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Habituation of unconditioned fear can be attenuated by the presence of a safe stimulus: Assessment using the neophobic response of the rat

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Abstract

Protection from extinction of conditioned fear has been demonstrated when a conditioned inhibitor of fear is presented during extinction treatment. The present study assessed if similar results could be obtained during the analogous habituation of unconditioned fear. The neophobic response typically elicited by the presentation of a novel flavor was used as a model of unconditioned fear. Consumption by rats was used to ascertain the impact of nonreinforced exposure to a novel flavor either alone, in compound with another novel flavor, or in compound with a safe flavor (i.e., a flavor previously trained as a conditioned inhibitor for illness). The presentation of the novel flavor alone in the absence of illness reduced neophobia. However, exposure to the novel flavor in compound with the safe flavor reduced habituation of neophobia. This effect was not observed when the novel flavor was exposed in compound with another novel flavor. These results suggest that removing safe stimuli from the therapeutical environment might improve the effectiveness of exposure therapy in the treatment of unconditioned fear.

Keywords: Neophobia; Learned safety; Conditioned taste aversion

1. Introduction

Current research on experimental extinction performed in both human and animal learning identifies important boundaries of exposure therapy as an efficient technique to eliminate unwanted behaviors. For example, it is currently known that, after experimental extinction treatment has been effectively conducted, a conditioned response can be recovered by means of interposing a time interval between extinction training and test (i.e., spontaneous recovery, Pavlov, 1927) or by presenting the conditioned stimulus (CS) in a context different from that in which extinction treatment was conducted (i.e., renewal; Bouton and Bolles, 1979). These and other experimental findings provide important insights about possible sources of relapse after exposure therapy treatments (e.g., Bouton, 2000, 2002; Bouton and Swartzentruber, 1991).

One experimental treatment that has been found to be effective in reducing the impact of extinction treatment consists of the concurrent presentation of a Pavlovian conditioned inhibitor during CS-alone exposures (e.g., Chorazyna, 1962; Lovibond et al., 2000; Rescorla, 2003; Soltysik et al., 1983). In these studies, a CS, B, was first trained as a Pavlovian conditioned inhibitor by means of a treatment consisting of interspersed pairings of a CS, A, with the unconditioned stimulus (US) and nonreinforced presentations of A in compound with B (i.e., A → US and AB → noUS trials). This treatment typically endows CS B with the potential to inhibit responding appropriate to the US (i.e., because the animal learns that the US, which is expected to occur based on the presence of CS A, does not occur whenever CS B is present). Subsequently, a target CS, X, received conditioning (X → US pairings) followed by extinction treatment (X → noUS presentations). When CS X is presented in nonreinforced compound with CS B (BX → noUS presentations), extinction of the conditioned response elicited by X is attenuated relative to a condition in which X is presented alone during extinction treatment. This finding can be explained by traditional associative theories (e.g., Rescorla and Wagner, 1972) by assuming that the presence of the conditioned inhibitor, B, during nonreinforcement of X reduces the expectation of the absent US elicited by X and, because the absent US must be expected in order for extinction to take place, B protects X from

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Table 1
Design of the experiment

Group	Inhibitory conditioning training with B	Exposure to X	Test of X
CI	3 A → US/9 AB → noUS	2 BX → noUS	1 X
CN	3 A → US/9 AB → noUS	2 CX → noUS	1 X
E	3 A → US/9 AB → noUS	2 X → noUS	1 X

Note. A: sucrose solution; B and C: vinegar and coffee solutions, counterbalanced; X: salt solution; BX: compound presentation of flavors B and X in a single solution; CX: compound presentation of flavors C and X in a single solution; US: LiCl injection; noUS: no LiCl injection. " → " means "immediately followed by" and "/" means that the trial types were interspersed within a single phase. The numbers denote the number of presentations of each trial type in each phase. See text for further details.

extinction. This finding has implications for the treatment of a conditioned fear: The presence of a safety signal (i.e., the conditioned inhibitor) during exposure therapy might dramatically impair the effectiveness of the therapy in eliminating unwanted behavior. For example, the presence of the parents (putative safe stimuli) during exposure treatment of a child's conditioned (or unconditioned) fear to dogs (CS) could interfere with extinction (or habituation) of the fear response.

