

Protection from extinction by concurrent presentation of an excitor or an extensively extinguished CS

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One conditioned taste aversion experiment with rats assessed the impact of extinguishing a target conditioned stimulus (CS), S, in compound with a second CS, A, upon conditioned responding elicited by CS S when presented alone at test. Following initial conditioning treatment with CSs A and S, the experiment manipulated number of extinction trials with CS A alone (i.e., 0, 5, or 10 trials) prior to AS compound treatment. In addition, two control groups received either extinction trials with S alone or no extinction treatment with S. Conditions receiving either 0 or 10 extinction trials with CS A prior to nonreinforced exposures to AS showed results indicating that aversion elicited by CS S was protected from extinction, whereas a condition receiving 5 extinction trials with CS A prior to AS trials showed unprotected extinction of aversion elicited by CS S. Current associative models are challenged in accounting for this pattern of results.

Pairing a conditioned stimulus (CS, e.g., tone or light) with an unconditioned stimulus (US, e.g., food or footshock) results in the development and strengthening of a conditioned response (CR, e.g., salivation or fear) to the CS, in anticipation to the occurrence of the US. Since Pavlov's (1927) early studies, it is well known that the CR produced by a CS can be reduced in several ways, one of them consisting of merely presenting the CS repeatedly in the absence of the US – an effect typically referred to as extinction. The study of extinction is one of the most fertile areas of research in animal conditioning, due to its great theoretical and clinical implications (for a recent review, see Bouton, 2002). An issue of relevance in the study of extinction regards the interactions among CSs with different prior associative histories that arise when they are presented in

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compound during nonreinforced exposures. Several studies have shown that extinction of the CR elicited by a target CS (X) can be either enhanced or prevented when this CS receives extinction treatment in compound with a second CS (A), depending on the associative status of A. Specifically, when CS A only received pairings with the US (i.e., A→US trials) prior to nonreinforced AX trials (i.e., when A acquired an excitatory status prior to extinction treatment), extinction of the CR elicited by X can be enhanced (Rescorla, 2000; for related results, see Rescorla, 2006). Conversely, when CS A is trained as a Pavlovian conditioned inhibitor (i.e., B→US trials interspersed with BA trials) prior to AX trials (i.e., when A acquired an inhibitory status prior to extinction treatment), CS X can be protected from extinction (e.g., Chorazyna, 1962; Lovibond, Davis, & O'Flaherty, 2000; Rescorla, 2003; Soltysik, Wolfe, Nicholas, Wilson, & Garcia-Sanchez, 1983).

Enhancement of extinction and protection from extinction can be viewed as symmetrical to CS interactions that arise from presenting two CSs (A and X) in a reinforced compound (AX→US trials). In this case, when CS A was previously trained as an excitor, AX→US trials result in CS X eliciting a weak CR (i.e., blocking, Kamin, 1968, 1969). Conversely, when CS A received prior inhibitory training, AX→US trials result in CS X eliciting an extraordinarily strong CR (i.e., superconditioning, Rescorla, 1971; Taukulis & Revusky, 1975; Wagner, 1971). That is, the addition of an excitor can enhance extinction of the CR elicited by the target CS, whereas it can attenuate the acquisition of the CR by the target CS. Conversely, the addition of an inhibitor can protect the target CS from extinction of the CR, whereas it can enhance the acquisition of the CR by the target CS.

Beyond this interesting symmetry, the study of CS interactions during an extinction treatment provides a useful framework for ascertaining the processes involved in extinction. This approach was followed by Calton, Mitchell, and Schachtman (1996) in a series of experiments devoted to ascertaining whether extinction of a previously conditioned taste aversion can endow an extinguished CS with the same properties of a conditioned inhibitor. Calton et al. (see also Denniston & Miller, 2003; but see Brooks, Bowker, Anderson, & Palmatier, 2003) showed that, after extensive extinction, a CS can pass both retardation and summation tests (i.e., the tests commonly accepted for the assessment of Pavlovian conditioned inhibition, see Rescorla, 1969). More importantly for our purposes, Calton et al.'s Experiment 4 attempted to assess whether an extinguished CS behaved as a net inhibitor, by studying its ability to interfere with extinction of a second CS. The rationale of Calton et al. was that, if an extinguished CS behaved as a Pavlovian conditioned inhibitor, it should also be able to

protect a second CS from extinction. Their results supported this prediction, and extinction of the target CS was weaker in a group given extinction treatment in compound with a previously extinguished CS than in a group given extinction treatment with the target CS alone.

