THE EFFECTS OF WORRY ON EMOTIONAL AVOIDANCE IN GENERALIZED ANXIETY DISORDER:

PHYSIOLOGICAL REACTIVITY AND SUBJECTIVE EMOTIONAL EXPERIENCE

A Thesis in
Psychology
by
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ABSTRACT

The present study examined the effect of worry versus relaxation on physiological and subjective responding to subsequent emotion-inducing stimuli. Participants selected based on their GAD status engaged in five worry, relaxation, or neutral thought inductions. Each induction was followed by exposure to one of five film clips, presented in counter-balanced order, designed to elicit a range of both positive and negative emotions. Results indicate that worry led to reduced vagal tone in the GAD group, higher negative affect and anxiety levels, and less physiological and subjective responding to the fearful exposure. Worry also led to a reduction in negative affect and anxious responding to the sad clip. Results suggest that worry facilitates avoidance of negative emotions, but not positive emotions. Implications for the role of worry in emotion dysregulation are discussed.
### TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>List of Tables</td>
<td>v</td>
</tr>
<tr>
<td>List of Figures</td>
<td>vi</td>
</tr>
<tr>
<td>Introduction</td>
<td>1</td>
</tr>
<tr>
<td>Goals and Hypotheses</td>
<td>8</td>
</tr>
<tr>
<td>Research Design and Methods</td>
<td>9</td>
</tr>
<tr>
<td>Participants</td>
<td>10</td>
</tr>
<tr>
<td>Measures</td>
<td>11</td>
</tr>
<tr>
<td>Selection Criteria</td>
<td>11</td>
</tr>
<tr>
<td>Emotion-Eliciting Stimuli</td>
<td>12</td>
</tr>
<tr>
<td>Self-Report Emotion Measures</td>
<td>13</td>
</tr>
<tr>
<td>Physiological Measures</td>
<td>15</td>
</tr>
<tr>
<td>Procedure</td>
<td>16</td>
</tr>
<tr>
<td>Results</td>
<td>18</td>
</tr>
<tr>
<td>Participant Parameters</td>
<td>18</td>
</tr>
<tr>
<td>Demographics</td>
<td>18</td>
</tr>
<tr>
<td>Depression</td>
<td>18</td>
</tr>
<tr>
<td>Manipulation Check</td>
<td>19</td>
</tr>
<tr>
<td>Physiological Measures</td>
<td>19</td>
</tr>
<tr>
<td>Baseline</td>
<td>19</td>
</tr>
<tr>
<td>Induction Period</td>
<td>20</td>
</tr>
<tr>
<td>Emotional Exposure</td>
<td>22</td>
</tr>
<tr>
<td>Self-Report Measures</td>
<td>25</td>
</tr>
<tr>
<td>PTEQ</td>
<td>25</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>25</td>
</tr>
<tr>
<td>Positive Affect</td>
<td>28</td>
</tr>
<tr>
<td>Anxiety Levels</td>
<td>29</td>
</tr>
<tr>
<td>Discussion</td>
<td>32</td>
</tr>
<tr>
<td>Physiological Measures</td>
<td>32</td>
</tr>
<tr>
<td>Induction Period</td>
<td>32</td>
</tr>
<tr>
<td>Emotional Exposures</td>
<td>33</td>
</tr>
<tr>
<td>Self-Report Emotional Responding.</td>
<td>35</td>
</tr>
<tr>
<td>Positive Emotions</td>
<td>37</td>
</tr>
<tr>
<td>Limitations</td>
<td>38</td>
</tr>
<tr>
<td>Figure 1</td>
<td>40</td>
</tr>
<tr>
<td>Table 1</td>
<td>41</td>
</tr>
<tr>
<td>Table 2</td>
<td>42</td>
</tr>
<tr>
<td>Table 3</td>
<td>43</td>
</tr>
<tr>
<td>Table 4</td>
<td>44</td>
</tr>
<tr>
<td>Table 5</td>
<td>45</td>
</tr>
<tr>
<td>References</td>
<td>46</td>
</tr>
</tbody>
</table>
LIST OF TABLES