It has been suggested that not all fear responses arise from conditioning episodes (Rachman, 1977). Additionally, there are reasons to assume that the mechanisms operating in exposure therapy that are effective in extinguishing a previously conditioned fear also play an important role in the treatment of unconditioned fears (e.g., Lovibond, 1993; Reiss, 1991). If exposure to an unconditioned fear-eliciting stimulus results in habituation of the unconditioned fear owing to the same mechanisms that mediate extinction of a conditioned fear, one might expect the presence of a safety signal to also impair the habituation of an unconditioned fear. To our knowledge, this possibility has not yet been tested. Unfortunately, finding unconditioned fear-eliciting stimuli is not easy: many stimuli that are believed to produce so-called innate fear in humans and monkeys (e.g., spiders and snakes) are better understood from the point of view of preparedness or innately facilitated conditioning (i.e., Cook and Mineka, 1990; Seligman, 1971). However, the neophobic response that is elicited by exposure to a novel flavor in rats and other omnivores (including humans, see Pliner et al., 1995) might provide a model for the study of unconditioned fear.

Neophobia refers to low consumption of a novel flavor, relative to consumption of the same flavor after repeated presentations. When a rat is first presented with a novel flavor, the animal shows reluctance to consume it. This wariness serves to avoid the ingestion of a potentially toxic substance. Hence, it improves the likelihood of survival and, as a consequence, this wariness will be more likely transmitted to the next generation. Although this wariness of novel foods can be observed in different omnivores (including humans), it is of critical relevance for survival in species, like the rat, that cannot vomit (Parker, 2003). The strength of the neophobic reaction is known to vary depending on factors such as the quality of the flavor (e.g., sweet and salty flavors are initially preferred over bitter and sour flavors, at least in the rat and the guinea pig), as well as on the concentration of the solution (for a review, see Miller and Holzman, 1981). However, regardless of the intensity of this initial avoidance, it is well documented that the neophobic reaction normally dissipates as the animal receives further exposure to the flavor in the absence of any noxious consequences (Domjan, 1976; Siegel, 1974). This reduction of neophobia as a function of exposure can be viewed as an instance of long-term habituation (e.g., Wagner, 1979). Alternatively, the reduction in neophobia can be explained as learning that the flavor is safe (Kalat and Rozin, 1973). This learned safety account assumes that the rat is actively trying to discriminate which foods are safe and which are poisonous. Importantly, in the framework of this view, the presentation of a safe stimulus during exposure to a novel flavor could disrupt the reduction of neophobia that would otherwise be observed. Specifically, exposure to a novel flavor alone (followed by no illness) should result in a decrease in the neophobic reaction because the animal has the chance to learn that the flavor is safe. By contrast, when a novel flavor is exposed together with a stimulus that was previously trained as a conditioned inhibitor for illness (i.e., a safe stimulus, Best, 1975), the absence of illness following the ingestion of the novel flavor could be readily attributed to the presence of the inhibitor. As a consequence, there is a little chance for the animal to learn that consuming the novel flavor is also safe by itself. The experiment reported here attempted to assess this possibility.

2. Experiment

The experiment was performed using a conditioned taste aversion preparation in which different flavors served as CSs that were paired with illness induced by an injection of lithium chloride (LiCl), which served as the US. Table 1 summarizes the design of the experiment. The first phase of the experiment was designed to produce inhibitory conditioning to flavor B. During this phase, the three groups received identical treatment consisting of three pairings of flavor A with LiCl (i.e., A → US trials) interspersed with nine nonreinforced presentations of flavors A and B in a simultaneous compound (i.e., AB → noUS trials).[1] During the second phase, the three groups received two

[1] The present study did not include control conditions for Pavlovian conditioned inhibition (e.g., groups given either A/US or A/B unpaired presentations, instead of A → US and AB → noUS trials, respectively). Also, this study did not directly assess the status of flavor B as a conditioned inhibitor using the conventional summation and retardation tests (i.e., Rescorla, 1969). However, the acquisition of inhibitory control by flavor B could be inferred from the pattern in the consumption of flavor A and the AB compound solution during Phase 1, as well as from a higher consumption of the BX compound solution (group CI) relative to consumption of CX and X solutions (groups CN and E, respectively), in Phase 2 (see text for further details).

nonreinforced presentations of a novel flavor, X, but differed on whether flavor X was presented elementally (group Elemental [E]), in compound with a novel flavor, C (group Compound Novel [CN]), or in compound with the putative conditioned inhibitor, B (group Compound Inhibitor [CI]). Finally, all groups received a single presentation of flavor X during testing. If inhibitory conditioning treatment effectively endows flavor B with the properties of a safe stimulus, then (1) based on Best's (1975) study, consumption of a solution containing the safe flavor, B (i.e., BX solution in group CI) should be greater than consumption of a solution that does not contain flavor B (i.e., CX and X solutions in groups CN and E, respectively) and, more importantly, (2) exposing flavor X in compound with B (group CI) should result in impaired learning of X as a safe stimulus. Therefore, when first presented alone at test, X should be consumed less in group CI, relative to consumption in those conditions in which flavor X was previously exposed either alone (group E) or in compound with another novel flavor (group CN).

