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Second-order conditioning during a compound extinction treatment

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Abstract

Two conditioned taste aversion experiments with rats were conducted to establish if a target taste that had received a prior pairing with illness could be subject to second-order conditioning during extinction treatment in compound with a flavor that also received prior conditioning. In these experiments, the occurrence of second-order conditioning was indicated by protection from extinction of the aversion elicited by the target taste. This possibility, although intuitive, deserves attention because current associative models [e.g., Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and nonreinforcement. In A. H. Black & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory* (pp. 64–99). New York: Appleton-Century-Crofts.] predict exactly the opposite outcome, namely, that compound extinction of two CSs should result in enhanced extinction.

Keywords: Second-order conditioning; Protection from extinction; Conditioned taste aversion

Pairing a relatively neutral stimulus or conditioned stimulus (CS, e.g., a light or a tone) with a stimulus of an intrinsic biological significance or unconditioned stimulus (US, e.g., food or a mild shock) is known to result in the development of a conditioned response to the CS. Once conditioned responding to this CS (CS_1) is established by means of first-

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order conditioning (i.e., direct pairings of CS_1 with the US), CS_1 can support conditioning to a second CS (CS_2) during their joint presentation in either simultaneous (i.e., $CS_1 + CS_2$) or serial (i.e., $CS_2 \rightarrow CS_1$) compound, an effect known as second-order conditioning (Pavlov, 1927). Second-order conditioning (SOC) encouraged a vast amount of research in the 1970's and 1980's and, together with other higher-order conditioning effects (e.g., sensory preconditioning, Brodgen, 1939), critically shaped learning theory (for a review, see Rescorla, 1980). SOC is also of considerable applied/clinical importance because it addresses the effective reinforcement of human behavior using stimuli that lack intrinsic biological significance, such as money, points, tokens, or even some social rewards (e.g., flattering remarks) or punishments (e.g., reproach).

Despite some recent notable exceptions (e.g., Stout, Escobar, & Miller, 2004; Winterbauer & Balleine, 2005), interest in the study of the mechanisms involved in *positive mediation effects* (i.e., effects in which the response potential of CS_2 is directly correlated with the response potential of CS_1, e.g., SOC and sensory preconditioning) has diminished somewhat in recent years. This decrease might be due to the great amount of attention devoted to their counterparts, namely, *negative mediation effects* (i.e., effects in which the response potential of CS_2 is negatively correlated with the response potential of CS_1, e.g., stimulus competition phenomena such as overshadowing and blocking). In fact, most current models of learning have been exclusively developed to account for negative mediation phenomena and, thus, are unable to explain positive mediation effects. This is true for traditional associative models (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981), recent revisions of such traditional associative models (e.g., Dickinson & Burke, 1996; Van Hamme & Wasserman, 1994), and even performance-focused models, such as statistical models (e.g., Allan, 1980; Cheng, 1997; Cheng & Novick, 1992; Spellman, 1996), and comparator theory (Miller & Matzel, 1988; see also Denniston, Savastano, & Miller, 2001).

Despite the widespread tendency of learning models to focus on *negative mediation effects*, research from the early 1980's found that *positive mediation effects* (e.g., SOC and sensory preconditioning) are much more pervasive than previously acknowledged. Speers, Gillan, and Rescorla (1980) demonstrated that SOC can take place even when the $CS_1 + CS_2$ compound presentations are followed by the US. Importantly, a SOC treatment in which the $CS_1 + CS_2$ compound is paired with the US (i.e., $CS_1 \rightarrow$ US trials followed by $CS_1 + CS_2 \rightarrow$ US trials) closely resembles a *blocking* treatment (Kamin, 1969), in which CS_2 is expected to produce weak conditioned responding due to its being paired with the US in the presence of CS_1. That is, CS_1 is expected to support conditioning to CS_2 when the $CS_1 + CS_2$ compound is never paired with the US (i.e., the traditional SOC procedure) but to prevent conditioning to CS_2 when the $CS_1 + CS_2$ compound is paired with the US. However, there is no *a priori* reason why the presence or absence of the US in a similar treatment should result in contrary behavioral outcomes. Instead of being blocked by CS_1 in its acquisition of conditioned responding, CS_2 could benefit from its pairings with both the US and CS_1, acquiring strong excitatory control of behavior (i.e., the target CS could become both a first-order and second-order CS, see Dickinson, Nicholas, & Mackintosh, 1983; Holland, 1982; Rudy, 1982). As pointed out by Speers et al. (see also Rescorla & Durlach, 1981), this kind of learning might complicate our understanding of some experimental results involving treatments like blocking. A failure to observe blocking could be due to CS_2 having developed a strong association with the US or, alternatively, this failure could be due

to CS_2 having acquired a weak association with the US, but a strong association with CS_1 (i.e., SOC).

Other experimental designs involving treatments with compound CSs have also been found to produce either positive or negative mediation. One such design involves the joint presentation of two CSs followed by the US (i.e., $CS_1 + CS_2 \rightarrow$ US trials). This treatment usually results in the target CS_2 eliciting a weak conditioned response compared to a condition in which CS_2 receives pairings with the US on its own, an effect known as *overshadowing* (Pavlov, 1927). Conversely, it has been also found that, in this procedure, CS_2 can come to produce a strong conditioned response after $CS_1 + CS_2 \rightarrow$ US trials, relative to a condition given $CS_2 \rightarrow$ US pairings, an effect referred to as *potentiation* (e.g., Rusiniak, Hankins, Garcia, & Brett, 1979). Potentiation can be explained within the same theoretical frameworks that were developed to explain higher-order conditioning, such as SOC and sensory preconditioning (e.g., Batsell, Trost, Cochran, Blankenship, & Batson, 2003; Durlach & Rescorla, 1980).

The present experiments examined the possibility that a target CS (CS_2) that was previously paired with the US can be subject to SOC when presented in a nonreinforced compound with another first-order CS (CS_1), that is, during a compound extinction treatment. According to associative models of learning (e.g., Rescorla & Wagner, 1972)[1], the presentation of two CSs in a nonreinforced compound should result in enhanced extinction of responding to each separate CS (Rescorla, 2000). The typical treatment that results in enhanced extinction involves the initial separate conditioning of two stimuli (i.e., $CS_1 \rightarrow$ US and $CS_2 \rightarrow$ US trials), which are later extinguished in compound (i.e., $CS_1 + CS_2$ trials). In this framework, enhanced extinction is expected to occur during nonreinforced presentations of the $CS_1 + CS_2$ compound because, based on their prior separate pairings with the US, the joint presentation of CS_1 and CS_2 elicits a large expectation of the absent US, which results in each CS undergoing a large loss of associative strength relative to if either CS had been nonreinforced in isolation. Importantly, the critical difference between this treatment and a SOC treatment consists of whether CS_2 is paired with the US on its own prior to $CS_1 + CS_2$ compound extinction or receives no prior treatment. As mentioned above regarding reinforcement of the $CS_1 + CS_2$ compound, there is no *a priori* reason to expect that reinforcing CS_2 elementally prior to nonreinforced presentations of the $CS_1 + CS_2$ compound should result in negative mediation (i.e., enhanced extinction), rather than positive mediation (i.e., SOC). If SOC can be effectively produced with this treatment, extinction of the conditioned response elicited by CS_2 should be attenuated. That is, the transfer of excitatory control of behavior from CS_1 to CS_2 through a SOC process would attenuate the impact of the nonreinforcement of CS_2 and, thus, CS_2 would maintain a high excitatory status.

