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Extinction Cues do not Reduce Recovery of Extinguished Conditioned Fear in Humans

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Abstract

We evaluated whether an extinction cue can reduce (or prevent) the recovery of previously extinguished fear conditioning using an ABC renewal design in humans. Two experiments were carried out. In Experiment 1, two groups were presented with geometric shapes as conditioned stimulus (CS), followed by a small electric shock as unconditioned stimulus (US) during the acquisition phase. Conditioned fear was measured as ratings of US expectancy and changes in skin conductance response (SCR). During the extinction phase, both groups received presentations of the CS without the US, while an extinction cue (EC) was presented. Both groups were tested in both the extinction context (extinction test) and a new context (renewal test) immediately and 48 hours after the end of the extinction phase (spontaneous recovery). Half of the subjects were tested in the presence of the EC (Group Extinction cue) while the other half were tested in the presence of a neutral cue (Group Neutral cue). The results suggested that the EC reduced the recovery of fear produced by a context change, but that this reduction was not maintained over time. Experiment 2 increased the salience of the EC and the contexts, however, results showed that the EC was unable to reduce the renewal of fear conditioning. These results are discussed as a function of the experimental manipulations performed, and their theoretical and practical implications.
Key words: Pavlovian conditioning, techniques to reduce recovery, human learning, translational research, exposure therapy.

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Novelty and Significance

What is already known about the topic?

- Extinction is an effective procedure to reduce conditioned fear responses, however, the extinguished response may reappear under certain conditions (e.g., a change in context after extinction).
- Extinction cues are stimuli part of the extinction context that help to recover the memory of extinction when the subject is in a different context, preventing the return of fear.
- Previous research suggests that extinction cues are sometimes effective in reducing the recovery of fear response after extinction.

What this paper adds?

- Extinction cue enhances extinction learning, but its effect in reducing fear response recovery after extinction is limited.
- The development of behavioral techniques to prevent post-extinction response recovery is important to improve the long-term effects of exposure therapy.

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Pavlovian conditioning, and specifically fear conditioning, has helped understand the etiology and treatment of anxiety disorders from a behavioral perspective (Barlow, Bullis, Comer, & Ametaj, 2013; Bouton, Mineka, & Barlow, 2001; Craske, Liao, Brown, & Vervliet, 2012; Dibbets, Poort, & Arntz, 2012; Laborda, McConell, & Miller, 2011; Lissek *et alii*, 2005; Vervliet, Craske, & Hermans, 2013). Authors propose that learned fear is a product of an association between a neutral stimulus and an aversive stimulus (unconditioned stimulus-US) which produces a non-learned fear response (unconditioned response-UR). After sufficient pairings, the previously neutral stimulus produces fear responses; the neutral stimulus becomes a conditioned stimulus (CS) that is capable of eliciting a conditioned response (CR; Watson & Rayner, 1920).

Under this paradigm, presentations of the CS without the US can reduce the acquired response, a phenomenon known as extinction (Pavlov, 1927). This procedure is the keystone of exposure therapies, which have shown to be effective in the treatment of fears and anxiety disorders (Chambless & Ollendick, 2001; Emmelkamp, 2013). The experimental evidence, however, shows that the CR reappears under certain conditions, such as changes in the physical or temporal contexts, and presentations of the US after extinction (Bouton, 1993, 2004). Based on these findings, researchers have argued that extinction does not eliminate the CS-US association, but corresponds to a new association that competes with the initial learning and suppresses its expression (Aristizabal, Callejas Aguilera, Ogallar, Pellón, & Rosas, 2015; Rosas, García Gutiérrez, & Callejas Aguilera, 2007; for a review, see Laborda & Miller, 2012). This is problematic in terms of guaranteeing the maintenance of the benefits of treatments based on exposure, since neither the magnitude of fear reduction during the exposure sessions nor the level of fear at the end of the exposure sessions appear to be good predictors of treatment outcomes. These findings reveal the need to improve existing treatment techniques and to further understand the extinction process (Barlow *et alii*, 2013).

Various experimental manipulations to prevent the recovery of the CR after extinction have been developed in the laboratory (Laborda, McConnell, & Miller, 2011). These techniques include the use of a large number of extinction trials (massive extinction), performing the extinction procedure in multiple contexts, presentations of the US alone during extinction, and the introduction of new stimuli during extinction (extinction cues). The present study focuses on this last technique.

An extinction cue is a distinctive stimulus part of the extinction context, that aids the recovery of the extinction memory when it is presented outside of this context during test (Brooks, Vaughn, Freeman, & Woods, 2004). A number of experiments with animal models (Brooks & Bouton, 1994) and humans (Collins & Brandon, 2002; Culver, Stoyanova, & Craske, 2011; Dibbets, Havermans, & Arntz, 2008; Dibbets & Maes, 2011; Dibbets, Moor, & Voncken, 2013; Vansteenwegen, Vervliet, Hermans, Beckers, Baeyens, & Eelen, 2006) have shown the efficacy of extinction cues in preventing the recovery of the fear response. However, methodological limitations in studies conducted with humans, caution about the extension and generalization of the results. In a critical analysis of human renewal studies, Vervliet, Baeyens, van den Bergh, & Hermans (2013) proposed that the increase in fear to an extinguished stimulus presented in a different context may be explained by an incomplete extinction. Based on these analyses, they suggest evaluating possible changes in the extinguished response without a contextual change. Similarly, the studies of Dibbets *et alii* (2008, 2011), using an ABA renewal design (where acquisition occurs in context A, extinction in B, and renewal in A; Bernal Gamboa *et alii*, 2012), proposed that if an association between the context and the US is

formed during the acquisition phase, the increase in the fear response during the renewal test could be explained by the sum of the response produced by both context A and the CS, and not only due to the response produced by the CS. To avoid this problem, Vervliet *et alii* (2012) suggested performing the renewal test in a new context (C), thereby avoiding any contextual associations that may produce conditioned responding.

Following Vervliet *et alii* (2012) suggestions, the aim of the present study was to experimentally evaluate the efficacy of extinction cues in preventing the renewal and spontaneous recovery of an extinguished conditioned fear response in humans. In order to incorporate the suggestions of Vervliet *et alii* (2012), both experiments include a within-subject ABB extinction control in an ABC renewal design. In addition, we equated the exposure time to contexts B and C before testing. Equal exposure to the testing context avoids changes in the conditioned response due to its novelty (Lovibond, Preston, & Mackintosh, 1984). The results of this study would help to evaluate the efficacy of the ECs in the attenuation of the recovery of extinguished conditioned fear, and contribute to the development of more effective strategies for the treatment of anxiety disorders, addressing one of the most important limitations in the literature. We also expect to contribute to the methodological design of fear conditioning studies in humans, which by their nature present important challenges to researchers.

