

The HKU Scholars Hub

# The University of Hong Kong



Title	The role of stimulus specificity and attention in the generalization of extinction
Author(s)	Barry, TJ; Griffith, JW; Vervliet, B; Hermans, D
Citation	Journal of Experimental Psychopathology, 2015, v. 7 n. 1, p. 143- 152
Issued Date	2015
URL	http://hdl.handle.net/10722/244345
Rights	This work is licensed under a Creative Commons Attribution- NonCommercial-NoDerivatives 4.0 International License.

Running Head: Generalization of Extinction

# The role of stimulus specificity and attention in the generalization of extinction

Tom J. Barry<sup>1\*</sup>

James W. Griffith<sup>2</sup>

Bram Vervliet<sup>1</sup>

Dirk Hermans<sup>1</sup>

<sup>1</sup>Centre for Learning Psychology and Experimental Psychopathology, University of Leuven,

Leuven, Belgium

<sup>2</sup>Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

<sup>\*</sup>Corresponding Author:

Tom J. Barry

Centre for Learning Psychology and Experimental Psychopathology,

Psychology Faculty, University of Leuven

Tiensestraat 102 - Bus 3712

3000 Leuven, Belgium

#### Abstract

Exposure therapy for anxiety is effective but fear can still return afterward. This may be because the stimuli that people are exposed to are dissimilar from the stimuli to which fear was originally acquired.

After pairing an animal-like image (A) with a shock stimulus (US), a perceptually similar stimulus (B) was presented without the US in extinction. Participants were then shown A (ABA), a second generalization stimulus (ABC) or B (ABB).

Groups ABA and ABC evidenced a return of US expectancy relative to participants who were shown B (ABB). Participants in group ABC who self-reported high levels of attentional control evidenced greater return of expectancy relative to participants low in attentional control. Participants with a high level of attentional control also showed steeper extinction gradients.

Attentional control may influence perceptions of similarity and the learning that follows. Making note of such differences may be valuable in exposure treatment for anxiety.

TOM J. BARRY

#### 1. Introduction

Exposure therapy is one of the most efficacious remedies for anxiety disorders such as specific phobia and social anxiety disorder (Craske & Mystkowski, 2006). However, inhibitory models of the learning that is thought to take place during exposure therapy, suggest that expectancy of an aversive event, and the fear response that accompanies this, can return after the fears are extinguished or treated (Vervliet, Craske & Hermans, 2013). It is therefore crucial to better understand the mechanism by which expectancy of aversive events can be extinguished and can return so that exposure therapy can be improved and the chances of clinical relapse be reduced.

Return of fear following extinction can occur when the stimuli (Vervliet, Vansteenwegen, Baeyens, Hermans & Eelen, 2005; Rowe & Craske, 1998) or contexts (Bouton, 2004; Culver, Stoyanova & Craske, 2011) that were present in extinction, (e.g., during exposure therapy) are not identical to those that were present when fear was originally acquired. In classical conditioning models of anxiety and exposure therapy, a conditional stimulus (CS; e.g., a dog) that elicits expectancy of a previously associated aversive unconditional stimulus (US; e.g., a dog bite) is presented repeatedly without the US until expectancy and the accompanying anticipatory fear response extinguish. Exposure to a CS without the anticipated US leads to the development of a new association between the CS and the absence of the US. This inhibitory association suppresses the previous, fear-eliciting, association between the CS and the US. However, original CSs are often inaccessible in the clinic and so exposure often involves stimuli that share some features of the CS - and so evoke expectancy of the US and a fear response - but which also have some of their own unique features (generalization stimuli; GS). These new stimulus features, which have never been paired with an aversive event, might impair the generalization of fear from the CS to the GS and they may be used to explain why the US does not occur. Any inhibitory learning that

3

subsequently develops may be dependent on the presence of these unique GS features; if the CS is encountered after treatment then expectancy of the US and fear can return and this return differs as a function of the similarity between the extinction stimulus and the acquisition stimulus (Vervliet, et al., 2005; Vervliet, Vansteenwegen & Eelen, 2006). For example, if someone is bitten by a black, long-haired dog, and the resulting fear for dogs is then treated by exposure to a blonde, long-haired dog, this change in hair colour might suggest that blonde dogs – rather than all dogs, or even dogs with long hair irrespective of colour – are safe. Subsequent encounters with black, long-haired dogs after treatment might lead to a return of fear.

