

An examination of the effectiveness of inflation and deflation treatments in detecting within-compound learning of a taste aversion

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Abstract

Two conditioned taste aversion experiments with rats assessed the relative effectiveness in providing evidence of within-compound learning of different procedures that involve the initial compound presentation of two stimuli, A and X, with the unconditioned stimulus (i.e., AX+). In Experiment 1, following a single AX+ trial, groups A+ and B+ received an additional conditioning trial (i.e., inflation treatment) with A and B, respectively, whereas group A– received an extinction trial (i.e., deflation treatment) with A. The results showed a reduction in the aversion elicited by the target stimulus, X, in group A– relative to both groups A+ and B+, which did not differ. Experiment 2 further investigated the failure of group A+ to increase the aversion to X relative to control group B+ by pairing A or B with either the same unconditioned stimulus that was previously paired with AX (groups A+ and B+) or with a stronger unconditioned stimulus (groups A* and B*). The results showed increased aversion to X in group A* relative to group B*, but not in group A+ relative to group B+. These results are interpreted as indicative of extinction of the within-compound association during the treatment with A, which could likely impair the detection of within-compound learning following an inflation, but not a deflation treatment.

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One of the most fruitful areas of research in associative learning concerns the change in the associative status of a stimulus, X, that is achieved by performing direct manipulations on the associative status of another stimulus, A, which was previously presented in compound with X. In within-compound learning effects, following presentations of the AX compound, pairing A with the unconditioned stimulus (US) (i.e., an inflation treatment), results in the development or increase of conditioned responding to X, an effect that is represented by sensory preconditioning (e.g., Archer and Sjöden, 1982; Brodgen, 1939; Lavin, 1976; Rescorla and Cunningham, 1978; Rizley and Rescorla, 1972) and by representation-mediated acquisition (e.g., Holland, 1981). By contrast, presentations of A without the US following conditioning treatment with this stimulus (i.e., a deflation treatment) weakens conditioned responding to X, an effect that is represented by extinction of sensory preconditioning (e.g., Rizley and Rescorla, 1972), extinction of

taste-mediated odor potentiation (e.g., Durlach and Rescorla, 1980), and representation-mediated extinction (e.g., Holland and Forbes, 1982).

The present article aims to contrast the relative effectiveness of inflation and deflation treatments in yielding evidence of within-compound learning. Comparable inflation and deflation treatments involve initial pairings of the AX compound with the US (i.e., AX+ trials), followed by additional presentations of A, either with the US (i.e., A+ trials, inflation treatment) or alone (i.e., A– trials, deflation treatment). The deflation treatment (i.e., AX+/A– procedure) has been consistently found to yield evidence of within-compound learning (e.g., Durlach and Rescorla, 1980; Nakajima and Kawai, 1997; Schnelker and Batsell, 2006; Trost and Batsell, 2004; Westbrook et al., 1983). In contrast, the inflation treatment (i.e., AX+/A+ procedure) seems to be poorly effective in the detection of within-compound learning. Studies using this procedure in a conditioned taste aversion paradigm have found mixed results. Some researchers successfully found increased aversion to X in the AX+/A+ procedure (e.g., Batsell et al., 2003; Schnelker and Batsell, 2006), whereas others failed to observe a change in the aversive response elicited by X after such treatment (e.g., Nakajima and Kawai, 1997; Westbrook et

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al., 1983). Thus, it seems that within-compound learning can generally be better detected by a deflation treatment than by an inflation treatment. This impression is supported by the study of Nakajima and Kawai (1997), who directly contrasted both AX+/A+ and AX+/A– procedures in a single experiment and found that, following an AX+ trial, an additional A+ did not appreciably increase aversive responding to X, whereas a single additional A– trial was enough to decrease the aversion to X.

