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What to Expect When it's Unexpected:

A Multi-Theoretical Approach to Exposure Therapy

A dissertation submitted in partial satisfaction

of the requirements for the degree

Doctor of Philosophy in Psychology

by

Aaron Scott Baker

2012

ABSTRACT OF THE DISSERTATION

What to Expect When it's Unexpected:

A Multi-Theoretical Approach to Exposure Therapy

by

Aaron Scott Baker

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2012

Professor Michelle Craske, Chair

Cognitive Behavioral Therapy is the most widespread and effective intervention used for anxiety disorders. Our understanding of the underlying mechanisms is somewhat limited by divergent theories, as the most widespread theory, Emotional Processing Theory (Foa & Kozak, 1986), has not been empirically supported, and in fact, has been regularly refuted when tested. An alternative can be drawn from associative learning theory by employing the Rescorla-Wagner model (Rescorla & Wagner, 1972) to model change during exposure therapy. This model postulates that after a strong dose of exposure therapy to a target stimulus, the use of secondary exciter in conjunction with a target stimulus would allow additional learning to accrue to the target stimulus. Study 1 applied this model by treating specific phobia of spiders with a 40-minute prolonged exposure to two spiders on day one, then providing an additional day of exposure where the control group again received exposure to the two spiders, and the experimental group received exposure to one spider

and worms concurrently. Results indicated that the intended manipulation was not effective at increasing expectancy due to the inclusion of worms; however, process analyses revealed a relationship of between session activation leading to better treatment outcomes. Study 2 again applied this model by inflating the outcome expectancy belief on a second day of exposure, by providing the experimental group with information about the spiders being more aggressive than usual. In a pilot sample the results were mixed as to whether the intervention was effective at increasing expectancy of an adverse event occurring, though it did provide important information about the feasibility of utilizing scripts to inflate outcome expectancy without risking inflated drop-out. While limited support was found for the model in these two studies, the large number of limitations warranted future work to be done with more acutely anxious samples. Further, future studies should look to employ more powerful forms of secondary exciters to reveal effects above and beyond the already powerful intervention.

The dissertation of Aaron Scott Baker is approved.

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2012

This work is dedicated to my wife Stephanie, who has always stood by me and provided the support and encouragement I needed to achieve what needs to be done.

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PUBLICATIONS

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Layne, C. M., Olsen, J. A., **Baker, A.**, Legerski, J., Isakson, B., Pašalić, A., Duraković-Belko, E., Đapo, N., Ćampara, N., Arslanagić, B., Saltzman, W. R., & Pynoos, R. S. (2010). Unpacking trauma exposure risk factors and differential pathways of influence: Predicting postwar mental distress in bosnian adolescents. *Child Development, 81*(4), 1053-1076.

Baker, A., Mystkowski, J., Culver, N., Yi, R., Mortazavi, A., & Craske, M. (*in press*) Disconfirmation of Expectancy in Exposure Therapy for Acrophobia. *Behavior Research & Therapy*

Vervilet, B., **Baker, A.**, Craske, M. (*in press*) Fear conditioning in animals and humans. In N.M. Seel & D. Quinones (Eds.) Encyclopedia of the sciences of learning. New York, NY: Springer Science+Business Media.

Baker, A., Bjork, R.A. & Craske, M. (*submitted*) Retrieval induced forgetting with traumatic imagery: An exploration of clinical utility. *Cognition and Emotion*

Kirkanski, K., Mortazavi, A., Castriotta, N., **Baker, A.**, Mystkowski, J., Yi, R., & Craske, M. (in prep). Stimulus and Emotion Variability in Exposure Therapy for Contamination Fears. *Journal of Behavior Therapy and Experimental Psychiatry*

Arch, J., Ayers, C., ***Baker, A.****, & Craske, M. (in prep)* Randomized Clinical Trial of MBSR and CBT in group treatment of anxiety disorders in the VA.

Baker, A.*, Arch, J., Ayers, C., Espejo, N., Niles, A.,& Craske, M. (in prep)* Differential treatment effects of MBSR and CBT in attentional bias paradigm.

PRESENTATIONS

Aikins, D.E., Borelli, J.L., & **Baker, A.** (October 2004) Phasic heart period reactions to cued affective stimuli in combat-related posttraumatic stress disorder. Symposium, 44th Annual Meeting of the Society for Psychophysiological Research, Santa Fe, NM.

Baker, A., Mystkowski, J, Craske, M (2007) Emotional Processing Theory in the Treatment of Acrophobia. Poster, 41st Annual Association for Behavioral & Cognitive Therapies Convention, Philadelphia, PA.

Baker, A., Mystkowski, J, Craske, M (2008) Exposure Therapy for Acrophobia: Does Within-session Habituation Matter? Poster, 116th American Psychological Association Annual Conference, Boston, MA.

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Baker, A., Mystkowski, J, Craske, M (2009) Exposure Therapy for Acrophobia: Does Within-session Habituation Matter? Symposium Presentation, 43rd Annual Association for Behavioral & Cognitive Therapies Convention, New York, NY.

Baker, A. & Craske, M (2010) Forgetting Trauma: Decreasing Trauma Memory Strength Through Retrieval Induced Forgetting. Poster, 44th Annual Association for Behavioral & Cognitive Therapies Convention, San Francisco, CA.

Baker, A., Castriotta, N., & Craske, M. (2010) Emotional Processing Theory in Treatment of OCD. Poster, 44th Annual Association for Behavioral & Cognitive Therapies Convention, San Francisco, CA.

Baker, A., Mortazavi, A., Loerinc, A., Craske, M. (2011) Exposure therapy and surprise: Using the rescorla-wagner in clinical treatment. Symposium Presentation, 45th Annual Association for Behavioral & Cognitive Therapies Convention, Toronto, ON, Canada.

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What to Expect When it's Unexpected: A Multi-Theoretical Approach To Exposure Therapy

Cognitive Behavioral Therapy

Cognitive behavioral therapy (CBT) is an effective form of treatment for a variety of psychological disorders. It has been judged to be an efficacious treatment for anxiety, due to the plethora of support it has received in the treatment of various anxiety disorders. In fact, it is considered to have the most extensive evidence for effectiveness among all psychological treatments (Roth & Fonagy, 1996). Additionally it is one of the most common modalities of treatment currently being practiced in the United States. A randomly sampled survey of 591 APA members indicated that 45.4% regarded themselves as CBT in theoretical orientation (Stewart & Chambless, 2007), exceeding all other theoretical orientations, including psychodynamic (21.9%), eclectic (19.8%), humanistic/experiential (4.4%), family systems (3.9%) and other (4.6%)

While there is variation in the way that CBT is practiced (Stobie, Taylor, Quigley, Ewing, & Salkovskis, 2007), and the inclusion of certain components (e.g. breathing retraining) can vary, at its base CBT is a collection of strategies that target two specific domains: cognitions (i.e. cognitive restructuring) and behaviors (i.e. exposure). The cognitive component initially came as part of the 'second wave' of behavioral therapy, and focuses upon restructuring maladaptive cognitions in order interrupt the effects of negative thoughts on emotion (Craske, 1999). While this has become a popular and widely practiced portion of CBT, support for the cognitive component independently improving treatment outcome has been mixed, and at best the causal effects of cognitive interventions are confounded with other treatment factors (Rachman, 1993; Arch & Craske, 2009).

There is evidence that behavioral interventions alone are just as effective as cognitive interventions or combined CBT interventions (e.g. Borkevec, Newman, Pincus, & Little, 2002). This finding calls into question the efficacy of including additional time-consuming components

like cognitive strategies in interventions, due to the additional financial burden. It is incumbent upon researchers to develop treatments that not only provide the best treatment possible, but also do so at the lowest cost (in terms of both financial and non-financial resources) possible in order to encourage wide dissemination of treatment procedures.

Some have even argued that the cognitive strategies are in fact watered-down forms of exposure (Boyd & Levis, 1983; Marks, 1987). If this is in fact the case, it may be more efficient to engage in straight-forward exposure techniques, rather than second-order cognitive techniques even if they accomplish similar results.

Return of Fear

Regardless of what the active ingredients in cognitive and behavioral strategies are, treatments still fall somewhat short, as not all people respond to treatment (reports of non-responders vary from 10-30%, see Craske, 1999 for a review), and people who do respond generally to not achieve a complete reduction in fear (Rachman, 1989). While these two scenarios are troubling, they are not nearly as problematic as the phenomenon of return of fear (ROF; Rachman, 1979), where subjects experience a recovery of their fear response after treatment has completed. ROF is operationalized as showing a fear response after treatment is completed, that is greater than the fear response evidenced at the end of treatment. Rates of ROF vary; Craske, Sanderson, & Barlow (1987) found that 19% of subjects evidenced ROF after treatment for Agoraphobia, while Rachman & Lopatka (1988) found that 62% of a sample of snake phobics showed ROF one month after treatment. The varying rates are likely a function of the relatively arbitrary distinction used to classify ROF, thus it has been suggested that it may be more useful to simply describe the degree of ROF via change scores (Craske, 1999). By addressing the problem of ROF, we may be able to address all three shortcomings of behavioral approaches to anxiety

disorders mentioned above. If it is possible to make treatments more effective in the first place, it may be possible to consolidate the long term gains that result from treatment. In order to develop strategies to do this, we must first gain a better understanding of the mechanism by which exposure is purported to work.

This task is a bit more complicated than it seems, as even within the relatively narrow spectrum of behavioral explanations there has been some contentious debate over the proper conceptualization of how exposure therapy functionally changes outcome. There are two major behavioral traditions, one that draws upon the non-associative behavioral tradition (combined with some cognitive explanations), and one that draws upon associative behavioral conditioning. Herein, 'conditioning' and 'learning' will be used interchangeably to describe the strengthening of the association between two stimuli or events.

Non-Associative Theory

Non-associative conditioning pertains to processes whereby observable behaviors (responses {R}) resultant from a stimulus (S) change due to repeated presentations of the S. Two non-associative learning processes have been identified: Sensitization and Habituation. Sensitization refers to a process where a response to a given stimulus increases over multiple presentations, while habituation refers to a decrease in responding to a given stimulus over multiple presentations. These two processes are understood to occur at the same time, and work in opposition to one another (Groves & Thompson, 1970). While there have been other theories about the underlying mechanism of these processes (e.g. Wagner, 1976), the current understanding is that the changes occur along a simple S-R neuronal pathway (Mackintosh, 1987). Sensitization is purported to be driven by activation over the state system (i.e. non-specific arousal), whereas habituation reflects a decrease in the efficiency of communication along the S-R system (Groves &

Thompson, 1970). Sensitization, while possibly a relevant issue pertaining to return of fear, is not particularly relevant to the discussion of fear reduction, as its effects are generated from non-specific arousal characteristics. Habituation on the other hand is a process that may be relevant to exposure therapy as a potential mechanism of fear reduction. When habituation occurs, subjects show a decrement in response to a S due to repeated presentations of the S. For example if a loud noise (S) is played, people will show a startle response (R); however over repeated presentation of the loud noise, the startle response will decrease in magnitude. Related to the concept of habituation is the opponent mechanism referred to as dishabituation, where the response is recovered in the presence of a second distracting stimulus (Thompson & Spencer, 1966). This phenomenon may be related to return of fear post-treatment if habituation is the process underlying treatment effects.

This concept of habituation was combined with the concept of 'corrective learning' to explain the effects of exposure therapy in the widely known "emotional processing" theory (EPT), proposed by Foa and Kozak (1986) and subsequently revised (Foa & McNally, 1996). EPT purports that the effects of exposure therapy derive from activation of a 'fear structure' and integration of information that is incompatible with it, resulting in the development of a non-fear structure that replaces (Foa & Kozak, 1986) or competes with (Foa & McNally, 1996) the original one. A 'fear structure', as first put forth by Lang (1971), is a set of propositions about a stimulus (e.g., spider), response (e.g., racing heart) and their meaning (e.g., "I will be poisoned") that are stored in memory. The fear structure is posited to be activated by inputs that match part of the structure (such as a spider, a racing heart, or a thought about poisoning), which generalizes to activate other parts of the structure.

Once activated, corrective learning is purported to occur through integration of information that is incompatible with the structure. Incompatible information derives from two primary sources. The first is within-session habituation (WSH) where fear responding reduces with prolonged exposure to the fears stimulus. WSH is considered a necessary pre-requisite for the second piece of incompatible information, which derives from between-session habituation (BSH) over repeated occasions of exposure. BSH is purported to form the basis for long term learning, and to be mediated by changes in the meaning proposition, in the form of lowered probability of harm (i.e., risk) and lessened negativity (i.e., valence) of the stimulus.