3. Method

3.1. Subjects

The subjects were 24 Wistar, naïve, young adult female rats, obtained from the breeding colony at the University of Seville. Rats were 90–100 days old at the beginning of the experiment, and their body weights ranged from 186 to 245 g ($M = 219$, SEM = 3.02). The animals were housed individually in 36 cm × 20 cm × 14 cm clear plastic cages on a 12:12-h light:dark cycle (from 07:00 to 19:00 h), with all the experimental sessions occurring during the light period. Subjects had free access to food in the home cage. Prior to initiation of the experiment, water availability was progressively reduced to 30 min per day. For 2 weeks prior to initiation of the experiment and until its termination, subjects were handled for 30 s two or three times a week.

3.2. Apparatus

All the 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.

Four distinct flavors were employed in this study (see Table 1). A 3.4% (w/v, 0.10 M) sucrose solution (Fluka Chemie GmbH, Buchs, Switzerland) served as flavor A. A 1% (v/v) apple cider vinegar solution (Prima, Spain) and 1% (w/v) decaffeinated coffee solution (Marcilla, Sara Lee Southern Europe, S.L., Barcelona, Spain), served as flavors B and C, counterbalanced. Finally, a 0.58% (w/v, 0.10 M) NaCl solution (Sigma–Aldrich Chemie GmbH, Steinheim, Germany) served as the target flavor, X. All solutions were made using tap water. The US was a 10 ml/kg of body weight intraperitoneal (i.p.) injection of 0.10 M lithium chloride (LiCl; ICN Biochemicals Inc., Aurora, OH, USA), which was administered using a 5-ml syringe with a 0.6 mm × 25 mm needle.

3.3. Procedure

Prior to the start of the experiment, subjects were assigned to one of three experimental groups. All subjects were given a single 20-min experimental session per day, which started at approximately 09:00 h. Also, all subjects received additional 10-min access to tap water soon after the session. Consumption during each session was recorded.

3.3.1. Pre-training

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

3.3.2. Phase 1: Inhibitory conditioning training with B

Inhibitory conditioning training with B was conducted on Days 5–19. The treatment of Phase 1 was conducted in three blocks of trials, each block containing one A → US pairing followed by three AB → noUS presentations. All animals received one pairing of flavor A with the US on each of Days 5, 10, and 15. The presentation of flavor A was followed immediately by an i.p. injection of LiCl (US), after which the animals were returned to the home cage. Each conditioning day was followed by one recovery day (i.e., Days 6, 11, and 16), on which tap water was presented with the glass bottle, while allowing the subjects to recover from the impact of LiCl. On each of the 3 days following recovery (i.e., Days 7–9, 12–14, and 17–19), all subjects were given a presentation of the AB compound solution, followed by no LiCl injection. These nonreinforced presentations of the AB compound solution were intended to establish flavor B as a conditioned inhibitor for the US prior to exposure to X.

3.3.3. Phase 2: Exposure to X

On each of Days 20 and 21, all groups received a presentation of flavor X, followed by no LiCl injection. On these days, group CI received presentations of X in compound with the conditioned inhibitor, B (BX solution). Group CN received presentations of X in compound with a novel flavor, C (CX solution). Group E received presentations of X alone.

3.3.4. Test of X

Test of X took place on Day 22, on which all groups received a presentation of the solution X by itself. An alpha level of $p < .05$ was adopted for all statistical analyses. All post-hoc comparisons (i.e., on results from training) were performed using Bonferroni's procedure, whereas planned comparisons (i.e., on results from testing) used the overall error term from the analysis of variance (ANOVA).