An explanation for the superiority of the deflation treatment over the inflation treatment in the detection of within-compound learning regards the extinction of the A–X within-compound association. Although treatments can be performed with the specific aim of weakening the within-compound association (e.g., Rescorla and Freberg, 1978), such extinction of the within-compound association can also be a collateral, unintended consequence of certain treatments. This is the case of inflation and deflation treatments with A, which necessarily imply the presentation of A in the absence of X and, thus, might weaken the A–X association. According to this view, although the A+ trial in an AX+/A+ procedure could increase the aversion elicited by A, its effectiveness could be completely counteracted by the extinction of the A–X association. That is, although A could become more aversive during the A+ trial, the presentation of X at test would be less able to activate the memory of A due to the A–X association having undergone extinction. As a consequence, the aversion produced by A would have less of an impact on the aversive response elicited by X. Furthermore, this viewpoint anticipates the relatively consistent success of the AX+/A– procedure in reducing aversive responding to X because, according to this view, the extinction of the A–X association should enhance, rather than counteract, the effectiveness of the deflation treatment. Specifically, after a deflation treatment, weak aversive responding to X should be expected not only because of the reduction in the aversion elicited by A, but also because of X's poor ability to retrieve the memory of A (i.e., due to a weakened A–X association). From the previous discussion it also follows that an inflation treatment involving the pairing of A with an intense US (i.e., A* trial, AX+/A* procedure) could succeed in detecting within-compound learning, provided that the inflation of A was not completely counteracted by the extinction of the A–X association.

The experiments in this report sought to assess the effectiveness of the AX+/A+, AX+/A–, and AX+/A* procedures in yielding evidence of within-compound learning. Based on the previous discussion, it was anticipated that the AX+/A+ procedure would fail to detect evidence of within-compound learning, whereas the AX+/A– procedure and, perhaps, the AX+/A* procedure could prove effective in yielding such evidence.

1. Experiment 1

Experiment 1 compared AX+/A+ and AX+/A– procedures in their potential to detect within-compound learning, relative to a control AX+/B+ procedure. Based on previous reports (e.g., Nakajima and Kawai, 1997), it was expected that the AX+/A– procedure would prove effective in reducing the aver-

sive response elicited by X, whereas the AX+/A+ procedure was expected to produce, if anything, a weak increase in the aversion to X.

2. Method

2.1. Subjects

The subjects were 24 Wistar, naïve, young adult rats (12 males and 12 females), obtained from the breeding colony at the University of Seville. The rats were approximately 95 days old at the beginning of the experiment, and their body weights ranged from 288 to 349 g ($M = 322.00$, $SEM = 5.57$) for the males and from 201 to 257 g ($M = 223.91$, $SEM = 5.82$) for the females. The animals were housed individually in 36 cm × 20 cm × 14 cm clear plastic cages in a colony room on a 12:12-h light:dark cycle (lights on at 07:00), with all 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 20 min/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.

2.2. 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 each liquid presentation.

Three distinct stimuli, two flavors and one taste, were employed in this study. The flavors were a 1% (v/v) apple cider vinegar solution (Prima, Spain) and a 1% (w/v) decaffeinated coffee solution (Marcilla, Sara Lee Southern Europe, S.L., Barcelona, Spain), which served as flavors A and B, counterbalanced. The taste was a 1% (w/v, 0.03 M) sucrose solution (Fluka Chemie GmbH, Buchs, Switzerland), which served as taste X. Solutions were made using tap water. The US was a 4 ml/kg of body weight intraperitoneal (i.p.) injection of 0.15 M lithium chloride¹ (LiCl, Sigma–Aldrich Chemie GmbH, Steinheim, Germany), which was administered using a 5-ml syringe with a 0.6 mm × 25 mm needle.

2.3. Procedure

The design of Experiment 1 is summarized in Table 1. Twenty-four subjects were assigned to one of three experimental groups, matched for body weight ($n = 8$, each group containing

¹ This volume for the LiCl injection was borrowed from a parametrical study by Nachman and Ashe (1973), which showed that a 4 ml/kg of body weight i.p. injection of 0.15 M LiCl produced incomplete aversion to a 15% sucrose solution.