Hence, according to this theory success is indexed by initial fear activation (IFA), WSH and BSH of the fear response. EPT clearly guided the focus of exposure therapy upon initial elevation followed by within- and between-session reductions in reported fear and physiological arousal, as continuation of those responses was presumed to represent erroneous evaluation of the probability of risk and negative valence. While this theory is enticing in its face validity, support for it has been inconsistent at best. Recent attempts to test the theory have found minimal support for the components of the theory, and no support for the complete theoretical framework as a whole (Baker, Mystkowski, Culver, Yi, Mortazavi, & Craske, 2010). A broader review of the literature concerning this theory by Craske et al. (2008) showed that there is very little support for any of assumptions of EPT. The extant evidence neither consistently supports nor refutes IFA effects. Some studies have found partial support for IFA relating to amount of change post-treatment, notably when heart rate is used as the index of IFA (e.g. Lang, Melamed, and Hart, 1970; Kozak, Foa, & Steketee, 1988; Pitman, Altman, & Longpre, 1996a), though other studies fail to demonstrate such a relationship (e.g. Foa, , Steketee, Doppelt, Turner, & Latimer, 1983; Pitman, Altman, & Longpre, 1996b; Telch, Valentiner, Ilai, Young, Powers, & Smits, 2004), and further

there is no evidence supporting self-report of skin conductance indices of IFA relating to outcome IFA (e.g. Lang et al., 1970; Kozak et al., 1988; Pitman et al., 1996a). Several studies have shown the opposite of EPT's assumption about WSH, with subjects showing *greater* improvement when exposure was terminated before WSH occurred (e.g. Emmelkamp & Mersch, 1982; Rachman, Craske, Tallman, & Solyom, 1986). The majority of studies do not find support for WSH as a predictor of outcome with either physiological (e.g. Pitman et al., 1996b) or verbal (e.g. Pitman et al., 1996a) indices. The evidence for BSH is limited to heart rate (e.g. Kozak et al., 1988) and indeed its effects are contraindicated by pre to post-treatment improvements in the absence of significant reductions in physiological fear indices over days of exposure in subjects that are fearful of spiders (Rowe & Craske, 1998), heights (Lang & Craske, 2000) and public speaking (Tsao & Craske, 2000).

Associative Theory

An alternative to EPT is drawn from the basic science on associative conditioning (i.e. excitatory and inhibitory learning). Associative conditioning (e.g. Pavlov, 1927) supposes that when an unconditioned stimulus (US), which causes an unconditioned response (UR) regardless of previous learning, is paired with a neutral stimulus (conditioned stimulus {CS}), the neutral stimulus will be begin to elicit a response (conditioned response {CR}) due to that association. For example, if a loud noise (US), which causes a subject to show a startle response (UR), is paired with a tone (CS); then the subjects will begin to show the startle reaction (CR) to the tone alone (CS). This is called excitatory conditioning, whereby responding is increased due to the association. This process has long been used as an explanatory mechanism for the acquisition of fear.

There is also associative learning which decreases responding to a conditioned stimulus; this

is known as inhibitory learning. Inhibitory learning occurs when a CS (that has been previously paired with a US) is presented in the absence of a US, resulting in a decrease in the CR. This is the process that underlies the phenomena of extinction, which has been used as an explanatory mechanism for the effects of exposure therapy (i.e. the extinction of fear responding). It is important to note, the while these processes are presupposed to underlie the processes of fear acquisition and extinction, these models have been most thoroughly examined in animal models, and less so with human subjects.

Initially it was theorized that the mere co-occurrence of the CS and US is what drove learning (Contiguity Theory; Guthrie, 1935), but this theory was later refined to focus more on the contingency between the two events (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972). The driving force behind this refinement was a number of results that indicated that contiguity between the CS and US was necessary, but *not* sufficient for conditioning to occur (e.g. Kamin, 1968, Rescorla, 1969; Wagner, 1969). To further explain, the simplest and most potent experiment will be outlined here. The curious result referred to as Kamin's blocking experiment (Kamin, 1968, 1969) uncovered a phenomena where it is possible to keep a CS from becoming associated with a given US despite the CS receiving many pairings with the US. In the procedure, Kamin first conditioned a stimulus (A) with a US (+) until learning reached asymptote. In the second phase, the previously conditioned stimulus was presented in compound with a second stimulus (B) and paired with the same US. At test, subjects showed a large CR to stimulus A, but no CR to stimulus B.

Phase 1	Phase 2	Test
A+	AB+	B

In effect, the previous conditioning to stimulus A 'blocked' the potential conditioning to stimulus B. This result was troubling to contiguity theory, which postulated that a CS and US co-occurring in time was both necessary and *sufficient* for learning to take place.

This result, among others, led to the development of contingency theory, which postulated that when a given stimulus *predicted* the greater likelihood of the occurrence of US learning can occur. The most prevalent theory for how this mechanism works is the Rescorla-Wagner model (RWM; Rescorla & Wagner, 1972; Wagner & Rescorla, 1972). The RWM relies on the assumption that subjects take in cues (CSs) from the environment around them and uses them to calculate the excitatory and inhibitory consequences (USs) of their situation. The assumption here is that if the subject expects something bad to happen and it does not occur, then inhibitory learning will occur and will result in a reduction (i.e. extinction) of the fear response (CR). Conversely if a more negative experience occurs than expected, excitatory conditioning will occur, leading to an increase (i.e. Acquisition) of the fear response. This model has accounted for a number of difficult to explain phenomena in animal conditioning models (Pearce & Bouton, 2001), but has been highly under-utilized in guiding human research. The RWM also posited a linear mathematical relationship between expectancy and learning, which will be elaborated upon later.

A model based on inhibitory learning may be the most promising in exploring the concept of return of fear, as many phenomena relating to the strength of inhibitory learning dovetail with the observations of return of fear. One example is reinstatement (Rescorla & Heth, 1975), where a fear response returns following an excitatory experience with another stimulus (ostensibly another US). This process could be most easily confused with dishabituation, the process by which non-associative theories of exposure (e.g. EPT) use to explain ROF. Dishabituation differs from reinstatement, as dishabituation supposes that the expression of habituation (the non-associative learning) is *interrupted* by a distracting stimulus, whereas reinstatement assumes that when a new US incurs *new* learning occurs that compete with and potentially over-rides the inhibitory learning. The fundamental difference is that learning that causes reinstatement can happen long before the

expression of a fear response, while dishabituation relies on a stimulus at the time of exposure to the CS in order to allow the fear response to be expressed.

Another phenomena that has been observed in associative conditioning which may explain ROF observations is spontaneous recovery (Baum, 1988), whereby the fear response returns due to the simple passage of time. This phenomenon supposes that inhibitory associations are much more fragile than excitatory associations, and degrade more rapidly over time. While this theory may lack an easily observable mechanism, it has broad applications to the phenomenon of ROF.

Recently there has been more intense exploration of a third phenomena, renewal (Bouton, 1993), whereby inhibitory learning is associated with a context (in addition to the CS) which limits the full expression of the inhibitory learning outside the context where the inhibitory learning was accrued. In a sense context acts as the B stimulus in Kamin's (1968,1969) blocking experiment, so that when a subject encounters the fear stimulus (A) at a later time, they express fear, because the context (B) is not there to inhibit responding. Contexts have a broad, almost a-theoretical definition and encompass visible, auditory, olfactory, temporal, thermal, and internal (i.e. bodily sensations: e.g. nausea) cues (among others). Context renewal has been shown to occur in humans post exposure therapy (Rodriguez, Craske, Mineka, & Hladek, 1999; Minka, Mystkowski, Hladek, & Craske, 1999; Mystkowski, Craske, & Echiverri, 2002) though effect sizes have varied.

Rescorla-Wagner Model

Context may provide the most compelling explanation of what may underlie ROF, and if it does, the solution to the problem may be simpler than expected. The reason for this is the relative simplicity of the Rescorla-Wagner Model (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972) in explaining how context renewal occurs. Rescorla and Wagner themselves admitted that their explanations were surprisingly simple, yet powerfully explanatory, as they accounted for a number

of perplexing results in the animal literature (e.g. Kamin's (1968, 1969) blocking experiment). The RWM, hinges on a mismatch between expectancy and result, which Kamin (1969) had referred to as surprise. This was not entirely different from previous models that postulated that learning (herein referred to as associative strength, and symbolized by 'V') resulted from the product of a learning rate (Φ) and the difference between what was already learned (V_A), and what was left to be learned (λ) (e.g. Hull, 1943; Spence, 1956):

$$\Delta V_A = \Phi(\lambda - V_A)$$

The revolutionary aspect to the RWM was that it assumed that subjects used all the cues available (ΣV), and not simply the cue of interest. Previously, it had been assumed that the change in associative strength for a particular cue on a given trial occurred regardless of the other cues available, but this theory postulated that the amount left to be learned was limited by the associative strength of not only the cue of interest (V_i), but the sum of all cues present ($\Sigma V = V_{i1} + V_{i2} + \ldots$). Additionally, the model split the learning rate into two individual aspects, the salience of the CS (α) and the salience of the US (β). Finally, the total amount learned was set to be equal to that of the intensity of the US (λ), such that more intense USs were able to yield more intense CRs. The resultant formula was:

$$\Delta V_i = \alpha_i\beta(\lambda - \Sigma V)$$

Another crucial aspect of the RWM, is the mathematical relationship postulated to exist during inhibitory conditioning (extinction training). It was assumed that when extinction took place there was no US, thus the value for $\lambda = 0$ (though not well explained, it is assumed that β took on a relatively small value, but did not equal 0, despite the lack of an extant US). By setting $\lambda = 0$, if there is a net excitatory associative strength among the cues present (ΣV), the formula computes a negative number, which is thought to be indicative of inhibitory conditioning. The same additive

properties apply to inhibitory stimuli as to excitatory stimuli, such that the presence of a strong excitatory stimulus could result in no CR, if presented with an equally strong inhibitory stimulus (e.g. A = 2, B = -2; AB → No CR). This function of the formula likely explains why attempts to over-learn in exposure had yielded disappointing results (Farchione, 2002), as once learning reaches asymptote, further trials yield no additional decrement in responding because the stimulus of interest is blocking itself from acquiring further inhibitory conditioning.

The final key aspect to this formula relevant to this discussion is that all cues acquire associative strength simultaneously, on a per trial basis. For example a given single trial with X number of variables present would yield the following learning opportunities (i.e. changes in associative strength):

$$\Delta V_A = \alpha_A \beta(\lambda - \Sigma V)$$
$$\Delta V_B = \alpha_B \beta(\lambda - \Sigma V)$$
$$\vdots$$
$$\Delta V_X = \alpha_X \beta(\lambda - \Sigma V)$$

This assumption has major implications for exposure trials, and specifically aspects related to renewal, as contextual cues are essentially secondary stimuli that gain associative strength with each trial. On subsequent trials, the presence of these contextual cues limit the amount left to be learned ($\lambda - \Sigma V$) and essentially block complete conditioning to the cue of interest.

Mathematically this formula has a number of potential applications for exposure therapy, most of them looking for ways to optimize the amount of inhibitory learning to a target stimulus. The key aspect of the theory as it relates to this is best summarized here:

> "… the degree of decrement in associative strength and the amount of inhibition that may accrue to a stimulus as a result of its nonreinforced presentation depends systematically upon the associative strength of the compound in which it is

embedded." (Wagner & Rescorla, 1972)

Thus, one way to enhance inhibitory learning is to manipulate the associative strength of a set of cues, rather than the target cue itself, when conducting exposures. This line of questioning echoes the exploration of over-expectation in animal models (Rescorla, 1970; Wagner, 1971), where two stimuli trained separately with a US (A+, B+) are presented in compound with the same US (AB+), resulting in inhibitory conditioning. This occurs because the expected US is twice that which occurs due to the additive nature of associative strengths. The effects of using an over-expectation procedure to incur greater inhibitory learning would likely be most potent if such a manipulation occurred after extinction to a target stimulus had reached asymptote (no further learning was possible). This would allow the maximal amount of inhibitory conditioning to occur to the target stimulus via treatment as usual, and then give the inflation of ΣV an opportunity to truly enhance the limits of inhibitory learning opportunities.

Study 1

The application in the study below identified people who were fearful of two stimuli (worms & spiders) in order to mimic the over-expectation experiments. The difference was that the target stimulus (a spider) underwent extinction until it reached asymptote, and then was presented in compound with a stimulus that had experienced no inhibitory conditioning (worms) in order to provide the target stimulus with additional opportunities to acquire inhibitory strength. In order to control for spurious multiple stimuli effects, initial exposure took place with two target stimuli in the initial stage of the experimental group, and in both stages of the control group. To clarify, here are the experimental groups, where A = a spider, and B = worms:

Group	Exp #1	Exp #2	Test
1	AA	AB	A
2	AA	AA	A

In the procedure outlined above it was hypothesized that group 1 would show lower fear to a spider

at post-test than group 2, as exposure # 2 allowed group 1 more opportunities to learn even though learning had reached asymptote in exposure #1. The procedures for this proposal are outlined in detail under the heading study 1.

Cognitive-Expectancy Application of Rescorla-Wagner Model

While the RWM model primarily operates at an unconscious level, as organisms passively take in cues (CSs) to transform into expectancy for the US, there may be more explicit ways to model this same relationship. Exploration of ways to explicitly shift expectancies in order to augment exposure may be a fruitful way to explore these more 'cognitive' applications. Expectancies regarding the likelihood of aversive events are central to human fear conditioning. For example, contingency awareness (i.e., knowledge that a specific CS predicts a US), although of debatable *necessity* for conditioned responding (e.g., Lovibond & Shanks, 2002, versus Ohman & Mineka, 2001) is a strong correlate of conditioned responding. Differential autonomic conditioning in particular is strongly associated with verbal measures of contingency knowledge (e.g., Purkis & Lipp, 2001). Extinction is posited to follow from a mismatch between the expectancy of an aversive event and the absence of its occurrence (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972), or from the perception of a negative change in the rate at which aversive events are associated with the CS (Gallistel & Gibbon, 2000).