Although there is clear value in testing whether conditional responding can return after extinction if a CS is encountered again, CSs to which fear was originally acquired may not be encountered after treatment either. In the previous example, it might also be the case that encountering another GS after treatment that possesses some CS features that were not present in extinction (e.g., a black short-haired dog) could also lead to a return of US expectancy and fear, and clinical relapse might occur. As such, research must now explore whether it is possible that fear and US expectancy can return after they have been extinguished, in treatment or otherwise, when stimuli that possess only some CS properties are subsequently encountered. From this it will be possible to explore the factors that contribute towards this return and to prevent it. This issue has been examined to some extent by Kalish and Haber (1963) who trained pigeons to peck at a disk illuminated by a light with a 550-micrometre wavelength (mµ). The pigeons then received extinction with lights of 550-, 540-, 530-, 520-, 510- or 490- mµ. They found that extinction with one of the generalization wavelengths (e.g., 520) led to a return of the pecking response if a second generalization wavelength was presented after extinction that was somewhere between the acquisition wavelength and the extinction wavelength (e.g., 530). Several studies using rats and humans

have also shown that fear can return after extinction if novel contexts (Bouton, 1988; Thomas, Larsen & Ayres, 2003) or stimuli (Rowe & Craske, 1998) are encountered. However, there has yet to be an examination of the role of perceptual similarity between acquisition, extinction and subsequently encountered stimuli, in the return of US expectancy or fear.

It is also important to examine how variability in the degree of this return might be influenced by individual differences. Differences in attention to the features of extinction stimuli that are in common with the original CS and which have previously been associated with threat may determine the extent of the return of US expectancy and fear after extinction. Anxious people have often been shown to attend preferentially to threat-relevant stimuli (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg & van IJzendoorn, 2007). Moreover, healthy people have shown preferential attention towards stimuli associated with aversive stimuli that have been experimentally manipulated (Raes, Koster, Van Damme, Fias & De Raedt, 2010). People with high levels of anxiety tend to show broader gradients of generalization from stimuli associated with threat to other related stimuli (Lissek, Rabin, Heller, Lukenbaugh, Geraci, Pine, Grillon et al., 2010). This may be due to increased attention to the properties of related stimuli that have previously been associated with threat and relative inattention to the unique properties of stimuli. Attending more to the features in common between an extinction GS and the original CS and any other similar stimuli (e.g. number of legs on a dog) rather than to its unique features (e.g., black hair vs. blonde hair) might increase CS/GS generalization and make any extinction learning that occurs with that GS more robust and therefore less susceptible to return.

Deficits in the control of attention and a tendency for attention to be captured by threat might be associated with attention to common features that have previously been associated with threat at the expense of attending to unique features that have never been 5

experienced along with an aversive stimulus. Paradoxically, this would mean that deficits in attentional control (AC), characteristic of the development and maintenance of anxiety disorders (Derryberry & Reed, 2002; Bar-Haim et al., 2007), would then be associated with more generalization of fear from an original CS to extinction GS and subsequently more robust extinction learning. People low in AC would then be expected to show less return of US expectancy and fear after extinction, and perhaps also exposure treatment. These effects would also be reflected in a rapid extinction of US expectancy at the start of extinction for people high in AC, as they perceive the extinction GS as dissimilar from the CS, and greater return of expectancy at test.

As a first step in investigating these issues, we tested whether extinction with a GS is sufficient to prevent a return of US expectancy after extinction when presented with a second GS that has some features of the original CS that were not present in extinction. We also examined whether individual differences in AC can predict return of expectancy after extinction and what the effects of these differences were on generalization and the progress of extinction.

### 2. Methods

#### 2.1 Participants

Participants were undergraduate students at the University of Leuven (Mean age: 21.2; *SD*: 3.2) who were given course credit for participation (*N*: 48; Females: 33). All participants provided written informed consent before the experiment began, and were informed that they were free to withdraw at any point.