Table 1
Design of Experiment 1

Group	Phase 1	Phase 2	Test 1	Test 2
A+	1 AX+	1 A+	1 X	2 A
A–	1 AX+	1 A–	1 X	2 A
B+	1 AX+	1 B+	1 X	2 A

Note: A and B = vinegar and coffee solutions, counterbalanced; X = sucrose solution; AX = simultaneous presentation of A and X; + = 4 ml/kg of body weight LiCl i.p. injection; – = 4 ml/kg of body weight saline i.p. injection. The numbers denote the number of presentations of each trial type in each phase. See text for further details.

four males and four females). All subjects were given a single 10-min experimental session per day, initiated at 14:00 h. Also, all subjects received additional 10-min access to water soon after the session. Consumption during each session was recorded.

2.3.1. Pretraining

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

2.3.2. Phase 1

On Day 5 all groups received a presentation of the AX simultaneous compound solution, 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 tap water was presented with the glass bottle, while allowing the subjects to recover from the impact of the LiCl injection.

2.3.3. Phase 2

On Day 7, groups A+ and A– received a presentation of flavor A, whereas group B+ was given a presentation of an alternative flavor, B. These fluid presentations were followed immediately by an i.p. injection of either LiCl for groups A+ and B+, or saline for group A–, after which the animals were immediately returned to the home cage. Day 8 consisted of a recovery day, on which tap water was presented with the glass bottle, while allowing the subjects in groups A+ and B+ to recover from the impact of the LiCl injection.

2.3.4. Testing

All subjects were tested for consumption of taste X (Day 9) and flavor A (Days 10 and 11). An alpha level of $p < .05$ was adopted for all statistical analyses and pairwise comparisons were performed using independent t -tests.

3. Results

The mean consumption of the AX compound solution during Phase 1 was 8.35 ml (SEM = 1.06), 7.63 ml (SEM = 1.21), and 8.18 ml (SEM = 0.84) for groups A+, A–, and B+, respectively. A one-way analysis of variance (ANOVA) detected no significant difference among groups, $p > .88$. In Phase 2, group B+ consumed a mean of 8.28 ml (SEM = 0.46) of flavor B, whereas

groups A+ and A– consumed a mean of 4.40 (SEM = 0.59) and 4.23 (SEM = 0.75), respectively, of flavor A. A one-way ANOVA found significant differences among groups, $F(2, 21) = 13.67$, $p < .01$. The consumption of flavor A was comparable in groups A+ and A–, $p > .86$, and both groups consumed less of this flavor than group B+ consumed of flavor B, $t_s(14) > 4.53$, $p < .01$. An additional 3 (group) \times 2 (day) ANOVA showed a main effect of day, $F(1, 21) = 19.22$, $p < .01$, and a significant Group \times Day interaction, $F(2, 21) = 5.29$, $p < .05$. The main effect of group was marginally significant, $p > .07$. Overall, these analyses indicate that: (1) all groups consumed comparable amounts of the AX compound solution (a condition that was necessary in order to compare consumption at test of X among groups), (2) the AX+ pairing of Phase 1 was effective in conditioning aversion to flavor A and, (3) conditioning of A was not appreciably generalized to flavor B.

The results of test of X in Experiment 1 are depicted in the top panel of Fig. 1. As can be appreciated from this panel, consumption of taste X was higher in group A– than in both groups A+ and B+, which did not appreciably differ. These impressions were confirmed by a one-way ANOVA, which showed significant differences, $F(2, 21) = 5.65$, $p < .05$. Planned comparisons revealed that the consumption of taste X was higher in group A– than in both group A+, $t(14) = 3.39$, $p < .01$, and group B+, $t(14) = 2.55$, $p < .05$. Also, groups A+ and B+ did not differ in their consumption of taste X, $p > .57$. Hence, extinction of flavor A in Phase 2 (group A–) proved effective in reducing the aversion elicited by taste X, but the additional conditioning trial with flavor A (group A+) did not increase the aversion to X, relative to a group given an additional conditioning trial with an alternative flavor (group B+). These results confirmed our expectations, according to which within-compound learning could be