The duration for which exposure to the CS continues, therefore, may be critical in the process of extinction, since durations that exceed the temporal expectancy for the US may serve as potent mismatches. Rodent research with mice indicates that extinction is more effective when individual CS presentations are massed, and blocks of massed CSs are spaced apart (Cain, Blouin, & Barad, 2003). These data have been interpreted to suggest that durations of a continuous CS presentation during extinction that exceed the length of the CS during acquisition induces extinction

learning most effectively by violating the temporal expectancy of the US, and that once induced, extinction learning is best consolidated with spaced training. There has been some support for the effectiveness of temporal violations in the explorations of extinction of instrumental conditioning, the results of extended CS duration during extinction was confounded with total exposure time (Drew, Yang, Ohyama, & Balsam, 2004).

In accord with Cain et al. (2003), several studies of human phobic samples indicate that a single massed exposure is more effective than a series of short exposures of the same total duration, such as one 60 minute duration versus three, 20 minute durations of exposure (e.g., Marshall, 1985). Conceivably, the lengthier (massed) exposure is more effective by virtue of providing sufficient time to learn that aversive outcomes do not occur (i.e., to disconfirm negative outcome expectancies). On the other hand, other studies indicate that exposure therapy is effective even when each exposure trial is terminated at the point of elevated or unduly high anxiety, presumably before violation of aversive expectancies has taken place (e.g., Emmelkamp & Mersch, 1982; Rachman, Craske, Tallman, & Solyom, 1986). However, none of these studies directly manipulated the duration, or number, of exposure trials with respect to the disconfirmation of fear-based expectancies. One study demonstrated that exposure therapy designed to disconfirm catastrophic beliefs was more effective than habituation based exposure (staying in the feared situation until fear declines) for individuals with panic disorder and agoraphobia (Salkovskis, Hackmann, Wells, Gelder, & Clark, 2007). However, again, disconfirmation of temporal expectancies was not directly manipulated.

In an unpublished study by Baker and colleagues[1], time was used as a means to violate expectancy. This study asked height phobic individuals to estimate how long they could be exposed to a feared situation (i.e. the top of a parking structure) until their feared outcome (e.g. fall off) would occur. The study utilized two groups, one group where exposures were limited to lengths of time that did not violate the subjects' expectancy, and one where exposures were designed to last longer than the length of time subjects estimated. The study did not yield a significant effect of purposely violating a subject's expectancy for when a negative event would occur, though the study confounded number of exposures (i.e. early session cues) with the experimental design which may have negated our results (Brooks & Bouton, 1993).

So while temporal expectancy parameters may moderate extinction in rodents (Cain et al., 2003), this mechanism may be more complicated in humans who are able utilize verbal and abstract thinking to update expectancies in real time. Further, fear learning in a human sample may be more complicated in the richness of cues available than in animal corollaries, which would limit the accuracy of any participant-generated expectancy to a given cue (e.g. time).

Finally, it is conceivable that temporal expectancies for occurrence of a negative event are less relevant than other forms of expectancy, such as expectancy for the valence of negative events or likelihood that a given CS will result in a US. This may be especially relevant since the many common aversive outcomes to feared stimuli have likely never been experienced by subjects (e.g. in a height phobic situation, 'I will fall off the ledge'), thus subjects are unlikely to consider themselves accurate judges of the probability of outcomes. If this is true, then the effect of disconfirming the expectancy of an aversive outcome may not be achieved, since subjects do not place any authority on their judgments made pre-exposure. Instead subjects may engage in online

[1] Data from this study was published looking at a different set of hypothesis given that the experimental hypothesis was not fruitful (Baker, Mystkowski, Culver, Mortazavi, & Craske, 2010)

expectancy generation during exposure, constantly updating their expectancy of the aversive outcome as they experience the aversive situation.

A different potentially fruitful avenue to take would be to manipulate a subject's expectancy of the likelihood of threat from a CS in a given exposure. For example, if participants were told that the exposure stimulus was more dangerous than in the previous exposure (and thus had a higher likelihood of causing the occurrence of US), then they would experience an increased expectation of threat. This would differ from Study 1 in that the size of the CS (e.g. spider) is not being physically manipulated. Instead this would target the danger cue of a CS (e.g. a spider), and inflate the expectancy that the CS could produce the feared US. This paradigm would attempt to change the perceived value of the predictive cue through a cognitive mechanism.

The efficacy of this approach relies on the theory that a given stimulus is comprised of multiple CSs that are integrated into a single expectancy. For example, while a spider is traditionally considered a CS in its own right; it is actually a collection of CSs (e.g. eight-legs, hair, color, size, species (spider), name (tarantula), and context (i.e. in a cage)). If this is true and all these cues are integrated into a single expectancy, then when a participant is told that the spider has been more aggressive, that information is integrated with the other CSs associated with the spider to create a single expectancy. Using this approach after a prolonged exposure, like study 1, would allow for additional learning to accrue for the target stimulus beyond asymptote.

There have been a number of studies that have looked at the effects of cognitions during fear conditioning, but the area that is most relevant in exploring this subject is outcome expectancy beliefs. Outcome expectancy beliefs describe the network of cognitions related to possible outcomes that form the basis for outcome expectancy judgments (Davey, 2006). Put simply, they are the thoughts that play into a person's expectation that a CS will produce a given outcome.

While there is no doubt that people develop cognitions related to objects and how aversive they are, there is evidence that anxious individuals are more likely to access thoughts that support negative outcomes than non-anxious individuals (e.g. Cavanagh & Davey, 2003). Further, there is evidence that greater verbal expectation of threat correlates with higher conditioned responding (Biferno & Dawson, 1977; Dawson, Schell, & Banis, 1986; Ohman & Soares, 1998). The application of manipulative cognitions to inflate the expectancy of a negative outcome has even been tested, when Davey (1992) demonstrated that warning participants of an aversive outcome before a conditioning paradigm caused inflated expectancy of aversive outcomes at the beginning of conditioning. While these effects were shown to be temporary, and other studies have displayed that negative expectancy biases remit with treatment (de Jong, Merckelbach, Arntz, & Nijman, 1992), they could provide an additional opportunity to inflate expectancy after conditioning to asymptote.

There is also evidence that expectancy for risk and valence of the CS at the conclusion of extinction training predict how much fear is expressed at re-test (Dirikx, Hermans, Vansteenwegen, Baeyens, & Eelen, 2004; Hermans et al., 2005). However, the degree to which changes in those expectancies predict improvement from pre-test to post-extinction re-test has not been evaluated. Instead of manipulating subjective approximations of when a given event could occur, participants could be given the opportunity to violate an increased expectancy of threat from an already conditioned CS during an additional exposure. This would have an advantage over the temporal expectancy design, as participants are more likely to consider themselves accurate judges of what discrete experiences they could handle, rather than how long they could handle a situation.

An additional benefit of this design would come from the fact that it will, in a sense, be a component analysis, allowing for an exploration of the incremental effects of cognitively processing the procedure of exposure. Groups can be equated on all aspects of exposures; it would

merely be the cognitive expectancy of the stimuli that will differ between groups. This experiment could provide insight into the cognitive aspects of CBT, as it uses a purely cognitive inflation of expectancy.

Study 2

The design for study 2 directly explored the question of inflating expectancy of a negative outcome using cognitive inflation of the likelihood of a negative outcome occurring. In this study, the participants in both groups went through a standard exposure paradigm to a phobic stimulus (e.g. Spider), and returned for a second exposure session. Both groups completed a second exposure that repeated the procedures from the first exposure session, but the experimental group was read a script that indicated that the spiders had been more aggressive that day. The inclusion of this script was intended to inflate the expectancy of a negative outcome (that that spider will attack them), without actually changing anything about the stimuli presented. This procedure allowed for exploration of the effects of cognitive inflation of expectancy in isolation.

Summary

The Rescorla-Wagner model has proven to be a powerful explanatory theory within the realm of behavioral psychology. Further, there is strong evidence that this model could explain a variety of processes that have been observed in human fear learning. By focusing on the idea of violation of expectancy the effectiveness of exposure-based interventions could be greatly improved and return of fear could be reduced. The two studies outlined below use both a behavioral and a cognitive approach to understanding how this phenomenon can be applied towards the end of improving the effectiveness of exposure.

Study 1 Hypotheses

-The inflated expectancy exposure group (Spider-Worm) will show less fear at post-test to the target stimulus than the standard exposure group (Spider-Spider).

-The Spider-Worm group will also show greater protection from ROF compared to the Spider-Spider group.

-These findings will be explored by subjective self-report of fear, objective physiological measures of fear, and behavioral measures of approach to feared stimuli.

Study 2 Hypotheses

-The inflated expectancy exposure group will show less fear at post-test than the control group.

-The inflated expectancy group will also show greater protection from ROF compared to the control group.

-These findings will be explored by subjective self-report of fear, objective physiological measures of fear, and behavioral measures of approach to feared stimuli.

Study 1 Methods

Participants

Participants were 61 UCLA undergraduate students enrolled in an introduction to psychology class, who participated for course credit. Participants were given Spider and Worm fear questionnaire (adapted from the SNAQ and the SPQ (Klorman, Weerts, Hastings, Melamed, & Lang, 1974), see "Fear of Spiders and Worms Questionnaire" in Appendix A) during a mass testing session in order to identify students with an intense fear of spiders (scored in top quartile of their testing cohort on spider fear), and a mild to intense fear of worms (scored above the first quartile on worm fear). Exclusion criteria for study entry included any heart, respiratory, or neurological

problems, current pregnancy, and previous advice by physician to avoid stressful situations. Following study entry, exposure treatment refusal, failure to complete exposure in the maximum time allotted, and insufficient levels of fear during an initial behavioral avoidance test (SUDS < 40; n = 12) are additional criteria for exclusion. The remaining 49 participants (37 female and 12 male) had a mean age of 19.32 (SD = 1.57) and were of primarily Asian background (i.e., 53%). Due to equipment failure the physiological data was incomplete for 12 of the study completers, so the physiological analyses were limited to that subset of the sample (n = 37)

Design

A 2-level between-subjects design was used. Participants were randomly assigned to one of two treatment conditions: Spider-Spider or Spider-Worm. The treatment conditions indicate the combination of stimuli used in the second day of exposure; the Spider-Spider group were exposed to two similar spiders in tandem on day 2 of exposure, whereas the Spider-Worm group were exposed to a single spider and worms in tandem on day 2 of exposure.

Therapists

Highly trained undergraduate research assistants as well as research coordinators with bachelor's degrees (5 females and 3 male) served as experimenters. Each experimenter received extensive training (four, 2-hour training sessions for a period of 1 month) using a standardized procedures manual. Each experimenter also treated at least two practice participants prior to running experimental participants.

Phobic Stimuli

Six spiders were used during exposure procedures they were all adult fully matured Chilean Rose-Haired tarantulas *(Phrixotrichus spatulata;* leg-span approximately 6 inches, or 15.2 cm). The spiders varied in their role as exposure stimuli and BAT stimuli, but were kept consistent

within participants. Each participant was exposed to 3 of the 6 spiders, 2 as exposure stimuli and 1 as a BAT stimulus. The worms used were common earthworms (Lumbricus terrestris; approximately 4-8 inches in length), and only participants in the Spider-Worm condition were exposed to them.

Dependent Variables

Behavioral Avoidance Test (BAT). BATs were conducted at baseline, post, and 2-week follow-up assessment, and involved rapidly approaching and remaining directly in front of a caged spider. Participants (Ps) were instructed to go through a hierarchy of 10 approach tasks. Participants were asked to take the next step on the BAT every 30 seconds; if they refused, they remained at their current distance and were asked to take the next step again after another 30 seconds. The undeclared maximum duration of the BAT was five minutes. This procedure allows for the collection of behavioral data, as well we subjective and objective indices of fear. Subjective Units of Distress (SUDS; 0-100) were collected before the task, and then at the beginning and end of each step, as well as every 30-second interval the participant stayed at a step for greater than 30 seconds. Psychophysiological data was collected continuously throughout the baseline, anticipatory, and BAT procedures as an additional index of fear responding. See "BAT Worksheet" in Appendix B for additional information on steps and procedures.

Self Report Questionnaires. Participants completed the SPQ (Klorman et al., 1974). The SPQ is a 31-item scale where participants indicate whether 31 spider-related statements applied to them on a True/False distinction (e.g., "I shudder when I see spiders."). Though this measure has demonstrated inconsistent internal consistency (0.62-.90; Muris & Merckelbach, 1996), it has demonstrated excellent test-retest reliability over three weeks (r = 0.94; Muris & Merckelbach, 1996) and one year (r = .87; Fredrickson, 1983). It has also demonstrated good convergent validity,

discriminating spider phobics from snake phobics, and correlating with aversiveness ratings while watching slides of spiders (Fredrickson, 1983), as well as other measures of spider-related fear and avoidance (Muris & Merckelbach, 1996). Also, the SPQ has been shown to be sensitive to treatment effects (Hellstrom & Öst, 1995, Murrs & Merckelbach, 1996).

Participants also completed a modified version of the 27-item Snake Questionnaire (SNAQ; Klorman, Weerts, Hastings, Melamed, & Lang, 1974), see "Fear of Spiders and Worms Questionnaire" in Appendix A) as well as the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) as secondary measures at baseline, post, and 2-week follow-up assessment. To measure fear of worms, the SNAQ was modified by replacing the word "snake" with the word "worm" in each question, and removing one question which did not pertain to worms. Participants indicated whether 26 worm-related statements applied to them on a True/False distinction (e.g., "when I see a worm I feel tense and restless"). The original snake measure has demonstrated high internal consistency (0.78-.90) and test-retest reliability ($r = 0.84$). It has also demonstrated good convergent validity, discriminating snake phobics from spider phobics, and correlating with aversiveness ratings while watching slides of snakes (Fredrickson, 1983; Klorman et al., 1974). While the SNAQ has been shown to be sensitive to treatment effects (Öst, 1978), it has tended to yield false positives (Klieger, 1987).