EXPERIMENT 1

The objective of Experiment 1 was to evaluate the effect of an EC in the reduction of renewal and spontaneous recovery of the conditioned fear response in humans. In this ABC renewal design, two groups differed in the type of cue presented (neutral cue or extinction cue) during a series of testing trials after extinction; extinction, renewal, spontaneous recovery, and renewal and spontaneous recovery combined (Table 1). We expected that the subjects tested in the presence of an EC would show a smaller recovery of the conditioned fear response compared to subjects tested in the presence of a neutral cue that was not present during training. A summary of the design can be found on Table 1.

Table 1. Experimental Design for Experiment 1.

Group	Acquisition	Extinction	Retention 1	Extinction and Renewal Test	Expected response	Retention 2	Spontaneous Recovery+ Renewal Test	Expected response
Extinction Cue	A (X+) A (Y+) A (Z-)	B (EC→X-) C (Z-)	5 min	C (EC→X-)	cr	48 hrs	C (EC→X-)	Cr
				B (EC→X-)	cr		B (EC→X-)	cr
Neutral Cue				C (NC→X-)	CR		C (NC→X-)	CR
				B (NC→X-)	cr		B (NC →X-)	Cr

Notes: A, B and C represent three contexts of different colors; X, Y and Z are three different conditioned stimuli, followed (+), or not (-) by the unconditioned stimulus; EC= extinction cue; NC= neutral cue; a horizontal arrow represents the serial presentation. To represent the response expected in the tests, cr= weak conditional responding; Cr= moderate conditional responding; CR= strong conditional responding.

METHOD

Participants

Forty-seven University students (31 women) with a mean age of 21.78 years (range 18-33, *SD*= 3.08) participated in the study and received a coupon for photocopies or a ticket to a movie theater as reward for their collaboration. All participants recruited

were within the cohort range score in the psychological dimensions evaluated by the Symptom Check List SCL-90-R (Derogatis, 1992) or did not have a medical history that justified exclusion (evaluated by a medical checklist). Before the experiment, all participants signed an informed consent approved by the local Ethics Committee of our institution.

Instruments

The Symptom Check List SCL-90-R (Derogatis, 1992) Is a questionnaire for self-reporting of psychological distress experimented by a person and assesses nine symptomatic dimensions: somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation and psychoticism. Gempp and Avedaño (2008) obtained Chilean normative data and characterized the psychometric properties that were used for this study.

The State-trait Anxiety Inventory STAI (Spielberger, Gorsush & Lushene, 1970). Is an anxiety questionnaire that assesses two components: state and trait. We used Vera Villarroel, Celis Atenas, Córdova Rubio, Buela Casal, & Spielberger (2007) version of the scale, which has Chilean normative data and adequate psychometric properties.

Stimuli

Three black geometric figures, a square (5 cm per side), an equilateral triangle (5 cm a side) and a circle (6 cm diameter), were presented on a computer screen and served as conditioned stimuli (X, Y and Z, counterbalanced). An ampersand (&) and hash (#) served as extinction and neutral cues (counterbalanced); these appeared in the four extremes of the computer screen at a mean distance of 5.3 cm from the upper and lower borders, and a mean of 4.4 cm from the sides. A, B and C contexts were different background screen colors on which the figures and cues were shown. Context A was a blue background (RGB: 102, 153, 204), and remained constant for all participants. Both a red (RGB: 195, 63, 30) and a green (RGB: 34, 177, 76) background were used as contexts B and C (counterbalanced). The US was a mild but uncomfortable electric shock on the left forearm, with a duration of 200 ms and intensity varying from 8 to 24 mA. The intensity of the US was determined by each participant; participants were asked to select an intensity that was unpleasant and demanded some effort to tolerate (Vansteenwegen, Ibérico, Vervliet, Marescau, & Hermans, 2008; Spoormaker *et alii* 2010).

Apparatus

The delivery of the electric shock was controlled by a Digitimer DS7A Constant Current Stimulator (Hertfordshire, UK) via a pair of steel disk electrodes of 8mm diameter with 30 mm spacing. We recorded the skin electrical conductance response (ECR) during all tests using Ag/AgCl electrodes (1 cm diameter) attached to the distal surfaces of the medial phalanges of the index and ring finger of the non-dominant hand. The I-330-C2 biofeedback system uses an exo-somatic measure and provided a constant 2 μA across electrodes. The analog signal was passed through a 16 bit AD-converter from 4s prior to the onset of the conditional stimulus until 4 s after conditional stimulus offset (Vansteenwegen *et alii*, 2008). To reduce signal noise, the experimenter cleaned the hands of the subject with a saline solution prior to the attachment of the electrodes.

The expectancy of an incoming US was measured in real time during each

stimulus presentation using a visual analog scale (VAS), located below the stimulus at the bottom of the screen. By clicking and dragging with the mouse, the indicator could be set anywhere in an horizontal line, for which the far left end meant that the shock was "certainly not expected" (1), and the far right end meant that the shock was "certainly expected" (100). The experiment was conducted on a Hewlett-Packard desktop computer interfaced with an *Arduino© Uno* board for shock delivery, and programmed in the E-prime software (*Psychology Software Tools*, http://www.pstnet.com).

Procedure

The experiment was carried out in an acoustically isolated room equipped with an armchair and a desk where the computer, the electric shock generator and the apparatus to measure the SCR were placed. The day before the experiment began, each participant filled an online version of the SCL-90 survey and listed any medical conditions that had been diagnosed. This allowed the experimenter to determine whether the participant fulfilled the inclusion criteria. If a participant score in the SCL-90 was above the cohort limit or had a medical contraindication related to the experiment, the principal investigator proceeded to interview the participant, and indicated the reasons for exclusion. If it seemed necessary, the principal investigator suggested potential actions to solve the problem. All remaining participants were randomly assigned to one of the two groups (Extinction cue or Neutral cue).

Pretraining. Before initiating the experiment, each participant signed the informed consent and answered the STAI in a room adjacent to the experimental room. The participant was then led to the experimental room and seated in a comfortable armchair, while the experimenter connected the electrodes to measure SCR and to provide the electric shock. Then, the experimenter administered an electric shock of 8 mA and asked the participant if the shock was "uncomfortable and demanded some effort to tolerate"; if the answer was no, the shock intensity was increased in increments of 1 mA until the response was yes. After the intensity was selected, the experimenter instructed the participant on the use of the VAS, and invited the subject to rate the chances of the US coming in three trials with each of the conditional stimuli that were used in the next phase; no electric shocks were given in those trials.