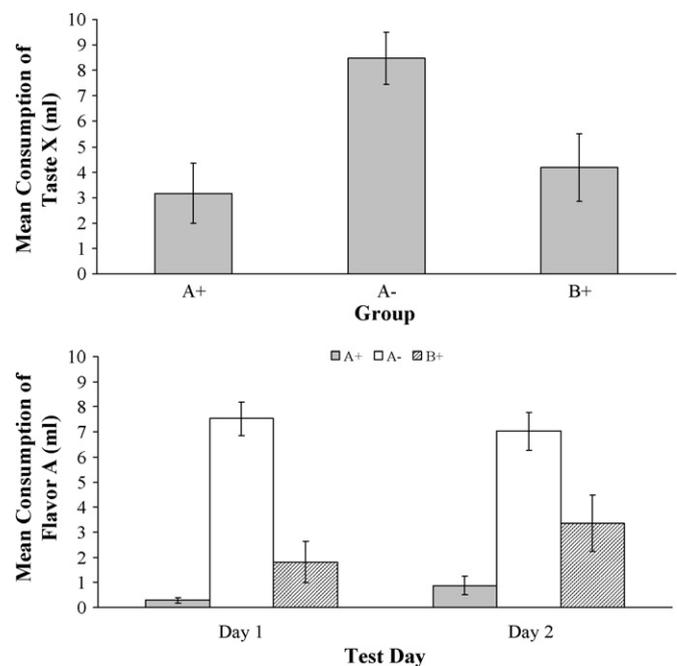


Fig. 1. Mean consumption during test of X (top panel) and test of A (bottom panel) in Experiment 1. Error bars depict standard error of the means.

detected by the AX+/A– procedure, but not by the AX+/A+ procedure.

The results of the test of A in Experiment 1 are depicted in the bottom panel of Fig. 1. As can be seen in this panel, on both test days with flavor A, group A– consumed the highest amounts, followed by group B+, and finally by group A+. A 3 (group) \times 2 (test day) ANOVA yielded only a main effect of group, $F(2, 21) = 29.57$, $p < .01$. Neither the main effect of day nor the Group \times Day interaction were significant, $ps > .14$. Because of the lack of a main effect of day and of a significant interaction, planned comparisons were performed among groups, pooling the consumption scores from both test days. These comparisons showed that the consumption of flavor A was higher in group A– than in both group A+, $t(14) = 13.16$, $p < .01$, and group B+, $t(14) = 4.35$, $p < .01$. Also, group A+ consumed less of flavor A than group B+, although this latter difference fell short of significance, $p > .06$.

In sum, the nonreinforced presentation of flavor A (group A–) effectively extinguished the aversion to A relative to group B+, a result that is consistent with the weak aversion to X found in group A– relative to group B+. Importantly, further conditioning of flavor A (group A+), was also effective in increasing the aversion to A relative to group B+ (a result that must be considered with caution given the marginal significance of this difference). This later result is at odds with the failure to observe an increase in the aversive response elicited by X in group A+ relative to group B+, and indicates that this failure cannot be attributed to the A+ treatment being unable to further increase aversion to flavor A.

4. Experiment 2

Experiment 2 aimed to ascertain if the effectiveness of the AX+/A+ procedure could be enhanced by pairing A with a US that was stronger than that previously paired with AX. This treatment proved unsuccessful in a study by Westbrook et al. (1983, Experiment 5). These researchers found that an AX+/A+ procedure involving the presentation of a stronger US on the trial with A (i.e., AX+/A* procedure) was effective in increasing the aversive response elicited by X. However, a control AX+/B* treatment also produced increased aversion to X. Although Westbrook et al. interpreted this result as due to a strengthening of the US representation, it might also be possible that, as proposed by LoLordo and Droungas (1989), the tastes Westbrook et al. used for A and B (quinine and hydrochloric solutions) were perceived as highly similar, in which case the treatment with B could be strongly generalized to A, thereby affecting responding to X. From the rationale of LoLordo and Droungas, it follows that Westbrook et al.'s AX+/A* procedure might have yielded the expected results if A and B were correctly discriminated. In this experiment, we relied on the results of Experiment 1 (specifically, the consumption scores of flavors A and B during Phase 2), which indicated that the flavors we used for A and B (i.e., vinegar and coffee solutions) were readily discriminated by our animals, in order to ascertain if the AX+/A* procedure could yield evidence of within-compound learning.