The present experiments were performed using a conditioned taste aversion 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. In both Exper-

[1] The model by Rescorla and Wagner (1972) explains extinction as due to unlearning of the previously acquired CS–US association, a notion that has been extensively discredited by more recent research (see Bouton, 1993, for a review). However, it is important to emphasize here that the prediction of enhanced extinction by compound extinction is not exclusive to the Rescorla–Wagner model; rather, it is shared by most associative models, even by those that explain extinction based on the formation of an inhibitory CS–US association (e.g., Pearce & Hall, 1980; Wagner, 1981).

iments 1 and 2, the target CS_2 (i.e., a sucrose solution) was first paired with the US. The critical treatment in both experiments consisted of the extinction treatment with CS_2, which took place either with CS_2 occurring by itself or in compound with a nontarget CS_1 (i.e., a vinegar or coffee solution). Also, prior experience with CS_1 was manipulated in both experiments. Experiment 1 manipulated the occurrence of $CS_1 \rightarrow US$ pairings and of $CS_1 + CS_2$ presentations during conditioning and extinction, respectively, as a means of assessing the role that SOC might have on conditioned aversive responding following nonreinforced compound presentations of pretrained excitors. In Experiment 2, both $CS_1 \rightarrow US$ and the $CS_1 + CS_2$ pairings were provided, but CS_1-alone trials were given either before or after $CS_1 + CS_2$ pairings in order to assess if extinction of CS_1 can attenuate SOC to CS_2.

Experiment 1

Experiment 1 aimed to ascertain if SOC could be observed when the target CS received a pairing with the US prior to nonreinforced compound presentations with the nontarget CS. Towards this purpose, this experiment used a procedure similar to those typically used in studies of SOC with a neutral target stimulus (e.g., Rizley & Rescorla, 1972). The design of Experiment 1 is summarized in Table 1. All groups received a single pairing of the target CS, sucrose, with the US. Group PP (paired–paired) first received a pairing of the nontarget CS, vinegar, with the US and then received extinction treatment consisting of nonreinforced compound presentations of vinegar and sucrose. Control groups included subjects that received explicitly unpaired presentations of vinegar and sucrose during extinction treatment (group PU) or an explicitly unpaired presentation of vinegar and the US (group UP). If this experiment obtained a SOC effect, a stronger aversion at test on sucrose should occur for group PP than for groups PU and UP. Groups PP-E and NE were additional control conditions. In group PP-E (paired–paired–extinction), as in group PP, vinegar was first paired with the US and then vinegar was presented in a nonreinforced compound with sucrose. However, in this group nonreinforced vinegar + sucrose presentations were interspersed with vinegar-alone presentations. Extinction of the conditioned aversion to vinegar could be expected to reduce its impact on the extinction of sucrose, in which case this group could consume the sucrose solution in an amount comparable to control groups PU and UP. Finally, group NE (no extinction) received a pairing of vinegar with the US. However, no nominal CS was presented

Table 1
Design of Experiment 1

Group	Conditioning	Extinction	Test 1	Test 2
PP	1 V → US, 1 S → US	2 VS / 2 W	1 S	1 V
PU	1 V → US, 1 S → US	2 S / 2 V	1 S	1 V
UP	1 V / 1 US, 1 S → US	2 VS / 2 W	1 S	1 V
PP-E	1 V → US, 1 S → US	2 VS / 2 V	1 S	1 V
NE	1 V → US, 1 S → US	4 W	1 S	1 V

Note. V, vinegar solution; S, sucrose solution; VS, simultaneous presentation of vinegar and sucrose; W, water; US, LiCl. '→' 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.

during the extinction phase (this group received only water in this phase). This group therefore provided a baseline for conditioned aversion to both sucrose and vinegar at test in order to determine the degree to which extinction was protected in group PP.

Method

Subjects

The subjects were 40 Wistar, naïve, young adult rats (20 males and 20 females), obtained from the breeding colony of the University of Seville. The rats were about 70 days old at the beginning of the experiment, and their body weights ranged from 251 to 344 g ($M = 287.35$, SEM = 5.32) for the males and from 171 to 250 g ($M = 212.90$, SEM = 5.04) for the females. The animals were housed individually in $36 \times 20 \times 14$ cm clear plastic cages in a colony room 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 experimental manipulations were conducted in the home cages. Daily access to water was provided in 500-ml plastic bottles fitted with stainless steel spouts, attached to the front of each cage. In the experimental sessions, fluids were provided at room temperature in glass 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.

Two distinct stimuli, one flavor and one taste, were employed in this study (see Table 1). The flavor was a 1% (v/v) apple cider vinegar solution (Prima, Spain) and the taste was a 1% (w/v, 0.03*M*) sucrose solution (Fluka Chemie GmbH, Buchs, Switzerland). Solutions were made using tap water. The US was a 10 ml/kg of body weight intraperitoneal (i.p.) injection of 0.15*M* lithium chloride (LiCl, ICN Biochemicals Inc., Aurora, Ohio, USA), which was administered using a 5-ml syringe with a 0.6 mm × 25 mm needle.

Procedure

Forty subjects were assigned to one of five experimental groups, matched for body weight ($n = 8$, each group containing 4 males and 4 females). All subjects were given a single 20-min experimental session per day, initiated between 14:00 and 15:00 h. Also, all subjects received additional 10-min access to water soon after the session. Consumption during each session was recorded.

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

Conditioning. Conditioning treatment took place on Days 5–9. On Day 5, subjects in groups PP, PU, PP-E, and NE received a presentation of the vinegar solution, whereas subjects in group UP received water. These fluid presentations were followed immediately by an i.p. injection of LiCl, after which the animals were immediately returned to the home cage. Day 6 consisted of a recovery day, on which water was presented in a glass bottle, while allowing the subjects to recover from the impact of the LiCl injection. On Day 7, group UP received a presentation of the vinegar solution, whereas the rest of the groups

were given a presentation of water. On Day 8, all groups received a presentation of the sucrose solution, immediately followed by an i.p. injection of LiCl. Day 9 was a recovery day to allow the subjects to recover from the impact of the LiCl injection.

Extinction. On Days 10–13, extinction treatment (i.e., presentations of flavored solutions or water, followed by no LiCl injection) took place. In this phase, groups PP and UP received 2 presentations of a vinegar + sucrose simultaneous compound solution. The presentation of the compound solution occurred on Days 10 and 13 for half of the subjects in each group, and on Days 11 and 12 for the other half of the subjects (this occurred in order to match the presentations of the vinegar + sucrose compound solution in these groups to the presentations of the sucrose solution in group PU as described below). On the remaining days of this phase (either Days 11 and 12, or Days 10 and 13), these groups received a presentation of water. Group PU received alternated daily presentations of vinegar and sucrose solutions, following an ABBA counterbalanced order (i.e., half of the subjects in group PU received presentations of the vinegar solution on Days 10 and 13, and presentations of the sucrose solution on Days 11 and 12; the other half of the subjects received presentations of the vinegar solution on Days 11 and 12, and presentations of the sucrose solution on Days 10 and 13). Group PP-E received a treatment similar to that of group PU; the only difference was that the former group was given presentations of the vinegar + sucrose compound solution, instead of presentations of the sucrose solution. On each day of extinction phase, the subjects in group NE received an exposure to water.