Table 1:
Means and Standard Errors of Change Scores of Power in the HF Variable from Induction to Emotional Exposure .......................... 63

Table 2:
Means and Standard Errors of Power in the HF Variable from Induction to Emotional Exposure .................................................. 65

Table 3:
Means and Standard Errors of PTEQ Scores for the GAD and Control Groups .............................................................. 65

Table 4:
Means and Standard Errors of Change Scores in NA, PA, and Anxiety Levels from Induction to Emotional Exposure ........................................ 67

Table 5:
Means and Standard Errors of NA, PA, and STAI-S Scores from Induction to Emotional Exposure .................................................. 67
LIST OF FIGURES

Figure 1: Interaction of Induction Type and Group on HF Power During Total Induction Period. 62
The Effects of Worry on Emotional Avoidance in Generalized Anxiety Disorder: 

Physiological Reactivity and Subjective Emotional Experience 

Introduction 

According to Cicchetti, Ackerman, and Izard (Cicchetti, Ackerman et al. 1995), the ability to experience emotions is paramount to both individual as well as social functioning. Emotions, they suggest, operate in terms of motivating and organizing behavior (e.g., interest leads to exploration; anger leads to assertive thoughts, identification of obstacles, and motivation to overcome these obstacles) as well as communication, both internal (informing the self regarding the external world) and external (motivating communication with others). Blumberg and Izard (Blumberg and Izard 1986), who studied emotions among school children, suggest that the cycle of emotions such as interest and joy may motivate pro-social behaviors such as play. Finally, Rachman (Rachman 1980) suggests that full emotional experiencing can lead to successful emotion processing, facilitating the absorption and subsequent decline of emotional disturbances, and allowing for everyday experiences and behaviors to proceed undisrupted. 

Numerous studies provide support for the positive impact of emotional experiencing. For example, attention to internal experiencing (including emotions) was positively correlated with, as well as predictive of, therapeutic success (Gendlen, Beebe et al. 1968). Furthermore, two studies found that asking college students to write about a traumatic experience led to a decrease in health center visits and an increase in grades point averages over the following months as compared to writing about a superficial topic (Pennebaker and Beall 1986; Pennebaker and Francis 1996).
In addition, focusing on acceptance of emotions during a biological challenge resulted in less anxiety and more willingness to participate in a future challenge when compared to suppression of emotion for individuals with panic disorder (Levitt, Brown et al. 2004).

Conversely, a wealth of research documents the detrimental effects of emotional avoidance. Two studies found that emotion suppression in dyadic conversations led to disrupted communication, magnified blood pressure response, reduced rapport, and inhibited relationship formation (Butler, Egloff et al. 2003; Huang 2004). In terms of coping with trauma, it was found that harboring an avoidant reaction towards a past trauma was positively correlated with subjective distress (Solano, Zoppi et al. 2001). In addition, the use of emotion-suppressing defenses in response to trauma led to physical as well as psychological stress symptoms (Wastell 2002).

In addition to evidence on the detrimental nature of emotional avoidance in general, several theorists have focused on problems associated with the avoidance of fear specifically. Mower (Mowrer 1947) posited the two-stage theory of fear and avoidance, stating that classically conditioned fear of a non-threatening stimulus leads to operantly conditioned (e.g., negatively reinforced) avoidance of internal fear cues. Thus, distinct from an external stimulus, the internal experience of fear itself becomes threatening. Furthermore, Borkovec and Sides (Borkovec and Sides 1979) theorized that the avoidance of fear may in fact lead to increased anxiety associated with this emotion, which is congruent with the James-Lange theory (e.g., if I avoid something, it must be dangerous) (James 1890). Additionally, it has been suggested that the
avoidance of fear precludes the benefits of emotion-based learning, as in exposure therapy (Foa and Kozak 1986). According to Foa and Kozak (1986), the internal fear structure must first be accessed before it can be modified; therefore, the avoidance of fear leads to the maintenance of anxiety. Based on these theories, it follows that avoidance of fear may lead to threatening associations, maintained (and even heightened) anxiety associated with feared stimuli, and an impetus for future emotional avoidance.