4. Results

Fig. 1 depicts consumption of the different flavored solutions during experimental treatment and test. Consumption of flavor

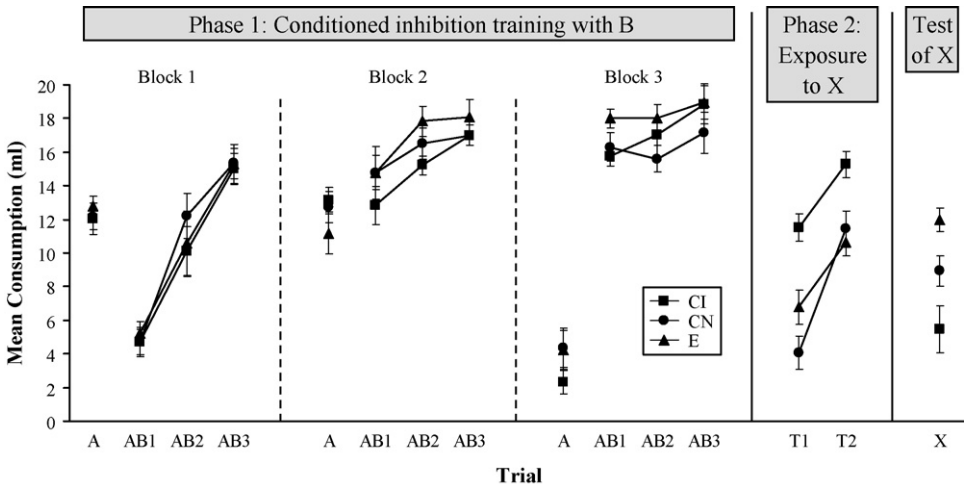

Fig. 1. Mean consumption during the experimental treatment and test. Consumption during Phase 1 (conditioned inhibition training with B) is depicted in the three left panels, with each panel depicting consumption within each block of trials. Consumption during Phase 2 (exposure to X) is depicted in the middle panel. Consumption at test of X is depicted in the right panel. Error bars depict SEMs.

A and the AB compound solution during Phase 1 (conditioned inhibition treatment with B) is depicted in the three left panels, with each panel representing a block of trials that include one A → US pairing, followed by three AB → noUS presentations. Groups did not appreciably differ in their consumption during conditioned inhibition treatment, but the pattern of consumption of flavor A and the AB compound solution importantly changed from Block 1 to Block 3. A 3 (Group: CI vs. CN vs. E) × 3 (Block: 1–3) × 4 (Trial: A vs. AB1 vs. AB2 vs. AB3) ANOVA on the mean consumption confirmed this impression, by showing main effects of block, $F(2, 42) = 51.49$, MSE = 8.90, $p < .01$, and trial, $F(3, 63) = 122.78$, MSE = 6.34, $p < .01$, as well as a Block × Trial interaction $F(6, 126) = 91.80$, MSE = 5.07, $p < .01$. Importantly, this ANOVA yielded no main effect of group nor any interaction involving group as a factor, all Fs < 1.48, all ps > .14. The source of the Block × Trial interaction was further examined by post-hoc comparisons. As suggested by the figure, consumption of A progressively decreased over blocks, whereas consumption of AB increased over blocks. In Block 1, consumption of A was higher than consumption in the first presentation of AB, suggesting that conditioned aversion previously acquired to A was, at least, partially transferred to the AB compound. Consumption of AB increased from its first presentation to its third presentation within Block 1, suggesting acquisition of an A vs. AB discrimination and/or extinction of aversion to flavor A. In Block 2, consumption of A did not differ from consumption of AB in its first presentation, suggesting that flavor B already counteracted the impact of aversive responding elicited by flavor A. In Block 3, subjects clearly mastered the discrimination between the A and AB solutions, as shown by the lower consumption of flavor A relative to consumption of the AB solution on its first presentation.

Consumption of solutions BX (group CI), CX (group CN), and X (group E) during Phase 2 (exposure to X) is depicted in the middle panel of Fig. 1. Group CI drank more of the BX solution than groups CN and E drank of the CX and X solutions, respectively. A 3 (Group: CI vs. CN vs. E) × 2 (Trial: T1 vs. T2) ANOVA on the mean consumption yielded main effects of group, $F(2, 21) = 14.63$, MSE = 9.87, $p < .01$, and trial, $F(1, 21) = 87.28$, MSE = 3.43, $p < .01$, as well as a Group × Trial interaction, $F(2, 42) = 4.98$, MSE = 3.43, $p < .05$. Separate one-way ANOVAs among groups showed significantly different consumption on both Trial 1, $F(2, 21) = 15.87$, MSE = 7.14, $p < .01$, and Trial 2, $F(2, 21) = 7.81$, MSE = 6.16, $p < .01$. Post-hoc comparisons showed that, on both trials, group CI consumed more than both groups CN and E, which consumed their corresponding solutions in comparable amounts. The higher consumption of the BX solution relative to consumption of solutions CX and X suggests that flavor B was learned as a safe stimulus, consistent with the study of Best (1975).