Testing. On Days 14 and 15, all subjects were tested for consumption of solutions sucrose and vinegar, respectively. An α level of $p < .05$ was adopted for all statistical analyses and planned comparisons used the overall error term from the analysis of variance (ANOVA).

Results

Conditioning

The left panel of Fig. 1 depicts the results from conditioning in Experiment 1. As can be seen in the figure, there were no appreciable group differences in the consumption of the vinegar (Day 5 or Day 7) and sucrose solutions (Day 8), but all groups consumed more of the sucrose solution than of the vinegar solution. These impressions were confirmed by a 5 (group) $\times$ 2 (solution: vinegar vs. sucrose) ANOVA on the consumption scores, which yielded only a main effect of solution, $F(1, 34) = 9.12$, $p < .01$, but no main effect of group or an interaction, $ps > .45$ (the consumption of vinegar by one subject from group PU could not be recorded due to experimenter error.) Also, a one-way ANOVA showed that groups did not differ in their consumption of water (Day 5 for group UP; Day 7 for groups PP, PU, PP-E, and NE), $p > .86$.

Extinction

The right panel of Fig. 1 depicts the results from extinction. Consumption of solutions containing sucrose (i.e., vinegar + sucrose compound for groups PP, UP, and PP-E; and sucrose-alone for group PU) is shown separately from consumption of vinegar (i.e., groups PU and PP-E). Regarding the consumption of the vinegar + sucrose or the sucrose-alone solution for the four groups receiving such trials, the most striking result is the high consumption of the vinegar + sucrose compound solution in group UP,

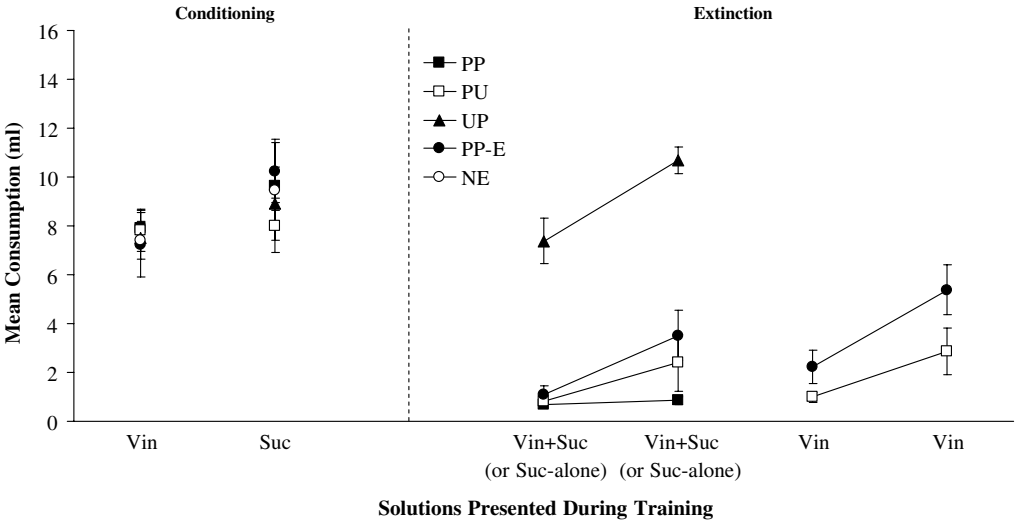

Fig. 1. Experiment 1: mean consumption during conditioning (left panel) and extinction (right panel). Error bars depict standard error of the mean.

compared to consumption in the other groups. Although unexpected, this result might suggest that vinegar acquired responding indicative of inhibition due to its single explicitly unpaired presentation with the LiCl injection. Alternatively, this finding might result from latent inhibition or more rapid habituation of neophobia occurring for vinegar during the conditioning phase for group UP (i.e., latent inhibition or such habituation for vinegar might also elevate consumption of this flavor for this group). A 4 (group) × 2 (day) ANOVA confirmed these impressions yielding main effects of both group, $F(3, 28) = 40.26$, $p < .01$, and day, $F(1, 28) = 29.49$, $p < .01$, as well as a group × day interaction, $F(3, 28) = 3.76$, $p < .05$. Pairwise comparisons showed that consumption on the first trial of either the vinegar + sucrose or the sucrose-alone solutions was comparable in groups PP, PU, and PP-E, ps $> .60$, and significantly lower than consumption of the vinegar + sucrose compound solution in group UP, Fs$(1, 28) > 72.55$, ps $< .01$. Consumption in group UP was still higher than consumption in the rest of the groups on the second trial, Fs$(1, 28) > 37.92$, ps $< .01$, but consumption in group PU did not significantly differ from consumption of groups PP and PP-E, ps $> .19$. Also, group PP-E consumed the vinegar + sucrose solution in a larger amount than group PP, $F(1, 28) = 5.26$, $p < .05$, a result that could be attributed to group PP-E receiving additional nonreinforced presentations of vinegar-alone interspersed with the nonreinforced presentations of the vinegar + sucrose compound solution. Finally, groups PU, UP, and PP-E significantly increased the consumption of their corresponding solution from the first to the second presentation, Fs$(1, 28) > 5.24$, ps $< .05$, an increase that was not observed in group PP, $p > .81$.

The data in the figure indicate that group PP-E consumed vinegar in slightly larger amounts than group PU on the vinegar-alone trials, a result that is consistent with group PP-E receiving, not only nonreinforced presentations of vinegar-alone (i.e., as group PU), but also nonreinforced presentations of vinegar in compound with sucrose. This impression was partially supported by a 2 (group) × 2 (day) ANOVA, which yielded only a main

effect of day, $F(1, 14) = 19.36$, $p < .01$. The main effect of group fell short of significance, $F(1, 14) = 3.89$, $p > .06$. Also, no interaction was found, $p > .29$.

Test

The results of the test of sucrose and vinegar are depicted in Fig. 2. As can be seen in the figure, groups PP and NE showed the lowest consumption of both sucrose and vinegar, whereas consumption of these solutions was moderately high in groups PU, UP, and PP-E. These impressions were supported by the following analyses. A one-way ANOVA showed that consumption of sucrose differed among groups, $F(4, 35) = 5.47$, $p < .01$. Pairwise comparisons showed that groups PP and NE consumed comparable amounts of sucrose, $p > .99$. Also, groups PP and NE consumed less sucrose than groups PU, UP, and PP-E, Fs$(1, 35) > 4.82$, ps $< .05$, which did not differ, ps $> .10$. Thus, extinction of conditioned aversion to sucrose for group PP was not only attenuated (i.e., lower consumption in group PP than in control groups PU and UP), but completely prevented (i.e., virtually identical consumption of sucrose in groups PP and NE). Also, nonreinforced presentations of the vinegar-alone solution interspersed with nonreinforced presentations of the vinegar + sucrose solution resulted in moderate extinction of sucrose, as indicated by the high consumption of group PP-E.