Table 2
Design of Experiment 2

Group	Phase 1	Phase 2	Test 1	Test 2
A+	1 AX+	1 A+	1 X	2 A
B+	1 AX+	1 B+	1 X	2 A
A*	1 AX+	1 A*	1 X	2 A
B*	1 AX+	1 B*	1 X	2 A

Note: A and B = vinegar and coffee solutions, counterbalanced; X = sucrose solution; AX = simultaneous presentation of A and X; + = 4 ml/kg of body weight LiCl i.p. injection; * = 16 ml/kg of body weight LiCl i.p. injection. The numbers denote the number of presentations of each trial type in each phase. See text for further details.

5. Method

5.1. Subjects and apparatus

The subjects were 32 Wistar, naïve, young adult rats (16 males and 16 females), obtained from the breeding colony at the University of Seville. The rats were approximately 95 days old at the beginning of the experiment, and their body weights ranged from 268 to 375 g ($M = 329.81$, $SEM = 9.48$) for the males and from 185 to 249 g ($M = 221.62$, $SEM = 4.12$) for the females. The animals were housed and maintained as in Experiment 1. The apparatus was identical to that of Experiment 1, with the exception that, in Experiment 2, two different volumes (4 and 16 ml/kg of body weight)² were used for the i.p. injections of 0.15 M LiCl that provided the USs (see Procedure in Section 5.2 for details).

5.2. Procedure

The design of Experiment 2 is summarized in Table 2. Thirty-two subjects were assigned to one of four experimental groups, matched for body weight ($n = 8$, each group containing four males and four females). All subjects were given a single 10-min experimental session per day, initiated at 14:00 h. Also, all subjects received additional 10-min access to water soon after the session. Consumption during each session was recorded.

5.2.1. Pretraining

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

5.2.2. Phase 1

On Day 5 all groups received a presentation of the AX simultaneous compound solution, followed immediately by a 4 ml/kg of body weight 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 tap water was presented with the

² The 16 ml/kg of body weight i.p. injection of 0.15 M LiCl was borrowed from Nachman and Ashe (1973), because this dosage produced an aversion that was significantly stronger than that produced by the 4 ml/kg of body weight injection.

glass bottle, while allowing the subjects to recover from the impact of the LiCl injection.

5.2.3. Phase 2

On Day 7, groups A+ and A* received a presentation of flavor A, whereas groups B+ and B* were given a presentation of an alternative flavor, B. These fluid presentations were followed immediately by an i.p. injection of LiCl for all groups. For groups A+ and B+, the volume of the injection was 4 ml/kg of body weight (i.e., the US-intensity was the same as in Phase 1), whereas for groups A* and B*, the volume of the injection was 16 ml/kg of body weight (i.e., the US was stronger than in Phase 1). Day 8 consisted of a recovery day, on which tap water was presented with the glass bottle, while allowing the subjects to recover from the impact of the LiCl injection.

5.2.4. Testing

All subjects were tested for consumption of taste X (Day 9) and flavor A (Days 10 and 11).