### 2.2 Stimuli and Measures

CS/GS were *Fribbles*, artificial, three-dimensional, combinations of shape, colour and texture similar to real-world animals (Barry, Griffith, De Rossi & Hermans, 2014). Separate species of Fribble were used for the experimental (+) and control (-) stimuli (see Figure 1)

TOM J. BARRY

and were counterbalanced between participants. These control stimuli featured in every phase of the experiment and were never associated with the US. They differed from one another to the same extent as the experimental stimuli. This controlled for non-associative effects on US expectancy ratings (see Vervliet, Vansteenwegen & Eelen, 2004).

AC was measured using the emotional Attentional Control Scale (eACS), a 14-item self-report measure of modulation of AC by emotions such as fear, where responses are given on a 4-point scale from 1 (*almost never*) to 4 (*always*) (Barry, Hermans, Lenaert, Debeer & Griffith, 2013). The items of the eACS assess individual differences in focusing and shifting of attention in the presence of emotion. For example, 'My attention easily shifts to my emotions' and 'I am able to put my feelings aside when I need to focus'. In this study, Cronbach's alpha was .88. A high score on the eACS represents good attentional control and vice versa.

## 2.3 Procedure

Prior to the experiment starting, informed consent was obtained and the eACS was administered. During the experiment, the CS+ (A+) and CS- (A-) were first presented once each without the US during the habituation phase. In acquisition, A+ was paired with electrocutaneous stimulation (US; individually set at an "uncomfortable but not painful" level) six times and the A- was presented six times without the US. In extinction, there were 12 trials of the GS+ (B+) without the US and 12 GS- (B-) trials. Participants were then immediately tested with six trials of either A+ again (Group ABA; n = 16), B+ again (Group ABB; n = 16), or a second GS (C+) that shared some features of both A+ and B+ (Group ABC; n = 16)(see Figure 1 for stimuli) and the equivalent control stimulus was also presented six times.

In each of the 24 trials the CS/GS replaced a blank screen after 1s and it remained onscreen for 8s. If the trial included the US, this was delivered at 7.5s after stimulus onset.

There was an inter-CS/GS-interval of 18s +/- 2s (see Figure 2). During each CS presentation, prior to the US, participants were asked to rate their expectancy by clicking on a scale from zero ('certainly no shock') through five ('not sure') to ten ('certainly a shock'). Our focus on US expectancy rather than physiological indices of fear was due to technical error in the measurement of skin conductance and startle reflex. Nevertheless, US expectancy is an important component of the fear response; it acquires, extinguishes and returns along with fear (Boddez, Baeyens, Luyten, Vansteenwegen, Hermans & Beckers, 2013).

Larger US expectancy scores for the CS/GS+ relative to the CS/GS- was used as evidence of the acquisition of conditioning at the end of the acquisition phase and generalization of this conditioning to the start of the extinction phase. The absence of a difference at the end of the extinction phase was used as evidence of extinction. Finally, the return of greater expectancy for the CS/GS+ relative to the CS/GS-, and relative to the size of this discrimination at the end of extinction, was used as evidence of the return of expectancy at test. The size of this difference was expected to differ between groups as a function of the stimulus that was presented to each group.

### 2.4 Data Analysis

Between-group differences were analysed using Group (3 levels: ABA; ABC; ABB)  $\times$  CS (2 levels: CS/GS+; CS/GS-)  $\times$  Trial mixed ANCOVA with eACS scores entered as a covariate. Separate ANOVAs were conducted with the first and last trials of each phase to test the progression of learning within and between phases. Planned comparisons using Fisher's LSD test were used to examine within and between group differences in the size, and change between trials, in the discrimination between CS/GS+/- (see Vervliet, Vansteenwegen & Eelen, 2006). Because we had focused hypotheses concerning acquisition of a discrimination between A+ and A- and the generalization and eventual extinction of this discrimination to B+ and B-, planned *t* tests were used to compare the extent of these discriminations within and between each of the groups. Expectancy scores for the CS/GSfrom the first trial of the test phase were subtracted from that for the CS/GS+ to form a difference score that was used in correlational analyses. We also computed for each participant the percentage change in expectancy for B+ in each trial of extinction relative to the first extinction trial (e.g., the first trial of the B+ was considered 100%). This was then used to model the slope of change across extinction in terms of Area Under the Curve with respect to decrease (AUC<sub>d</sub>) using the percentage score for the last trial of extinction as the baseline to account for individual differences in the intercept of the extinction curve. Alpha level was set at .05.