The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item scale where participants indicate the extent to which depression related symptoms outlined in the DSM-IV (e.g. inappropriate guilt, sleeping problems, etc.) apply to them. Nineteen items lie on a 4-point likert scale ranging from 0-3, indicated presence and intensity of depression symptoms'. The other 2 items (relating to sleep and appetite) allow respondents to indicate whether they have noticed a change in target behaviors. Though this measure has demonstrated excellent

internal consistency (.93), it has demonstrated excellent test-retest reliability (r = 0.93). It has also demonstrated good convergent validity, correlating highly with the Hamilton Rating Scale for Depression (r = .71; Beck, Steer, & Garbin, 1988).

Psychophysiology Psychophysiological data was collected continuously throughout the 2-minute anticipatory phase and the BAT using a Polar Vantage NV. The polar vantage NV provided wireless heart-rate monitoring with ECG-accurate continuous measurement, sampling once every five seconds. The monitor consists of an elastic belt that attaches around the chest and a wrist-watch receiver, where data are stored and later downloaded into a computer for analysis. This data was collected as a means to monitor sympathetic activity as an index of fear: Simple HR is not an ideal index of sympathetic activity, due to the mixture of sympathetic and parasympathetic influences on HR, but it does allow for rudimentary exploration.

Procedure

All participants came to laboratory for 3 sessions: Pre-Assessment/Treatment Day #1 (Session 1), Treatment Day #2/Post Assessment (Session 2), Follow-up Assessment (Session 3).

During session 1 all participants were escorted to the assessment room by the experimenter. Participants were first given an informed consent form and asked basic demographic information. Following this, participants were fitted with the ambulatory physiological recording device, and given instructions on how to use the SUDS scale. After a 5-min acclimation period to the HR equipment, participants were given instructions for the pretreatment BAT (BAT#l). The experimenter then administered the BAT, and gave the participants the questionnaires shortly afterward (modified-SNAQ#1, SPQ#1, BDI#1).

After completing the assessment phase, participants were escorted to the treatment area where the experimenter informed the participants of the rationale for exposure treatment, explained

the treatment procedures, and answered nay questions. The treatment was a two-session graduated in vivo exposure treatment based on a standard fear hierarchy (Öst, 1997). Treatment deviated from typical exposure procedures in that it incorporated two stimuli (either two spiders, or a spider and worms, depending on session number and experimental condition).

Specifically, treatment consisted of a series of graduated tasks of progressively increasing difficulty. Based on previous procedures, participants rated their expected SUDS on 14 pre-determined approach steps (Mystkowski et al., 2002) in order to develop a specific hierarchy for each participant. Participants went through a series of tasks of increasing difficulty (e.g. Task 1 on the hierarchy involved standing 6 feet away from the spiders in a closed container, Task 7 involved touching the spider five times with a small paintbrush while it moved around its cage, and Task 14 would involved the participants handling the spider with their bare hands; see Table 1). For each of the steps during the exposure therapy, the experimenter recorded the following: initial fear level, end-of-task fear level, task duration, and spider movement. The experimenter also recorded the participants SUDS rating at every minute during the exposure. Task engagement at each step in the hierarchy continued until participants were able to perform the task for a set period of time (2 minutes) or until the participants' fear began to decrease, at which point treatment progressed to the next step in the hierarchy. Fear level alone was not used as a basis for step termination, as it has been shown that fear level during extinction is not critical for long-term fear reduction (Rescorla, 2006). If participants refused the next step they would remain engaged in the step they were on and gently encouraged to try the next step every minute. Treatment was terminated once all steps were completed, or the participant refused to complete additional steps after 5 minutes at a single step. The time allotted for exposure was 40 minutes, and the total time for session 1 was two hours. The two groups do not differ in any procedures during session 1.

Session 2 took place two-six days after Session 1 (mean = 3.92 days). Sessions were spaced this close together to limit the amount of spontaneous recovery that could occur between sessions, since the manipulation aimed to increase activation, and any spontaneous recovery would create additional noise in the analysis. Participants were again outfitted with the physiology monitor and then immediately escorted to the treatment area. Participants in the spider-spider condition were presented with the same two spiders from the previous day; participants in the spider-worm condition were presented with one of the spiders from the previous day, as well as a box containing 10-20 worms. Participants were then asked to engage in the most difficult task they completed on the previous day of exposure. If participants declined to complete that task, they were asked to complete the most difficult task on the hierarchy they were able to. They were not allowed to move beyond the steps they undertook during the previous day of exposure; since if they were allowed to do so, they would in effect experience a different CS, thus inflating expectancy and confounding the results. Treatment followed the same procedure as the previous day of exposure, except that subjects were not allowed to move beyond the highest level of exposure from the previous day. Additionally, the exposure time in this session was limited to 20 minutes. This treatment phase was shorter in order to control for disappearance of conditioned inhibition over excessive trials with no exciter present (Wagner & Rescorla, 1972).

At conclusion of the treatment phase of session 2, subjects were escorted to the assessment room for a 5-minute rest period. After that, participants completed another BAT (BAT#2) and questionnaires (modified-SNAQ#2, SPQ#2, BDI#2). Total time for session 2 was approximately one hour.

Participants returned for session 3 approximately two weeks after session 2 (mean = 14.62 days) and were escorted to the assessment area. Participants were refitted with the physiology

equipment, underwent 5-min adaptation to the physiology equipment, and then engaged the follow-up BAT (BAT#3), followed by the questionnaires (modified-SNAQ#3, SPQ#3, BDI#3). Participants were then debriefed on the aims of the study. Total time for session 3 was one half-hour.

Overall, the two groups only differed in terms of the stimuli they are presented with on the second day of exposure, and were matched on every other aspect of treatment. Matched variables include: assessment battery, stimuli during first day of exposure, total days of exposure, total exposure time, exposure trials, and number of exposure stimuli.

Study 1 Results

Data Analytic Strategy

A 2 x 3 mixed design ANOVA was used, using assessment time (pre, post, and follow-up) as a repeated measure, and group assignment (Spider-Spider and Spider-Worm) as the between-subjects factor. This analysis was carried out with subjective (i.e. SUDS) and objective (i.e. heart rate) data collected during the BAT tasks, as well as the self-report questionnaire data (modified-SNAQ, SPQ, and BDI).

Pre-Manipulation Characteristics

The mean SPQ score was 19.34 (SD = 5.18), which is below clinical norms (M = 23.76, SD = 3.8). The two groups did not differ on non-manipulation variables of age ($t(49) = 0.04$, $p > .50$), BDI ($t(47) = 0.84$, $p > .10$), BAT1-SUDS Average ($t(47) = 0.78$, $p > .10$), BAT1-HR average ($t(37) = 1.02$, $p > .10$), or Exposure 1 HR average ($t(37) = .83$, $p > .10$), Exposure 1 SUDS Average ($t(47) = 0.10$, $p > .50$). The groups were found to have pre-manipulation differences on exposure steps completed ($t(47) = 2.87$, $p < .01$), with the S-S group completing 12.6 steps (SEM = .34) and the S-W group completing 10.8 steps (SEM = .56); as well as trend towards differences on BAT1 steps

completed ($t(47) = 1.68$, $p < .10$), SPQ1 ($t(47) = 1.78$, $p < .10$), modified-SNAQ1 ($t(47) = 1.80$, $p < .10$). Means and standard errors are available in table 2.

Outcome Analyses

Overall the treatment was found to be effective, with participants showing significant change in the subjective outcome variables of SPQ (F (2, 90) = 55.11, $p < .001$, $\eta^2 = .454$), modified-SNAQ (F (2, 90) = 15.56, $p < .001$, $\eta^2 = .262$), average SUDS during the BAT (F (2, 90) = 298.55, $p < .001$, $\eta^2 = .869$), and steps completed on the BAT (F (2, 90) = 13.05, $p < .001$, $\eta^2 = .225$). A significant effect was not found in average heart rate on the BAT (F (2, 52) = 1.18, $p > .10$, $\eta^2 = .044$). Means can be found in table 2.

No condition by time interactions were found on any of the outcome variables, including SPQ (F (2, 90) = 0.12, $p > .10$, $\eta^2 = .006$) and SUDS during the BAT (F (2, 90) = 0.88, $p > .10$, $\eta^2 = .003$), and steps completed on the BAT (F (2, 90) = 2.01, $p > .10$, $\eta^2 = .043$), or average heart rate on the BAT (F (2, 52) = 1.46, $p > .10$, $\eta^2 = .053$). Means can be found in table 2.

Process Analyses

Process analyses were conducted to explore whether the two experimental conditions differed on measures of expectancy during the exposure. A series of 2x2 (Condition x Exposure Occasion) ANOVAs were conducted to explore this question. It was found that the two groups did not differ on objective measures (HR average during exposure (F (1, 33) = 0.89, $p > .10$, $\eta^2 = .026$), though non-significant trends showed that subjective measures (SUDS average during exposure (F (1, 47) = 2.98, $p < .10$, $\eta^2 = .060$), 1st minute of SUDS during exposure (F (1, 47) = 3.09, $p < .10$, $\eta^2 = .063$)), and 1st minute of heart rate during exposure (F (1, 33) = 2.84, $p < .10$, $\eta^2 = .079$)) of expectancy were increased in the spider-worm group during exposure. Means in table 3.

Change scores were calculated to capture the increase in activation at the beginning of the second day of exposure (between session increase; BSI), which was the original purpose of the experimental manipulation. The objective measure of BSI (HR-BSI) was calculated by subtracting the average heart rate during the 1st minute of the second day of exposure from the average heart rate during the last minute of the first day of exposure. The subjective measure of BSI (SUDS-BSI) was calculated by subtracting the first SUDS rating on the second day of exposure (collected after 1 minute) from the final SUDS rating on the first day of exposure. These measures were correlated with change scores looking at improve at post, follow, and between post and follow. Of note is HR-BSI significantly correlated with a decrease in average heart rate on the BAT (r (31) = -.569, p < .001, R^2 = .32), though the effect reversed itself on post to follow (r (27) = .466, p < .05, R^2 = .22). HR-BSI also had non-significant trend predicting SPQ at follow up (r (34) = -.244, p = .17, R^2 = .06). SUDS-BSI showed more inconsistent findings, predicting worse outcome on the SPQ at post (r (49) = .322, p < .05, R^2 = .10), while predicting better improvement post to follow on the SPQ (r (49) = -.314, p < .05, R^2 = .10). SUDS-BSI also showed non-significant trends predicting better outcome on average heart rate during the BAT at post (r (34) = -.204, p = .25, R^2 = .04) and follow (r (31) = -.158, p = .396, R^2 = .03). The full correlation table can be found in table 4.

Study 1 Discussion

Results from study 1 were not consistent with the original hypotheses. While the treatment was found to be very effective, with large and robust treatment effects on the self-report measures, the study failed to reveal any interactions between the experimental manipulation and treatment outcome. While the experimental manipulation was not successful, this may be due to the fact that the random assignment failed to produce similar groups, which is a major limitation for

interpretability. This along with the other limitations outlined below necessitate the need to interpret with the findings caution.

Process analyses revealed that the treatment manipulation was not effective at increasing activation during the exposures on objective measures (heart rate), though there was some support for the intervention increasing participants' subjective activation (SUDS). Exploration of the processes variables and how they related to outcome revealed inconsistent results, but generally showed a higher activation at the beginning of exposure session 2 was related to better overall outcomes. Maintenance of heart rate reactivity in the second session yielded more consistent and robust effects than subjective measures, which indicated short-term effects in the opposite direction, then positive effects from post to follow. This set of analyses was highly underpowered, and mild effects were not significant due to high variability in measurement. This could addressed with a larger sample size, though the ceiling/floor effects discussed earlier likely limit the ability to accurately capture these effects

The pre-intervention differences created statistical issues as mentioned earlier, but the higher pre-intervention difference also led to worse performance during the first day of exposure, where the participants in the spider-worm group completed fewer of the 14 steps in the exposure hierarchy. The design of the study was such that participants could not progress past the exposure steps they completed on the first day of exposure, so the spider-worm group received a lower intensity dose of exposure than the spider-spider group, further complicating the results.

Results were also likely affected by the fact that this was a sub-clinical, non-treatment seeking sample. The participants were not screened for meeting criteria for specific phobia, and the mean score on the SPQ was well below the mean for a clinical population. This is likely due to drawing the sample from a college population, which includes young and generally high

functioning individuals. This effect is also accentuated by the tendency for the most anxious people refusing treatment due to high fear. The population, coupled with the large treatment effects for the intervention created severe floor/ceiling effects that may have prevented the groups from revealing significant treatment differences through the manipulation and process variables. This is a problem that is inherent in any study that attempts to compare two active and highly effective treatments, as the effect sizes are generally small and require large sample sizes to reveal. The non-clinical population used in this study likely worsened this problem, as the treatment had less fear to treat with the intervention.

