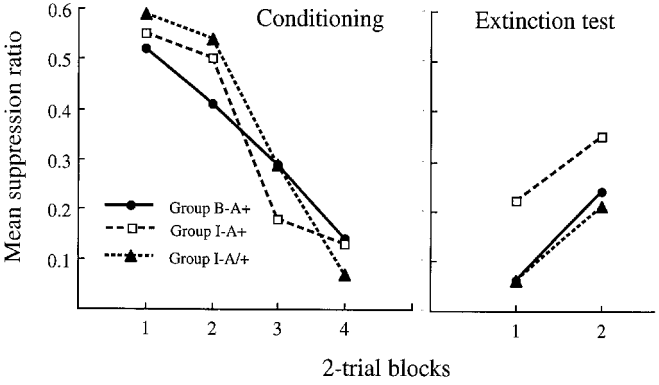

FIG. 2. Experiment 2: Group mean suppression ratios for conditioning trials in which Stimulus B was paired with shock and for the test in which Stimulus B was presented in extinction. The I groups had received pre-exposure to alternating presentations of the stimulus compounds AX and BX; group B had received a block of AX trials followed by a block of BX trials during pre-exposure. The A+ groups received a reinforced trial with Stimulus A prior to conditioning with B; the A/ + group experienced Stimulus A and the reinforcer unpaired.

non-significant, (Fs < 1). The groups had similar pre-CS response rates during this phase of training: 12.25 rpm for group B–A+, 11.25 rpm for group I–A+, and 15.49 rpm for group I–A/+ (F < 1).

Again as in Experiment 1, group differences became evident during the extinction test trials. As the right-hand panel of Figure 2 shows, group I–A+ showed less suppression than did the other two groups, which did not differ from each other. An analysis of variance showed there to be a significant effect of group, $F(2, 21) = 4.29$, a significant effect of block, $F(1, 21) = 33.85$, and no interaction between these two variables. Pairwise comparisons among the groups using Duncan's test showed that group I–A/+ differed significantly from each of the other two groups, which did not themselves differ. Pre-CS response rates during this test were 14.25 rpm for group I–A+, 17.95 rpm for Group A–I/+, and 12.25 for group B–A+. These rates did not differ significantly, (F < 1).

The results for groups I–A+ and B–A+ exactly match those for the equivalent groups of Experiment 1. Although no difference was apparent during the reinforced trials of the test, group I–A+ showed less suppression during the extinction test. This outcome suggests that reinforcement was relatively ineffective in endowing Stimulus B with excitatory associative strength in group I–A+ and is thus consistent with the proposition that for this group, Stimulus B possessed inhibitory strength at the start of the test phase. The test results for group I–A/+ support this interpretation. The difference between groups I–A+ and B–A+ in their test performance could conceivably be a consequence of a difference in the extent to which excitation acquired by Stimulus A during conditioning generalized to Stimulus B. But group I–A+ also differed from group A–I/+—a group that received no excitatory conditioning to Stimulus A. That group A–I/+ did not differ on test from group B–A+ indicates that direct generalization from the A+ trials contributed little to the suppression governed by Stimulus B on the test. The performance shown by group I–A+ is thus best interpreted as supporting the view that A+ conditioning after intermixed pre-exposure to AX and BX will render Stimulus B inhibitory.

EXPERIMENT 3

In Experiments 1 and 2, the properties acquired by Stimulus B were assessed by means of a retardation test. It has become customary, however, to employ both retardation and summation tests when evaluating the inhibitory properties of a stimulus. Accordingly in Experiment 3 rats (groups I and B) were given either intermixed and blocked pre-exposure, (the procedures used being identical to those described for Experiment 2). After reinforcement of A, the inhibitory properties of B were assessed in a summation test. This involved shock-reinforced presentations of a novel stimulus, Stimulus C (a flashing jewel light) followed by a test comparing the suppression controlled by Stimulus C alone and the compound stimulus, Stimulus BC. If the effects demonstrated in the retardation tests of Experiments 1 and 2 indeed reflect the fact that Stimulus B is inhibitory in group I but not in group B, we can expect to find in Experiment 3 that Stimulus B will be more effective at alleviating the suppression evoked by the test excitor, Stimulus C, in group I than in group B.

Method

The subjects were 16 experimentally naive male hooded Lister rats with a mean free-feeding weight of 355 g (range: 335–380 g). They were maintained in the manner described for the subjects of Experiment 1. The apparatus was that used in the previous experiments.

The initial phases of training were identical to those described for Experiment 2. Briefly, after the baseline response had been established, the rats were randomly assigned to one of two equal-sized groups (group I or group B) for the 12 daily sessions of pre-exposure. In each session subjects in group I received four presentations of AX and four of BX in an alternating sequence. Subjects in group B received eight presentations of AX in each of the first six sessions and eight presentations of BX in each of the last six sessions. All then received a single conditioning trial with Stimulus A. As this trial produced a loss of baseline responding in several subjects, all animals received a recovery session the next day in which neither stimuli nor shocks were presented but during which responding continued to be reinforced on the VI-60 schedule. Similar recovery sessions followed all subsequent conditioning sessions.