Studies supporting these theories have found that cognitively avoiding the experience of fear can lead to incomplete emotional processing. One such study demonstrated that cognitive distraction from a fear-eliciting stimulus results in less subjective fear during in vivo exposure as compared to cognitively focusing on the feared stimulus (Craske, Street et al. 1991). This suggests that the experience of fear has been avoided, along with any opportunity to learn from the exposure exercise. A second study found that cognitive avoidance of an imagined fear stimulus precludes the effects of exposure and leads to the maintenance of fear associated with the stimulus (Grayson and Borkovec 1978).

A recent line of research suggests that worry, the cardinal feature of GAD, may facilitate cognitive distraction from, or avoidance of, emotional processing (Borkovec and Inz 1990; Borkovec, Alcaine et al. 2004; McLaughlin and Mennin 2005). Indeed, in two separate studies, a survey on the perceived functions of worry found that the item categorizing worry as a distraction from unwanted emotions was the only item distinguishing persons with GAD from those with sub-clinical anxiety.
and non-anxious controls (Freeston, Rheaume et al. 1994; Borkovec and Roemer 1995).

Additional experimental evidence indicates that it is the abstract verbal/linguistic nature of worry that may function to inhibit imagery (Borkovec and Inz 1990), which in turn may lead to reduced emotional processing (Tucker and Newman 1981; Vrana, Cuthbert et al. 1986; Borkovec and Hu 1990; Borkovec, Lyonfields et al. 1993; Peasley Miklus and Vrana 2000). In a study of induced worry, Borkovec and Inz (1990) found that individuals reported a predominance of verbal/linguistic thought over imagery during worry periods, and the reverse was found during relaxation periods. Furthermore, Vrana and colleagues (1986) found that individuals showed lower autonomic activation to silent (verbal) repetition of arousing fearful scenes than to imagery of those scenes. According to Foa and Kozak (1986), this indicates the limited emotional processing capacity of verbal/linguistic thought, and therefore worry as well. In fact, Shearer and Tucker (1980) found that individuals spontaneously engage in verbalization to inhibit, and imagery to facilitate, emotional experiencing when exposed to arousing photographs. Similarly, Tucker and Newman (1981) found that verbalization reduced sympathetic responses to negatively arousing images.

If, as these findings indicate, the verbal/linguistic nature of worry can lead to diminished emotional processing, it would follow that worry could preclude the therapeutic effects of exposure, which relies on the ability of individuals to emotionally respond to phobic stimuli. Indeed, Borkovec and Hu (1990) demonstrated that engaging in worrisome thought (vs. relaxation) prior to imaginal
exposure to a feared stimulus resulted in lower cardiovascular response and higher subjective anxiety ratings across several presentations. Peasley-Miklus and Vrana (2000) also found that worrisome (vs. relaxed) thinking prior to imaginal exposure led to reduced cardiovascular responding to fearful imagery. Moreover, whereas amount of worrisome thinking was negatively correlated with cardiac arousal to a fearful image, amount of relaxed thinking was positively correlated with cardiac arousal to the image (Borkovec et al., 1993). Taken together, these data suggest that it is the combination of verbal/linguistic thoughts and the worrisome nature of these thoughts that mutes cardiac responding, which in turn reduces emotional processing and precludes the beneficial therapeutic effects of exposure.

Given what we know about the importance of undisrupted emotional processing, it is likely that utilizing worry as an avoidance technique could lead to negative emotional consequences. In fact, two studies showed that worrying immediately after watching a gruesome film (as opposed to image rehearsal or calming down) led to increased cognitive intrusions over a subsequent three day period (Butler, Wells et al. 1995; Wells and Papageorgiou 1995).