Finally a major limitation of this study is that it assumed that a tarantula and worms elicited the same US representation. This may have been a faulty assumption, since spiders are generally assumed to elicit a fear reaction, but worms may elicit a disgust response. There is certainly evidence that all specific phobias, including spider phobia, elicit disgust (Matchett & Davey, 1991; Mulkens, de Jong, & Merckelbach, 1996), but it is possible that the spiders and the worms elicited distinct US associations form participants. The disgust literature hypothesizes a disease avoidance model underlying phobic avoidance, which would be distinct from a fear model, which generally identifies pain as the expected US. If this is the case then the results from the experimental manipulation are rendered moot, since the CS-US association being inflated would be distinct from the target CS-US association.

The analyses of the process variables yielded inconsistent, but promising results for supporting the Rescorla-Wagner theory utility in exposure therapy. Still, these effects need to be interpreted with care due to the limitations described above, as well as a potential confound of using difference scores. The ability to find an increase in reactivity in day 2 from day 1 is confounded with treatment effects on day 1, as larger treatment effects on day 1 allow for larger increases in

reactivity on day 2 due to expanded range availability. For example: A participant who had a final SUDS score of 10 on day 1 can attain a difference score between -10 and 90, whereas a participant who had a final score of 50 can only attain a difference score between -50 and 50. This means that the participants that showed greater increases in reactivity in the second day of exposure are also likely to be the participants that showed the greatest treatment effects during exposure session.

While this paradigm failed to produce the expected results, there are different approaches to this question that could yield more promising findings. As mentioned earlier, the match of the expected US across multiple exciters is imperative for the functionality of the Rescorla-Wagner model. A snake may be a more appropriate secondary CS to use to inflate the US expectancy during an exposure to a spider, since snakes likely also elicit fear through an associate with pain/bites as the US. Another potentially fruitful avenue would be to inflate expectancy by using internal cues (e.g. increased heart rate) that are associated with the fear response. An intervention could focus on increasing activation through a mechanism like hyperventilation to increase expectancy via interoceptive cues. Future studies may benefit by working with participants to identify and verbalize the feared outcome (US) before an exposure to make the relationship more explicit and then ensure that the secondary exciter elicits the same expected outcome. While this may be a fruitful avenue clinically, it is unclear how this could applied in a research setting due to the variability of participants' perceptions of what might happen in a feared situation.

Study 2 Methods

Participants

Participants were 7 UCLA undergraduate students enrolled in an introduction to psychology class, who participated for course credit. Participants were given the SPQ (Klorman et al., 1974) during a mass testing session in order to identify students with an intense fear of spiders (scored in

top quartile of their testing cohort on spider fear). Exclusion criteria for study entry included any heart, respiratory, or neurological problems, current pregnancy, and previous advice by physician to avoid stressful situations. Following study entry, exposure treatment refusal, failure to complete exposure in the maximum time allotted, and insufficient levels of fear during an initial behavioral avoidance test (SUDS < 40, n=0) were additional criteria for exclusion. The remaining 7participants (4 female and 3 male) had a mean age of 19.71 (*SD* = 1.60).

Design

A 2-level between-subjects design was used. Participants were randomly assigned to one of two treatment conditions: Control or Inflation. The treatment conditions differed by the script read on day 2 of exposure. The inflation group was read a script that indicated that the spider had been more aggressive than usual that day, whereas the control group was read a script that emphasized that the spider was behaving as usual.

Therapists

Highly trained undergraduate research assistants as well as research coordinators with bachelor's degrees (2 females and 2 males) served as experimenters. Each experimenter received extensive training (four, 2-hour training sessions for a period of 1 month) using a standardized procedures manual. Each experimenter has also participated as experimenters in study 1.

Phobic Stimuli

Six spiders were used during exposure procedures; they were all adult fully matured Chilean Rose-Haired tarantulas *(Phrixotrichus spatulata;* leg-span approximately 6 inches, or 15.2 cm). The spiders varied in their role as exposure stimuli and BAT stimuli, but were kept consistent within participants. Each participant was exposed to 2 of the 6 spiders, 1 as an exposure stimulus and 1 as a BAT stimulus.

Dependent Variables

Behavioral Avoidance Test (BAT). BATs were conducted at baseline, post, and 2-week follow-up assessment, and involved rapidly approaching and remaining directly in front of a caged spider. Participants (Ps) were instructed to go through a hierarchy of 10 approach tasks. Participants were asked to take the next step on the BAT every 30 seconds; if they refused, they remained at their current distance and were asked to take the next step again after another 30 seconds. The undeclared maximum duration of the BAT was five minutes. This procedure allows for the collection of behavioral data, as well we subjective and objective indices of fear. Subjective Units of Distress (SUDS; 0-100) were collected before the task, and then at the beginning and end of each step, as well as every 30-second interval the participant stayed at a step for greater than 30 seconds. Psychophysiological data was collected continuously throughout the baseline, anticipatory, and BAT procedures as an additional index of fear responding. See "BAT Worksheet" in Appendix B for additional information on steps and procedures.

Self Report Questionnaires. Participants completed the SPQ (Klorman et al., 1974). The SPQ is a 31-item scale where participants indicate whether 31 spider-related statements applied to them on a True/False distinction (e.g., "I shudder when I see spiders."). Though this measure has demonstrated inconsistent internal consistency (0.62-.90; Muris & Merckelbach, 1996), it has demonstrated excellent test-retest reliability over three weeks ($r = 0.94$; Muris & Merckelbach, 1996) and one year ($r = .87$; Fredrickson, 1983). It has also demonstrated good convergent validity, discriminating spider phobics from snake phobics, and correlating with aversiveness ratings while watching slides of spiders (Fredrickson, 1983), as well as other measures of spider-related fear and avoidance (Muris & Merckelbach, 1996). Also, the SPQ has been shown to be sensitive to treatment effects (Hellstrom & Öst, 1995, Murrs & Merckelbach, 1996).

Participants also completed the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) as secondary measure at baseline, post, and 2-week follow-up assessment. The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item scale where participants indicate the extent to which depression related symptoms outlined in the DSM-IV (e.g. inappropriate guilt, sleeping problems, etc.) apply to them. Nineteen items lie on a 4-point likert scale ranging from 0-3, indicated presence and intensity of depression symptoms'. The other 2 items (relating to sleep and appetite) allow respondents to indicate whether they have noticed a change in target behaviors. Though this measure has demonstrated excellent internal consistency (.93), it has demonstrated excellent test-retest reliability (r = 0.93). It has also demonstrated good convergent validity, correlating highly with the Hamilton Rating Scale for Depression (r = .71; Beck, Steer, & Garbin, 1988).

Psychophysiology. Psychophysiological data was collected continuously throughout the 2-minute anticipatory phase and the BAT using a Polar Vantage NV. The polar vantage NV provided wireless heart-rate monitoring with ECG-accurate continuous measurement, sampling once every five seconds. The monitor consists of an elastic belt that attaches around the chest and a wrist-watch receiver, where data are stored and later downloaded into a computer for analysis. This data was collected as a means to monitor sympathetic activity as an index of fear: Simple HR is not an ideal index of sympathetic activity, due to the mixture of sympathetic and parasympathetic influences on HR, but it does allow for rudimentary exploration.

Procedure

All participants came to laboratory for 3 sessions: Pre-Assessment/Treatment Day #1 (Session 1), Treatment Day #2/Post Assessment (Session 2), Follow-up Assessment (Session 3).

During session 1 all participants were escorted to the assessment area by the experimenter. Participants were first given an informed consent form and asked basic demographic information. Following this, participants were fitted with the ambulatory physiological recording device, and given instructions on how to use the SUDS scale. After a 5-min acclimation period to the HR equipment, participants were given instructions for the pretreatment BAT (BAT#l). The experimenter then administered the BAT, and gave the participants the questionnaire shortly afterward (SPQ#1).

After completing the assessment phase, participants were escorted to the treatment area where the experimenter informed the participants of the rationale for exposure treatment, explained the treatment procedures, and answered any questions. The treatment was a two-session graduated in vivo exposure treatment based on a standard fear hierarchy (Öst, 1997).

Specifically, treatment consisted of a series of graduated tasks of progressively increasing difficulty. Based on previous procedures, participants rated their expected SUDS on 14 pre-determined approach steps (Mystkowski et al., 2002) in order to develop a specific hierarchy for each participant. In the graduated exposure procedure subjects went through a series of tasks of increasing difficulty (e.g. Task 1 on the hierarchy involved standing 6 feet away from the spider in a closed container, Task 7 involved touching the spider five times with a small paintbrush while it moved around its cage, and Task 14 would involved the participants handling the spider with their bare hands; see Table 1). For each of the steps during the exposure therapy, the experimenter recorded the following: initial fear level, end-of-task fear level, task duration, and spider movement. Task engagement at each step in the hierarchy continued until participants were able to perform the task for a set period of time (2 minutes) or until the participants' fear began to decrease, at which point treatment progressed to the next step in the hierarchy. Fear level alone was not used, as a

basis for step termination, as it has been shown that fear level during extinction is not critical for long-term fear reduction (Rescorla, 2006). If participants refused the next step they would remain engaged in the step they were on and gently encouraged to try the next step every minute. Treatment was terminated once all steps were completed, or the participant refused to complete additional steps after 5 minutes at a single step. The time allotted for exposure was 40 minutes, and the total time for session 1 was two hours. The two groups do not differ in any procedures during session 1.

Session 2 took place two-six days from Session 1 (mean = 4.58 days). Sessions were spaced this close together to limit the amount of spontaneous recovery that could occur between sessions, since the manipulation aimed to increase activation, and any spontaneous recovery would create additional noise in the analysis. Participants were again outfitted with the physiology monitor and then immediately escorted to the treatment area. Once they were in the treatment area they were read one of two scripts depending on their group assignment. The content of the scripts can be found in Appendix B.

Participants in both conditions were presented with the same spider from the previous day of exposure. Participants were then asked to engage in the most difficult task they completed on the previous day of exposure. If participants declined to complete that task, they were asked to complete the most difficult task on the hierarchy they were able to. They were not allowed to move beyond the steps they undertook during the previous day of exposure; since if they were allowed to do so, they would in effect experience a different CS, thus inflating expectancy and confounding the results. Treatment followed the same procedure as the previous day of exposure, except that subjects were not allowed to move beyond the highest level of exposure from the previous day. Additionally, the exposure time in this session was limited to 20 minutes. This treatment phase was

shorter in order to control for disappearance of conditioned inhibition over excessive trials with no exciter present (Wagner & Rescorla, 1972).

At conclusion of the treatment phase of session 2, participants were escorted to the assessment area for a 5-minute rest period. After that, participants completed another BAT (BAT#2) and questionnaire (SPQ#2). Total time for session 2 was approximately one hour.

Participants returned for session 3 approximately two weeks after session 2 (mean = 14.62 days) and were escorted to the assessment area. Participants were refitted with the physiology equipment, underwent a 5-min adaptation to the physiology equipment, and then engaged the follow-up BAT (BAT#3), followed by the questionnaire (SPQ#3). Participants were then debriefed on the aims of the study. Total time for session 3 was one half-hour.

Overall, the two groups only differed in the script they were read on the second day of exposure, and were matched on every other aspect of treatment. Matched variables include: assessment battery, stimuli during first day of exposure, total days of exposure, total exposure time, exposure trials, and number of exposure stimuli.

Study 2 Results

Data Analytic Strategy

A 2 x 3 mixed design ANOVA was used, using assessment time (pre, post, and follow) as a repeated measure, and group assignment (Control or Inflated) as the between-subjects factor. This analysis was carried out with subjective (i.e. SUDS) and objective (i.e. Physiological Data) data collected during the BAT tasks, as well as the self-report questionnaire data (SPQ).

Pre-Manipulation Characteristics

The mean SPQ score was 18.25 (*SD* = 2.18), which is below clinical norms (*M* = 23.76, *SD* = 3.8). The two groups did not differ on non-manipulation variables, though it would not be

expected that they would given that this is a pilot sample and is highly underpowered for detecting those effects.

Outcome Analyses

There was a significant treatment effect across the entire sample for average SUDS during the BAT (F (2, 10) = 24.08, $p < .01$, $\eta^2 = .923$), and non-significant trends for SPQ (F (2, 10) = 1.68, $p = .24$, $\eta^2 = .251$), and steps completed on the BAT (F (2, 10) = 1.75, $p = .22$, $\eta^2 = .26$). There was no positive finding for average HR on the BAT (F (2, 10) = 0.26, $p = .78$, $\eta^2 = .060$). Means can be found in table 5.

There were non-significant trends for group by factor interactions for steps completed on the BAT (F (2, 10) = 1.75, $p = .22$, $\eta^2 = .26$) indicating a larger increase in steps completed for the inflated group; however, there were significant ceiling effects that likely make the result spurious. There was also a non-significant trend for average heart rate on the BAT (F (2, 10) = 4.19, $p = .135$, $\eta^2 = .736$), which indicated that the inflation group experienced decreased HR on the BAT at post treatment, though that effect was not sustained long term. There were no significant findings for SPQ (F (2, 10) = 0.38, $p = .69$, $\eta^2 = .071$) or SUDS during the BAT (F (2, 10) = 0.82, $p = .50$, $\eta^2 = .290$). Means can be found in table 6.