A recent study from our laboratory (Pineño, Zilski, & Schachtman, 2007) found results that appear to contradict Calton et al.'s (1996) finding of protection from extinction by an extinguished CS. In Pineño et al.'s experiments, extinction of a conditioned aversion to a target CS (S) was attenuated by a second, nontarget CS (A) that only received prior conditioning treatment. That is, contrary to Rescorla's (2000; also see Rescorla, 2006) finding of enhanced extinction, Pineño et al. observed that the joint nonreinforced presentation of two excitors protected the target CS from extinction (also see Lovibond et al., 2000; Pearce & Wilson, 1991). Moreover, Pineño et al. found that extinguishing the aversion elicited by CS A prior to (but not after) extinction treatment with the AS compound allowed the aversive CR elicited by S to extinguish more strongly. These results were interpreted by Pineño et al. as due to a putative second-order conditioning process taking place during compound treatment. That is, according to these authors presenting two excitors in a nonreinforced compound might have resulted in extinction of the CR elicited by S being counteracted by the strengthening of the association between S and the CR, which was elicited by the presence of A. In accordance with this interpretation, extinguishing the CR of A prior to AS presentations prevented S from strengthening its association with the CR elicited by A (i.e., the putative second-order conditioning process would be, if anything, weak) which, in turn, resulted in better extinction of the CR elicited by S.

A possible reconciliation between the results of Calton et al. (1996) and those of Pineño et al. (2007) is related to the number of extinction trials received by the nontarget flavor prior to compound extinction treatment: in Calton et al.'s experiment, subjects received 9 extinction trials with the nontarget flavor, whereas in Pineño et al.'s experiment 5 extinction trials were given. The number of extinction trials with the nontarget flavor seems especially critical in light of a study by Brooks et al. (2003), who replicated some of Calton et al.'s results when 9, but not 3, extinction trials were given. Although a moderate number (3 or 5) of nonreinforced trials can be enough to produce detectable extinction, extending this extinction treatment could perhaps endow a CS with the properties of a conditioned inhibitor (as proposed by Calton et al.). Thus, different results could be anticipated to occur after extinction of a target CS in compound with either a moderately or an extensively extinguished CS. The objective of the present experiment was to replicate the results of Pineño et al.'s study while trying to ascertain whether extending extinction treatment with the nontarget flavor could

produce, in accordance with Calton et al.'s finding, protection from extinction. Towards this goal, the nontarget CS (A) received either 0, 5 or 10 extinction trials prior to compound extinction. If the results of both Pineño et al.'s and Calton et al.'s studies are conjointly observed in the present experiment, the target CS (S) should be protected from extinction due to its presentation in a nonreinforced compound with a second CS, A, that previously received either 0 or 10, but not 5, extinction trials. That is, protection from extinction could be found to occur when the target CS is nonreinforced in the presence of either an excitor or an extensively extinguished CS (a putative inhibitor, according to Calton et al.), but not in the presence of a CS that received a moderate amount extinction treatment.

EXPERIMENT

The experiment was performed using a CTA preparation in which different flavors and tastes provided the CSs that were paired with illness induced by an injection of lithium chloride (LiCl), which served as the US. Table 1 summarizes the design of the experiment. In all experimental groups, CSs A, B, and S were first paired with the US. Following conditioning, two extinction phases took place. In a first extinction phase, all groups were given nonreinforced presentations with either CS A, CS B, or both. Specifically, group 0A-2AS received 0 A-alone presentations, group 5A-2AS received 5 A-alone presentations, and group 10A-2AS was given 10 A-alone presentations (i.e., the first number in the group names represents the number of extinction trials with CS A). In order to equate the number of total extinction trials during this phase, group 0A-2AS received 10 extinction trials with CS B, group 5A-2AS received 5 extinction trials with CS B, whereas group 10A-2AS received no presentation of CS B during Extinction Phase 1. In a second extinction phase, these three groups were given 2 extinction trials with CS S in compound with CS A (i.e., AS trials). Two control groups were included in the experiment. Group 0A-2S was given 2 presentations of CS S-alone during Extinction Phase 2. Group 0A-0S was given no presentation of CS S during this phase. Rather, this group received mere exposure to a novel taste, NaCl. Control groups also received extinction treatment with one of the nontarget CSs, B, during Extinction Phase 1. Following extinction treatment, all groups were given two test sessions, one with CS S and one with CS A.

