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Does Habituation Matter? Emotional Processing Theory and Exposure Therapy for Acrophobia

Aaron Baker, M.A.[*],
University of California, Los Angeles

Jayson Mystkowski, Ph.D.,
University of California, Los Angeles

Najwa Culver, M.A.,
University of California, Los Angeles

Rena Yi,
Claremont Graduate University

Arezou Mortazavi, and
University of California, Los Angeles

Michelle G. Craske, Ph.D.
University of California, Los Angeles

Abstract

Clinically, there is wide subscription to emotional processing theory (EPT; Foa & Kozak, 1986) as a model of therapeutic effectiveness of exposure therapy: EPT purports that exposure is maximal when (1) fear is activated (IFA), (2) fear subsides within sessions (WSH), and (3) fear subsides between sessions (BSH). This study examined these assumptions, using *in vivo* exposure therapy for 44 students scoring high on Acrophobia measures. Results indicated that no EPT variables were consistently predictive of treatment outcome. No support was found for IFA or WSH; measures of BSH were predictive of short-term change, but these effects were attenuated at follow-up. Furthermore, EPT variables were not predictive of each other as previously hypothesized, indicating the variables are not functionally related.

Keywords

Anxiety Disorders; Exposure Therapy; Habituation; Emotional Processing Theory; Phobias; Acrophobia

[*]Correspondence is to be sent to Aaron S. Baker, M.A., Department of Psychology, 405 Hilgard Ave., Los Angeles, CA 90095-1563, USA, asbaker@ucla.edu.

[1]In conducting heart rate (HR) analyses, the average HR during the initial 5-min adjustment period for session one, as well as the square of that value, were entered as covariates. The first entry corrects for individual differences in baseline HR, while the second entry adjusts for errors in underestimation of HR (Cacioppo & Petty, 1982). Since there was a positive correlation between baseline and task HR across BATs, the HR data was analyzed using a repeated measures approach, as opposed to a reactivity approach using change scores (Russell, 1990).

Repeated, non-reinforced exposure is a core component of treatment for phobias and anxiety disorders. However, not every recipient of exposure therapy benefits (non-response rates roughly vary from 10 to 30% depending on the anxiety disorder; see Craske, 1999). Also, those who do benefit do not typically achieve *complete* fear reduction, and some respondents experience a subsequent return of fear (e.g., Rachman, 1989). Thus, further exploration of the mechanisms behind this method is warranted in order to improve therapeutic outcomes.

The concept of habituation (e.g., Groves & Thompson, 1970) 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. As reviewed by Craske, Kircanski, Zelikowsky, Mystkowski, Chowdhury, & Baker (2008), the extant evidence neither consistently supports nor refutes IFA effects, the majority of studies do not find support for WSH as a predictor of outcome, and the evidence for BSH is limited and indeed its effects are contrandicated by pre to post-treatment improvements in the absence of significant reductions in physiological fear indices over days of exposure (e.g., Kozak, Foa, & Steketee, 1988).

The current study reports on the premises of EPT.

Methods

Participants and Sample Characteristics

Forty-nine moderate to extremely acrophobic participants (35 females; 14 males), were recruited from several UCLA Introduction to Psychology classes for a study identified as a treatment study for individuals with the fear of heights in return for course credit. Participants were administered the Acrophobia Questionnaire (AQ) (Baker, Cohen & Saunders, 1973) during a mass-testing session conducted for course requirement; those scoring in the top quartile were recruited. 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 (n=5) were additional criteria for exclusion. The remaining 44 participants (32 female and 12 male) were college freshmen with a mean age of 18.88 (SD = .99) of primarily Asian background (i.e., 73%).

Treatment Administrators

Highly trained undergraduate research assistants as well as research coordinators with bachelors degrees (3 females and 1 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. Training was limited to running the protocol for this study alone, and not a broader training on exposure based treatment or cognitive strategies.