The right panel of Fig. 1 depicts the critical results of the experiment, namely, the consumption of flavor X at test. Inspection of this panel suggests that group CI drank the least amount of the X solution, followed sequentially by groups CN and E. A one-way ANOVA among groups on the consumption of flavor X at test detected differences, $F(2, 21) = 9.77$, MSE = 8.66, $p < .01$. Planned comparisons showed that group CI consumed less of flavor X than group CN, $F(1, 21) = 5.53$, MSE = 8.66, $p < .05$, and group E, $F(1, 21) = 19.51$, MSE = 8.66, $p < .01$. Also, consumption of flavor X was numerically lower in group CN than in group E, but this difference fell short of significance, $F(1, 21) = 4.26$, MSE = 8.66 $p > .051$.

Finally, a comparison showed a difference in group E between the consumption of flavor X on Trial 1 of Phase 2 and the consumption at test, $t(7) = -4.46$ $p < .01$. Therefore, the two exposures to X alone during Phase 2 resulted in increased consumption and, hence, in significant reduction of neophobia in group E. Ideally, the present experiment should have included a fourth group given no exposure to flavor X prior to testing. Consumption of flavor X at test in such a group would have provided a point of comparison to assess whether the neophobic response elicited by flavor X in groups CI and CN was significantly reduced. However, although not perfect, comparisons can be made with group E's consumption of flavor X on Trial

1 of Phase 2 as a substitute for this control group. These comparisons showed that groups CI and CN consumed flavor X at test in amounts comparable to that of group E on Trial 1 of Phase 2, $ts < 1.55$, $ps > .44$. Thus, although these comparisons must be considered with caution, they suggest that the neophobic reaction was not significantly reduced in groups CI and CN.

5. Discussion

The experiment reported here found a reduction in a neophobic response to a novel flavor, salt (flavor X), due to exposure to the flavor in the absence of any noxious consequence (Domjan, 1976; Siegel, 1974). More importantly, this reduction of neophobia was prevented by the presentation of a safe stimulus, B (i.e., a putative conditioned inhibitor for illness; see Best, 1975), in compound with flavor X. The addition of another novel flavor, C, produced a detectable, but insignificant attenuation of the reduction of neophobia. Because of this latter finding, the results of this experiment do not preclude a generalization decrement account. That is, the response acquired during exposure to the compound solution (BX or CX) could have incompletely generalized to the stimulus presented at test (X) due to removal of the added flavor (B or C), thereby resulting in little reduction of neophobia in both groups CI and CN. A generalization decrement is most likely to have occurred in group CN, due to this group having received in Phase 2 a solution formed by a mix of two novel flavors, C and X, which might have promoted configural processing of the CX compound (e.g., Pearce, 1987, 1994). However, a generalization decrement account fails to explain the observation that the reduction in neophobia was more strongly prevented by a conditioned inhibitor (group CI) than by a novel flavor (group CN).

In our experiment, we used the reduction of a neophobic response to assess whether protection from habituation of an unconditioned fear could be achieved by the concurrent presentation of a safe stimulus. Several points in our approach might be found controversial. First, it could be claimed that neophobia does not involve fear at all. Although the neophobic reaction resembles an avoidance response (the rat retreats from the bottle containing the novel flavored solution), and although neophobia has been found to be enhanced by fear in both rodents (Minor, 1990) and humans (Pliner et al., 1995), commonality with the mechanism underlying fear cannot be unequivocally asserted. Instead, neophobia might be viewed merely as reflecting wariness or reluctance, rather than real fear. In any case, we must remark on our use of neophobia *as a model* for unconditioned fear. Second, we have discussed the reduction of a neophobic response as an instance of habituation of unconditioned fear. The idea that the same mechanisms that operate in the extinction of a conditioned fear might also be involved in the reduction of an unconditioned fear is not new (e.g., Lovibond, 1993; Reiss, 1991). However, from a strict point of view, extinction of any response requires previous conditioning and, thus, the reduction of an unconditioned fear might be viewed as a process resembling (but different from) extinction, that is, habituation. Future research should further address the relationship of habituation to extinction. Finally, if the idea that neophobia involves actual fear is accepted, another question arises: in order for a safe stimulus (i.e., the conditioned inhibitor for illness) to impair the reduction of neophobia during exposure to a novel flavor, the safe stimulus must not only inhibit aversive responses but also fear. In this regard, Parker (2003) has proposed that conditioned fear (avoidance) might play an important role in results traditionally viewed as indicative of conditioned nausea (aversion). Thus, our conditioned inhibition treatment could have endowed the safe stimulus with the ability to counteract not only aversion but also fear. If so, the safe stimulus might have prevented the reduction of neophobia by reducing fear elicited by the encounter of a novel flavor.