METHOD

Subjects. The subjects were 60 Wistar, naïve, young adult rats (30 males and 30 females), obtained from the breeding colony of the University of Seville. Rats were 70-90 days old at the beginning of the experiment, and their body weights ranged from 275 to 376 g ($M = 312.86$, $SEM = 4.64$) for the males and from 174 to 245 g ($M = 202.53$, $SEM = 3.37$) for the females. The animals were housed individually in 36 x 20 x 14 cm clear plastic cages on a 12:12-h light:dark cycle (from 07:00 to 19:00 h), with all the experimental sessions occurring during the light period. Subjects had free access to food in the home cage. Prior to initiation of the experiment, water availability was progressively reduced to 30 min per day. For two weeks prior to initiation of the experiment and until its termination, subjects were handled for 30 s 2-3 times a week.

Apparatus. All the experimental manipulations were conducted in the home cages. Daily access to water was provided in 500-ml bottles fitted with stainless steel spouts, attached to the front of each cage. In the experimental sessions, fluids were provided at room temperature in bottles fitted with stainless steel spouts containing ball bearings and attached to the front of each cage. The amount of liquid intake was measured by weighing the bottles before and after the liquid presentations.

Four distinct stimuli, two flavors and two tastes, were employed in this study (see Table 1). The flavors were a 1% (vol/vol) apple cider vinegar solution (Prima, Spain) and a 1% (wt/vol) decaffeinated coffee solution (Marcilla, Sara Lee Southern Europe, S. L., Barcelona, Spain), which served as solutions A and B, counterbalanced within groups. The tastes were a 1% (wt/vol, 0.17M) NaCl (N) solution (Sigma-Aldrich Chemie GmbH, Steinheim, Germany), and a 1% (wt/vol, 0.03M) sucrose (S) solution (Fluka Chemie GmbH, Buchs, Switzerland and ICN Biochemicals Inc., Aurora, Ohio, USA, in Replications 1 and 2, respectively). All solutions were made using tap water. The US was a 10 ml/kg of body weight intraperitoneal (i.p.) injection of 0.15M lithium chloride (LiCl, ICN Biochemicals Inc., Aurora, Ohio, USA), which was administered using a 5-ml syringe with a 0.6 mm x 25 mm needle.

Procedure. The experiment was run in two separate replications. In the first replication, 36 subjects were assigned to one of five experimental groups (see Table 1), matched for body weight ($n = 8$, 4 males and 4 females, for each of groups 0A-2AS, 5A-2AS, 10A-2AS, and 0A-2S, and $n = 4$, 2 males and 2 females, for group 0A-0S). In the second replication, 24

subjects were assigned to one of three experimental groups (i.e., groups 0A-2AS, 5A-2AS, and 10A-2AS), also matched for body weight ($n = 8$, each group containing 4 males and 4 females). Thus, after pooling data from both replications, groups 0A-2AS, 5A-2AS, and 10A-2AS consisted of 16 subjects each, whereas groups 0A-2S and 0A-0S consisted of 8 and 4 subjects, respectively. Unless explicitly stated, all subjects were given a single 20-min experimental session per day, which started between 11:00 h and 12:00 h. Also, all subjects received additional 10-min access to tap water soon after the session. Consumption during each session was recorded.

Table 1: Design of the experiment.

Group	Conditioning	Extinction		Test 1	Test 2
		Ext. Ph. 1	Ext. Ph. 2		
0A-2AS	1 A→US / 1 B→US, 1 S→US	10 B	2 AS	1 S	1 A
5A-2AS	1 A→US / 1 B→US, 1 S→US	5 A / 5 B	2 AS	1 S	1 A
10A-2AS	1 A→US / 1 B→US, 1 S→US	10 A	2 AS	1 S	1 A
0A-2S	1 A→US / 1 B→US, 1 S→US	10 B	2 S	1 S	1 A
0A-0S	1 A→US / 1 B→US, 1 S→US	10 B	2 N	1 S	1 A

Note. Ext. = Extinction; Ph. 1 = Phase 1; Ph. 2 = Phase 2. A & B = vinegar and coffee solutions, counterbalanced; S = sucrose solution; AS = simultaneous presentation of A and S; US = LiCl i.p. injection. '→' means 'immediately followed by' and '/' means that the trial types were interspersed within a single phase. The numbers denote the number of presentations of each trial type in each phase. See text for further details.