Process Analyses

Process analyses were conducted to explore whether the two experimental conditions differed on measures of expectancy during the exposure. A series of 2x2 (Condition x Exposure Occasion) ANOVAs were conducted to explore this question. It was found that the two groups did not differ on objective measures (HR average during exposure (F (1, 5) < 1, $p > .50$, $\eta^2 < .01$), and while there was not a significant effect for the first minute of heart rate (F (1, 5) < 1, $p > .10$, $\eta^2 = .151$) due to pre-manipulation differences, there appears to be a greater preservation of initial heart

rate reactivity in exposure 2 (M = 82.62, SEM = 3.43) from session 1 (M = 80.58, SEM = 7.03) for the inflated group than the control group (Exposure 2: M = 89.21, SEM = 9.94; Exposure 1: M = 84.71, SEM = 4.86). There was also no significant effect on peak heart rate (F (1, 5) < 1, p > .10, η^2 = .045), though again there appears to be a greater preservation of peak heart rate reactivity in exposure 2 (M = 96.00, SEM = 7.87) from session 1 (M = 93.25, SEM = 2.79) for the inflated group than the control group (Exposure 2: M = 99.5, SEM = 11.13; Exposure 1: M = 102.00, SEM = 3.95). There was no significant effect change in SUDS average during exposure (F (1, 5) < 1, p > .5, η^2 < .01) due to the experimental manipulation.

Study 2 Discussion

This study was a pilot sample of seven non-clinical, non-treatment-seeking spider phobics, and thus the results need to be interpreted carefully. Given the small sample size, significant results are very difficult to find, and even when they are found, the effects could be spurious. The outcome analyses yielded two non-significant trends indicating greater treatment effects for the inflated condition. The effect on number of BAT steps completed is likely spurious due to a ceiling effect where the control condition could not improve from baseline, which the inflated condition had the ability to improve. The effect on average heart rate during the BAT did not sustain at follow-up and in fact reversed itself, limiting the interpretability of this finding.

Process analyses did not reveal any significant effects of the manipulation, though there does seem to be a trend towards the intervention causing a retention of both the initial heart rate activation and the peak heart rate activation in the inflation group. While this result is not significant, it does suggest that the intervention was effective at increasing objective measures of expectancy. These results were not replicated with the subjective measures, though the large

treatment effect on SUDS from exposure 1 to exposure 2 made it especially difficult to parse out group differences.

These results are preliminary, but they do provide some important information on the feasibility of this intervention. This pilot study indicated that the experimental manipulation did not cause an increase in participant refusal to continue, which was a significant concern before the study. This finding means that future studies may be able to increase the intensity in cognitively enhancing the expectancy during exposures without risking high dropout rates. Future studies could provide more severe information (e.g. "One of the spider bit a participant earlier in the week") before exposures start, or include statements during exposures to maintain the inflated expectancy throughout the exposure.

The results of this study differed from study 1, in that the change in process variables due to the intervention was more concordant with the objective measures of expectancy. This non-significant finding could indicate that the verbal inflation of expectancy was tapping into the same initial US as the spider alone, and thus could be a better test of the Rescorla-Wagner model than adding exposure to worms. Still, the results need to be more consistent to draw any reliable conclusions.

This study has some of the same limitations as study 1, in that this was a sub-clinical, non-treatment seeking sample. The participants were not screened as meeting criteria for specific phobia, and the mean score on the SPQ was well below the mean value for a clinical population. Also, like study 1, this study is comparing two active and highly effective treatments, thus effect sizes are likely small and difficult to detect even with a larger sample. This study also assumes that the expected US when a spider phobic is exposed to a tarantula is that the spider will become aggressive with them, and then presumably attack. While this may be true, and fits with the model

put forth in this paper, others argue that spider phobia elicits a disease avoidance model that is distinct from fear associations (Davey & Matchett, 1991). If this were true, than the attempt to inflate the expectancy of the US would be ineffective, since the experiment focused on inflating the expectancy of physical danger outcomes, rather than disease outcomes, when inflating expectancy.

Conclusions

These two studies yielded disappointing results for the study hypotheses, and failed to produce significant treatment by condition interactions, though both studies also failed to produce comparable groups through random assignment. Future studies may benefit from using matched samples after the initial day of exposure to ensure that the intervention compares groups that perform equally well in the exposures that occur before the experimental manipulation. Another factor to take into account is that this experiment was trying to improve upon an already highly effective treatment, thus the treatment effects being sought through manipulation are inherently small and difficult to detect, especially with a small sample size.

Manipulation checks on the interventions also yielded inconsistent results on the efficacy of inflating expectancy. This could be due to an ineffective manipulation, or poor measurement of the manipulation variable. It is likely the case that both these factors were at play here. The studies did not clearly pre-identify the expected US for the CSs used in exposure, thus it is unclear if the inflation attempts during the study accurately targeted the inflation of the expected US for each participant, the inflation of a different US, or if they served a function of being generally activating (i.e. sensitization). This also assumes that a single US-CS association is at play for specific phobia, which may not be the case given the conflicting reports on the underlying mechanism for phobic avoidance (i.e. fear vs. disgust; see Mulkens, de Jong, & Merckelbach, 1996 for review). If both

mechanisms play a role in the maintenance of phobias, then the treatment effect being sought could be even smaller, and thus even more difficult to detect in a study like this.

While these studies did not find overwhelming support for application of the Rescorla-Wagner model to guide practices during exposure treatment, there were some indications that increased expectancy during a second exposure leads to better treatment outcomes. These findings, along with the large number of limitations inherent in the studies, indicate that further exploration of the model is warranted. The largest issue in the above studies was that the manipulations failed to produce consistent change in the underlying variable being targeted for a Rescorla-Wagner guided exposure intervention. Still, findings here, as well as other published studies support sustained activation during exposure as a potential mechanism of change during exposure (Rowe & Craske, 1998; Lang & Craske, 2000; Tsao & Craske, 2000). More powerful forms of inflation, as well as participant-specific inflation goals would likely produce more robust manipulation of the underling variables, and thus would more accurately test the efficacy of using this model to guide treatment.

Treatment applications of the Rescorla-Wagner model are still not well elucidated and will require further exploration to develop. These studies experimented with two methods to inflate expectancy: use of a secondary exciter and cognitively influencing the perceived likelihood of the CS causing a US, but there are likely other ways to exploit the relationship between expectancy and learning. While these studies focused on external cues, internal cues (i.e. physiology) could be manipulated to inflate expectancy since internal cues would have secondary associations with feared outcomes. This could take the form of having participants hyperventilate, engage in physical activity, or ingesting a stimulant (e.g. caffeine) to manipulate physiological correlates of fear.

Developing procedures that build upon the Rescorla-Wagner model and validating them could be instrumental in making exposure-based treatments more effective and ultimately, more efficient. The most widespread explanatory model in use today (Foa & Kozak, 1986) encourages treatment providers to prolong exposure treatments and wait until fear responding has significantly decreased. This procedure has not been supported experimentally (see Craske et al., 2006), yet it has a significant impact on the application of resources due to its high impact in the field. The treatment procedures based on it encourage extended length exposures and suggests that clinicians should not move past an exposure until there is a significant decrement in subjective ratings of fear (e.g. Prolonged Exposure {Foa, Hembree, & Rothbaum, 2006}, which requires exposures that last 45 minutes and requires a 50% decrement in subjective fear before moving past a given exposure). This could be an incredible waste of resources if the best way to achieve learning is create more intense and sustained expectancy during exposure. Instead, treatment could focus on repeated, shorter length exposures that become increasingly more difficult and allow participants to challenge themselves more and learn more quickly.

TABLE 1
SPIDER PHOBIA TREATMENT HIERARCHY

1. Stand 5 ft from the spider in a closed container.
2. Stand l ft from the spider and look down at it in the closed container.
3. Place the palm of your hand against the closed container near the spider.
4. Stand at a close distance to the spider's closed container and place the palm of your hand on either side of the container with your face 1 ft from the container.
5. Focus on the spider as you look down at it with the container top open.
6. Watch the spider as it crawls around a washing bin. (Tasks 6 to 14 use washing bin.)
7. Direct the spider's movement with a small paintbrush 5 times.
8. Touch the spider with a heavily gloved hand 5 times.
9. Let the spider walk over a heavily gloved hand.
10. Touch the spider with a latex-gloved hand 5 times.
11. Let the spider walk over a latex-gloved hand.
12. Touch the spider with bare finger 5 times.
13. Let the spider walk on bare hand with arm covered.
14. Let the spider walk on bare hand with arm uncovered.

TABLE 2
STUDY1 OUTCOME VARIABLE MEANS

		Spider-Spider			Spider-Worm			Combined		
		Pre	Post	Follow	Pre	Post	Follow	Pre	Post	Follow
SPQ	M	17.83	13.75	12.71	20.44	16.91	15.96	19.13	15.33	14.33
	SEM	1.09	1.27	1.22	1.11	1.30	1.25	0.78	0.91	0.87
Modified-SNAQ	M	12.92	11.38	11.29	15.44	12.65	12.22	14.18	12.01	11.76
	SEM	0.96	1.11	1.12	0.98	1.13	1.15	0.68	0.79	0.80
BDI	M	10.56	7.41	6.96	9.21	6.14	6.31	9.88	6.77	6.64
	SEM	1.64	1.49	1.40	1.58	1.44	1.35	1.14	1.04	0.98
BAT Steps	M	8.83	9.71	9.67	8.00	9.52	10.04	8.00	8.42	9.62
	SEM	0.40	0.12	0.30	0.41	0.13	0.31	0.41	0.29	0.09
BAT - SUDS	M	62.35	20.89	18.10	68.05	25.53	21.66	68.05	65.20	23.21
	SEM	3.81	3.30	3.14	3.89	3.37	3.21	3.89	2.72	2.36
BAT - HR	M	89.81	90.37	89.53	94.49	87.36	89.47	94.49	92.15	88.86
	SEM	2.94	3.07	2.91	2.74	2.86	2.71	2.74	2.01	2.10
Exposure 1 - HR	M	82.24			93.39			87.82		
	SEM	4.36			5.04			3.33		
Exposure 1 - SUDS	M	53.03			52.51			52.77		
	SEM	3.64			3.71			2.60		
Exposure 1 - Steps	M	12.70			11.60			11.69		
	SEM	0.31			0.53			0.35		

TABLE 3
STUDY 1 PROCESS VARIABLE MEANS

		Spider-Spider		Spider-Worm	
		Exp1	Exp2	Exp1	Exp2
HR - Average	M	82.24	88.98	93.39	92.69
	SEM	4.36	2.27	5.04	2.62
HR - 1st Minute	M	82.55	88.74	94.80	91.34
	SEM	3.73	2.70	4.30	3.12
SUDS - Average	M	53.03	30.36	52.51	39.24
	SEM	3.64	4.54	3.71	4.64
SUDS - 1st Minute	M	42.21	22.24	37.00	29.43
	SEM	4.06	3.46	4.06	3.46

TABLE 4
STUDY1 PROCESS VARIABLE CORRELATIONS

		SPQ Post	SPQ Follow	SPQ Post-Follow	BAT Steps Post	BAT Steps Follow	BAT Steps Post-Follow
BSI-HR	r	-0.11	-0.24	-0.23	0.27	0.28	0.12
	p	>.05	0.17	0.19	0.11	0.11	0.49
	n	35	34	34	35	34	34
BSI-SUDS	r	.322*	0.16	-0.31*	-0.01	-0.03	-0.03
	p	<.05	0.28	<.05	>.05	>.05	>.05
	n	49	47	47	49	47	47
		BAT SUDS Post	BAT SUDS Follow	BAT SUDS Post-Follow	BAT HR Post	BAT HR Follow	BAT HR Post-Folow
BSI-HR	r	-0.09	-0.13	0.03	-0.57***	-0.07	.47**
	p	>.05	0.45	>.05	<.001	>.5	<.01
	n	35	34	34	31	27	27
BSI-SUDS	r	-0.05	-0.16	-0.16	-0.20	-0.16	0.04
	p	>.05	0.27	0.29	0.25	0.40	>.05
	n	49	47	47	34	31	32

TABLE 5
STUDY 2 OUTCOME VARIABLE MEANS (ACROSS GROUPS)

		Pre	Post	Follow
SPQ	M	18.21	16.79	15.17
	SEM	0.83	1.40	2.27
BAT Steps	M	9.63	10.00	10.00
	SEM	0.28	0.00	0.00
BAT - SUDS	M	45.70	15.63	14.74
	SEM	8.01	6.96	5.52
BAT - HR	M	85.00	86.86	84.95
	SEM	2.74	5.03	5.23

TABLE 6
STUDY 2 OUTCOME VARIABLE MEANS

		Control			Inflation		
		Pre	Post	Follow	Pre	Post	Follow
SPQ	M	19.67	19.33	16.33	16.75	14.25	14.00
	SEM	1.25	2.12	3.43	1.08	1.84	2.97
BAT Steps	M	10.00	10.00	10.00	9.25	10.00	10.00
	SEM	0.43	0.00	0.00	0.37	0.00	0.00
BAT - SUDS	M	47.117	19.467	15.583	44.275	11.787	13.888
	SEM	12.112	10.523	8.344	10.489	9.113	7.226
BAT - HR	M	90.755	96.38	88.795	79.245	77.333	81.11
	SEM	4.472	8.209	8.534	3.162	5.805	6.034

Appendix A – Self-Report Measures

Fear of Spiders of Worm Questionnaire

For the following statements, please answer each in terms if whether it is true or false for you. Circle **T for true** and **F for false**.

T F 1. I would feel some anxiety holding a toy spider in my hand.