The test excitor, Stimulus C, was provided by the two jewel lights situated on either side of the food tray. These were operated at 24 V and were turned on and off at 0.5-sec intervals to create a flashing stimulus. In the next session all subjects received a single 60-sec presentation of Stimulus C, designed to attenuate any unconditioned suppression produced by this novel and salient stimulus. Stimulus C was presented for 60 sec exactly half-way through the session. In each of the next three sessions Stimulus C was again presented once but was followed immediately by presentation of a 0.5-sec, 0.5-mA shock.

The next two sessions constituted the summation test. In each session the animals received two non-reinforced presentations of Stimulus C and two of the compound stimulus, Stimulus BC. The order of presentation of the stimuli was counterbalanced so that in the first session half the subjects in each group received BC, C, C, BC and the other half C, BC, BC, C. On the second test day the order was reversed. The stimuli were presented for 60 sec and the ITI was 384 sec. Response rates were recorded separately during the CS presentations and during the 60-sec pre-CS period.

Results and Discussion

Informal examination of the data collected during the initial phases of training revealed that all the subjects responded readily throughout the pre-exposure sessions and that the recovery session successfully restored the loss of baseline responding that occurred at the introduction of shock-reinforced trials with A and C. By the final trial of the conditioning with Stimulus C, all subjects were showing complete suppression in the presence of this stimulus; both groups had a mean suppression ratio of 0.

Baseline responding was well maintained during the summation test, and there were no differences between the groups. The mean response rates for the pre-CS periods prior to BC trials were 23.25 rpm for group I and 16.90 rpm for group B, $F(1, 14) = 1.82$. The pre-CS rates prior to C trials were 22.90 rpm for group I and 17.20 rpm for group B, $F(1, 14) = 1.11$.

Figure 3 shows the group mean suppression ratios governed by stimuli C and BC on both days of the summation test. For each subject the responses emitted during both trials of a given type were pooled before the suppression ratio for that day was calculated. It is evident that on the first trial block both groups showed substantial suppression to both C and the BC compound. On the second block, however, BC controlled less suppression

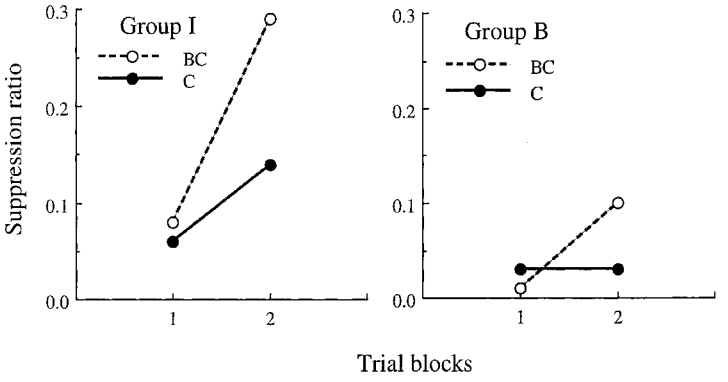

FIG. 3. Experiment 3: Group mean suppression ratios to stimuli C and BC in the summation test. Group I received intermixed pre-exposure to stimulus compounds AX and BX, and group B received a block of AX trials followed by a block of BX trials before all subjects were given a single shock-reinforced presentation of Stimulus A. Both groups were then conditioned to Stimulus C before beginning the summation test.

than did C—an effect that was particularly marked in group I. Statistical analysis largely confirmed these impressions. An analysis of variance was performed on the data summarized in Figure 3, with group as a between-subject variable and trial and stimulus as within-subject variables. The analysis showed that there were significant main effects of group, $F(1, 14) = 5.29$, stimulus, $F(1, 14) = 17.09$, and trial, $F(1, 14) = 17.49$, and significant interactions between group and stimulus, $F(1, 14) = 5.37$, group and trial, $F(1, 14) = 5.45$, and stimulus and trial, $F(1, 14) = 5.83$. There was no significant three-way interaction, $F < 1$. Further analyses were conducted to identify the source of the theoretically critical interaction between group and stimulus. Analysis of simple main effects showed that there was a significant difference between the groups with respect to stimulus BC, $F(1, 14) = 8.21$, but not Stimulus C, $F(1, 14) = 2.38$; furthermore, there was a significant effect of stimulus in group I, $F(1, 14) = 20.81$, but not in group B, $F(1, 14) = 1.65$. In summary, the groups were equivalently suppressed to Stimulus C; presenting B in compound with C alleviated this suppression in group I but not in group B. We conclude that by this measure, Stimulus B had acquired inhibitory properties in group I, thus confirming the conclusion based on the retardation tests of Experiments 1 and 2.