6. Results

The mean consumption of the AX compound solution in Phase 1 was 7.78 ml (SEM=0.85), 7.41 ml (SEM=1.02), 7.28 ml (SEM=0.63), and 7.76 ml (SEM=0.67) for groups A+, B+, A*, and B*, respectively. A 2 (Phase 2 flavor: A vs. B) \times 2 (Phase 2 US: + vs. *) ANOVA yielded no main effect or interaction, $ps > .60$. In Phase 2, groups A+ and A* consumed less of flavor A than groups B+ and B* consumed of flavor B. The mean consumption during Phase 2 was 3.60 ml (SEM=0.45), 6.59 ml (SEM=0.70), 3.43 ml (SEM=0.44), and 7.84 ml (SEM=0.87) for groups A+, B+, A*, and B*, respectively. A 2 (Phase 2 flavor: A vs. B) \times 2 (Phase 2 US: + vs. *) ANOVA yielded only a main effect of Phase 2 flavor, $F(1, 28) = 32.87$, $p < .01$. Neither the main effect of Phase 2 US nor the interaction were significant, $ps > .28$. Finally, an additional 2 (Phase 2 flavor: A vs. B) \times 2 (Phase 2 US: + vs. *) \times 2 (day) ANOVA showed a main effect of Phase 2 flavor, $F(1, 28) = 12.68$, $p < .01$, a main effect of day, $F(1, 28) = 18.39$, $p < .01$, and a significant Phase 2 Flavor \times Day interaction, $F(1, 28) = 12.71$, $p < .01$. The main effect of Phase 2 US and the other interactions was not significant, all $ps > .29$. As in Experiment 1, these analyses indicate that: (1) all groups consumed comparable amounts of the AX compound solution, (2) the AX+ pairing of Phase 1 was effective in conditioning aversion to flavor A and, (3) conditioning of A was not appreciably generalized to flavor B.

The results of test of X in Experiment 2 are depicted in the top panel of Fig. 2. Inspection of this panel suggests that groups A+ and B+ consumed comparable amounts of taste X, whereas group A* appreciably consumed less of taste X than group B*. A 2 (Phase 2 flavor: A vs. B) \times 2 (Phase 2 US: + vs. *) ANOVA revealed a main effect of Phase 2 flavor, $F(1, 28) = 5.72$, $p < .05$, but no effect of Phase 2 US, $p > .70$. The Phase 2 Flavor \times Phase 2 US interaction fell short of significance, $p > .09$. The lack of a significant interaction might be due to groups A+ and B+ consuming taste X in an intermediate amount that did not significantly differ from the consumption of groups A* and B*. Despite

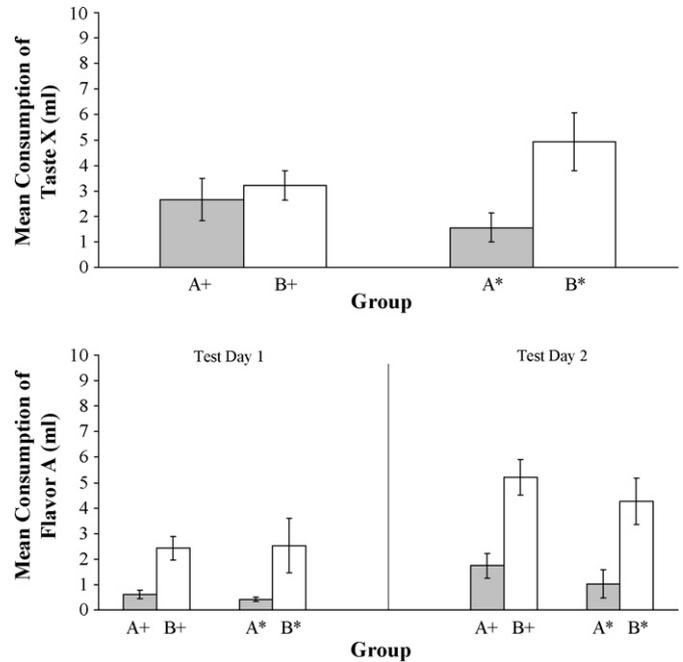


Fig. 2. Mean consumption during test of X (top panel) and test of A (bottom panel) in Experiment 2. Error bars depict standard error of the means.

the lack of a significant interaction (which calls for caution in the interpretation of the results of test of X in the present experiment), planned comparisons were conducted and showed that groups A+ and B+ consumed taste X in similar amounts, $p > .59$, and, more important, that group A* consumed less of taste X than group B*, $t(14) = 2.63$, $p < .05$. Therefore, these results replicate and extend the results of Experiment 1. As in Experiment 1, a treatment involving the pairing of AX and A with the same US (AX+/A+ procedure) failed to detect within-compound learning. However, pairing A with a substantially stronger US than that previously paired with AX (AX+/A* procedure) did yield evidence of within-compound learning.