Acquisition. The acquisition phase was identical for both groups. In context A, the X and Y stimuli were presented individually for 8s, and were both followed by the electric shock for 200 ms (X+ and Y+). Presentations of the Z stimulus were not followed by the electric shock (Z-). The presence of Z- allowed controlling the orientation and habituation effects that could be reflected in the galvanic response (Vansteenwegen et alii, 2006). Each stimulus was presented 6 times in a random order. The inter-trial interval (ITI) varied randomly between 4 and 6s (M= 5s; Dibbets & Maes, 2011).

Extinction. The extinction phase was equivalent for both groups. During this phase, stimuli X and Z were presented without the electric shock (X- and Z-) in contexts B and C, respectively, which allowed equal exposition time for the extinction (B) and renewal (C) contexts. Both the stimulus duration and the ITI were identical to the ones used in the acquisition phase. The EC lasted for 4s and was presented in all the X- trials, ending 1s before the beginning of X.

Extinction and renewal tests. The first round of testing trials occurred 5 min after the end of the extinction phase. Three trials of X in its extinction context (Extinction Test) and 3 trials of X in a novel but familiar context (Renewal Test) were given in 2 three-trial blocks (counterbalanced within groups). The presentations of X were preceded by either the EC (Group Extinction Cue) or a new-novel cue (Group Neutral Cue).

The interval between trials and between stimuli was identical to the one used during previous phases. No US was presented in these trials.

Spontaneous recovery and renewal tests. Approximately 48 hours after the first round of testing, a second, identical round of testing was carried, to test for spontaneous recovery and spontaneous recovery+renewal in all participants.

RESULTS

Only the data of the participants who fulfilled both of the following criteria were analyzed: (1) in the last acquisition trial, the US expectancy ratings for X and Y were 70 points or higher, and the US expectancy for Z was 30 points or less; and (2) in the last extinction trial, the US expectancy with X and Z was 30 points or less, in order to analyze the effects of EC only in participants that acquired and extinguished correctly. Under these criteria, the data of 15 participants were eliminated from the analyses. The final sample included 32 participants, 16 in each group, balanced by sex. The STAI and SCL-90 scores were contrasted for both groups using Analysis of Variance (ANOVA). No significant differences were found for STAI Trait [$F(1,30) < 1$], STAI State [$F(2,30) = 1.1$, $p > .05$], or SCL-90 sub-scales (all ps >.05). Furthermore, there were no significant differences by sex in STAI Trait [$F(1,30) < 1$], STAI State [$F(1,30) = 1.6$, $p > .05$], or SCL-90 sub-scales (all *ps* >.05) before they were exposed to the experimental conditions.

The mean US expectancy for X, Y and Z during the acquisition phase are shown in Figure 1A. As the figure suggests, both groups of participants learned to differentiate between the CS that were paired with the electric shock (X and Y) and the one that was not (Z). We assessed the reliability of these results using a repeated measures ANOVA with Stimulus (X, Y and Z) and Trial (1-6) as within-subject factors, and Cue (neutral or extinction) as a between-subject factor. The results showed a main effect of the Stimulus, $F(2,29) = 269.2$, $MSE = 1545.401$, $p < .05$, $\eta^2 = 0.94$, 95% CI [.89, .96], and a main effect of the Trial, $F(5,26) = 21.5$, $MSE = 624.946$, $p < .05$, $\eta^2 = 0.80$, 95% CI [.57, .85], along with a Stimulus x Trial interaction, $F(10,21) = 39{,}1$, $MSE = 748.738$, $p < .05$, $\eta^2 = 0.72$, 95% CI [.50, .81]. Most importantly the overall US expectancy did not reliably differ between groups, $F(1,30) < 1$. Pairwise comparisons showed that the US expectancy scores between X and Y were not significantly different ($p > .05$) but were reliably higher than that for Z (both *ps* <.05).

The mean US expectancy for X and Z, across extinction trials, are presented in Figure 1B. Although the US expectancy scores for X were higher than those for Z at the beginning of extinction, these progressively decreased; by trial 6, the US expectancy scores for these cues were practically identical. These results were analysed using a repeated measures ANOVA, with Stimulus (X or Z) and Trial (1-12) as within-subject factors, and Cue (neutral or extinction) as the between-subject factor. The results showed a main effect of the Stimulus, $F(1,28) = 23.7$, $MSE = 597.379$, $p < .05$, $\eta^2 = 0.45$, 95% CI [.17, .62], and of Trial, $F(11,18) = 58.6$, $MSE = 774.545$, $p < .05$, $\eta^2 = 0.67$, 95% CI [.43, .77]. More importantly, the introduction of the extinction cue did not reliably affect the overall US expectancy levels, as shown by non-significant effect of Cue, $F(1,28) = 1.9$, $MSE = 48.716$, $p > .05$. Furthermore, initial US expectancy levels were higher for X ($M = 77.92$) than for Z ($M = 44.90$), which led to greater levels of extinction for X, as indicated by the significant Stimulus x Trial interaction, $F(1,18) = 8.0$, $MSE = 976.608$, $p < .05$, $\eta^2 = .22$, 95% CI [.01, .43]. No other significant interactions were found (all *ps* >.05).

The mean US expectancy for X during the first round of testing are shown in Figure 1C. To assess these scores, we performed a mixed ANOVA, with Trial (1-3)

and Context (B or C for extinction and renewal tests, respectively) as within-subject factors and Cue (extinction or neutral) and Test order (extinction-renewal or renewal-extinction) as between-subject factors. Testing order did not affect the overall US expectancy, $F(1,28)= .06$, $MSE= 22204.8$, $p > .05$. Response recovery was observed in the first of either extinction or renewal trials, but quickly decreased, as shown by a significant effect of Trial, $F(2,27)= 26.9$, $MSE= 28091.0$, $p < .05$, $\eta^2= .36$, 95% CI [.16, .58]. Furthermore, response recovery was larger when testing was carried out in a novel but familiar context (renewal) than when it was carried out in the extinction context, as shown by a significant effect of Context, $F(1,28)= 20.8$, $MSE= 7113.9$, $p < .05$, $\eta^2= .42$, 95% CI [.18, .60]. However, although the mean US expectancy was always smaller in the Extinction Cue group than in the Neutral Cue group, the main effect of Cue was not significant, $F(1,28)= 27.7$, $MSE= 31525.4$, $p > .05$, $\eta^2= .49$, 95% CI [.21, .65]. Both a Context x Cue, and a Trial x Context interactions were found $F(1,28)= 4.5$, $p < .05$, $\eta^2= .14$, 95% CIs [.09, .22], and $F(2,27)= 7.2$, $p < .05$, $\eta^2= .34$, 95% CIs [.26, .42], respectively. No other interactions were significant (all *ps* >.05).