A one-way ANOVA showed that consumption of vinegar differed among the groups, $F(4, 34) = 13.70$, $p < .01$. (The consumption of one subject from group PU could not be recorded due to experimenter error.) Pairwise comparisons showed that groups PP and NE consumed comparable amounts of vinegar, $p > .34$. Groups PP and NE consumed the vinegar solution in smaller amounts than groups PU, UP, and PP-E, Fs$(1, 34) > 5.51$, ps $< .05$. Consumption by group PP-E was comparable to that in groups PU and UP, ps $> .16$, but consumption of vinegar in group UP was higher than in group PU, $F(1, 34) = 7.73$, $p < .01$. Similar to the analyses on consumption of sucrose at test, these analyses on the consumption of vinegar are indicative of prevention of extinction of aversion to vinegar in group PP based on its low consumption relative to groups UP and PU, as well as on its consumption being comparable to that of group NE. Moreover, the finding that group UP consumed larger amounts of vinegar than group PU suggests

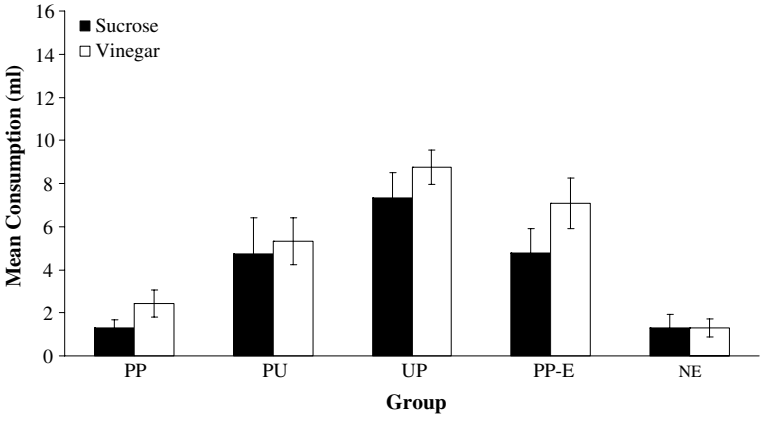

Fig. 2. Experiment 1: mean consumption at test of sucrose and vinegar. Error bars depict standard error of the mean.

that the explicitly unpaired presentation of vinegar and the US in group UP was effective in preventing vinegar from becoming an excitor. If anything, and considering the higher consumption of the vinegar + sucrose solution in group UP during extinction treatment (see Fig. 1), vinegar could have been conditioned as an inhibitor during the explicitly unpaired procedure.

Preliminary ANOVAs showed that trial order during extinction had no effect, nor interacted with group, on the consumption at either test of sucrose, $ps > .94$, or vinegar, $ps > .61$. More importantly, a 5 (group) × 2 (test solution: sucrose vs. vinegar) ANOVA on the consumption during test showed main effects of group, $F(4, 34) = 11.60$, $p < .01$, and solution, $F(1, 34) = 4.69$, $p < .05$, but no interaction, $p > .57$. The lack of an interaction is indicative of similar patterns in the consumption of sucrose and vinegar across groups. Supporting this claim, a positive correlation was found between consumption of sucrose and vinegar at test, *Spearman* $R = 0.77$, $p < .01$. This positive correlation, together with the main analyses on the consumption of sucrose and vinegar at test, indicates that our treatment (i.e., presenting vinegar and sucrose in a nonreinforced compound for two trials) resulted in positive mediation (i.e., the response potential of sucrose directly correlated with the response potential of vinegar). In the present case, positive mediation refers to a protection-from-extinction effect potentially occurring through a SOC mechanism.

One problem with the present results concerns the similarity between consumption of sucrose at test and consumption of solutions containing sucrose (i.e., vinegar + sucrose compound or sucrose-alone solutions) on the second presentation in the extinction phase. In fact, a strong positive correlation was found between these two consumption values in groups PP, PU, UP, and PP-E (group NE was only given water presentations during extinction), *Spearman* $R = 0.78$, $p < .01$. Therefore, it could be claimed that differences found at test of sucrose are merely due to differences in consumption of sucrose during extinction: the more sucrose was consumed during extinction, the greater extinction of aversion to sucrose and, thus, the more sucrose was consumed at test. In this view, one does not need to assume that extinction of sucrose was attenuated by vinegar by means of a process resembling SOC. Rather, the presence or absence of vinegar during extinction treatment with sucrose could be seen as merely modulating the amount of sucrose consumed which, in turn, would result in different levels of extinction for sucrose. The present experiment offers no means by which to discard this alternative, simpler explanation. However, Experiment 2 will, in part, address this issue.

Experiment 2

Experiment 1 obtained a protection-from-extinction effect (group PP vs. control groups PU and UP). These results suggest that a first-order CS (sucrose) can be subject to SOC due to its pairings with another first-order CS (vinegar) in a nonreinforced compound. Interestingly, group PP-E received alternating nonreinforced presentations of vinegar-alone with nonreinforced presentations of the vinegar + sucrose compound, and this treatment resulted in a weak aversion to sucrose relative to group PP (which did not receive vinegar-alone presentations), and a comparable aversion to that of the control groups (PU and UP).

The results of group PP-E in Experiment 1 are potentially interesting because studies on SOC have shown that extinguishing the nontarget CS (CS_1) does not always attenuate SOC. Specifically, Rizley and Rescorla (1972; see also Archer & Sjödén, 1982; Holland

& Rescorla, 1975a, 1975b; Nairne & Rescorla, 1981) found that extinguishing CS_1 prior to its pairings with the target CS_2 (i.e., $CS_1 + CS_2$ compound presentations) effectively attenuated SOC, whereas such attenuation of SOC was not observed when extinction of CS_1 took place after $CS_1 + CS_2$ trials. This finding, although not observed in all studies (see Rashotte, Griffin, & Sisk, 1977; but also see Nairne & Rescorla, 1981, for a possible reconciliation of these discrepant results), has been viewed as indicating that SOC is due to the formation, during $CS_1 + CS_2$ compound presentations, of an association between CS_2 and the conditioned response[2] elicited by the presence of CS_1. In this framework, CS_1 should maintain a strong excitatory status during its pairings with CS_2 so that a strong conditioned response is evoked. Thus, extinction of CS_1 prior to (but not after) $CS_1 + CS_2$ presentations can be expected to reduce SOC.

Although this differential effect of pre- and post-compound extinction of CS_1 cannot be taken as a certain method for assessing the occurrence of SOC, an experiment showing such a differential impact of this treatment would illuminate the process involved in the results of Experiment 1. There are two mechanisms by which SOC might occur for CS_2, and extinction of CS_1 can be used to experimentally evaluate these mechanisms. SOC may occur because a CS_2-response association is formed during compound exposure. Alternatively, a CS_1-CS_2 association may be formed during compound exposure. Extinction of CS_1 can impact each of these putative associations differently depending on whether it occurs before or after $CS_1 + CS_2$ presentations (Archer & Sjödén, 1982; Holland & Rescorla, 1975a, 1975b; Nairne & Rescorla, 1981; Rizley & Rescorla, 1972).

Regarding the CS_2-response mechanism, in group PP of Experiment 1, extinction of aversion to CS_2 (sucrose) could have been opposed during extinction treatment by the strengthening of the sucrose-response association. Specifically, the presence of CS_1 (vinegar), would elicit a conditioned response, which would become associated with CS_2 (sucrose) during the $CS_1 + CS_2$ (vinegar + sucrose) compound presentations. In this case, as in the studies mentioned above, extinguishing the aversion to vinegar could be expected to reduce protection from extinction of sucrose only if vinegar-alone trials were given prior to the vinegar + sucrose presentations.