Dependent Variables

Behavioral Avoidance Test (BAT)—BATs were conducted at baseline, immediately following the final exposure, and 2-week post assessment, and involved approaching and remaining directly in front of a 12-inch gap in a 4-foot-high cement wall surrounding the roof of a six-storey parking structure and looking down towards the group. Participants (Ps) were instructed: "Approach the gap, look over the edge, and stay there as long as you can, and let me know if you want to stop." The undeclared maximum duration of the BAT was five minutes, and the length of time the Ps remained at the gap was recorded. Subjective Units of Distress (SUDS; 0–100) and self efficacy (0–100) (confidence in being able to complete the task successfully) were each rated after a 2-minute anticipatory period and before initiating the BAT. Throughout the BAT, SUDS were rated at minute 0 and each minute thereafter. At the same time, Ps rated the perceived likelihood of their most feared event occurring, using a 0–100 Likelihood of Adverse Event (LAE) scale developed for the purposes of this study with the following anchors: 0 = extremely unlikely, 25 = unlikely, 50 = uncertain, 75 = likely, and 100 = extremely likely. Heart rate data were collected continuously throughout the 2-minute anticipatory phase and the BAT using a Polar Vantage NV that provides 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.

Self Report Questionnaires—Ps completed the 20-item Anxiety subscale of the Acrophobia Questionnaire (AQ; Baker et al., 1973) at baseline, immediately following the final exposure, and 2-week post assessment. Participants rated how anxious they would feel, on a 0 to 6 point scale, in 20 different height situations (e.g., "riding a Ferris wheel," "on the roof of a ten story apartment building"). This Anxiety subscale of the AQ has demonstrated adequate internal consistency (split-half reliability $r = 0.82$) and good test-retest reliability ($r = 0.86$; Baker et al., 1973). It has also demonstrated good convergent validity, significantly correlating with the Fear Survey Schedule ($r = 0.46$, $p < 0.01$) and with an Acrophobia Behavioral Approach Test ($r = -0.32$, $p < 0.01$; Cohen, 1977).

Procedure

Data were collected over four sessions; baseline; first exposure session; second exposure session, followed by an immediate assessment; after the final exposure and a 2-week post assessment. Three locations served as the experimental contexts: (1) an instruction room; (2) a behavioral assessment location, as described; and (3) an exposure location, approaching and putting one foot through a 6-inch gap in the 4-feet-high cement wall surrounding the roof on the opposite side of the 6-story parking lot used for the BAT. The BAT and exposure locations

provided very different outlooks, one looking out towards a street and buildings, the other looking out towards a wooded area.

During Session 1, Ps were escorted by the experimenter to the instruction location, where they were informed about the study and gave informed consent. Next, Ps were instructed in the SUDS scale. Ps were then fitted into the wireless heart-rate monitor. After a 5-minute resting period to adjust to the monitor, Ps were given instructions for the baseline BAT. Then they were led to the assessment location, where after a 2-minute anticipatory period, they rated their anticipatory SUDS and self efficacy before engaging in the BAT. Throughout the BAT, SUDS ratings were obtained each minute. After the BAT, participants stepped away from the edge of the roof and completed the AQ. If the Ps did not report a level of fear of 70 or greater on the SUDS at any time during the BAT, they were excused from the study due to insufficient level of fear.

The first exposure session was scheduled one week after the baseline BAT, and the second exposure session was scheduled one week later. At both exposure sessions, Ps were re-fitted with the heart rate monitor, underwent a 5-minute acclimation period, and then answered questions relating to how likely a feared outcome was to occur, based on time pre-determined time intervals. These questions were used to determine the length of the treatment session that day, in accordance with the research aims of the over-arching study. The over-arching study specifically aimed to evaluate how expectancy of negative events relates to treatment outcome in exposure base treatment. Single trial lengthy exposure sessions were compared to multi-trial shorter duration exposures. No significant differences were found between the two groups, so they were collapsed for the purposes of this paper.

Exposure sessions were conducted in the treatment location, with ongoing measurement of heart rate as well as SUDS ratings which were recorded at the start of each exposure and every minute thereafter. Treatment administrators were positioned to the side of the P and, aside from prompting for subjective ratings, remained silent during the exposure trials. At completion of the second exposure session, the immediate BAT was completed followed by the AQ. The post BAT and questionnaires were completed two weeks later.

Results

Pre-treatment Variables

The mean acrophobia fear score on the AQ was 62.63 (SD = 13.33) (maximum score = 120), which is comparable to a clinical sample (61.30, SD = 15.85; Cohen, 1977). The average exposure duration across both sessions was 21.27 minutes (SD = 17.25), though there was a large range in total exposure time, so it was used as a covariate in all analyses.