Pretraining. On Days 1-4, tap water was presented with the glass bottle. This treatment acclimated subjects to drinking from the lick tubes at the daily treatment time.

Conditioning. Conditioning treatment took place on Days 5-10. On Day 5, half of the subjects in each group received a presentation of the A solution, whereas the other half of the subjects received a presentation of the B solution. On Day 7, the assignment of the A and B solutions was reversed: the subjects that were given the A solution on Day 5 now received a presentation of the B solution, whereas the subjects that were given the B solution on Day 5 now received a presentation of the A solution. On Day 9, all subjects received a presentation of the S solution. The presentations of the A, B, and S solutions on Days 5, 7, and 9 were followed immediately by an i.p. injection of LiCl, after which the animals were immediately returned to their home cage. Each conditioning day was followed by one recovery

day (i.e., Days 6, 8, and 10), on which tap water was presented, while allowing the subjects to recover from the impact of the LiCl injection.

Extinction. On Days 11-26, extinction treatment (i.e., A, B, S, AS, or NaCl (N) solutions followed by no LiCl injection) took place. On each day of Extinction Phase 1 (Days 11-20), groups 0A-2AS, 0A-2S, and 0A-0S received a presentation of the B solution, whereas group 10A-2AS received a presentation of the A solution. Group 5A-2AS received 5 presentations of the A solution, interspersed with 5 presentations of the B solution in a pseudorandom order, with the only restriction being that no more than 2 consecutive trials of the same type could be given. Extinction Phase 2 occurred on Days 21-26, with all groups receiving treatment only on Days 23 and 26¹. Groups 0A-2AS, 5A-2AS, and 10A-2AS received access to the AS compound solution. Groups 0A-2S and 0A-0S received access to the S and N solutions, respectively.

Testing. On Days 27 and 28, all subjects were tested for consumption of solutions S and A, respectively. An alpha level of $p < .05$ was adopted for all statistical analyses.

RESULTS

Two separate 3 (group: 0A-2AS vs. 5A-2AS vs. 10A-2AS) x 2 (replication) ANOVAs were performed on the consumption at test of S and at test of A, and yielded neither main effect of replication nor a Group x Replication interaction on either test, $ps > .15$. Therefore, data from both replications were pooled.

Figure 1 depicts the results from conditioning and extinction. As can be appreciated from the left panel of the figure, consumption of the S solution during conditioning was apparently lower than that of the A solution. These impressions were confirmed by a 5 (group) x 2 (flavor) ANOVA on mean consumption, which showed a main effect of flavor, $F(1, 55) = 6.03$, $p < .05$, but no main effect of group, $p > .19$, nor a Group x CS interaction, $p > .08$.

The results of Extinction Phase 1 are depicted in the middle panel of Figure 1. Both groups 5A-2AS and 10A-2AS increased their consumption of flavor A during this phase. However, group 5A-2AS increased its consumption more rapidly than group 10A-2AS, as shown by a 2 (group) x

¹ The treatment of Extinction Phase 2 was conducted in alternate days because, in the original experiment, additional groups were given 6 extinction trials during this phase. Because groups 0A-2AS and 5A-2AS are otherwise identical to conditions of Pineño et al.'s (2007) experiment, in which compound extinction treatment occurred on two consecutive days, the present experiment will indicate whether sparse AS trials have an impact comparable to that of consecutive AS trials.

5 (day) ANOVA on the consumption of flavor A on Days 1-5, which yielded a main effect of day, $F(4, 120) = 74.55, p < .01$, and a Group \times Day interaction, $F(4, 120) = 2.72, p < .05$, but no main effect of group, $p > .13$. This faster rate of extinction in group 5A-2AS than in group 10A-2AS over Days 1-5 can be due to the 5 A-alone trials of group 5A-2AS being spread over 10 days and interspersed with 5 B-alone trials, which might have produced some generalization of extinction from B to A. However, group 10A-2AS increased its consumption of flavor A between Day 6 and Day 10, $F(4, 60) = 8.02, p < .01$, with the result that, on the last day of Extinction Phase 1, groups 5A-2AS and 10A-2AS consumed comparable amounts of flavor A, $p > .93$.