The results of test of A in Experiment 2 are depicted in the bottom panel of Fig. 2. The most obvious result in this panel is the lower consumption of flavor A in groups A+ and A* relative to groups B+ and B*, respectively, on both test days. A 2 (Phase 2 flavor: A vs. B) \times 2 (Phase 2 US: + vs. *) \times 2 (test day) ANOVA confirmed this impression, by showing a main effect of Phase 2 flavor, $F(1, 28) = 27.37$, $p < .01$. This ANOVA also yielded a main effect of test day, $F(1, 28) = 17.15$, $p < .01$, and a marginally significant Phase 2 Flavor \times Test Day interaction, $p > .07$. The main effect of Phase 2 US and the rest of the interactions were not significant, all $ps > .30$. Due to this marginally significant Phase 2 Flavor \times Test Day interaction, separate analyses were performed on each test day. Two independent 2 (Phase 2 flavor: A vs. B) \times 2 (Phase 2 US: + vs. *) ANOVAs showed a main effect of Phase 2 flavor on both Test Day 1, $F(1, 28) = 11.33$, $p < .01$, and Test Day 2, $F(1, 28) = 24.56$, $p < .01$, but no main effect of Phase 2 US nor a Phase 2 Flavor \times Phase 2 US interaction on either day, all $ps > .23$. Consumption of flavor A was lower in group A+ than in group B+ on both test days, $t_s(14) > 3.73$, $ps < .01$. Although consumption of flavor A in group A* was

also lower than consumption of group B* on the second test day, $t(14) > 3.07$, $p < .01$, this difference was only marginally significant on the first test day, $p > .06$. Finally, the lack of a Phase 2 Flavor \times Phase 2 US interaction indicates that we failed to observe a lower consumption of flavor A in group A* relative to group A+, a difference that was expected given that A was paired with a stronger US in group A* than in group A+. This failure, however, might simply reflect a floor effect, given the low consumption of A in both groups A+ and A*.

In sum, as in Experiment 1, an additional pairing of A with the same US that was previously paired with the AX compound solution was effective in increasing the aversion elicited by A (group A+ vs. group B+). Although both groups A+ and A* were more reluctant to consume flavor A than groups B+ and B*, respectively, only the additional pairing of A with a stronger US modified the aversive response elicited by X (group A* vs. group B*).

7. General discussion

Experiment 1 showed, in accordance with previous reports (e.g., Nakajima and Kawai, 1997), that the AX+/A+ procedure is less effective than the AX+/A– procedure in obtaining results indicative of within-compound learning. In this experiment, a group given the AX+/A– treatment (group A–) showed weaker conditioned aversion to X than a control condition given an AX+/B+ treatment (group B+), whereas a condition that received the AX+/A+ treatment (group A+) did not show an increased aversion to X, relative to group B+. Experiment 2 further investigated this failure of the AX+/A+ procedure in yielding evidence of within-compound learning by manipulating the magnitude of the US paired with AX and A. As in Experiment 1, a group that received the same US in the AX+/A+ treatment (group A+) showed no evidence of increased aversion to X, relative to a group receiving the same US in the AX+/B+ treatment (group B+). However, within-compound learning was evident when the inflation treatment with A employed a stronger US than that previously used on the AX+ trial: a group given AX+/A* treatment (group A*) showed stronger conditioned aversion to X than a group given AX+/B* treatment (group B*). The AX+/A* procedure, which failed in prior studies (e.g., Westbrook et al., 1983, Experiment 5), was successful in Experiment 2 perhaps because of our having used highly discriminable flavors for A and B (see LoLordo and Droungas, 1989).