Given the quick decrease in US expectancy across trials -explained, by the testing trials being not followed by the US- we assessed US expectancy in the first trial of each test. This subset of observations was again not affected by the order of testing $F(1,28) < 1$. US expectancy was again reliably larger in the renewal context than in the extinction context, $F(1,28)= 17.5$, $MSE= 143.187$, $p < .05$, $\eta^2= 0.38$, 95% CI [.10, .56]. The mean US expectancy was higher for the Neutral cue group than for the Extinction cue group in both extinction (26.09 and 12.48, respectively) and renewal tests (59.01 versus 37.92 respectively). However, this time the effect of Cue was nonsignificant, $F(1,28)= 3.6$, $MSE= 1409.281$, $p= .06$. No other interactions were significant (all *ps* >.05).

The mean US expectancy for X in both tests performed after 48 hours are shown in Figure 1D. Time did not produce an appreciable recovery in the response. An ANOVA identical to the one used to assess the previous results revealed that the US expectancy reliably decreased across testing trials, $F(2,27)= 7.8$, $MSE= 4160.3$, $p < .05$, $\eta^2= .29$, 95% CIs [.18, .36], and that testing order did not affect US expectancy $F(1,28)= .23$, $MSE= 22204.8$, $p > .05$. US expectancy was not significantly different between groups, $F(1,28) < 1$. or between contexts, $F(1,28)= 20.8$, $MSE= 4653.8$, $p > .05$. No interactions were found (all *ps* >.05).

In order to assess whether or not spontaneous recovery took place, we compared the US expectancy during the first trial of testing in the extinction context and the first trial in the renewal context, after the 48 hrs. -the instance that should produce the highest level of response recovery. An ANOVA using those scores revealed no differences between the two test conditions $F(1,30) < 1$, and no differences between groups, $F(1,30) < 1$. This finding confirms that time did not affect the recovery of the US expectancy. An illustration of the results of US expectancy is shown in Figure 1.

To analyze the skin conductance response (SCR) we calculated the Second Interval Response (SIR); that is, the mean conductance during seconds 5-8 of the CS presentation minus the mean conductance during the 4s before the onset of the CS. Negative values were registered as 0. Finally, the data were square root transformed (Prokasy & Kumpfer, 1973; Effting & Kindt, 2007). The results were analyzed using the same methods as in the analysis of US expectancy.

A repeated measures ANOVA was performed, with Stimulus and Trial as within-subject factors, and Cue as between-subject factor. The results showed a Trial effect, $F(5,26)= 12.9$, $MSE= 7.329$, $p < .05$, $\eta^2= .30$, 95% CIs[.06, .51], Stimulus, $F(2,29)= 14.8$,

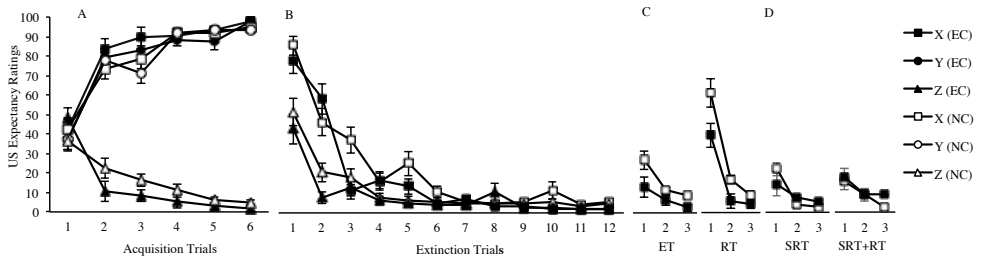

Figure 1. Mean US expectancy across the different phases of Experiment 1. A rating of 1indicates security that the electric shock will not occur, and a rating of 100 indicates that the electric shock will surely occur. Closed and open symbols represent the extinction cue and neutral cue groups, respectively. (A) Means US expectancy produced by X, Y and Z during the acquisition phase. (B) Mean US expectancy produced by X and Z during the extinction phase. (C) Mean US expectancy produced by X during the first round of testing, 5 minutes after extinction training ended; ET= Extinction Test; RT= Renewal Test. (D) Mean US expectancy produced by X during the second round of testing, 48 hours after extinction training ended; SRT= Spontaneous Recovery Test; SRT+RT= Spontaneous Recovery plus Renewal Test.

MSE= 3.880, p= .15, η^2= .33, 95% CIs [.07, .53], and a Trial x Stimulus interaction, $F(10,21)$= 5.9, *MSE*= 7.329, p <.05, η^2= .17, 95% CI [.00, .39]. More importantly, there was no effect of the Cue, $F(1,30)$ <1. However, pairwise comparisons between the stimuli revealed that SCR for X was higher than those of Y and Z (both *ps* <.05), and that the SCR for Y was greater than of Z (p <.05). Given the failure to find a reliable differential conditioned SCR for the training stimuli, no further analyses using this measure are reported.

DISCUSSION

In experiment 1, participants successfully learned to discriminate between CS that predicted the occurrence of the US (X and Y) and a CS predicted the absence of the US (Z). The resulting differential US expectancy between X and Z was successfully extinguished in the subsequent extinction treatment phase. During testing, the US expectancy for X was recovered when tested in a context different to the use during extinction, as expected in a renewal situation. However, time did not produce an appreciable nor reliable recovery of the extinguished response. It is very likely that the renewal and extinction tests acted as further extinction trials, enhancing the extinction memory and thus preventing the occurrence of spontaneous recovery. The lack of robust spontaneous recovery reduces the odds of detecting a reliable effect of the EC.

The high variability in the results of SCR suggests the need to modify the training protocol in order to stabilize the response after the presentation of the CSs. Moreover, it is important to note that the renewal of responding was partially, but not significantly, impaired by the presence of the extinction cue; an increased salience of the extinction cue might allow it to have a greater impact in response recovery. Those were the aims of Experiment 2.

EXPERIMENT 2

Experiment 2 evaluated whether increasing the salience of the EC and the contexts would increase the ability of the EC to decrease the renewal of an extinguished fear response. The experimental design was identical to Experiment 1, except that the present Experiment lacked the test 48 hours after the end of the extinction phase. Additionally, to provide a reliable physiological measure, we included a habituation phase previous

to the acquisition phase, and changed certain parameters, including the number of trials of acquisition and extinction, and the ITI, all which is described below. The design is summarized in Table 2.