#### 3. Results

A 3 × 2 × 2 (Group × CS × Trial) ANCOVA using the first and last trial of acquisition showed a significant main effect of CS, F(1, 42) = 10.80, p = .002,  $\eta_p^2 = .21$  and a CS × Trial interaction, F(1, 42) = 18.68, p < .001,  $\eta_p^2 = .31$ , with no other main effects or interactions. Planned comparisons showed that each group displayed no difference in expectancy between the CS+ and CS- at the start of the phase but then acquired a significant discrimination by the end of the phase (p < .001) with greater expectancy for the CS+ than the CS- (see Table 1).

Another  $3 \times 2 \times 2$  ANCOVA using the last trial of acquisition and the first trial of extinction showed that all groups displayed evidence of generalization of US expectancy, with a main effect of CS, F(1, 42) = 29.49, p < .001,  $\eta_p^2 = .41$ , and no main effects or interactions with Trial or Group. Groups ABB and ABC showed significantly greater expectancy for B+ than B- (p < .001), but group ABA did not show a significant discrimination. However, this effect can be explained by greater expectancy for B- in group ABA relative to the other groups, particularly group ABC (p = .026) whereas all groups showed similar expectancy to B+.

A  $3 \times 2 \times 2$  ANCOVA using the first and last trials of extinction showed significant main effects of CS, F(1, 42) = 7.45, p = .009,  $\eta_p^2 = .15$ , and Trial, F(1, 42) = 6.17, p = .017,  $\eta_p^2 = .13$ , and a CS × Trial interaction, F(1, 42) = 9.26 p = .004,  $\eta_p^2 = .18$ . There were no main effects or interactions with Group. All groups showed a significant decrement in expectancy for B+ from the first trial to the last trial of extinction (p < .001). Groups ABB and ABC did not show a significant discrimination between B+ and B- at the end of extinction. Group ABA now showed a moderately significant discrimination (p = .04). However, there were no significant differences in expectancy ratings for either B+ or Bbetween any of the groups. All groups showed similar levels of extinction of their US expectancy by the end of the extinction phase.

Finally, a 3 × 2 × 2 ANCOVA using the last trial of extinction and the first trial of test, showed significant Group × CS, F(2, 42) = 4.02, p = .025,  $\eta_p^2 = .16$ , Group × CS × Trial, F(2, 42) = 6.46, p = .004,  $\eta_p^2 = .24$ , interactions. There were significant increases in expectancy from B+ to A+ in Group ABA (p < .001), and from B+ to C+ in Group ABC (p < .005), and no increase for group ABB. Group ABA showed a significant difference between A+ and A- (*Mean Difference*: 5.55; *SE*: .98; p < .001) and group ABC showed a significant difference between C+ and C- (*Mean Difference*: 2.01; *SE*: .94; p < .05) whereas group ABB showed no difference between B+ and B- (*Mean Difference*: .79; *SE*: .79). There was greater expectancy for A+ in group ABA and C+ in group ABC than B+ in group ABB (p < .001). There was also a significant difference between groups ABA and ABC for these stimuli (p < .05). There were also significant Group × CS × eACS, F(2, 42) = 5.33, p = .009,  $\eta_p^2 = .20$ , and Group × CS × Trial × eACS, F(2, 42) = 5.33, p = .009,  $\eta_p^2 = .20$ , and Group × CS × Trial × eACS, F(2, 42) = 5.33, p = .009,  $\eta_p^2 = .20$ , and Group × CS × Trial × eACS, F(2, 42) = 5.33, p = .009,  $\eta_p^2 = .20$ , interactions to test was influenced by Group membership and also by individual differences in eACS scores.