Alternatively, the results of group PP in Experiment 1 could be viewed as due to the formation of a within-compound association between CS_1 and CS_2 (vinegar and sucrose, respectively) during the compound extinction trials. In this case, vinegar + sucrose compound presentations would result in the formation of a bidirectional association between both CSs, which would allow the subsequent presentation of one of these CSs to retrieve the representation of the other, absent CS. In this framework, the aversion to sucrose could extinguish uneventfully during the nonreinforced vinegar + sucrose compound presentations. However, when sucrose is presented at test, it would retrieve, not only the US representation (although perhaps weakly, due to extinction treatment), but also the representation of vinegar which, in turn, would also weakly retrieve the US representation. As a consequence of the US representation being (weakly) retrieved by both CSs, a stronger aversion could be expected to occur at test of sucrose (or vinegar) in group PP. From this viewpoint, extinguishing the aversion to vinegar should have a comparable effect

[2] Alternatively, an association could be formed between CS_2 and a static representation of the US, which is elicited by CS_1 during $CS_1 + CS_2$ trials. A static US representation refers to a US representation that is relatively unaffected by subsequent direct manipulations on the value of the US. This possibility, first proposed by Rescorla (1980), received support in a recent study by Winterbauer and Balleine (2005).

regardless of whether vinegar-alone trials are given either before or after vinegar + sucrose presentations (assuming the formation of the within-compound association is not affected by this order). What matters from this perspective is that, at the time of testing, vinegar no longer can retrieve the US representation (see, e.g., Archer & Sjödén, 1982; Rescorla & Durlach, 1981; Rescorla & Freberg, 1978; Rizley & Rescorla, 1972). Moreover, from the within-compound perspective vinegar-alone presentations could be expected to be more effective in reducing the aversion to sucrose when given after presentations of the vinegar + sucrose compound. Specifically, although vinegar-alone presentations could result in similar extinction of the aversion to vinegar regardless of trial order, presenting vinegar without sucrose after the vinegar + sucrose compound trials could also extinguish the vinegar–sucrose within-compound association. Thus, during the sucrose test, not only would vinegar weakly retrieve the US representation, but also sucrose would weakly retrieve the vinegar representation.

In order to distinguish among these processes, Experiment 2 used a treatment similar to that of group PP-E in Experiment 1 involving vinegar + sucrose and vinegar-alone during the extinction phase. As can be seen in the design of Experiment 2 (see Table 2), all groups received identical conditioning treatments, consisting of a single pairing of each of flavors A and B (vinegar and coffee solutions, counterbalanced), as well as of sucrose, with the US. Group S received extinction treatment with sucrose-alone, whereas group NE received no presentation of sucrose during the extinction phase (an alternative taste, salt, was given instead). More importantly, groups A-AS, AS-A, and AS received the same number of nonreinforced presentations of flavor A in compound with sucrose. The critical difference among these groups was their experience with flavor A. Group A-AS received the flavor A-alone trials before A + sucrose trials. Conversely, group AS-A received the flavor A-alone trials after A + sucrose trials. Finally, group AS did not receive any additional treatment with flavor A. Rather, this group (similar to groups S and NE) received comparable treatment with the alternative flavor, B. If extinction of the aversion to sucrose is protected by flavor A maintaining a high excitatory status during the A + sucrose compound presentations (i.e., stimulus-response account), weaker aversion to sucrose should be observed in group A-AS relative to both groups AS-A and AS. By contrast, if extinction of the aversion to sucrose is protected by stronger retrieval at test of the US mediated by a sucrose-flavor A within-compound association, the aversion to sucrose at test should be weaker in both groups A-AS and AS-A than in group AS. Moreover, according to a within-compound view, it is also quite conceivable that the aversion to sucrose will be even weaker in group AS-A than in groups A-AS and AS because nonreinforced A-alone presentations can be also presumed to extinguish the within-compound (A-sucrose) association when given after A + sucrose presentations (group AS-A), but not before A + sucrose presentations (group A-AS).

Method

Subjects and apparatus

The subjects were 40 Wistar, naïve, young adult rats (20 males and 20 females), obtained from the breeding colony of the University of Seville. The rats were about 95-105 days old at the beginning of the experiment, and their body weights ranged from 323 to 452 g ($M = 396.35$, SEM = 7.60) for the males and from 179 to 262 g ($M = 233.25$, SEM = 4.48) for the females. The animals were housed and maintained as

Table 2
Design of Experiment 2

Group	Conditioning	Extinction			Test 1	Test 2
		Ext. Ph. 1	Ext. Ph. 2	Ext. Ph. 3		
A-AS	1 A→US / 1 B→US, 1 S→US	5 A	2 AS	---	1 S	1 A
AS-A	1 A→US / 1 B→US, 1 S→US	---	2 AS	5 A	1 S	1 A
AS	1 A→US / 1 B→US, 1 S→US	5 B ---	2 AS	--- 5 B	1 S	1 A
S	1 A→US / 1 B→US, 1 S→US	5 B ---	2 S	--- 5 B	1 S	1 A
NE	1 A→US / 1 B→US, 1 S→US	5 B ---	2 N	--- 5 B	1 S	1 A

Note. A and B, vinegar and coffee solutions, counterbalanced; S, sucrose solution; AS, simultaneous presentation of flavor A and sucrose; N, NaCl solution; US, LiCl. '→' 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. Split treatments in Extinction Phases 1 and 3 indicate half the subjects in the group received the treatment above the line and half the subjects in the group received the treatment below the line.

in Experiment 1. The apparatus was identical to that of Experiment 1, with the following exceptions. First, sucrose was obtained from a different provider (ICN Biomedicals Inc., Cleveland, Ohio, USA). Second, two additional solutions (a taste and a flavor) were used. The taste consisted of a 1% (w/v, 0.17*M*) NaCl (salt) solution (ICN Biochemicals Inc., Cleveland, Ohio, USA) and the flavor consisted of a 1% (w/v) decaffeinated coffee solution (Marcilla, Sara Lee Southern Europe, S. L., Barcelona, Spain). Vinegar and coffee solutions served as flavors A and B, counterbalanced within groups.

Procedure

Forty subjects were assigned to five experimental groups, matched for body weight ($n = 8$, each group containing 4 males and 4 females). Unless explicitly stated, all subjects were given a single 20-min experimental session per day, initiated between 14:00 h and 15:00 h. Also, unless explicitly stated all subjects received additional 10-min of access to water soon after the session. Consumption during each session was recorded.

Pretraining. On Days 1–4, water was presented in a 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 flavor A, whereas the other half of the subjects received a presentation of flavor B. On Day 7, the assignment of flavors A and B was reversed: the subjects that were given flavor A on Day 5 now received a presentation of flavor B, 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 sucrose solution. The presentations of the A, B, and sucrose 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 the home cage. Each conditioning day was followed by one recovery day (i.e., Days 6, 8, and 10), on which water was presented in a glass bottle while the subjects recovered from the impact of the LiCl injection.