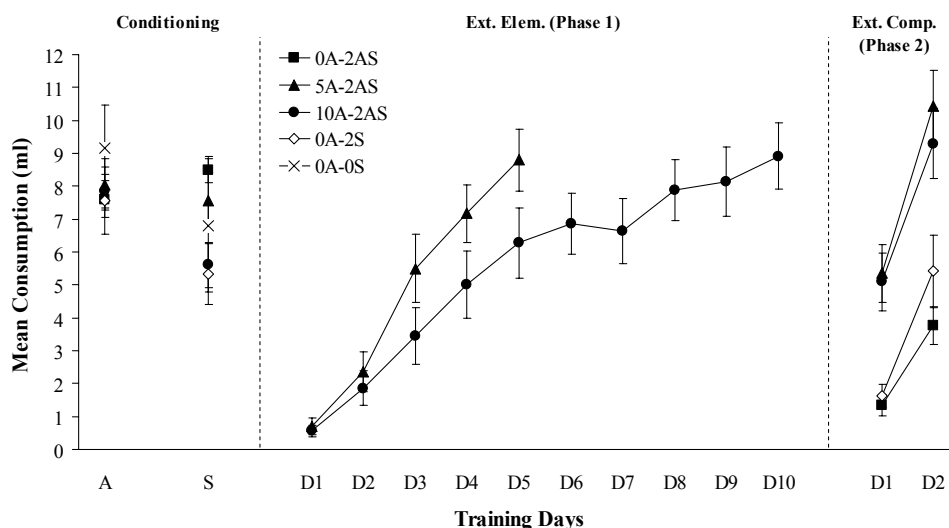


Figure 1. Mean consumption during conditioning (left panel) and extinction (middle and right panels). Error bars depict standard error of the means.

The results of Extinction Phase 2 are depicted in the right panel of Figure 1. All groups increased their consumption of either the AS compound solution (i.e., groups 0A-2AS, 5A-2AS, and 10A-2AS) or the S solution (i.e., group 0A-2S). However, those groups that received prior extinction treatment with flavor A (i.e., groups 5A-2AS and 10A-2AS) showed higher consumption of the AS solution than the group that received no prior extinction treatment with flavor A (i.e., group 0A-2AS) which, in turn consumed the AS solution in an amount comparable to consumption of

S in group 0A-2S). These impressions were confirmed by a 4 (group) x 2 (day) ANOVA, which showed main effects of group, $F(3, 52) = 11.13, p < .01$, and day, $F(1, 52) = 130.27, p < .01$, as well as a Group x Day interaction, $F(3, 52) = 3.32, p < .05$. In order to ascertain the source of this interaction, one-way ANOVAs were conducted on each daily consumption during Extinction Phase 2 and pairwise comparisons were performed using the error-term from each one-way ANOVA. Groups differed in their consumption on Day 1, $F(3, 52) = 8.60, p < .01$, and on Day 2, $F(3, 52) = 10.87, p < .01$. Groups 0A-2AS and 0A-2S consumed the AS and S solutions, respectively, in comparable amounts on both days, $ps > .30$. Groups 5A-2AS and 10A-2AS also consumed the AS solution in similar amounts, $ps > .39$. Moreover, groups 0A-2AS and 0A-2S consumed less than groups 5A-2AS and 10A-2AS on both days, $F_s(1, 52) > 5.85, ps < .05$.