#### **3.1** Generalization and attention

Groups ABA and ABB showed no correlations between the test discrimination and eACS scores. The discrimination between C+ and C- on the first trial of the test phase for group ABC showed a large correlation with scores on the eACS, r = .62, p = .010. A scatterplot of eACS scores against test discrimination scores revealed that the relationship was linear without outliers (Figure 4.). Group ABC participants who reported greater eAC in the presence of emotion showed greater return of expectancy after extinction. To test whether this effect was related to an association between eACS scores and extinction learning, a second correlation between eACS and the AUC<sub>d</sub> of percent change in expectancy for B+ across extinction was performed for group ABC. Higher eACS scores were associated with smaller AUC<sub>d</sub>, r = -.62, p = .011. Also, smaller AUC<sub>d</sub> was associated with higher test discrimination, r = -.54, p = .031, or less return of expectancy at test. In a regression model with test discrimination as the dependent variable and eACS and AUC<sub>d</sub> input as predictors, neither independently explained a significant amount of variance when input together but the overall model did,  $R^2 = .43$ , F(2, 15) = 4.80, p = .028. The model was no longer a significant predictor when the interaction between eACS and AUC<sub>d</sub> was entered,  $R^2 = .43$ , F(3, 15) =2.96, p = .075. Self-reports of emotional AC and the curve of extinction were independently associated with the return of US expectancy to a GS when extinction involved another GS.

### 4. Discussion

We investigated whether extinction of US expectancy by presenting a GS was sufficient at preventing a return of expectancy following extinction when the CS is presented again or a second GS that shared some non-extinguished CS features is presented. We also asked whether individual differences in AC might moderate this return of expectancy. The presence of a significant discrimination in US expectancy for A+ versus A- confirmed that participants acquired the contingency between A+ and US by the end of acquisition; that there was also greater US expectancy for B+ versus B- at the start of extinction confirmed that US expectancy had generalized perceptually from the CS+ to the GS+. However there was substantial decrement in expectancy, most notably in group ABA where no discrimination between B+ and B- was evident on the first trial of extinction. This was due to increased expectancy for the safe stimulus, B-. Importantly, there were also no differences between any of the groups in expectancy for B+.

Following extinction of this expectancy, the degree of return of expectancy between participants who were presented with the identical CS+ after extinction or a GS that shared some non-extinguished CS+ features was larger than the return for participants shown the extinction stimulus again. This finding contributes to other literature suggesting that fear can return after treatment if the therapeutic context (e.g., the room in which treatment is conducted) differs from an original conditioning context when clients subsequently enter the original conditioning context or a second novel context after treatment (Culver et al., 2011) and also when novel stimuli are presented after exposure for spider phobia (Rowe & Craske, 1998). It seems that the perceptual similarity between feared stimuli may contribute towards this process.

Perceptual similarity of the stimuli encountered after extinction relative to the original CS and extinction stimuli seems crucial in the prevention of return of US expectancy. Combining the findings of this investigation with those of Kalish and Haber (1963), it appears that stimuli that are more similar to the CS that are encountered after extinction with a GS should evoke greater return of US expectancy and perhaps also fear: the presence of more non-extinguished CS features in stimuli encountered after extinction with a GS, the greater the anticipation of the US. This has clinical implications because it suggests that expectancy of aversive events, previously associated with CSs but subsequently extinguished or treated through exposure therapy, may return after treatment if treatment stimuli are markedly dissimilar to CS or if stimuli are encountered after treatment that possess nonextinguished CS features.

The extent to which perceptual similarity of extinction stimuli influences return of expectancy, however, may differ as a function of individual differences in attention to CS features that are present in GSs. In our study, participants high in eAC who were presented with the second GS after extinction showed greater return of expectancy than participants low in eAC. That this effect was not present in group ABA may have been because there were so many non-extinguished CS+ features in the test stimulus in group ABA that there was robust return of US expectancy across all participants. With much of the variability in expectancy for A+ at test explained by the stimulus itself then there may have been little variance left to be explained by eAC.

It is also possible that the extent to which similarity and attention influence return of expectancy may also be influenced by the passage of time between the end of extinction, or exposure treatment, and encounters with previously feared stimuli. As our core hypotheses concerned the role of perceptual similarity and attention, we did not include a time gap between extinction and test as this may have brought additional confounds regarding individual differences in memory consolidation and retrieval. In clinical settings it is unlikely that treatment would be followed immediately by an encounter with a feared stimulus as in the current experiment. Although this might limit the clinical validity of our procedure - and may be the reason for the absence of return of expectancy in group ABB - we would expect that the extent of return of expectancy would only increase with greater time between the end of extinction and the test phase. This is because the extinction context is likely to acquire some inhibitory strength during the extinction phase. If there is a time gap between extinction and test, then the test phase may then represent a different context to the extinction context and this inhibitory strength will no longer limit return of US-expectancy (Vervliet et al.,

13

2013). Therefore, that we observe a return of expectancy in the ABA and ABC conditions suggests that these effects may be even greater in clinical settings with a gap between treatment and subsequent encounters with feared stimuli. Future research could examine the additional contribution of breaks between extinction and test on the return of US expectancy and fear.