Table 2. Experimental Design for Experiment 2.

Group	Habituation	Acquisition	Extinction	Retention 1	Extinction and Renewal Test	Expected response
Extinction Cue	D (X-)	A (X+)	B (EC→X-)	5 min	C (EC→X-)	cr
	D (Y-)	A (Y+)	C (Z-)		B (EC→X-)	cr
	D (Z-)	A (Z-)			C (NC→X-)	CR
Neutral Cue	D (EC-)					
	D (NC-)				B (NC→X-)	cr

Notes: A, B, C and D represent four contexts of different colors; X, Y and Z are three different conditioned stimuli, followed (+), or not (-) by the unconditioned stimulus; EC= extinction cue; NC= neutral cue; a horizontal arrow represents the serial presentation. To represent the response expected in the tests, cr= weak conditional responding; Cr= moderate conditional responding; CR= strong conditional responding.

METHOD

Participants

Forty-two University students participated in this experiment (26 women). The average age was 22.59 years (range 17-34, *SD*= 4.77). All participants were exposed to the same conditions as in Experiment 1.

Instruments, Stimuli and Procedure

The same instruments, stimuli, and procedures were used as in Experiment 1, unless otherwise noted. Extinction and Neutral cues were replaced by a pair of opposite figures, specifically, a colored cat and a mouse, of 6.38×4.03 cm and 3.60x5.18 cm respectively (counterbalanced), and their position in the screen varied between trials. As contexts, in addition to the background screen color, we also produced a change in the illumination color of the experimental room, matching the color of the screen background.

To decrease the variability of the response in the VAS we incorporated five perpendicular lines (1.2 cm of height, and 0.02 cm wide, separated by 2.4 cm) with values 1, 25, 50, 75 and 100, which defined ranges of expectancy of US occurrence (improbable, little probable, unpredictable, probable, very probable).

Pretraining. The pretraining phase was identical to the one described in Experiment 1.

Habituation. At the end of the pretraining phase and after the test trials, an habituation phase lasting 3 minutes, took place. This phase aimed to stabilize the basal electrical conductance of the skin to increase sensitivity to detect changes in the response during the acquisition phase. During these three minutes a yellow-colored context was presented, and the participants were asked to relax and try not to move.

Acquisition. We reduced the number of trials to 4 per stimulus, since in Experiment 1 we observed that the subjects reached asymptotic levels of response after 4 trials. The ITI varied trial to trial with a mean of 20 s (range 16-24) in order to allow more time for the conductance response to recover its baseline levels after the electric shock.

Extinction. The number of extinction trials was reduced to 10.

Extinction and renewal tests. This testing phase was identical to that of Experiment 1.

RESULTS

Data from 10 subjects were excluded from analyses because they did not fulfill the acquisition and extinction criteria used in Experiment 1. The final sample analyzed included 32 participants (17 women and 15 men). The number of subjects in both groups was 16. There were no differences in the STAI Trait [$F(1,30) < 1$], STAI State [$F(1,30) < 1$], or SCL-90 sub-scales (all *ps* >.05). Likewise, there were no sex differences in STAI Trait [$F(1, 30) < 1$], STAI State [$F(1,30) < 1$], or SCL-90 sub-scales (all *ps* >.05).

The mean US expectancy ratings for X, Y, and Z across acquisition trials are presented in Figure 2A. Performance was nearly identical to the one during the acquisition phase of Experiment 1. All the data from this and other phases were analyzed using the same model structures used to assess Experiment 1's data. The results showed a main effect of Stimulus, $F(2,29)= 195.8$, $MSE=.744$, $p < .05$, $\eta^2= 0.87$, 95% CI [.75, .91], and a main effect of Trial, $F(3,28)= 42.4$, $MSE= .403$, $p < .05$, $\eta^2= 0.59$, 95% CI [.33, .52] and a Stimulus x Trial interaction, $F(6,25)= 57.7$, $MSE= .409$, $p < .05$, $\eta^2= 0.93$, 95% CI [.01, .42]. Importantly, there was no difference between cue types, $F(1,30)= 1.6$, $MSE= 14429.3$, $p > .05$. Pairwise comparisons showed that the US expectancy for X and Y were not reliably different ($p > .05$), but both were higher than those for Z, (both *ps* <.05).

The mean US expectancy for X and Z across extinction trials is shown in Figure 2B. Again, performance was nearly identical to the one during extinction in Experiment 1. The results showed that the overall US expectancy was higher for X, $F(1,30)= 28.3$, $MSE= 2966.7$, $p < .05$, $\eta^2= .48$, 95% CI [.21, .64]. Overall US expectancy decreased across trials, $F(9,22)= 39.5$, $MSE= .744$, $p < .05$, $\eta^2= 0.56$, 95% CIs [.30, .70]. As found before, extinction of response was larger for X, as shown by a Stimulus x Trial interaction, $F(9,22)= 5.08$, $MSE= 105.5$, $p < .05$, $\eta^2= 0.14$, 95% CI [.00, .36]. A comparison between X and Z revealed that US expectancy for X was significantly higher at the start of the extinction, $F(1,30)= 28.3$, $MSE= 2.4$, $p < .05$, $\eta^2= 0.48$, 95% CI [.00, .22], but such difference disappeared towards the end of the extinction phase ($p > .05$). There were no differences between groups, $F(1,30)= 0.2$, $MSE= 135873.9$, $p= 0.88$, $\eta^2= 0.00$, 95% CI [.00, .22]. However, the Stimulus x Cue interaction was significant, $F(1,30)= 9.4$, $MSE= .432$, $p < .05$, $\eta^2= 0.23$, 95% CI [.02, .45]. An analysis of this interaction revealed that the overall US expectancy between cues was reliably different for the neutral cue group $F(9,7)= 17.5$, $MSE= 325.9$, $p < .05$, $\eta^2= 0.63$, 95% CI [.52, .71] but not for the extinction cue group $F(9,7)= 1.1$, $MSE= 338.9$, $p > .05$.

The mean US expectancy for X in extinction and renewal tests is shown in Figure 2C. As it can be seen, the extinction cue reduced the recovery of US expectancy in extinction testing trials, but such trend is not clear in the renewal testing trials. Testing order did not affect US expectancy scores, test $F(1,28) < 1$. The overall US expectancy decreased with testing, as shown by a main effect of trial, $F(2,27)= 20.9$, $MSE= 566.5$, $p < .05$, $\eta^2= .60$, 95% CI [.53, .67]. Responding was renewed; US expectancy was reliably higher in the novel context, $F(1,28)= 6.7$, $MSE= 371.2$, $p < .05$, $\eta^2= .19$, 95% CI [.12, .26]. However, the extinction cue did not affected responding, $F(1,28) < 1$. All other interactions were non-significant (all *ps* >.05).