The results of the test of S and A are depicted in Figure 2. As can be observed in this figure, consumption of S at test was lower in groups 0A-2AS and 10A-2AS than in group 5A-2AS. More important, comparisons of these three groups with control groups 0A-2S and 0A-0S indicate that groups 0A-2AS and 10A-2AS were protected from extinction of the aversion elicited by S, whereas group 5A-2AS extinguished its aversion to S uneventfully. The following analyses supported these impressions. A one-way ANOVA among groups on the consumption of S at test showed significant differences, $F(4, 55) = 4.80, p < .01$. Groups 0A-2AS and 10A-2AS consumed S in comparable amounts, $p > .69$. Also, group 5A-2AS consumed more of the S solution than both groups 0A-2AS, $F(1, 55) = 6.22, p < .05$, and 10A-2AS, $F(1, 55) = 8.31, p < .01$. Protection from extinction, as indicated by low consumption of S relative to group 0A-2S, was found in group 0A-2AS, $F(1, 55) = 4.29, p < .05$, and group 10A-2AS, $F(1, 55) = 5.71, p < .05$, but not in group 5A-2AS, $p > .97$. Also, comparisons with group 0A-0S, which received no extinction treatment with S, showed that protection from extinction was complete in both groups 0A-2AS, $p > .25$, and 10A-2AS, $p > .36$. Group 5A-2AS, by contrast, consumed more of the S solution than group 0A-0S, $F(1, 55) = 7.47, p < .01$. Finally, two S-alone presentations resulted in significant extinction of the aversion elicited by S, as shown by the higher consumption of group 0A-2S than that of group 0A-0S, $F(1, 55) = 6.35, p < .05$.

Consumption of flavor A also differed at test, as shown by a one-way ANOVA among groups, which yielded significant differences, $F(4, 55) = 7.05, p < .01$. Group 0A-2AS consumed less of flavor A than groups 5A-2AS, $F(1, 55) = 9.67, p < .01$, and group 10A-2AS, although this latter difference fell short of significance, $p > .06$. Groups 5A-2AS and 10A-2AS consumed comparable amounts of flavor A, $p > .21$. Also, groups 0A-2AS, 5A-2AS, and 10A-2AS consumed more of flavor A than group 0A-2S did,

$F_s(1, 55) > 5.21$, $ps < .05$, which indicate that the 2 AS presentations were enough to produce detectable extinction of aversion elicited by flavor A. These differences were not consistently found when consumption in groups 0A-2AS, 5A-2AS, and 10A-2AS was compared to that of group 0A-0S, perhaps due to one subject in this latter group consuming a large amount of flavor A and this group containing only 4 subjects in total (which resulted in high mean consumption and large variance in this group). Group 0A-0S consumed less of flavor A than group 5A-2AS, $F(1, 55) = 5.88$, $p < .05$, but it did not differ from groups 0A-2AS and 10A-2AS, $ps > .10$. Finally, groups 0A-2S and 0A-0S consumed comparable amounts of flavor A, $p > .23$.

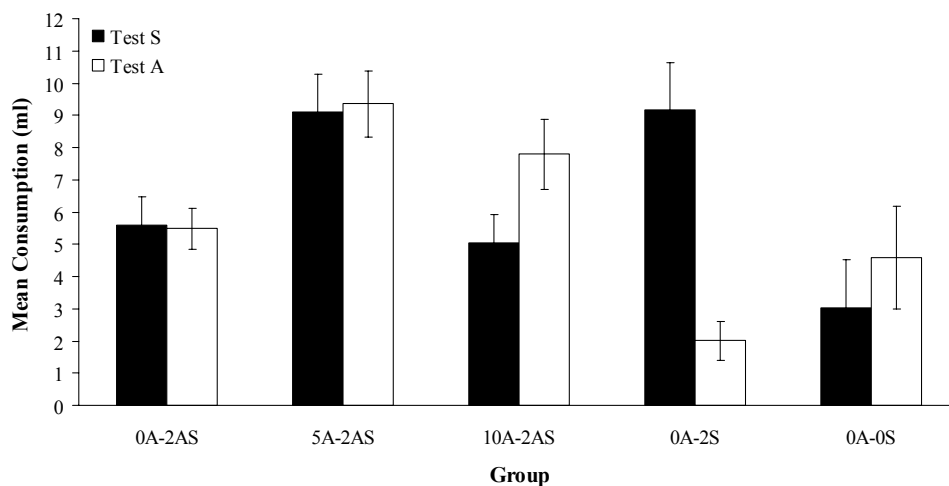


Figure 2. Mean consumption at test of S and A. Error bars depict standard error of the means.

DISCUSSION

The experiment in this report provided evidence of protection from extinction in a conditioned taste aversion preparation by performing extinction treatment with the target CS (S) conjointly with a second CS (A), consisting of either an excitator (group 0A-2AS) or an extensively extinguished CS (group 10A-2AS). Protection from extinction was not observed when CS S was presented in a nonreinforced compound with a moderately extinguished CS (group 5A-2AS). These results replicated the findings of previous studies by Pineño et al. (2007), who found protection from extinction in a condition identical to group 0A-2AS in the present

experiment, and unaffected extinction in a condition identical to the present group 5A-2AS. Also, the results of Calton et al. (1996), who found protection from extinction when a target CS received nonreinforced exposures conjointly with a CS that received previous extensive extinction treatment, are replicated in group 10A-2AS of the present experiment.