Higher eAC was also associated with more rapid extinction relative to low eAC. This may have been because people higher in eAC quickly shifted their attention from the common, threatening, features between A+ and the extinction B+ to the features of B+ that had never been paired with the US. High eAC participants then reduced their expectancy of the US immediately after the extinction phase began. People low in eAC might have had their attention captured and maintained by the common, threatening, features between A+ and B+ and so did not immediately attend to the unique features of B+ and continued to expect the US for longer. Deficits in disengaging attention from threat have previously been associated with low AC whereas people with good AC have shown increased ability to shift away from threat (Derryberry & Reed, 2002). The effects of this could have been that, compared with people high in eACS, people low in eAC did not recognize the extinction GS as being as dissimilar from the CS+. This made their extinction learning and inhibition of US expectancy more generalizable to other GSs that also shared some CS features. This suggests that having low eAC and perhaps also an attention bias towards threat might be beneficial in the treatment of clinical anxiety, and in particular specific phobia, in terms of preventing return of fear after exposure treatment, relative to having high eAC. Consistent with this, there is evidence suggesting that threat-related attention biases can be predictive of improved response to treatment for a range of anxiety disorders (e.g., Legerstee, Tullen, Dierckx, Treffers, Verhulst & Utens, 2010; Price, Tone & Anderson, 2011; Waters, Mogg & Bradley, 2012; Niles, Mesri, Burklund, Lieberman & Craske, 2013).

Future research must now address the limitations of the present study by replicating these findings with physiological measures of fear. Following this, research should use measures of gaze fixation to test our hypotheses regarding attention to stimulus features on extinction learning, generalization and return of fear and from there whether it is possible to reduce the return of US expectancy associated with extinction with GSs, by increasing the similarity between extinction stimuli with original CSs. Nevertheless, the present study provides a preliminary investigation of the ways in which learning about CS-US contingencies can generalize to perceptually similar stimuli, can then be extinguished and can return after extinction and the possible role of attention in these processes.

#### References

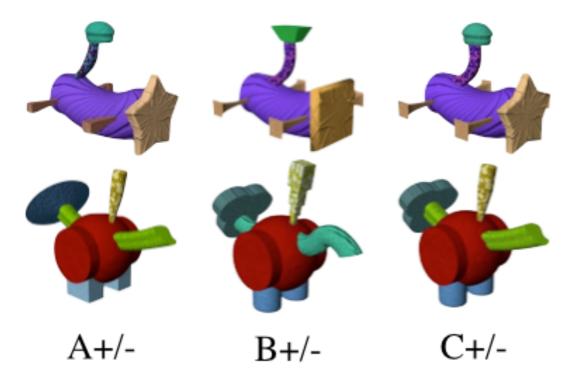
- Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: a meta-analytic study. Psychological Bulletin, 133, 1–24. doi:10.1037/0033-2909.133.1.1
- Barry, T. J., Hermans, D., Lenaert, B., Debeer, E., & Griffith, J. W. (2013). The eACS: Attentional control in the presence of emotion. *Personality and Individual Differences*, 55, 777–782. doi:10.1016/j.paid.2013.06.014
- Barry, T. J., Griffith, J. W., De Rossi, S., & Hermans, D. (2014). Meet the Fribbles: novel stimuli for use within behavioural research. *Frontiers in Psychology*, 5, 1–8. doi:10.3389/fpsyg.2014.00103
- Bouton, M. E. (1988). Context and ambiguity in the extinction of emotional learning:
  implications for exposure therapy. *Behaviour Research and Therapy*, 26, 137–149.
  doi:10.1016/0005-7967(88)90113-1
- Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning & Memory*, *11*, 485–494. doi:10.1101/lm.78804
- Craske, M. G., & Mystkowski, J. (2006). Exposure therapy and extinction: Clinical studies. In M. G. Craske, D. Hermans, & D. Vansteenwegen (Eds.), *Fear and learning: Basic science to clinical application* (pp. 213–233). Washington, DC: APA Books.
- Culver, N. C., Stoyanova, M., & Craske, M. G. (2011). Clinical relevance of retrieval cues for attenuating context renewal of fear. *Journal of Anxiety Disorders*, 25, 284–292. doi:10.1016/j.janxdis.2010.10.002