GENERAL DISCUSSION

The experiments reported here have replicated, for the conditioned suppression procedure, an effect previously demonstrated by Espinet et al. (1995) in a series of experiments using the flavour-aversion procedure. The present experiments showed that for animals given pre-exposure to the compound stimuli AX and BX presented in alternation, shock reinforcement of A endowed B with the properties of an inhibitor for shock, as assessed both by retardation tests (Experiments 1 and 2) and a summation test (Experiment 3). Experiment 2 allowed comparison with a control procedure in which the animals received intermixed pre-exposure to AX and BX but no conditioning with A. This comparison confirmed that reinforcement of A is necessary for the acquisition of inhibition by B. All

three experiments allowed comparison with a control condition in which the subjects received, during pre-exposure, a block of AX trials followed by a block of BX trials prior to conditioning with A. Stimulus B acquired no (or less) inhibition in this blocked control condition. The results are thus consistent with the suggestion made by Espinet et al. that the acquisition of inhibition by B is a consequence of the development of inhibitory links between A and B during pre-exposure—such links might be expected to develop in the intermixed but not in the blocked condition.

Although our results are consistent with the account proposed by Espinet et al. (1995) they do not compel its acceptance. Espinet et al.'s account depends on the proposition that the co-occurrence of an inhibited stimulus representation and a US will make that stimulus an inhibitor for the US. But a rather different principle for representation-mediated learning proposed by Dickinson and Burke (1996; see also Van Hamme & Wasserman, 1994) is also capable of providing an explanation for our findings. Specifically, Dickinson and Burke have suggested that the presentation of a US along with the *excitatory* associative activation of a CS representation will produce inhibitory learning, with the latter acquiring the power to inhibit the former. As we have already noted, the intermixed training procedure is likely to establish excitatory links between the components of the pre-exposed compounds A–X and B–X. (It may also establish inhibitory links between A and B, but these have no relevance for the present analysis.) The existence of the A–X link will mean that the representation of X will be activated on the trial on which A is reinforced and thus, according to the Dickinson and Burke hypothesis, X will acquire inhibitory strength. Because of the B–X link established in initial training, the representation of X will be activated on test trials with B, and X's inhibitory properties will play a part in determining the performance observed on the test.

There is nothing in the data presented here to allow a choice between these alternative interpretations. It may be pointed out, however, that there is no independent evidence (that is, no evidence apart from that provided by the effect demonstrated in experiments of the sort reported here) to support the validity of the learning rule proposed by Espinet et al. (1995), whereas Dickinson and Burke (1996) provide new experimental results that accord with their analysis. We should also acknowledge, however, that these new results come from a procedure (contingency judgement by human subjects) quite different from that used in the present experiments (or in the experiments by Espinet et al.) and that the learning rule proposed by Dickinson and Burke is, as yet, poorly supported by evidence from experiments using standard animal-conditioning procedures. Indeed, there are experimental results from studies using such procedures (e.g. Hall, 1996; Holland, 1981; Honey & Hall, 1991; Ward-Robinson & Hall, 1996) to suggest that, far from producing inhibitory learning, the associative activation of a stimulus representation in the presence of a US can result in excitatory conditioning. The issue thus remains unresolved for the time being.

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Apprentissage inhibitoire par voie de représentation dans une procédure de suppression conditionneé

Dans trois expériences des rats dans le groupe I (pour <<intermixed>>) furent présentés des stimuli composés AX et BX sans renforcement, où A et B représentaient des signaux auditifs différents et X un signal visuel. AX et BX furent présentés en alternation. Les rats dans le group B (pour <<blocked>>) furent présentés une bloc d'essais AX et par la suite un bloque d'essais BX; ni AX ni BX n'était renforcé. Par la suite, un renforcement de A avec choque a produit une force inhibitoire chez B dans le group I mesurée par des tests de retardation (expériences 1 et 2) et de sommation (expérience 3). Ce resultat confirme et approfondit les résultats de Espinet et al. (1995) et offre un autre example d'apprentissage indirecte dans lequel la force associative d'un stimulus est modifiée en fonction d'un apprentissage avec un autre stumulus avec lequel il est associé.

Aprendizaje inhibitorio mediado por representacíon en el procedimiento de supresíon condicionada

En tres experimentos, a las ratas del grupo I (alternada) se les dio una exposición no reforzada a dos estímulos compuestos, AX y BX, en los que A y B representan claves auditivas diferentes y X una clave visual. AX y BX se presentaron alternadamente. El grupo B (en bloques) recibió una exposición similar excepto en que primero experimentaron un bloque de ensayos AX y luego un bloque de ensayos BX. Se encontró que el reforzamiento posterior de A con una descarga dotó a B de fuerza inhibitoria en el grupo I, evaluándose con pruebas de retraso (experimentos 1 y 2) y sumación (experimento 3). Este resultado confirma y amplia los resultados de Espinet y cols. (1995) y constituye un ejemplo más del aprendizaje mediado en el que la fuerza asociativa de un estímulo se modifica como consecuencia del entrenamiento que se le da a otro evento con el que este estímulo está asociado.