Extinction. On Days 11–22, extinction treatment (i.e., presentations of flavored solutions followed by no LiCl injection) occurred. During Extinction Phase 1 (Days 11–15), group A-AS received presentations of flavor A, whereas group AS-A was given no solution. Also, half of the subjects in groups AS, S, and NE received presentations of flavor B, whereas the other half of the subjects received no solution. During Extinction Phase 2 (Days 16 and 17), groups A-AS, AS-A, and AS received presentations of the A + sucrose simultaneous compound solution. Groups S and NE were given presentations of the sucrose and salt solutions, respectively. During Extinction Phase 3 (Days 18–22), group AS-A received presentations of flavor A, whereas group A-AS was given no solution. Also, the subjects in groups AS, S, and NE that were given no solution during Extinction Phase 1 received presentations of flavor B, whereas those subjects that received presentations of flavor B during Extinction Phase 1 were now given no solution. Soon after the session, subjects that received a solution during the experimental session received additional 10-min access to water, whereas those subjects that were given no solution during the experimental session received 30-min access to water in order to equate the total exposure time to fluid.

Testing. On Days 23 and 24, all subjects were tested for consumption of the sucrose and A solutions, respectively.

Results

Conditioning

The results during the conditioning phase in Experiment 2 are depicted in the left panel of Fig. 3. Consumption of flavors A and B (Days 5 and 7) were similar, and slightly lower than consumption of sucrose (Day 9). A 5 (group) × 3 (solution: A vs. B vs. sucrose) ANOVA conducted on consumption showed a marginal effect of solution, $F(2, 70) = 3.07$, $p > .05$, and no main effect of group nor interaction, $ps > .38$. Despite the marginal effect of solution, pairwise comparisons were performed and showed that consumption of sucrose was higher than consumption of both flavors A and B, $Fs(1, 35) > 4.48$, $ps < .05$, which did not differ, $p > .98$.

Extinction

The middle and right panels of Fig. 3 depict the consumption during the extinction phases. Consumption values depicted in the middle panel are those from the elemental extinction treatment of Phases 1 and 3, which were pooled in our subsequent analyses. Pooling these data was possible because the consumption of flavor B was comparable in groups AS, S, and NE during Phases 1 and 3 (Days 11–15 and 18–22), as shown by a 3 (group) × 2 (extinction phase) × 5 (day) ANOVA, which showed no main effect of extinction phase, $p > .71$, no Group × Extinction Phase interaction, $p > .63$, a marginal Extinction Phase × Day interaction, $p > .07$, and no three-way interaction, $p > .53$. (Also, presenting flavor B in either Phase 1 or Phase 3 did not affect consumption of sucrose and flavor A at test, as shown by separate 3 (group) × 2 (extinction phase) ANOVAs, which yielded no main effect of extinction phase nor an interaction, $ps > .36$.)

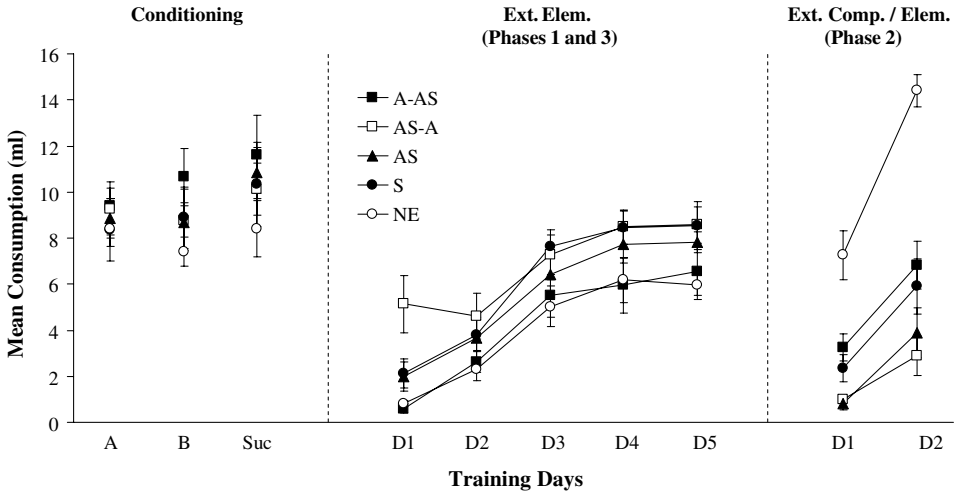

Fig. 3. Experiment 2: mean consumption during conditioning (left panel) and extinction (middle and right panels). Error bars depict standard error of the mean.

A 5 (group) × 5 (day) ANOVA on consumption during elemental extinction (Phases 1 and 3, Days 11–15 and 18–22) yielded main effects of group, $F(4, 35) = 3.45$, $p < .05$, and day, $F(4, 140) = 91.00$, $p < .01$, but no interaction, $p > .51$. Hence, all groups increased their consumption over the five days of extinction. Pairwise comparisons showed that group AS-A consumed flavor A more than group A-AS, $F(1, 35) = 8.07$, $p < .01$, a difference that can be attributed to group AS-A having received two exposures to flavor A in compound with sucrose prior to the first A-alone presentation. Also group AS-A consumed flavor A in an amount larger than the consumption of flavor B in group NE, $F(1, 35) = 9.32$, $p < .01$. Group S also consumed flavor B more than group NE did, $F(1, 35) = 5.16$, $p < .05$, and in an amount larger than that of consumption of flavor A in group A-AS, $F(1, 35) = 4.24$, $p < .05$. The rest of the differences were not significant, $ps > .11$.

During Phase 2 (Days 16 and 17), groups A-AS, AS-A, and AS received extinction treatment with the A + sucrose compound solution, whereas groups S and NE were given nonreinforced presentations of the sucrose-alone and salt-alone solutions. A 5 (group) × 2 (day) ANOVA on consumption during Phase 2 revealed main effects of both group, $F(4, 35) = 27.74$, $p < .01$, and day, $F(1, 35) = 85.31$, $p < .01$, as well as a group × day interaction, $F(4, 35) = 4.40$, $p < .05$. Pairwise comparisons showed that consumption of salt in group NE was higher than consumption of flavors received by the other groups on both days, $Fs(1, 35) > 21.61$, $ps < .01$. On both days of Extinction Phase 2, consumption of the A + sucrose compound solution in group A-AS was higher than in groups AS-A and AS, $Fs(1, 35) = 4.36$, $ps < .05$, and comparable to consumption of sucrose-alone in group S, $ps > .30$. Consumption of A + sucrose did not differ between groups AS-A and AS on either day, $ps > .48$, a result that was expected because these groups received identical treatments until this point. On the first day of Extinction Phase 2 (Day 16), consumption of sucrose in group S did not differ from consumption of the A + sucrose compound in groups AS-A and AS, $ps > .08$. On the second day of Extinction Phase 2 (Day 17), consumption of sucrose for group S did not differ from consumption of the A + sucrose

compound in group AS, $p > .16$, but it was higher than consumption of the A + sucrose compound by group AS-A, $F(1, 35) = 4.58$, $p < .05$.