Considering that experimental induction of worry can disrupt normal cardiac responding to emotional stimuli, it is possible that chronic worry may have lasting ramifications on cardiac functioning, including vagal functioning. Before exploring this idea further, it is necessary to lay down a connection between vagal (parasympathetic) activity and emotional responding. Levels of vagal activity are considered to reflect efferent communication between the brain and various bodily organs, including the heart, for the purpose of physiological regulation. Higher tonic
(or resting) levels of vagal activity represent greater heart rate variability (HRV), and as such reflect a physiological potential to respond to changes in the environment. Decreases in parasympathetic activity (or vagal withdrawal) in times of stress allow our cardiovascular system to respond with proper sympathetic activation. Correspondingly, HRV levels are found to increase with calming activities such as relaxation following yoga postures (Sarang and Telles 2006). As such, higher levels of resting vagal tone have been posited to be part of a healthy affect regulation system, allowing us to respond appropriately to our environment (Porges 1994).

Considering that inductions of worry can lead to reduced vagal activity in healthy controls (Lyonfields, Borkovec et al. 1995; Thayer, Friedman et al. 1996; Thayer and Lane 2002; Brosschot, Van Dijk et al. 2007), it follows that chronic worry should be associated with lower overall vagal tone. Indeed, research indicates that GAD is associated with lower levels of resting HRV (Hoehn-Saric and McLeod 1988; Hoehn-Saric, McLeod et al. 1989), suggesting a reduced potential to respond to environmental change. Together, this research indicates a diminished capacity to physically regulate affective experiences, or emotional responses to shifting environmental stimuli, in individuals with GAD.

Furthermore, interference with cardiac functioning has been implicated in increased health risks, as high levels of worry have been associated with increased risk of coronary heart disease in older men (Kubzansky, Kawachi et al. 1997). Additionally, individuals with comorbid GAD and Major Depressive Disorder (MDD) have an increased risk of mortality from coronary artery disease (Sevincok, Buyukozturk et al. 2001). These findings indicate a pressing need to better understand
the effects of worry, including its ability to disrupt healthy physiological and emotional functioning.

Recently, research has begun to address emotional problems in GAD, including anxious associations to emotions (McLaughlin and Mennin 2005; Mennin, Heimberg et al. 2005; Turk, Heimberg et al. 2005). Mennin and colleagues (2005) explored this issue by distributing questionnaires to both analogue and clinical GAD populations. They found that persons with GAD reported greater fear of the negative consequences of such emotions as anxiety, depression, anger, and even some positive emotions as compared to non-anxious controls. Persons with GAD also reported less perceived acceptance, clarity, control, and typicality of their emotional experiences. Similarly, Turk and colleagues (2005) found that individuals with GAD reported greater fear of a range of both positive and negative emotions than non-anxious controls, and reported greater fear of depression than individuals with social anxiety disorder. Mennin and colleagues (2005) then tested these findings experimentally. They divided 122 college students into analogue GAD and non-anxious groups using a median split, and then employed a musical mood induction of anxious, sad, and neutral states. Results showed that persons with GAD had greater self-reported physiological responding to the negative mood inductions than did controls. These results, however, were not tested through the observation of actual physiological responding, so we cannot definitively conclude that these individuals experienced more arousal. Nevertheless, results do suggest that persons with GAD report more subjective discomfort with their negative emotions.
A second study exploring emotional problems in GAD examined the effects of worry on negative emotional experiencing (McLaughlin & Mennin, 2005). In this study, individuals with GAD and non-anxious controls engaged in a worried, relaxed, or neutral induction, and were then exposed to a sad film clip. Results indicate that persons with GAD who received the worry induction experienced greater subjective intensity of emotional responding and less perceived ability to regulate this experience than persons with GAD in the two other conditions, as well as non-anxious controls in all conditions. Taken together, these studies lend support to the theory that persons with GAD experience greater subjective distress associated with negative emotions than do non-anxious controls, and also indicate that worry may play a role in this experience.