Overall the treatment was found to be effective, with participants showing significant change in the subjective outcome variables of AQ ($F(1,2) = 62.32$, $p < .001$) and SUDS during the BAT ($F(1,2) = 64.46$, $p < .001$). Though participants did not show significant decreases in average heart rate during the BAT ($F(1,2) < 1$, $p = .58$). Means can be found in table 1.

Outcome change scores were calculated by subtracting baseline measures from measures taken at immediate and at post assessments. Given the varying durations of exposure across Ps, 5-sec samples of HR and minute by minute SUDS values collected during exposure were split into quartiles for each exposure day. Values were weighted when quartiles included partial measurements (e.g. if a quartile lasted 1min and 15 secs, the SUDS value for minute 1 was weighted four times more than the SUDS value for minute 2). All analyses were performed via stepwise regression. After entering experimental condition as a variable, the variables were entered in the order of the causality assumed by emotional processing theory (Foa & Kozak,

1986), IFA, WSH, BSH. The significance of the variables was calculated by testing the change in the R^2 statistic when each variable was added to the model.

IFA was operationalized as the peak in HR or SUDS score in the first quartile, or minute whichever was longer, of the first exposure session. HR-IFA ranged from 70 to 135 bpm with a mean of 101.59; SUDS-IFA ranged from 25 to 100 with a mean of 68.8. Neither SUDS-IFA nor HR-IFA significantly predicted any outcome change measure, across baseline-immediate or baseline-post.

WSH was operationalized as the difference between with 1st quartile average and 4th quartile average of HR or SUDS ratings, averaged across the two exposure sessions. HR-WSH ranged from −14.5 to 19.50 with a mean of 2.59; SUDS-WSH ranged from 25–100 with a mean of 68.8. Results are presented in Table 2. Again, neither HR-WSH nor SUDS-WSH significantly predicted any outcome change measure, in either baseline-immediate or baseline-post.

BSH was operationalized as the difference between the peak (or maximal) HR and SUDS response from the first to the second exposure session. HR-BSH ranged from −21 to 39 with a mean of −.79; SUDS-BSH Ranged from −35 to 80 with a mean of 15.66. Full results are presented in Table 2. SUDS-BSH predicted change in AQ scores from baseline to immediate (ΔR^2 = .151, p = .009), but not from baseline to post (ΔR^2 = .039, p = .199). SUDS-BSH predicted change in SUDS-maximum during BAT from baseline to immediate (ΔR^2 = .273, p = .000) and from baseline to post (ΔR^2 = .187, p = .002). HR-BSH predicted change in HR during BAT from baseline to immediate (ΔR^2 = .307, p = .000), but not baseline to post (ΔR^2 = .001, p = .854).

Correlations among the six measures of emotional processing theory (see Table 3) yielded only two significant results: HR-IFA correlated significantly with HR-WSH (r = .462, p =.003), and SUDS-IFA correlated significantly with SUDS-WSH (r = .528, p =.000).

Discussion

Our analyses of the premises of EPT failed to produce compelling results to support the veracity of that theory. Neither IFA nor WSH during exposure had any relationship to outcomes. The lack of evidence for level of activation predicting treatment outcome may be due to the most fearful participants refusing to participate in the study, as well as designing exposure tasks that maximized IFA for all participants. These two factors limit the range of relationship within the sample and renders a complete analysis of the efficacy of activation in treatment of acrophobia impossible. The same, however, cannot be said of the other two factors of emotional processing theory. The lack of evidence for WSH is astounding, as none of the 8 analyses aimed at uncovering the efficacy of this process yielded significant results. Beyond that, the amount of unique variance that is explained by WSH was miniscule (ranging from 0 to 4.7%).

Some support was found for the role of BSH for predicting change from baseline to immediate, but the effects were restricted to the same response domain of measurement, such that reported anxiety predicted changes in reported anxiety and heart rate predicted changes in heart rate. Furthermore, for the most part, these effects did not extend to baseline to post outcomes. This inconsistent finding on BSH may be due to the fact that its measurement was confounded with the outcome measure due to the proximity in measurement schedule. In addition, no relationship was found between WSH and BSH. This is especially problematic for EPT, as WSH is presumed to be a necessary prerequisite for BSH.