Test

The results of the test of sucrose and flavor A are depicted in Fig. 4. The critical results in this experiment are those of the sucrose test. A quick inspection of the figure reveals that the aversion to sucrose was effectively extinguished in group S relative to group NE. Of more importance, although consumption of sucrose in groups A-AS, AS-A, and AS were appreciably lower than in group S, group A-AS consumed more sucrose than both groups AS-A and AS. To assess these impressions, a one-way ANOVA was conducted, which yielded a significant difference among groups, $F(4, 35) = 7.81$, $p < .01$. Pairwise comparisons showed that group S consumed more sucrose than group NE, $F(1, 35) = 16.59$, $p < .01$. More importantly, these comparisons showed higher consumption of sucrose in group A-AS than in both groups AS-A and AS, Fs$(1, 35) > 4.97$, ps $< .05$, and that groups AS-A and AS did not differ, $p > .80$. Group A-AS consumed less sucrose than group S, $F(1, 35) = 4.84$, $p < .05$, and marginally more than group NE, $F(1, 35) = 3.50$, $p > .06$. By contrast, groups AS-A and AS consumed less sucrose than group S, Fs$(1, 35) > 19.64$, $p < .01$, and in amounts comparable to that of group NE, ps $> .55$. In sum, the results of the sucrose test show that extinguishing flavor A (CS_1) facilitated extinction of sucrose (CS_2) only when A-alone trials preceded A + sucrose presentations. These results are only compatible with the aforementioned stimulus-response view of SOC, while posing a problem for the within-compound association account of SOC.

Regarding the results of test of flavor A, it is apparent from Fig. 4 that groups A-AS, AS-A, and AS did not differ, and that consumption of flavor A in these groups was higher than in groups S and NE which, in turn, did not appreciably differ. All these impressions were confirmed by a one-way ANOVA, which showed that groups differed in their consumption of flavor A, $F(4, 35) = 5.64$, $p < .01$. Pairwise comparisons showed comparable consumption in groups A-AS, AS-A, and AS, ps $> .26$, as well as similar consumption in groups S and NE, $p > .64$. Groups S and NE did not drink flavor A as much as groups

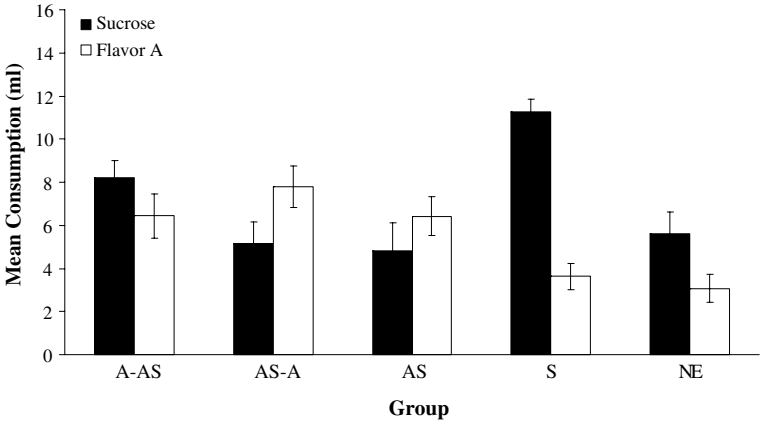

Fig. 4. Experiment 4: mean consumption at test of sucrose and flavor A (vinegar or coffee). Error bars depict standard error of the mean.

A-AS, AS-A, and AS, $Fs(1, 35) > 5.33$, $p < .05$. Therefore, it seems that, in the present experiment, just two extinction trials of the A + sucrose compound (group AS) were enough to produce extinction of the aversion to flavor A which was as strong as that of the groups that received five additional A-alone trials (groups A-AS and AS-A). The observation that, in Experiment 2, flavor A was not protected from extinction in group AS seems to contradict the results of group PP in Experiment 1, in which both flavors exposed in a nonreinforced compound (i.e., sucrose and vinegar) were protected from extinction. An inspection of the consumption on the first test trial (i.e., sucrose in both experiments) and on the second test trial (vinegar and flavor A in Experiments 1 and 2, respectively) shows a nonsignificant tendency to consume more on the second test trial than on the first test trial in both experiments. In Experiment 2, this trial order effect might have been facilitated by the longer interval between conditioning treatment and test (ten additional days, five in each of Extinction Phases 1 and 3), which could have produced some forgetting of the conditioning experience.

In the discussion of the results of Experiment 1, it was pointed out that the differential consumption of sucrose at test could be explained merely on the basis of the different consumption of sucrose during extinction treatment. That is, in that experiment the presence or absence of vinegar during nonreinforced presentations of sucrose could have modulated the amount of sucrose that was consumed, such that greater consumption resulted in greater extinction. The results of Experiment 2 cannot be fully explained by such a simple process. In this experiment, groups A-AS and S drank the A + sucrose and sucrose-alone solutions, respectively, in comparable amounts during Extinction Phase 2 (if anything, a nonsignificant tendency was found towards less consumption of sucrose-alone for group S than of A + sucrose for group A-AS). However, group S was found to drink significantly more sucrose at test than group A-AS. Such a difference could not be expected from an account relying exclusively on differential consumption of sucrose during extinction due to the presence or absence of flavor A.

General discussion

The experiments in this report found evidence that second-order conditioning (SOC), which is usually observed in experiments using an initially neutral target stimulus, can be also observed using a target stimulus that had previously been paired with the US. In experiments using a typical SOC treatment (i.e., $CS_1 \rightarrow US$ trials followed by $CS_1 + CS_2$ trials), SOC is assessed by the development of a conditioned response to CS_2 despite this CS never having been directly paired with the US. In contrast, in the treatment used in the present experiments (i.e., $CS_1 \rightarrow US$ and $CS_2 \rightarrow US$ trials followed by $CS_1 + CS_2$ trials), SOC was indicated by attenuated extinction of the conditioned response elicited by CS_2. Experiment 1 showed that such attenuation of extinction of responding elicited by CS_2 (i.e., sucrose) critically depended on CS_1 (i.e., vinegar) having been previously paired with the US as well as CS_2 having received extinction treatment in compound with CS_1. Moreover, in Experiment 1 aversive responding elicited at test by CS_2 in the condition given $CS_1 \rightarrow US$ and $CS_1 + CS_2$ trials (i.e., group PP) was comparable to that of a condition that received no extinction treatment with CS_2 (i.e., group NE), a result that indicates complete prevention of extinction in group PP. Thus, Experiment 1 showed that nonreinforced presentations of two CSs (i.e., $CS_1 + CS_2$ trials) can result in CS_2 being fully protected from extinction. These results cannot be accounted for by associative models of

learning (e.g., Rescorla & Wagner, 1972), according to which joint nonreinforced presentations of two CSs should result in enhanced extinction (Rescorla, 2000) instead of the presently observed protection from extinction. These models postulate that protection from extinction can occur when the target CS receives extinction treatment in compound with a Pavlovian conditioned inhibitor (e.g., Chorazyna, 1962; Lovibond, Davis, & O' Flaherty, 2000; Rescorla, 2003; Soltysik, Wolfe, Nicholas, Wilson, & Garcia-Sanchez, 1983; for related results also see Calton, Mitchell, & Schachtman, 1996), instead of a conditioned excitor, as shown in the present experiments.