T F 2. I dislike looking at pictures of spiders in a magazine.

T F 3. I am terrified at the thought of touching a harmless spider.

T F 4. If someone says that there are spiders about I become alert/on edge.

T F 5. When I see a spider I feel tense and restless.

T F 6. I don't believe anyone can hold a spider without some fear.

T F 7. I wouldn't take a course in Biology if I thought I might have to handle live spiders.

T F 8. Even if I was late for a very important appointment, the thought of spiders would stop me from taking a shortcut through an underpass.

T F 9. Not only am I afraid of spiders, but millipedes and caterpillars make me feel anxious.

T F 10. I would not go down into the basement to get something if I thought there might be a spider down there.

T F 11. I avoid going to parks or on camping trips because there may be worms about.

T F 12. I would feel some anxiety holding a toy worm in my hand.

T F 13. I dislike looking at pictures of worms in a magazine.

T F 14. I am terrified at the thought of touching a harmless worm.

T F 15. If someone says that there are worms about I become alert/on edge.

T F 16. When I see a worm I feel tense and restless.

T F 17. I wouldn't take a course in Biology if I thought I might have to dissect a worm.

T F 18. I think I am more afraid of worms than the average person.

T F 19. Even if I was late for a very important appointment, the thought of worms would stop me from taking a shortcut through and open field.

T F 20. Not only am I afraid of worms, but worms and most similar insects make me feel anxious.

Worm Questionnaire (Modified-SNAQ)

Answer each of the following statements either True or False as you feel they generally apply to you. If the statement is true most of the time or mostly true for you, you should answer **true.** If it is mostly false or false most of the time, mark it **false**. Indicate your answer by placing a mark (X) in the appropriate column.

TRUE	FALSE		
_____	_____	1.	I avoid going to parks or on camping trips because there may be worms about.
_____	_____	2.	I would feel some anxiety holding a toy worm in my hand.
_____	_____	3.	If a picture of a worm appears on the screen during a motion picture, I turn my head away.
_____	_____	4.	I dislike looking at pictures of worms in a magazine.
_____	_____	5.	Although it may not be so, I think of worms as slimy.
_____	_____	6.	I enjoy watching worms at the zoo.
_____	_____	7.	I am terrified by the thought of touching a harmless worm.
_____	_____	8.	If someone says that there are worms anywhere about, I become alert and on edge.
_____	_____	9.	I would not go swimming at the beach if worms had ever been reported in the area.
_____	_____	10.	When I see a worm, I feel tense and restless.
_____	_____	11.	I enjoy reading articles about worms and other insects.
_____	_____	12.	I feel sick when I see a worm.
_____	_____	13.	Worms are sometimes useful.
_____	_____	14.	I shudder when I think of worms.
_____	_____	15.	I don't mind being near a non-poisonous worm if there is someone there in whom I have confidence.
_____	_____	16.	Some worms are very attractive to look at.
_____	_____	17.	I don't believe anyone could hold a worm without some fear.
_____	_____	18.	The way worms move is repulsive.
_____	_____	19.	It wouldn't bother me to touch a dead worm with a long stick.
_____	_____	20.	If I came upon a worm in the woods I would probably run.
_____	_____	21.	I'm more afraid of worms than any other animal or insect.
_____	_____	22.	I would not want to travel "down south" or in tropical countries because of the greater prevalence of worms.
_____	_____	23.	I wouldn't take a course like biology if I thought I might have to dissect a worm.

_____ _____ 24. I have no fear of non-poisonous worms.

_____ _____ 25. Not only am I afraid of worms, but worms and most insects make me feel anxious.

_____ _____ 26. Worms are very graceful insects.

Spider Questionnaire (SPQ)

Answer each of the following statements either True or False as you feel they generally apply to you. If the statement is true most of the time or mostly true for you, you should answer **true.** If it is mostly false or false most of the time, mark it **false**. Indicate your answer by placing a mark (X) in the appropriate column.

TRUE	FALSE		
_____	_____	1.	I avoid going to parks or on camping trips because there may be spiders about.
_____	_____	2.	I would feel some anxiety holding a toy spider in my hand.
_____	_____	3.	If a picture of spider crawling on a person appears on the screen during a motion picture, I turn my head away.
_____	_____	4.	I dislike looking at pictures of spiders in a magazine.
_____	_____	5.	If there is a spider on the ceiling over my bed, I cannot go to sleep unless someone kills it for me.
_____	_____	6.	I enjoy watching spiders build webs.
_____	_____	7.	I am terrified by the thought of touching a harmless spider.
_____	_____	8.	If someone says that there are spiders anywhere about, I become alert and on edge.
_____	_____	9.	I would not go down to the basement to get something if I thought there might be spiders down there.
_____	_____	10.	I would feel uncomfortable if a spider crawled out of my shoe as I took it out of the closet to put it on.
_____	_____	11.	When I see a spider, I feel tense and restless.
_____	_____	12.	I enjoy reading articles about spiders.
_____	_____	13.	I feel sick when I see a spider.
_____	_____	14.	Spiders are sometimes useful.
_____	_____	15.	I shudder when I think of spiders.
_____	_____	16.	I don't mind being near a harmless spider if there is someone there in whom I have confidence.
_____	_____	17.	Some spiders are very attractive to look at.
_____	_____	18.	I don't believe anyone could hold a spider without some fear.
_____	_____	19.	The way spiders move is repulsive.
_____	_____	20.	It wouldn't bother me to touch a dead spider with a long stick.
_____	_____	21.	If I came upon a spider while cleaning the attic I would probably run.
_____	_____	22.	I'm more afraid of spiders than any other animal.
_____	_____	23.	I would not want to travel to Mexico or Central America because of the greater prevalence of tarantulas.

_____ _____ 24. I am cautious when buying fruit because bananas may attract spiders.

_____ _____ 25. I have no fear of non-poisonous spiders.

_____ _____ 26. I wouldn't take a course in biology if I thought I might have to handle live spiders.

_____ _____ 27. Spider webs are very artistic.

_____ _____ 28. I think that I'm no more afraid of spiders than the average person.

_____ _____ 29. I would prefer not to finish a story if something about spiders was introduced into the plot.

_____ _____ 30. Even if I was late for a very important appointment, the thought of spiders would stop me from taking a shortcut through an underpass.

_____ _____ 31. Not only am I afraid of spiders, but millipedes and caterpillars make me feel anxious.

Beck Depression Inventory (BDI)

Instructions: Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle the highest number for that group. *Do not choose more than one statement for any group.*

1.
0 I do not feel sad.
1 I feel sad
2 I am sad all the time and I can't snap out of it.
3 I am so sad and unhappy that I can't stand it.

2.
0 I am not particularly discouraged about the future.
1 I feel discouraged about the future.
2 I feel I have nothing to look forward to.
3 I feel the future is hopeless and that things cannot improve.

3.
0 I do not feel like a failure.
1 I feel I have failed more than the average person.
2 As I look back on my life, all I can see is a lot of failures.
3 I feel I am a complete failure as a person.

4.
0 I get as much satisfaction out of things as I used to.
1 I don't enjoy things the way I used to.
2 I don't get real satisfaction out of anything anymore.
3 I am dissatisfied or bored with everything.

5.
0 I don't feel particularly guilty
1 I feel guilty a good part of the time.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6.
0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7.
0 I don't feel disappointed in myself.
1 I am disappointed in myself.
2 I am disgusted with myself.
3 I hate myself.

8.
0 I don't feel I am any worse than anybody else.
1 I am critical of myself for my weaknesses or mistakes.
2 I blame myself all the time for my faults.
3 I blame myself for everything bad that happens.

9.
0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10.
0 I don't cry any more than usual.
1 I cry more now than I used to.
2 I cry all the time now.
3 I used to be able to cry, but now I can't cry even though I want to.

11.
0 I am no more irritated by things than I ever was.
1 I am slightly more irritated now than usual.
2 I am quite annoyed or irritated a good deal of the time.
3 I feel irritated all the time.

12.
0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.

13.
0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions more than I used to.
3 I can't make decisions at all anymore.

14.
0 I don't feel that I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel that there are permanent changes in my appearance that make me look unattractive.
3 I believe that I look ugly.

15.
0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.

16.
0 I can sleep as well as usual.
1 I don't sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

17.
0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18.
0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.

19.
0 I haven't lost much weight, if any, lately.
1 I have lost more than five pounds.
2 I have lost more than ten pounds.
3 I have lost more than fifteen pounds.

20.
0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains, or upset stomach, or constipation.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems that I cannot think about anything else.

21.
0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I have almost no interest in sex.
3 I have lost interest in sex completely.

Appendix B – Procedural Notes

BAT Worksheet

Completed?

_____ 1. Stand 9 feet from cage

_____ 2. Stand 6 feet from cage

_____ 3. Stand 3 feet from cage

_____ 4. Stand in front of cage with hands at sides

_____ 5. Hands on Side of cage

_____ 6. Hands on top of cage

_____ 7. Face pressed to top of cage

_____ 8. Hand over opening in cage

_____ 9. Hand in cage

_____ 10. Push Spider with a Q-Tip

SUDS Ratings

Step	BEG	END
AA		
1		
2		
3		
4		
5		
6		
7		
8		
9		
10		

Study 2 Scripts:

Experimental Script:

"Today we will be conducting a second exposure session that will closely mirror the exposure we completed the last time you were in the laboratory. During this exposure we will repeat all of the exposure steps you complete the last time you were in. You will not be asked to do anything with spider/worm that you were not asked to do last time.

Do you have any questions about what we are going to do today?"

(Answer any questions)

"I do want to let you know that the spider has been move active and aggressive today. They have not bitten anyone, but it is possible that they can get aggressive and potentially bite. If the spider becomes aggressive and attacks then the exposure will be immediately discontinued. Do you have any questions or concerns about this?"

(Address any questions/concerns about the spiders being aggressive, you can note that the spiders have not bitten anyone during the course of the studies and if they ask what it would feel like you can tell them that the spiders fangs are not large enough to break the skin, and it feels similar to a bee sting. You can also note that the spiders more likely to rear up and/or jab one of their legs at the participants hand rather than bite.)

Control Script:

"Today we will be conducting a second exposure session that will closely mirror the exposure we completed the last time you were in the laboratory. During this exposure we will repeat all of the exposure steps you complete the last time you were in. You will not be asked to do anything with spider that you were not asked to do last time.

Do you have any questions about what we are going to do today?"

(Answer any questions)

"The spider is the same as the one you worked with the last time you were in, and has been behaving the same as it was the last time you were in. Do you have any questions about the spider you will be working with today?"

(Address any questions/concerns about the spider/worm)

References

Arch, J. J., & Craske, M. G. (2009). First-line treatment: A critical appraisal of cognitive behavioral therapy developments and alternatives. *Psychiatric Clinics Of North America*, 32(3), 525-547.

Baker, A., Mystkowski, J., Culver, N., Yi, R., Mortazavi, A., & Craske, M. (2010) Disconfirmation of Expectancy in Exposure Therapy for Acrophobia. *Behavior Research & Therapy*, *48*(11), 1139-1143.

Baum, M. (1988). Spontaneous recovery from the effects of flooding (exposure) in animals. *Behaviour Research & Therapy*, *26*, 185–186.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561-571.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the beck depression inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8*(1), 77-100.

Biferno, M. A., & Dawson, M. E. (1977). The onset of contingency awareness and electrodermal classical conditioning: An analysis of temporal relationships during acquisition and extinction. *Psychophysiology*, *14*(2), 164-171.

Borkovec, T. D., Newman, M. G., Pincus, A. L., & Lytle, R. (2002). A component analysis of cognitive-behavioral therapy for generalized anxiety disorder and the role of interpersonal problems. *Journal of Consulting and Clinical Psychology, 70*(2), 288-298.

Boyd, T. L., & Levis, D. J. (1983). Exposure is a necessary condition for fear-reduction: A reply to de silva and rachman. *Behaviour Research and Therapy, 21*(2), 143-149.

Bouton, M. E. (1993). Context, time and memory retrieval in the interference paradigms of Pavlovian learning. *Psychological Bulletin*, *114*, 90–99.

Brooks, D. C., & Bouton, M. E. (1993). A retrieval cue for extinction attenuates spontaneous recovery. *Journal of Experimental Psychology: Animal Behavior Processes*, *19*(1), 77–89.

Cain, C. K., Blouin, A. M., and Barad, M. (2003). Temporally massed CS presentations generate more fear extinction than spaced presentations. *Journal of Experimental Psychology: Animal Behavior Processes, 29*, 323-333.

Cavanagh, K., & Davey, G. (2003). Access to information about harm and safety in spider fearful and nonfearful individuals: When they were good they were very very good but when they were bad they were horrid. *Journal Of Behavior Therapy And Experimental Psychiatry*, *34*(3-4), 269-281.

Craske, M. G. (1999). *Anxiety disorders: Psychological approaches to theory and treatment*. Boulder, CO, US: Westview Press.

Craske, M., Kircanski, K, Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A (2008) Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, 46(1), 5-27.

Craske, M. G., Sanderson, W. C., & Barlow, D. H. (1987). How do desynchronous response systems relate to the treatment of agoraphobia: A follow-up evaluation. *Behaviour Research and Therapy, 25*(2), 117-122.

Davey, G. C. (1991). Classical conditioning and the acquisition of human fears and phobias: A review and synthesis of the literature. *Advances in Behaviour Research and Therapy*, *14*(1), 29–66.