These results raise serious doubts about the role of within-session habituation as a mechanism of change, and while between-session habituation may have some limited effects immediately,

there appear to be other more important factors that play into long-term results of exposure therapy.

While this study failed to produce results that supported the hypotheses behind EPT as a moderator of improvement in exposure, this study is telling about the viability of the theory. EPT has found scant support over the twenty-plus years in existence (see Craske et al., 2008 for review), and this study was unable to produce any results that support the over-arching theory. This would seem to suggest that EPT is not a viable explanation of the mechanism of change in exposure therapy, and further research should look to uncover possible alternative theories to explain this phenomenon.

An alternative to EPT could come from advances in the basic science of fear learning (Craske et al., 2008). 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). Expectancies also are important for extinction; extinction is posited to follow from a mismatch between the expectancy of an aversive event and the absence of its occurrence (Rescorla & Wagner, 1972), or from the perception of a negative change in the rate at which aversive events are associated with the CS (Gallistel & Gibbon, 2000). Future research should build upon the wealth of research in animal models for fear conditioning in extinction in order to better understand the mechanisms of change in exposure base treatments.

One example of how this could be done is by looking to the animal research on temporal expectancies. The duration for which exposure to the CS continues 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, Blounin, & 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. Thus disconfirmation of temporal expectancy may be a means for enhancing learning through exposure therapy and should be fully explored to this end.

Another alternative would be to further explore potential cognitive mechanisms that may underlie the effectiveness of exposure based treatments. From one perspective the above potential mechanism of mismatches in expectancies is akin to cognitive models. The above the explanation merely works with the model in a way that does not demand that the cognitive processing needs to be explicit. That being said, more exploration in needed to uncover whether cognitive awareness has any mediating or moderating effects on the effectiveness of exposure based treatments.

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Table 1

Treatment Outcome Variables at Pre, Immediate, and Post.

	Pre	Immediate	Post
AQ	62.63	50.02	40.85
	(13.33)	(14.79)	(14.81)
BAT (Heart Rate)[a]	88.42	87.76	90.20
	(14.32)	(12.15)	(11.84)
BAT (SUDS)[b]	71.61	44.32	36.57
	(13.87)	(24.90)	(21.30)

Note. Scores reflect means values with standard deviations in parentheses

[a] Average heart rate during the procedure

[b] Peak SUDS score

Table 2

Emotional Processing Theory Variables Collected During Treatment Sessions and Outcome Measures

	SUDS			HR		
	IFA	**WSH**	**BSH**	**IFA**	**WSH**	**BSH**
AQ-BP[a]	.020	.001	.151**	.071	.047	.002
	(.346)	(.866)	(.009)	(.092)	(.160)	(.765)
AQ-BF[b]	.006	.000	.039	.029	.018	.018
	(.613)	(.968)	(.199)	(.314)	(.432)	(.432)
HR-BP[a]	.002	.002	.037	.006	.026	.307***
	(.822)	(.798)	(.281)	(.656)	(.335)	(.000)
HR-BF[b]	.001	.005	.016	.013	.005	.001
	(.864)	(.683)	(.480)	(.494)	(.680)	(.854)
SUDS-BP[a]	.014	.000	.273***	.006	.022	.005
	(.407)	(.950)	(.000)	(.621)	(.332)	(.633)
SUDS-BF[b]	.023	.005	.187*	.005	.018	.000
	(.302)	(.644)	(.002)	(.648)	(.400)	(.946)

Note. Scores reflect ΔR^2 values with p values in parentheses

[a]Measurement at immediate-test

[b]Measurement at post-test (2 weeks)

Table 3

Correlation Matrix of Emotional Processing Theory Variables Collected During Treatment Sessions

	HR IFA	HR WSH	HR BSH	SUDS IFA	SUDS WSH	SUDS BSH
HR IFA	---					
HR WSH	.275 (.081)	---				
HR BSH	.462** (.003)	.029 (.862)	---			
SUDS IFA	.034 (.834)	.281 (.068)	−.074 (.656)	---		
SUDS WSH	.018 (.912)	.175 (.262)	.049 (.767)	.528*** (.000)	---	
SUDS BSH	−.075 (.643)	.240 (.122)	.084 (.610)	.144 (.352)	−.038 (.808)	---

Note. Scores reflect r values with p values in parentheses