Following the same strategy used in Experiment 1, only the first of both trial types were analyzed. For these, there was no reliable effect of Context, $F(1,30)= 1.2$, $MSE= 2043.4$, $p < .05$, or cue, $F(1,30) < 1$. However, the Context x Cue interaction was significant, $F(1,30)= 4.4$, $MSE= 3018,1$, $p < .05$, $\eta^2= 0.12$, 95% CI [.00, .23]. Examination of this

interaction suggests that the extinction cue significantly reduced US expectancy during the extinction test, $F(1,30)= 4.1$, $p <.05$, but not during the renewal test, $F(1,30)= 0.4$, $p= .5$. Figure 2 illustrates the results of the US expectancy for the present Experiment.

Our procedure failed to produce differential changes in the SCR Acquisition. The ANOVA assessing these data revealed a main effect of Stimulus, $F(2,29)= 5.6$, $MSE= 8.81$, $p <.05$, $\eta^2= .15$, 95% CI [.00, .37], of Trial, $F(3,28)= 6.0$, $MSE= 7.5$, $p <.05$, $\eta^2= 0.16$, 95% CI [.00, .38], and a Trial x Cue interaction, $F(3,28)= 3.9$, $MSE= .98$, $p <.05$, $\eta^2= 0.11$, 95% CI [.00, .33]. No other interactions were significant (all *ps* >.05). There were no differences between groups, $F(1,30) <1$. However, pairwise comparisons between the stimuli showed that the overall SIR for X was different to the SIR for Z ($p <.05$), but that the SIRs between Y and Z, and X and Y were not different (both *ps* >.05). Since the analysis of conductance did not fulfill the criteria of a differential response between the excitatory (X and Y) and inhibitory stimuli (Z), no further analyses of these data were performed.

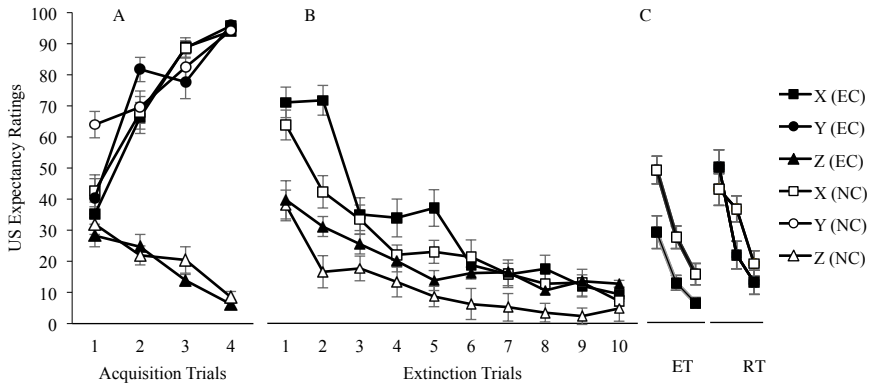

Figure 2. Means US expectancy across the different phases of Experiment 2. A rating of 1indicates security that the electric shock will not occur, and a rating of 100 indicates that the electric shock will surely occur. Closed and open symbols represent the extinction cue and neutral cue groups, respectively. (A) Mean US expectancy produced by X, Y and Z during the acquisition phase. (B) Mean US expectancy produced by X and Z during the extinction phase. (C) Mean US expectancy produced by X during the first round of testing, 5 minutes after extinction training ended; ET= Extinction Test; RT= Renewal Test.

Discussion

As in Experiment 1, in Experiment 2 participants learned to discriminate between the excitatory and inhibitory stimuli during acquisition. This difference in US expectancy disappeared across extinction. However, the EC was only effective in reducing the response recovery in the extinction context. Importantly, US expectancy among participants tested with a neutral cue was the same in both extinction and renewal contexts. Although these results suggest the absence of renewal, a comparison between experiments shows that what really occurred was a greater response recovery in the extinction context for the Neutral Cue Group compared to the Extinction Cue Group (see Figure 2C). This suggests that the cues acquired inhibitory properties during the extinction procedure (see General discussion). Finally, even with the introduction of long intervals between acquisition trials, and the introduction of an habituation phase, we did not manage to produce a differential SCR for the excitatory and inhibitory stimuli.

General Discussion

In Experiment 1, we observed that previously extinguished conditioned response (in the form of US expectancy) could be recovered after a context change. Furthermore, this recovery appears to be impaired by the presence of a cue presented during extinction. However, this impairment was not statistically reliable.

Since we compared performance to stimuli presented in a novel but familiar context with the performance to stimuli presented in their extinction context, both 5 minutes after extinction training was finished, we may conclude that renewal of responding in both groups was due only to the context change, and not to other factors such as an incomplete extinction (Vervliet *et alii*, 2012). This feature is particularly important; other studies in renewal of human fear conditioning have not considered that the low response in the last extinction trial may be due to non-associative factors. We also disregard the possibility that the response recovery was caused by the summation of the associative strength of the context and the CS; given the ABC design where our test occurred in a novel but familiar context, and not in the acquisition context as in the ABA renewal design (Vervliet *et alii*, 2012), which has been widely used in studies of human fear conditioning.

However, the tests performed 48 hours after extinction showed no time-induced recovery in any of the groups; and that response recovery caused by a context change vanished. This lack of response recovery makes it difficult to draw conclusions about the effectiveness of the extinction cue. One possible explanation of these findings is that the first round of testing resulted in the strengthening of the CS-nonUS association. After the first testing phase the participants had a considerably higher amount of training in which the CS was not followed by the US, compared to training of a CS-US association. Thus, testing the participants 48 hours later might have made them more prone to recover the extinction memory.

Given the transient nature of the extinction cue effect, response recovery was most strongly reduced on the first trial of testing. In Experiment 2 we increased the salience of the cue, leading to the extinction cue reducing the recovery of US expectancy in the cue's extinction context, but not doing so in a novel context.

Our failure to obtain a reliable reduction of renewal through the use of extinction cues is consistent with Culver *et alii* (2011) who reported that cues for the recovery of the extinction memory failed to reduce the renewal of the fear of speaking in public. Likewise, our results are similar to those found by Dibbets *et alii* (2013), who reported that extinction cues failed to reduce the renewal of arachnophobia and Laborda *et alii* (2016) who found no effect of the EC in public speaking fear. These findings undermine the efficacy of extinction cues to attenuate the renewal, and support the contextual dependence of extinction learning (Bouton, 1993, 2002, 2004).