These results are consistent with the view, anticipated in the Introduction, that the additional (inflation or deflation) treatment with A might extinguish, at least partially, the A–X within-compound association. From this viewpoint, the inflation treatment in the AX+/A+ procedure necessarily implies a trade-off: because the A–X association undergoes extinction during the A+ trial, the increase in the aversion to A should have less of an influence on the aversive response elicited by X. In accord with this prediction, in Experiments 1 and 2 the AX+/A+ procedure was found to be poorly effective. The finding that the AX+/A* procedure (Experiment 2) yielded evidence of within-compound learning could also be explained from this viewpoint by assuming that the inflation of the aversion to A on

the A* trial could not be completely counteracted by the extinction of the A–X association. Furthermore, because this view anticipated that the extinction of the A–X within-compound association should have an additive impact on the effectiveness of the deflation treatment, it also predicted the success of the AX+/A– procedure (Experiment 1) in yielding evidence of within-compound learning.

Although speculative at this point, the present results are also consistent with an alternative view, according to which presenting the AX compound and A with different outcomes enhances the impact of the treatment with A on the associative status of X. A common feature in studies showing evidence of within-compound learning is that, typically, the AX compound is not paired with the US in experiments using an inflation treatment (i.e., AX–/A+ procedure), a feature that can be found in experiments on sensory preconditioning (e.g., Archer and Sjöden, 1982; Brodgen, 1939; Lavin, 1976; Rescorla and Cunningham, 1978; Rizley and Rescorla, 1972) and representation-mediated acquisition (e.g., Holland, 1981). Conversely, the AX compound is typically paired with the US in experiments using a deflation treatment (i.e., the AX+/A– procedure; see Durlach and Rescorla, 1980; Nakajima and Kawai, 1997; Schnelker and Batsell, 2006; Trost and Batsell, 2004; Westbrook et al., 1983). It is thus possible that the AX+/A– and AX+/A* procedures were effective in the present experiments because the animals were able to detect that something changed between the presentation of AX and the presentation of A, regardless of whether this change consisted of the absence of an expected US (AX+/A– procedure) or the presentation of an unexpectedly strong US (AX+/A* procedure). By contrast, the AX+/A+ procedure might have been ineffective because both AX and A were followed by the same outcome.

A change in the outcome following the presentation of AX and A could enhance the detection of within-compound learning if it was assumed that, in order for the inflation or deflation treatment with A to affect the associative status of X, A must undergo a minimum amount of associative change. In this case, both AX+/A– and AX+/A* procedures could presumably involve a large discrepancy between the outcome that was predicted to occur based on the presence of A and the outcome that actually occurred. By contrast, if A had already acquired a strong association with the US on the AX+ trial, an additional A+ trial in the AX+/A+ procedure could be expected to provide A with additional associative strength, an increase that might be nevertheless insufficient to affect aversive responding to X. This hypothetical process can be better explained by assuming the existence of a threshold for the variation in associative strength undergone by the present stimulus, A, to be able to affect the associative status of the absent stimulus, X. Only when the associative strength of A changes above such a putative threshold, the associative status of X is updated. An alternative explanation might involve the surprisingness of the outcome accompanying A. If we assumed that the animals might more actively rehearse the stimuli that were present (and absent) previously to the occurrence of an unexpected outcome, the impact of the treatment with A on responding elicited by X might be enhanced by the AX+/A– and AX+/A* procedures, but not by the AX+/A+ procedure.

Because the results of the present experiments do not selectively support any of the above discussed accounts, we must favor the most parsimonious explanation based on a balance between the extinction undergone by the A–X within-compound association and the impact of the (inflation or deflation) treatment with A on the conditioned response produced by X. Future research may determine the merit of the rather “less conventional” viewpoint based on the facilitative impact of a change in the outcome accompanying AX and A in the detection of within-compound learning.

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