Davey, G. C. (1992). An expectancy model of laboratory preparedness effects. *Journal Of Experimental Psychology: General*, *121*(1), 24-40.

Davey, G. C. (2006) Cognitive mechanisms in fear acquisition and maintinence. In M. Craske, D. Hermans, & D. Vansteenwegen (Eds.) *Fear & Learning: From Basic Processes to Clinical Applications*. Washington D.C.: American Psychological Association.

Dawson, M. E., Schell, A. M., & Banis, H. T. (1986). Greater resistance to extinction of electrodermal responses conditioned to potentially phobic CSs: A noncognitive process?. *Psychophysiology*, *23*(5), 552-561.

de Jong, P. J., Merckelbach, H., Arntz, A., & Nijmam, H. (1992). Covariation detection in treated and untreated spider phobics. *Journal Of Abnormal Psychology*, *101*(4), 724-727.

Dirikx, T., Hermans, D., Vansteenwegen, D., Baeyens, F., & Eelen, P. (2004). Reinstatement of extinguished conditioned responses and negative stimulus valence as a pathway to return of fear in humans. *Learning and Memory*, *11*(5), 549–554.

Drew, M. R., Yang, C., Ohyama, T., & Balsam, P. D. (2004). Temporal specificity of extinction in autoshaping. *Journal of Experimental Psychology: Animal Behavior Processes, 30*(3), 163-176.

Emmelkamp, P. M., & Mersch, P. P. (1982). Cognition and exposure in vivo in the treatment of agoraphobia: Short-term and delayed effects. *Cognitive Therapy and Research*, *6*(1), 77–90.

Farchione, T. J. (2002). Effects of overlearning on return of fear. *Dissertation Abstracts International: Section B: The Sciences & Engineering*, *62*, 5371.

Foa, E. B., Grayson, J. B., Steketee, G. S., Doppelt, H. G., Turner, R. M., & Latimer, P. R. (1983). Success and failure in the behavioral treatment of obsessive-compulsives. *Journal of Consulting and Clinical Psychology*, *51*(2), 287–297.

Foa, E. B., and Kozak, M. J. (1986). Emotional processing of fear: Exposure of corrective information. *Psychological Bulletin, 99*, 20-35.

Foa, E. B., and McNally, R. J. (1996). Mechanisms of change in exposure therapy. In M. Rapee, (Ed.) *Current Controversies in the Anxiety Disorders* (pp. 329-343). New York: The Guilford Press, 329-343.

Foa, E.B., Hembree, E.A., & Rothbaum, B.O. (2006) *Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences.* New York: Oxford University Press.

Fredrikson, M. (1983). Reliability and validity of some specific fear questionnaires. *Scandinavian Journal of Psychology, 24*(4), 331-334.

Gallistel, C. R., and Gibbon, J. (2000). Time, rate, and conditioning. *Psychological Review, 107*, 289-344.

Goodman, W. K., Price, L. H., Rasmussen, S. A., & Delgado, P. L. (1989). Efficacy of fluvoxamine in obsessive-compulsive disorder: A double-blind comparison with placebo. *Archives of General Psychiatry, 46*(1), 36-44.

Goodman, W. K., Price, L. H., Rasmussen, S. A., & Mazure, C. (1989). The yale-brown obsessive compulsive scale: I. development, use, and reliability. *Archives of General Psychiatry, 46*(11), 1006-1011.

Groves, P. M., & Thompson, R. F. (1970). Habituation: A dual-process theory. *Psychological Review, 77*(5), 419-450.

Guthrie, E. R. (1935). *The psychology of learning*. Oxford, England: Harper.

Hellström, K., & Öst, L. (1995). One-session therapist directed exposure vs two forms of manual directed self-exposure in the treatment of spider phobia. *Behaviour Research and Therapy, 33*(8), 959-965.

Hermans, D., Dirkix, T., Vansteenwegen, D., Baeyens, F., Van den Bergh, O., & Eelen, P. (2005). Reinstatement of fear responses in human aversive conditioning. *Behaviour Research and Therapy*, *43*(4), 533–551.

Hull, C. L. (1943). *Principles of behavior: An introduction to behavior theory*. Oxford, England: Appleton-Century.

Kamin, L.J. (1968). "Attention-like" processes in classical conditioning. In M.R. Jones (Ed.). *Miami Symposium on the Prediction of Behavior*, 1967: Aversive Stimulation. Coral Gables, Florida: University of Miami Press.

Kamin, L.J. (1969a). Predictability, surprise, attention, and conditioning. In B. A. Campbell and R. M. Church (Eds.) *Punishment and Aversive Behavior*. New York: Appleton-Century-Crofts.

Klorman, R., Hastings, J. E., Weerts, T.C., Melamed, B. G., & Lang, P. J. (1974). Psychometric description of some specific-fear questionnaires. *Behavior Therapy*, *5*, 401-409.

Klieger, D. M. (1987). The snake anxiety questionnaire as a measure of ophidophobia. *Educational and Psychological Measurement, 47*(2), 449-459.

Kozak, M. J., Foa, E. B., and Steketee, G. S. (1988). Process and outcome of exposure treatment with obsessive-compulsives: Psychophysiological indicators of emotional processing. *Behavior Therapy, 19*, 157-169.

Lang, A. J., & Craske, M. G. (2000). Manipulations of exposure-based therapy to reduce return of fear: A replication. *Behaviour Research & Therapy*, *38*(1), 1–12.

Lang, P. J. (1971). The application of psychophysiological methods to the study of psychotherapy and behavior modification. In A. Bergin & S. Garfield (Eds.), *Handbook of psychotherapy and behavior change*. New York: Wiley.

Lang, P. J., Melamed, B. G., & Hart, J. (1970). A psychophysiological analysis of fear modification using an automated desensitization procedure. *Journal of Abnormal Psychology*, *76*, 220–234.

Lovibond, P. F., and Shanks, D. (2002). The role of awareness in Pavlovian conditioning: Empirical evidence and theoretical implications. *Journal of Experimental Psychology: Animal Behavior Processes, 28*, 3-26.

Mackintosh, N. J. (1987). Neurobiology, psychology and habituation. *Behaviour Research and Therapy, 25*(2), 81-97.

Marks, I. M. (1987). *Fears, phobias, and rituals: Panic, anxiety, and their disorders*. New York, NY, US: Oxford University Press.

Marshall, W. L. (1985). The effects of variable exposure in flooding therapy. *Behavior Therapy*, *16*, 117–135.

Matchett, G., & Davey, G. C. (1991). A test of a disease-avoidance model of animal phobias. *Behaviour Research And Therapy*, *29*(1), 91-94.

Mineka, S., Mystkowski, J., Hladek, D., & Rodriguez, B. (1999). The effects of changing contexts on return of fear following exposure treatment for spider fear. *Journal of Consulting and Clinical Psychology*, *67*, 599–604.

Mulkens, S. N., de Jong, P. J., & Merckelbach, H. (1996). Disgust and spider phobia. *Journal Of Abnormal Psychology*, *105*(3), 464-468.

Muris, P., & Merckelbach, H. (1996). A comparison of two spider phobia questionnaires. *Journal of Behavior Therapy and Experimental Psychiatry*, *27*, 241-244.

Mystkowski, J. L., Craske, M. G., & Echiverri, A. M. (2002). Treatment context and return of fear in spider phobia. *Behavior Therapy*, *33*, 399–416.

Öhman, A., & Soares, J. F. (1998). Emotional conditioning to masked stimuli: Expectancies for aversive outcomes following nonrecognized fear-relevant stimuli. *Journal Of Experimental Psychology: General*, *127*(1), 69-82.

Ohman, A., and Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review, 108*, 483-522.

Öst, L. G. (1978). Fading vs. systematic desensitization in the treatment of snake and spider phobia. *Behaviour Research & Therapy, 16*, 379-389.

Pavlov, I. (1927). *Conditioned reflexes*. New York, NY, US: Oxford University Press.

Pitman, R. K., Orr, S. P., Altman, B., & Longpre, R. E. (1996a). Emotional processing and outcome of imaginal flooding therapy in Vietnam Veterans with chronic post-traumatic stress disorder. *Comprehensive Psychiatry*, *37*(6), 409–418.

Pitman, R. K., Orr, S. P., Altman, B., & Longpre, R. E. (1996b). Emotional processing during eye movement desensitization and reprocessing therapy of Vietnam Veterans with chronic post-traumatic stress disorder. *Comprehensive Psychiatry*, *37*(6), 419–429.

Purkis, H. M., and Lipp, O. V. (2001). Does affective learning exist in the absence of contingency awareness? *Learning & Motivation, 32*, 84-99.

Rachman, S. (1979). The return of fear. *Behaviour Research and Therapy, 17*(2), 164-166.

Rachman, S. J. (1989). The return of fear: Review and prospect. *Clinical Psychology Review, 9*(2), 147-168.

Rachman, S. (1993). A critique of cognitive therapy for anxiety disorders. *Journal of Behavior Therapy and Experimental Psychiatry, 24*(4), 279-288.

Rachman, S., Craske, M., Tallman, K., & Solyom, C. (1986). Does escape behavior strengthen agoraphobic avoidance? A replication. *Behavior Therapy*, *17*(4), 366–384.

Rachman, S., & Lopatka, C. (1988). Return of fear: Underlearning and overlearning. *Behaviour Research and Therapy, 26*(2), 99-104.

Rescorla, R. A. (1970). Reduction in the effectiveness of reinforcent after prior excitatory conditioning. *Learning and Motivation*, *1*, 372-381.

Rescorla, R. A. (1974). Effect of inflation of the unconditioned stimulus value following conditioning. *Journal Of Comparative And Physiological Psychology*, *86*(1), 101-106.

Rescorla, R. A. (2006). Deepened extinction from compound stimulus presentation. *Journal of Experimental Psychology: Animal Behavior Processes*, *32*(2), 135–144.

Rescorla, R. A., & Heth, C. D. (1975). Reinstatement of fear to an extinguished conditioned stimulus. *Journal of Experimental Psychology: Animal Behavior Processes*, *104*, 88–96.

Rescorla, R. A., and Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A. H. Black, and W. F. Prokasy, (Eds.) *Classical conditioning II* (64-99), New York, Appleton-Century-Crofts.

Rodriguez, B. I., Craske, M. G., Mineka, S., & Hladek, D. (1999). Context-specificity of relapse: Effects of therapist and environmental context on return of fear. *Behaviour Research and Therapy*, *37*, 845–862.

Roth, A. & Fonagy, P. (1996). *What works for whom? A critical review of psychotherapy research*. London: Guilford.

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.

Salkovskis, P. M., Hackmann, A., Wells, A., Gelder, M. G., & Clark, D. M. (2007). Belief disconfirmation versus habituation approaches to situational exposure in panic disorder with agoraphobia: A pilot study. *Behaviour Research and Therapy, 45*(5), 877-885.

Spence, K. W. (1956). *Behavior theory and conditioning*. New Haven, CT, US: Yale University Press.

Steketee, G., Chambless, D. L., Tran, G. Q., & Worden, H. (1996). Behavioral avoidance test for obsessive compulsive disorder. *Behaviour Research and Therapy, 34*(1), 73-83.

Stewart, R. E. & Chambless, D. L. (2007). Does Psychotherapy Research inform treatment decisions in private practice? *Journal of Clinical Psychology, 63*(3), 267-281.

Stobie, B., Taylor, T., Quigley, A., Ewing, S., & Salkovskis, P. M. (2007). Contents may vary: a pilot study of treatment histories of OCD patients. *Behavioural and Cognitive Psychotherapy, 35*, 273-282.

Telch, M. J., Valentiner, D. P., Ilai, D., Young, P. R., Powers, M. B., & Smits, J. A. J. (2004). Fear activation and distraction during the emotional processing of claustrophobic fear. *Journal of Behavior Therapy and Experimental Psychiatry*, *35*(3), 219–232.

Thompson, R. F., & Spencer, W. A. (1966). Habituation: A model phenomenon for the study of neuronal substrates of behavior. *Psychological Review, 73*(1), 16-43.

Tsao, J. C. I., & Craske, M. G. (2000). Timing of treatment and return of fear: Effects of massed, uniform-, and expanding-spaced exposure schedules. *Behavior Therapy*, *31*(3), 479–497.

Wagner, A. R. (1971). Elementary associations. *Essays in neobehaviorism: A memorial volume to Kenneth W. Spence.* (1971). East Norwalk, CT, US: Appleton-Century-Crofts.

Wagner, A. R., (1976). Priming in STM: An information processing mechanism for self-generated or retrieval-generated depression in performance. In T. J. Tighe & R. N. Leaton (Eds.) *Habituation: Perspectives from child development, animal behavior, and neurophysiology* (pp. 95-128). Hillsdale, NJ: Lawrence Erlbaum.

Wagner, A. R., & Rescorla, R. A. (1972). Inhibition in Pavlovian conditioning: Application of a theory. In R. A. Boakes & M. S. Halliday (Eds.), *Inhibition and learning*. London: Academic Press.

Williams, S. L., Dooseman, G., & Kleifield, E. (1984). Comparative effectiveness of guided mastery and exposure treatments for intractable phobias. *Journal of Consulting and Clinical Psychology, 52*(4), 505-518.

Williams, S. L., Turner, S. M., & Peer, D. F. (1985). Guided mastery and performance desensitization treatments for severe acrophobia. *Journal of Consulting and Clinical Psychology, 53*(2), 237-247.