Considering the significant effect of the EC in the decrease of recovery in the extinction context, a potential mechanism for its effects could be the development of inhibitory properties during extinction. During extinction, the presence of the extinction cue, which also becomes associated with the absence of the US, partially prevents the extinction of the CS-US association. The extinction cue indicates the absence of the US, otherwise expected in the presence of the CS (Laborda & Miller, 2012).

When the CS was tested in its extinction context and in the absence of the extinction cue, the residual excitatory association after extinction might have expressed, increasing the CR. The opposite -a reduction of the CR- should occur when the extinction cue is

present, as it provides the inhibitory association acquired during extinction. This was precisely what we observed during the testing of the neutral and extinction cue groups in Experiment 2. However, note that this inhibitory association was not transferred to a novel context. As Dibbets *et alii* (2008) argued, if the extinction cue becomes a security signal due to conditioned inhibition, it should survive transfer; the extinction cue should decrease the response in the presence of a new context. We did not observe this in these experiment. In the same way, if the EC promotes the recovery of the extinction memory when the CS is presented during test, then this ability should transfer to other contexts than the one used during extinction (Laborda & Miller, 2012). One potential explanation to the failure in observing this effect in the present Experiments is the existence of an inhibitory configural association between the extinction cue and the extinction context (see Pearce, 1994). During test in a new context (i.e., C), this configuration present during test in B would be no longer present and the recovery would be caused by the presentation of a new configuration (Context C+EC) that was not presented during extinction. This explanation can be inferred from Nelson, San Juan, Vadillo Ruiz, Pérez, & León (2011) potential mechanisms to explain renewal. This configural association could have been enhanced by the change on the EC from Experiment 1 to Experiment 2, which would explain the decrease on the effectiveness of the cue from one Experiment to the other.

It is also important to emphasize that both of our experiments introduced experimental conditions that challenged the possibility of finding the effect of the cue, in contrast with other experiments in which this effect was found (Dibbets *et alii*, 2008, 2011; Vansteenwegen *et alii,* 2006, 2008; Vervliet *et alii,* 2010). We think this constitutes a contribution to the development of methods to study fear conditioning in humans.

Incorporating a neutral cue in the test allowed us to control the effect that the presence of a stimulus may itself have on the response. Introducing an extinction test allowed us to ensure that the renewal was not due to an incomplete extinction. Furthermore, the use of an ABC design allowed us to show that the renewal observed was not due to the contextual excitation, as may occur in ABA designs (Vervliet *et alii*, 2012). In addition, we controlled for differences in amount of exposure to the context of extinction and renewal, and that change compared to others human experiments in fear conditioning, as Lovibond *et alii* (1984) stated, makes the effect observed in absence of such control disappear.

Unfortunately, both experiments failed to find a reliable physiological measure. Even the increase of the interval between trials and the use of an habituation phase in Experiment 2 did not help to obtain stable responses. This failure to find reliable measures of conductance has been previously reported (Dibbets *et alii*, 2008).

From a clinical viewpoint, the lability of extinction learning undermines the long-term maintenance of the results of exposure therapy for anxiety disorders. Thus, an important challenge is to continue to work on the development of exposure/extinction procedures that will reduce the probability of relapse/reappearance of the fear after the procedure. It is highly likely that a single technique will not be sufficient to reduce the probability of response recovery, but rather several techniques must be combined to guarantee long-term results.

References

Aristizabal JA, Callejas Aguilera JE, Ogallar PM, Pellón R, & Rosas JM (2015). Context Specificity of Extinguished Schedule-Induced Drinking Within an ABA Renewal Design in Rats. *Psicologica, 36,* 337-366.

Barlow DH, Bullis JR, Comer JS & Ametaj AA (2013). Evidence-based psychological treatments: An update and a way forward. *Annual Review of Clinical Psychology, 9,* 1-27. Doi: 10.1146/annurev-clinpsy-050212-185629.

Bernal Gamboa R, Juarez Y, González Martín G, Carranza R, Sánchez Carrasco L, & Nieto J (2012). ABA, AAB and ABC renewal in taste aversion learning. *Psicologica, 33,* 1-13.

Bouton ME (1993). Context, time, and memory retrieval in the interference paradigm of Pavlovian learning. *Psychological Bulletin, 114*, 80-99. Doi: 10.1037/0033-2909.114.1.80.

Bouton ME (2002). Context, ambiguity and unlearning: sources of relapse after behavioral extinction. *Biological Psychiatry, 52,* 976-986. Doi: 10.1016/s0006-3223(02)01546-9.

Bouton ME (2004). Context and behavioral processes in extinction. *Learning and Memory*, *11*, 485-494. Doi: 10.1101/lm.78804.

Bouton ME, Mineka S & Barlow D (2001). A Modern learning theory perspective on the etiology of panic disorder. *Psychological Review, 108,* 4 -32. Doi: 10.1037/0033-295x.108.1.4.

Brooks DC & Bouton ME (1994). A retrieval cue for extinction attenuates response recovery (renewal) caused by a return to the conditioning context. *Journal of Experimental Psychology: Animal Behavior Processes, 20*, 366-379. Doi: 10.1037//0097-7403.20.4.366.

Brooks DC, Vaughn JM, Freeman AJ & Woods AM (2004). And extinction cue reduces spontaneous recovery of ataxic ethanol tolerance in rats. *Psychopharmacology, 176,* 256-265. Doi: 10.1007/s00213-004-1882-y.

Collins BN & Brandon TH (2002). Effects of extinction context and retrieval cues on alcohol cue reactivity among nonalcoholic drinkers. *Journal of Consulting and Clinical Psychology, 70(2,* 390-397. Doi: 10.1037/0022-006X.70.2.390.

Chambless DL & Ollendick TH (2001). Empirically Supported Psychological Interventions: Controversies and Evidence. *Annual Review of Psychology, 52*, 685-716. Doi: 10.1146/annurev.psych.52.1.685.

Craske M, Liao B, Brown L, & Vervliet B (2012). The role of inhibition in exposure therapy. *Journal of Experimental Psychopathology*, *3*(3), 322-345. Doi: 10.5127/jep.026511.

Culver NC, Stoyanova M, & Craske MG (2011). Clinical relevance of retrieval cues for attenuating context renewal of fear. *Journal of Anxiety Disorders*, *25*(2), 284-292. Doi: 10.1016/j.janxdis.2010.10.002.