Interestingly, interspersing nonreinforced presentations of CS_1 with nonreinforced presentations of $CS_1 + CS_2$ in Experiment 1 resulted in moderate extinction of CS_2. Experiment 2 systematically manipulated the order of presentation of the CS_1-alone and $CS_1 + CS_2$ trials, in an attempt to ascertain whether trial order (i.e., extinguishing CS_1 before or after extinction of the $CS_1 + CS_2$ compound) affected responding to CS_2 at test. This question was important because the view of SOC based on a stimulus-response association and the view based on SOC involving within-compound associations made different predictions regarding the outcome of this manipulation of trial order (see the introduction of Experiment 2 for a detailed explanation of these accounts). The results of Experiment 2 showed that extinguishing CS_1 after extinction treatment with the $CS_1 + CS_2$ compound had virtually no effect on extinction to CS_2 relative to a condition given no extinction treatment with CS_1. However, extinguishing CS_1 prior to extinction treatment with the $CS_1 + CS_2$ compound proved effective in releasing CS_2 from the impact of CS_1 mediation (i.e., aversive responding to CS_2 was better extinguished in this condition), a result that can only be accounted for by the stimulus-response account (for similar results, see Archer & Sjödén, 1982; Holland & Rescorla, 1975a, 1975b; Nairne & Rescorla, 1981; Rizley & Rescorla, 1972).

Alternative explanations of the present results can be ruled out. For example, presenting CS_2 in a nonreinforced compound with CS_1 could result in attenuated extinction of CS_2 merely by means of generalization decrement. This possibility, anticipated by configural models (e.g., Pearce, 1987), would imply that the strong aversion observed to CS_2 after our critical treatment would be due to the $CS_1 + CS_2$ compound only partially resembling CS_2, so that extinction of the compound would, at best, partially transfer to its elements, CS_1 and CS_2. However, this generalization decrement view fails to explain why, in Experiment 1, the aversion elicited by the $CS_1 + CS_2$ compound during extinction treatment was weaker in group UP than in both groups PP and PP-E. If the $CS_1 + CS_2$ compound was perceived as different from its elements (CS_1 and CS_2), then these three groups should have evidenced comparable aversion to the $CS_1 + CS_2$ compound regardless of their previous treatment with the elemental CSs. Moreover, in Experiment 2, CS_1-alone trials only affected extinction of CS_2 when given prior to $CS_1 + CS_2$ trials. Even if it was assumed that extinguishing CS_1 prior to $CS_1 + CS_2$ trials promoted the subsequent elemental processing of the compound, it would be reasonable to ask if such elemental processing could have been already warranted by the initial elemental conditioning of each CS. In a similar vein, it could be proposed that the nonreinforced compound presentation of two CSs resulted in protection from extinction due to each CS acting as an extinction context for the other. In this case, after $CS_1 + CS_2$ compound trials, separate presentations of CS_1 and CS_2 could result in renewal of the aversive response because the CSs are presented out of their corresponding extinction context (e.g., Bouton & Bolles, 1979). However, as was the case with the explanation in terms of a generalization decrement, this view fails to explain the impact

of the prior associative history of CS_1 (i.e., paired or unpaired with the US [Experiment 1], extinguished or not extinguished [Experiment 2]) on aversive responding elicited by CS_2 at test. That is, if the only role played by CS_1 during extinction in compound with CS_2 consisted of providing an extinction context for CS_2 (i.e., so that the memory of extinction of CS_2 would depend upon the presence of CS_1 for its retrieval), the associative history of CS_1 should have a negligible influence on its ability to modulate the response potential of CS_2.

As previously discussed, another alternative explanation concerns the possibility that consumption of sucrose during extinction treatment dictates consumption at test, in which case the only role played by CS_1 (i.e., vinegar and Flavor A, in Experiments 1 and 2, respectively) would be determining the animals' amount of exposure to CS_2 (i.e., sucrose) during the $CS_1 + CS_2$ presentations. This possibility is due to the intrinsic properties of the preparation used in the present experiments, namely, the conditioned taste aversion preparation, in which the degree to which animals consume a CS (taste or flavor) is not merely an index of the underlying aversion, but also a strong determinant of the animals' exposure to the CS itself. In our experiments, presenting two CSs in a simultaneous compound was expected to produce an overall stronger aversion relative to each of the elements of the compound (i.e., summation) which, in turn, was expected to result in a smaller consumption of the compound and, therefore, of each of its elements (i.e., protection from extinction). Thus, it is possible that presenting CS_1 attenuated extinction of CS_2 by merely reducing the intake of CS_2 during extinction treatment (i.e., the smaller the consumption of CS_2, the weaker the extinction of this CS). As previously noted, the difference in the consumption at test of CS_2 (sucrose) found between group A-AS and group S of Experiment 2 cannot be accounted on the basis of a differential consumption during extinction treatment. There are other reasons to reject this simple process as an explanation of our results. Pineño (2006) recently conducted an experiment showing evidence of both positive and negative mediation (i.e., effects in which the response potential of CS_2 correlates either positively or negatively with the response potential of CS_1), in which he used the same preparation and identical solutions for the CSs as in the present experiments. The fact that negative mediation was found between two CSs presented in compound during extinction is of special relevance here because, in negative mediation effects, the consumption observed at test does not positively correlate with the consumption observed during extinction. If anything, a negative correlation is usually observed (e.g., less consumption in extinction results in more consumption at test). In any case, replicating the present findings in a preparation in which prior experience with the CSs does not affect the amount of exposure to CS_2 during extinction would be desirable.

The fact that the present results are at odds with previous reports in the literature (e.g., Rescorla, 2000) might be interpreted as due to an idiosyncrasy of taste aversion conditioning. However, there are at least two previous reports indicating that compound extinction with two CSs can result in protection from extinction, rather than enhanced extinction. Lovibond et al. (2000) found, using a skin conductance response in a fear conditioning preparation with humans, that extinction of a CS was protected because it received nonreinforced exposure conjointly with either a Pavlovian conditioned inhibitor or an excitor. Because these authors expected the concurrent presentation of an excitor to enhance, rather than attenuate, extinction of the target CS, the implications of this finding were not considered in detail with respect to the present concerns. Rather, Lovibond et al. suggested that, because CSs with different associative histories (an inhibitor and an excitor)

produced similar effects on extinction of the target CS, protection from extinction could be better explained by generalization decrement, that is, as due to the elemental presentation of the target CS (during conditioning and testing) partially resembling the presentation of the target CS in compound with another CS (during extinction treatment). Another study, this one using an autoshaping preparation with pigeons, also reported protection from extinction by presenting two excitatory CSs in compound during nonreinforcement (Pearce & Wilson, 1991). Interestingly, this study directly addressed the possibility that the compound CS could be processed as a configural stimulus that resembles only partially the CSs forming it (see Pearce, 1987). Thus, similar to Lovibond et al., Pearce and Wilson explained this effect as an instance of generalization decrement. Importantly, these authors did not contemplate the possibility that SOC could play a role in their observation of protection from extinction.

The present results also have important implications for clinical/applied practice. According to the prevailing view of current models of learning (e.g., Rescorla & Wagner, 1972), presenting two CSs in a nonreinforced compound should result in enhanced extinction of each CS. Based on this expectation of negative mediation, one could easily conclude that compound exposures to, for example, two fear-eliciting CSs would be more effective than an identical number of separate exposures to each CS. These results challenge this expectation, suggesting that such therapeutical treatment could yield no benefit at all: the joint nonreinforced presentation of two CSs could result in protection from extinction, instead of enhanced extinction. Some caution is recommended in clinical practice until the mechanisms involved in the occurrence of either positive or negative mediation in treatments with compound stimuli are better understood (e.g., Pineño, 2006; Stout et al., 2004; Yin, Barnet, & Miller, 1994).

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