5.1. Explanation from a learned safety view and by the Rescorla–Wagner model

As anticipated in Section 1, a safety learning view can account for these results (Kalat and Rozin, 1973). In this framework, despite an initial wariness of a novel flavor, X, repeated exposure to flavor X in the absence of illness allows the animal to learn that it can be safely consumed. Therefore, a safety learning explanation for the reduction of neophobia provides a straightforward account for the observed protection from the decrease in the neophobic response that is produced by the addition of a stimulus previously trained as a safety signal. In this view, because the animal presumably expects no illness to occur in the presence of flavor B, it is not possible to ascertain that consuming flavor X is also safe *per se*. As a consequence, when first faced with flavor X alone, the animal is reluctant to consume it.

Despite our preference for the explanation provided by the learned safety account, the viability of an alternative explanation must be noted. The Rescorla and Wagner (1972) model predicts that pairing a novel CS, X, in compound with a conditioned inhibitor should result in the acquisition of excitatory associative strength by X, even if exposure to the BX compound occurs in the absence of reinforcement. This prediction of the Rescorla–Wagner model is based on its explanation of excitation and inhibition as seeming mirror images. That is, the Rescorla–Wagner model explains excitatory and inhibitory conditioning as positive and negative values, respectively, on a single continuum of associative strength. According to this model, pairing a neutral CS, X, in a nonreinforced compound with a conditioned inhibitor, B (i.e., BX → noUS trials) should result in CS X undergoing positive changes in associative strength because (1) the absent US cannot support any associative strength on those trials and (2) the total associative strength of the BX compound has a negative value. As a consequence, a positive change in associative strength occurs in order to reduce the error between the actual occurrence of the (absent) US (i.e., value of 0) and expected occurrence of the US based on the presentation of BX (i.e., negative value due to B being an inhibitor). In this sense, according to the Rescorla–Wagner model, CS X should undergo a positive increment in associative strength comparable to that resulting from a direct pairing with the US. However, in this latter case, the discrepancy would arise from the positive value of associative strength that the (present) US can support (i.e., value

of 1) and expected occurrence of the US based on the presence of X (i.e., value of 0).

This prediction of the Rescorla–Wagner model was initially supported by a study of Rescorla (1971; Experiment 2), but it was later rejected by Baker in a study including a critical control condition (Baker, 1974; Experiment 2). Also, it is strongly related to another prediction of this model, namely, that nonreinforced presentations of a conditioned inhibitor should extinguish its inhibitory associative strength, which did not pass empirical scrutiny (e.g., Zimmer-Hart and Rescorla, 1974). Therefore, although the present experiment cannot categorically reject the possibility that presenting flavor X in compound with flavor B in group CI resulted in the acquisition of conditioned aversion to CS X, previous studies call for caution in the consideration of the appropriateness of this explanation by the Rescorla–Wagner model.

5.2. Implications for exposure therapy

The results of this experiment have important implications for the clinical treatment of fear. If the neophobic response is viewed as a model for unconditioned fear, then these results suggest that the habituation of unconditioned fear can be impaired when a safe stimulus is present during exposure treatment. In this vein, the results of the present experiment are analogous to the finding that extinction of a conditioned response can be protected by a conditioned inhibitor (e.g., Chorazyna, 1962; Lovibond et al., 2000; Rescorla, 2003; Soltysik et al., 1983). The similarity of these results supports the idea that factors that can influence the effectiveness of exposure therapy in the treatment of conditioned fear might also be relevant during exposure therapy in the treatment of unconditioned fear. Although counterintuitive, these results add to a growing set of evidence suggesting that the effectiveness of exposure to a fear-eliciting stimulus could be critically enhanced by removing any signals for safety from the therapeutical environment.

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