- Derryberry, D., & Reed, M. A. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology*, *111*, 225–236. doi:10.1037//0021-843X.111.2.225
- Legerstee, J. S., Tulen, J. H. M., Dierckx, B., Treffers, P. D. a, Verhulst, F. C., & Utens, E.
  M. W. J. (2010). CBT for childhood anxiety disorders: differential changes in selective attention between treatment responders and non-responders. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *51*, 162–172. doi:10.1111/j.1469-7610.2009.02143.x
- Lissek, S., Rabin, S., Heller, R. E., Lukenbaugh, D., Geraci, M., Pine, D. S., & Grillon, C. (2010). Overgeneralization of conditioned fear as a pathogenic marker of panic disorder. *American Journal of Psychiatry*, *167*, 47–55.
  doi:10.1176/appi.ajp.2009.09030410
- Niles, A. N., Mesri, B., Burklund, L. J., Lieberman, M. D., & Craske, M. G. (2013).
  Attentional bias and emotional reactivity as predictors and moderators of behavioral treatment for social phobia. *Behaviour Research and Therapy*, *51*, 669–679.
  doi:10.1016/j.brat.2013.06.005
- Price, M., Tone, E. B., & Anderson, P. L. (2011). Vigilant and avoidant attention biases as predictors of response to cognitive behavioral therapy for social phobia. *Depression* and Anxiety, 28, 349–353. doi:10.1002/da.20791

Raes, A. K., Koster, E. H. W., Van Damme, S., Fias, W., & De Raedt, R. (2010). Aversive conditioning under conditions of restricted awareness: effects on spatial cueing. *Quarterly Journal of Experimental Psychology*, 63, 2336–2358.
doi:10.1080/17470218.2010.492995

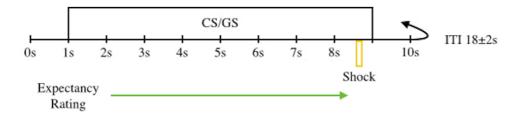
- Rowe, M. K., & Craske, M. G. (1998). Effects of varied-stimulus exposure training on fear reduction and return of fear. *Behaviour Research and Therapy*, *36*, 719–734. doi:10.1016/S0005-7967(97)10017-1
- Thomas, B. L., Larsen, N., & Ayres, J. J. (2003). Role of context similarity in ABA, ABC, and AAB renewal paradigms: Implications for theories of renewal and for treating human phobias. *Learning and Motivation*, 34, 410–436. doi:10.1016/S0023-9690(03)00037-7
- Vervliet, B., Craske, M. G., & 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
- Vervliet, B., Vansteenwegen, D., & Eelen, P. (2004). Generalization of extinguished skin conductance responding in human fear conditioning. *Learning & Memory*, 11, 555– 558. doi:10.1101/lm.77404.stimuli.
- Vervliet, B., Vansteenwegen, D., Baeyens, F., Hermans, D., & Eelen, P. (2005). Return of fear in a human differential conditioning paradigm caused by a stimulus change after extinction. *Behaviour Research and Therapy*, 43, 357–371. doi:10.1016/j.brat.2004.02.005
- Vervliet, B., Vansteenwegen, D., & Eelen, P. (2006). Generalization gradients for acquisition and extinction in human contingency learning. *Experimental Psychology*, 53, 132–142. doi:10.1027/1618-3169.53.2.132
- Waters, A. M., Mogg, K., & Bradley, B. P. (2012). Direction of threat attention bias predicts treatment outcome in anxious children receiving cognitive-behavioural therapy. *Behaviour Research and Therapy*, 50, 428–434. doi:10.1016/j.brat.2012.03.006