Goals and Hypotheses

An important absence from the literature at this point is a comparison between the effects of worry on physiological versus subjective responding to emotional exposures. Given the extant research on worry precluding autonomic arousal during fearful exposures, it is somewhat paradoxical that individuals with GAD report increased subjective responding to negative emotions. As such, it remains necessary to elucidate the apparent disconnect between reduced physiological and increase subjective responding to negative emotion inductions. Moreover, research has given reason to speculate that persons with GAD may be motivated to avoid positive as well as negative emotions, as they retrospectively report fear associated with their positive emotions (Mennin, Heimberg et al. 2005; Turk, Heimberg et al. 2005). In fact, a
recent study showed that persons with GAD exhibited a defensive cardiac reaction to presentations of positively-valenced images (Yamasaki, Behar et al. 2002). Therefore goal of the current study is to examine the effect of worry on responses to positive as well as negative emotion-inducing stimuli. Additionally, by observing the effects of experimentally induced worry on both physiological as well as subjective emotional responding, we hope to further elucidate the vicissitudes of worry and its relationship with emotional avoidance.

We predict that worrying prior to emotional exposures will lead to the preclusion of negative and positive emotional experiencing in terms of diminished autonomic arousal as compared to relaxation. We also predict that worry will lead to greater subjective intensity of emotional experiencing as well as subjective distress. The physiological element of this hypothesis will be tested through the observation of changes in vagal activity from worry (vs. relaxation) inductions to emotional exposure, with greater change representing greater levels of emotional responding. The subjective piece of this prediction will be studied by comparing changes in self-report emotional responding and anxiety levels between worry and relaxation from before to after each emotional exposure.

Research Design and Methods

Overall Design

A 2 (group: GAD vs. non-anxious) X 3 (induction type: worry, relax, or neutral) block design was used to explore the differential effects of worry, relaxation, and neutral inductions on exposure to 5 different emotional stimuli (happy, sad, fear,
contentment, and neutral) using both individuals with GAD and non-anxious controls in a between-subject design. The participants were randomly assigned to experience either a worried, relaxed, or neutral induction, followed by exposure to a series of emotional film clips, presented in a counter-balanced order. After each clip, participants were asked to rate the extent of emotion experienced and current anxiety levels, thereby assessing both subjective intensity of emotional experiencing as well as subjective distress. Before viewing the next clip, subjects first underwent a distraction task, and then experienced a 2 minute re-induction (worry, relax, or neutral) period. Heart-rate variability (HRV) was monitored throughout the experiment using a Biopac one-channel isolated amplifier to assess mean HRV differences between the induction and emotion-exposure periods. Vagal tone was estimated using the high frequency (HF) band components of the power spectral density of the HRV analysis.

**Participants**

Seventy-three participants (51 females; M age, 18.85 years, SD, .95 years) were recruited for this study from introductory psychology courses at the Pennsylvania State University. Students were given class credit as compensation for their participation in this research. The ethnic distribution of participants included in the study was 87.7% Caucasian, 4.1% Asian, 2.7% African American, 2.7% Hispanic, 1.4% Middle Eastern, and 1.4% Native American. These groups were equally distributed across conditions.
Participants were selected based on their scores on the Generalized Anxiety Disorder Questionnaire-IV (GAD-Q-IV; Newman, Zuellig, Kachin, Constantino, Przeworski, Erickson, & Cashman-McGrath, 2002) and the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). Scores from these measures were used to assign participants to either the analogue GAD or non-anxious control group. Individuals were included in the GAD group if they score above the cutoff of 5.7 on the GAD-Q-IV, and at least one standard deviation above the mean on the PSWQ. Individuals were included in the control group if they score sufficiently below the cutoff on the GAD-Q-IV, and within a standard deviation of the mean on the PSWQ. Groups did not differ in terms of gender, ethnicity, or age, and these factors were also balanced across conditions.

Depression is associated with a diminished capacity to habituate to anxious states, which may also hinder emotional processing (Foa and Kozak, 1986). Considering that GAD very frequently co-occurs with mood disorders such as depression (Sanderson, Beck et al. 1990), comorbidity was allowed, but was equalized across condition groups. Participants were tested for comorbid depressed mood using the Beck Depression Inventory (Beck, Rush et al. 1979), and completed this measure prior to the experiment.