Derogatis CR (1992). *SCL-90-R. Administration, scoring, and procedures. Manual for the R(evised) version and other instruments of the psychopathology rating scale series*. Baltimore: John Hopkins University School of Medicine.

Dibbets P, Habermans R & Arntz A (2008). All we need is a cue to remember: the effect of an extinction cue on renewal. *Behaviour Research and Therapy, 46, 1070-1077*. Doi: 10.1016/j.brat.2008.05.007.

Dibbets P & Maes J (2011). The effect of an extinction cue on ABA renewal: does valence matter? *Learning and Motivation, 42,* 133-144. Doi: 10.1016/j.lmot.2010.12.003

Dibbets P, Poort H & Arntz A (2012). Adding imaginery rescripting during extinction leads to less ABA renewal. *Journal of Behavior Therapy and Experimental Psychiatry, 43,* 614-624. Doi: 10.1016/j.jbtep.2011.08.006.

Dibbets P, Moor C, & Voncken MJ (2013). The effect of a retrieval cue on the return of spider fear. *Journal of Behavior Therapy and Experimental Psychiatry*, *44*, 361-367. Doi: 10.1016/j.jbtep.2013.03.005.

Effting M & Kindt M (2007). Contextual control of human fear associations in a renewal paradigm. *Behaviour Research and Therapy*, *45*, 2002-2018. Doi: 10.1016/j.brat.2007.02.011.

Emmelkamp PMG (2013). Behavior therapy with adults. In Lambert (Ed), *Bergin and Garfield's handbook of psychotherapy and behavior change (pp. 343-392)*. New Jersey: John Wiley & Sons, Inc.

Gempp R & Avendaño C (2008). Datos normativos y propiedades psicométricas del SCL-90-R en estudiantes universitarios chilenos. *Terapia Psicológica, 26,* 39-58. Doi: 10.4067/s0718-48082008000100004.

Laborda MA, McConnell BL, & Miller RR (2011). Behavioral techniques to reduce relapse after exposure therapy: Applications of studies of experimental extinction. In TR Schachtman & S Reily (Eds.), *Associative learning and conditioning theory: Human and non-human applications (pp. 79-103)*. New York: Oxford University Press.

Laborda MA & Miller RR (2012). Reactivated memories compete for expression after Pavlovian extinction. *Behavioural Processes*, *90*(1), 20-27. Doi: 10.1016/j.beproc.2012.01.012.

Laborda MA, Schofield CA, Johnson EM, Schubert JR, George-Denn D, Coles ME, & Miller RR (2016). The Extinction and Return of Fear of Public Speaking. *Behavior Modification*, *40,* 901-921. Doi: 10.1177/0145445516645766.

Lissek S, Powers AS, McClure EB, Phelps EA, Woldehawariat G, Grillon C, & Pine DS (2005). Classical fear conditioning in the anxiety disorders: A meta-analysis. *Behaviour Research and Therapy*, *43*, 1391-1424. Doi: 10.1016/j.brat.2004.10.007.

Lovibond PF, Preston GC, & Mackintosh NJ (1984). Context specificity of conditioning, extinction, and latent inhibition. *Journal of Experimental Psychology: Animal Behavior Processes*, *10*, 360-375. Doi: 10.1037/0097-7403.10.3.360.

Nelson JB, Sanjuan MDC, Vadillo-Ruiz S, Pérez J, & León SP (2011). Experimental renewal in human participants. *Journal of Experimental Psychology: Animal Behavior Processes*, *37*, 58-70. Doi: 10.1037/a0020519.

Pavlov I (1927). *Conditioned reflexes*. London: Oxford University Press.

Pearce JM (1994). Similarity and discrimination: A selective review and a connectionist model. *Psychological Review*, *101*, 587-607. Doi: 10.1037/0033-295X.101.4.587.

Prokasy WF & Kumpfer KL (1973). Classical conditioning. In WF Prokasy and DC Raskin (Eds.), *Electrodermal activity in psychological research* (pp. 157-202). New York: Academic Press.

Rosas JM, García Gutiérrez A, & Callejas Aguilera JE (2007). AAB and ABA renewal as a function of the number of extinction trials in conditioned taste aversion. *Psicologica, 28, 129*-150.

Spielberger C, Gorusch R, & Lushene R (1970). *Manual for the State Trait Anxiety Inventory*. Palo Alto, California: Consulting Psychologist Press.

Spoormaker VI, Sturm A, Andrade KC, Schröter MS, Goya Maldonado R, Holsboer F, Wetter TC, Sämann PG, & Czisch M (2010). The neural correlates and temporal sequence of the relationship between shock exposure, disturbed sleep and impaired consolidation of fear extinction. *Journal of Psychiatric Research, 44*, 1121-1128. Doi: 10.1016/j.jpsychires.2010.04.017.

Vansteenwegen D, Ibérico C, Vervliet B, Marescau V, & Hermans D (2008). Contextual fear induced by unpredictability in a human fear conditioning preparation is related to the chronic expectation of a threatening US. *Biological Psychology, 77,* 39 -46. Doi: 10.1016/j.biopsycho.2007.08.012.

Vansteenwegen D, Vervliet B, Hermans D, Beckers T, Baeyens F, & Eelen P (2006). Stronger renewal in human fear conditioning with an acquisition retrieval cue than with an extinction retrieval cue. *Behaviour Research and Therapy, 44,* 1717-1725. Doi: 10.1016/j.brat.2005.10.014.

Vera Villarroel P, Celis Atenas K, Córdova Rubio N, Buela Casal G, & Spielberger CD (2007). Análisis preliminar y datos normativos del Cuestionario de Ansiedad Estado-Rasgo en Adolescentes y Adultos de la Ciudad de Santiago de Chile. *Terapia Psicológica, 25,* 155-162. Doi: 10.4067/S0718-48082007000200006.

Vervliet B, Baeyens F, Van den Bergh O, & Hermans D (2013). Extinction, generalization, and return of fear: A critical review of renewal research in humans. *Biological Psychology*, *92*, 51-58. Doi: 10.1016/j.biopsycho.2012.01.006.

Vervliet B, Craske MG, & Hermans D (2013). Fear extinction and relapse: state of the art. *Annual Review of Clinical Psychology*, *9*, 215-248. Doi: 10.1146/annurev-clinpsy-050212-185542.

Watson JB & Rayner R (1920). Conditioned emotional reactions. *Journal of Experimental Psychology, 3,* 1-14. Doi: 10.1037/h0069608.

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