As in the study of Pineño et al. (2007), the finding that an excitor (group 0A-2AS) can protect the target CS from extinction can be interpreted as indicative of a putative second-order conditioning process. In this case, the presence of flavor A during AS compound trials could result in second-order conditioning to CS S due to S strengthening its association with the aversive CR, which is elicited by the presence of CS A. This interpretation therefore relies on the assumption that the response potential of CS S was positively mediated by the response potential of CS A, an assumption that is further supported by the results of group 5A-2AS, in which prior extinction of CS A allowed CS S to extinguish uneventfully. However, a contradiction arises when the results of group 10A-2AS are considered from this positive mediation view: assuming that extending extinction treatment with flavor A endowed this CS with the properties of a conditioned inhibitor (Calton et al., 1996; Denniston & Miller, 2003; but see Brooks et al., 2003), then one would expect enhanced extinction of the aversion to CS S to be found in group 10A-2AS, a result that would resemble second-order conditioned inhibition (Rescorla, 1976). Rather, the results of group 10A-2AS speak of CS A negatively mediating the response potential of CS S: in this group, due to CS A being extensively extinguished prior to extinction treatment with the AS compound, CS S maintained a strong excitatory status through compound extinction treatment.

These results pose a problem to associative models (e.g., Dickinson & Burke, 1996; Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Van Hamme & Wasserman, 1994; Wagner, 1981), which are only able to explain protection from extinction when the target CS is nonreinforced in compound with a conditioned inhibitor. According to these models, presenting a conditioned inhibitor (A) during extinction treatment with the target CS (S) should decrease the expectation of the absent US (i.e., based on a low total associative strength of the AS compound), thereby reducing the loss of associative strength undergone by CS S on each extinction trial (i.e., protection from extinction). By contrast, presenting an excitor (A) during extinction treatment with CS S should increase the expectation of the absent US (i.e., based on a high total associative strength of the AS compound), which would result in an extraordinary loss of associative strength by CS S (i.e., enhanced extinction). Therefore, these models could explain the protection from extinction found in group 10A-2AS by assuming that CS A became a net inhibitor prior to compound

exposures with CS S; but are unable to explain the results of group 0A-2AS because in this group enhanced extinction, instead of protection from extinction, was expected to occur.

In a similar vein, the present results cannot be explained from a configural theory (e.g., Pearce, 1987, 1994) as an instance of generalization decrement. From this configural view, compound treatment with A and S could result in processing the AS compound as a configural unit that resembles only partially its features, A and S. This claim of the configural view is especially reasonable when applied to the present experiment, because the simultaneous presentation of a taste and a flavor in a single cocktail might result in a solution with a unique palatability, which cannot be perceived when tasting each of its component features on their own. Strictly taken, a configural view would state that the AS configural unit was perceived as a completely different stimulus from its features, A and S. This view could explain the strong aversive response elicited by S at test following extinction treatment with the AS compound in groups 0A-2AS and 10A-2AS, because extinction treatment with AS would not transfer to S at all. However, this strict configural view would fail to explain the results of group 5A-2AS, which showed less aversion to S than groups 0A-2AS and 10A-2AS. Importantly, Pearce's (1987, 1994) configural model is flexible enough to allow treatment with A and S to be transferred to the AS configural unit and *vice versa* and, hence, predicts that some extinction should transfer from the AS configural unit to S (i.e., generalization from compound extinction to test). Also, Pearce's model would expect that extinction treatment with A should transfer to the AS configural unit (i.e., generalization from elemental to compound extinction treatments). If so, as flavor A received more extinction trials, A would transfer less excitatory associative strength to the AS configural unit which, in turn, would transfer less excitatory strength to S. If this was the case, according to Pearce's model, the test of S should yield a strong aversive response in group 0A-2AS, followed by a weaker aversion in group 5A-2AS and, finally, by the weakest aversion in group 10A-2AS. Our finding that groups 0A-2AS and 10A-2AS consumed less of S at test than group 5A-2AS is at odds with the predictions of Pearce's model.