# Figure 1.



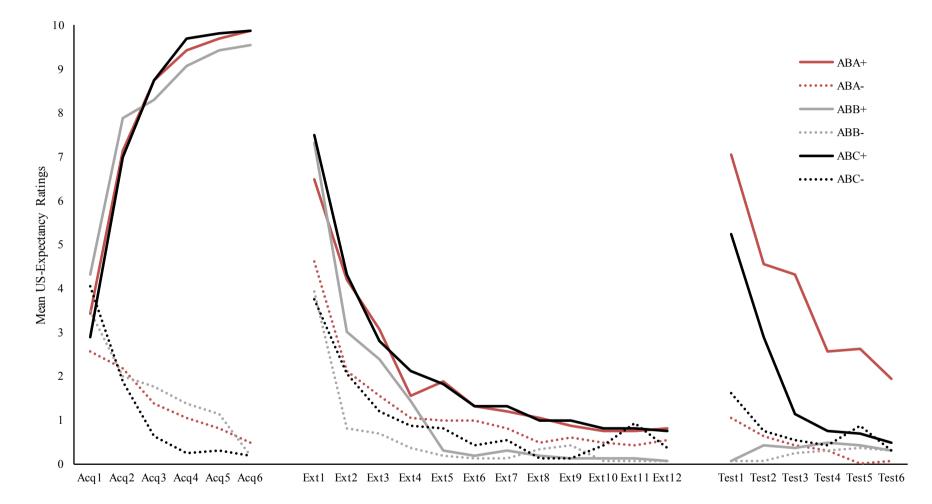
Note. Two species of artificial animal-like images, referred to as Fribbles, used as conditional stimuli (CS). Different species are used for CS+ and CS-. C includes two features unique to B and two features of A and one feature in common between all three–the central body.

# Figure 2.



Note. The flow of trials including conditional stimulus (GS) and generalization stimulus (GS) presentations. The shock is only present on 'conditional stimulus plus unconditional stimulus' (CS+) trials during the acquisition phase. Everything else remains the same for all stimuli in all other phases of the experiment.

# Figure 3.



Note. Mean unconditional stimulus (US) expectancy ratings in each trial of the experiment. Separate lines are shown for each of the conditional stimuli and generalization stimuli (CS/GS+ and CS/GS-) for each of the groups: ABA; ABB; and ABC

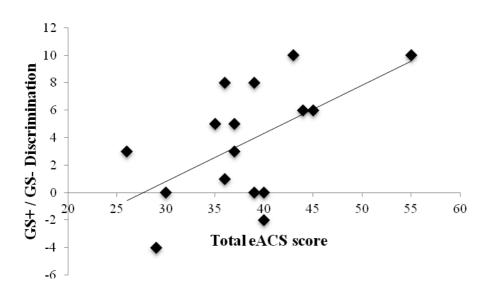


Figure 4.

Note. The relationship between total scores on the emotional Attentional Control Scale (eACS; Barry et al., 2013) and the return in unconditional stimulus (US) expectancy due to a stimulus change following extinction. The generalization stimulus (GS)+/- discrimination was calculated by subtracting scores for the GS- from scores for the GS+ on the first trial of each in the test phase.

# Table 1.

	Acquisition				Extinction				Test	
	Start		Start End		Start		End		Start	
	+	-	+	-	+	-	+	-	+	-
ABA	3.44 (2.34)	2.56 (1.83)	9.88(.34)	.50(.97)	6.50 (2.88)	4.63 (2.92)	.81 (1.94)	.56 (1.54)	7.06 (3.79)	1.06 (1.77)
ABB	2.88 (2.39)	4.06 (2.16)	9.88(1.09)	.19(1.47)	7.31 (1.96)	3.94 (2.74)	.06 (.25)	.06 (.25)	.06 (.25)	.06 (.25)
ABC	4.31 (1.93)	3.50 (2.32)	9.56(.50)	.81(.75)	7.50 (2.13)	3.75 (2.32)	.75 (1.24)	.37 (1.02)	5.25 (3.17)	1.63 (2.94)

Note. Mean unconditional stimulus (US) expectancy ratings at the start and end of each phase of the experiment for each stimulus and group. Values in parenthesis are one standard deviation.

## Acknowledgements

This research was supported by a research programme of the Research Foundation-Flanders (FWO) and by the Center of Excellence on Generalization Research (GRIP/TT; University of Leuven Grant PF/10/005). There are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.