Some studies (e.g., Stout, Escobar, & Miller, 2004; Yin, Barnet, & Miller, 1994; see also Gewirtz & Davis, 2000) found that interspersing pairings of a CS (A) with the US (i.e., A→US trials) with nonreinforced presentations of CS A in compound with a second CS (X, i.e., AX trials) can result in either second-order conditioning (positive mediation between the response potentials of A and X) or Pavlovian conditioned inhibition (negative mediation between the response potentials of A and X) as a function of the number of AX compound trials. Specifically, in these studies

when few AX compound trials were given, second-order conditioning was observed; whereas Pavlovian conditioned inhibition was found after extensive nonreinforced treatment with the AX compound. The results of the present experiment suggest that the number of nonreinforced presentations of CS A *per se*, instead of the number of nonreinforced presentations of the AX compound, might be the critical factor determining the transition from positive to negative mediation found in the studies by Stout et al. and Yin et al.

Although perhaps premature at this point, a speculative explanation could be outlined. If it was assumed that the amount of extinction undergone by the CR elicited by the target CS (S) depended on a balance between the strength of the excitatory and inhibitory potentials of this CS, the presence of CS A during extinction treatment with CS S could be able to attenuate extinction to CS S in two different ways. On one hand, when CS A consists of an excitor, its presence during nonreinforcement of CS S could increase the excitatory potential of CS S, perhaps by strengthening the S-CR association (second-order conditioning, as suggested by Pineño et al., 2007). In this case, CS S could uneventfully acquire an inhibitory potential during extinction treatment, but this inhibitory potential would be counteracted at test by the strengthened excitatory potential. On the other hand, when CS A consists of an inhibitor, its presence during nonreinforcement of CS S could prevent S from acquiring an inhibitory potential (blocking of inhibition; for related results see Suiter & LoLordo, 1971). In this case CS S would also maintain a strong net excitatory status despite extinction treatment. The first of these hypothetical processes would account for protection from extinction in group 0A-2AS, whereas the second hypothetical process would explain protection from extinction in group 10A-2AS. Furthermore, if a moderate amount of extinction treatment with CS A (group 5A-2AS) resulted in this CS being neither a strong excitor nor a strong inhibitor prior to compound treatment (i.e., CS A could be processed as a neutral stimulus), neither of these hypothetical processes should occur. Thus, in group 5A-2AS, CS S could have uneventfully extinguished its aversive CR despite the presence of CS A. Regardless of the merit of this speculative explanation, the results of the present experiment indicate that protection from extinction can be achieved, not only by a concurrent conditioned inhibitor (e.g., Chorazyna, 1962; Lovibond et al., 2000; Rescorla, 2003; Soltysik et al., 1983), but also by the concurrent presentation of an excitor (Pineño 2007; also see Vervliet, Vansteenwegen, Hermans, & Eelen, 2006, for a demonstration in fear conditioning with humans) or an extensively extinguished CS (Calton et al., 1996), during extinction treatment with the target CS. These results, thus,

claim for further research on the complex interactions that arise between CSs with different prior associative histories during an extinction treatment.

RESUMEN

Protección de la extinción por la presentación concurrente de un excitador o un EC extensivamente extinguido. Un experimento en aversión condicionada al sabor evaluó el impacto de la extinción de un estímulo condicionado (EC) crítico, S, en compuesto con un segundo EC, A, sobre la respuesta condicionada elicitada por el EC S durante su presentación en solitario en la prueba. Tras un tratamiento inicial de condicionamiento con los ECs A y S, el experimento manipuló el número de ensayos de extinción con el EC A en solitario (esto es, 0, 5, o 10 ensayos) previamente al tratamiento con el compuesto AS. Además, dos grupos de control recibieron ensayos de extinción con S en solitario o ningún tratamiento de extinción con S. Los resultados de las condiciones que recibieron 0 o 10 ensayos de extinción con el EC A previamente a las exposiciones no reforzadas de AS indicaron protección de la extinción de la aversión elicitada por el EC S, mientras que la condición que recibió 5 ensayos de extinción con el EC A antes de los ensayos con AS mostró una extinción desprotegida de la aversión elicitada por el EC S. Los modelos asociativos actuales tienen dificultades para explicar este patrón de resultados.

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