Measures

Selection Criteria

The GAD-Q-IV (Newman et al., 2002) is a 9-item self-report questionnaire reflecting the criteria for GAD as delineated in the Diagnostic and Statistical Manual
for Mental Disorders, 4th Edition (DSM-IV) (American Psychiatric Association 2000). Most items are dichotomous and measure the excessive and uncontrollable nature of worry as experienced by individuals with GAD. The GAD-Q-IV demonstrates internal consistency (Cronbach’s alpha = .94), 2-week test-retest reliability (92% of the sample showed stability across time), convergent and discriminant validity, and kappa agreement of .67 with a structured interview (Newman, Zuellig et al. 2002).

The PSWQ is a 16-item self-report inventory designed to assess trait worry and to measure the generality, excessiveness, and uncontrollability characteristics of pathological worry. Items are scored on a 5-point Likert-type scale. The PSWQ has demonstrated internal consistency and test-retest reliability (Meyer, Miller et al. 1990).

The BDI (Beck et al., 1979) assesses the presence and severity of affective, cognitive, motivational, vegetative, and psychomotor components of depression. Items include statements such as “I feel sad” and “I feel discouraged about the future” and are ranked on a scale of severity from 0 to 3. Test-retest reliabilities have been from good to very good, ranging from .48 for psychiatric patients after 3 weeks to .74 for undergraduate students after 3 months. The BDI has also been shown to have high concurrent validity with other measures of depression and there is evidence that it discriminates psychiatric from non-psychiatric patients (Beck, Steer et al. 1988).

Emotion-Eliciting Stimuli

Film clips were thought to be the most appropriate stimuli for this study in order to ensure consistency of emotional exposure across participants. Although not
personally relevant, exposure to film clips is less idiosyncratic than asking participants to recall individual emotion-memories. Also, compared with static imagery such as photographs, film clips allow emotions to be experienced in a more natural, gradually increasing manner (Gross and Levenson 1995; Sloan 2004).

Participants viewed five brief film clips (ranging in length from 120 to 180 s) representing positive (happiness and contentment), negative (sadness and fear), and neutral emotions. The film clips used in this study have been successful at eliciting the desired emotions in previous studies (Gross and Levenson 1995; Sloan 2004). These clips include scenes of slapstick comedy (happiness), scenes of nature (contentment), a plane crash (fear), people grieving over a dying man (sadness), and changing color bars (neutral). The clips were presented in counterbalanced order to prevent sequencing effects. However, in all trials, the neutral film clip was played first in order to establish a baseline reaction to neutral emotion.

**Self-Report Emotion Measures**

The Perceptions of Threat from Emotion Questionnaire (PTEQ: McCubbin and Sampson 2006) is a 72 item questionnaire designed to assess beliefs about the threat posed by seven basic emotions (including fear, sadness, happiness, and disgust) as well as ‘strong emotions in general’. Items on this scale include “Do you think it is dangerous to feel (emotion)?” and “Could (emotion) overwhelm you so that you are unable to function?” Individuals are asked to think about how they experience these emotions most of the time, and rate each item on a 5-point scale ranging from “not at all” to “definitely”. The PTEQ demonstrates good test-retest reliability, with a Pearson product moment correlation coefficient of $r = .830$, $P < .001$. Each PTEQ
subscale demonstrates satisfactory internal consistency, apart from the ‘happiness’ subscale (Cronbach’s alpha = .408). The PTEQ shows satisfactory divergent validity, as well as face and construct validity, in that items relate to each of five basic emotions described in the literature (McCubbin and Sampson 2006).

The Positive and Negative Affect Schedule (PANAS: Watson, Clark et al. 1988) is an index of self-report emotional reactivity designed to assess the extent of emotion experienced. Recently used to assess level of emotional reactivity to film clips (e.g., Sloan, 2004), the PANAS is a 20-item mood adjective checklist designed to measure both Positive Affect (PA) and Negative Affect (NA). Participants rate the extent to which their current emotional experience matches with each adjective on a five-point Likert scale ranging from “very slightly or not at all” to “extremely”. Internal consistency reliabilities for the PANAS scales are high, with the PA scale ranging from .86 to .90, and the NA scale ranging from .84 to .87. Test-retest reliability is variable, with the PA scale ranging from .47 to .68, and the NA scale ranging from .39 to .71. Convergent validity of both scales ranges from .89 to .95 whereas discriminant correlations are low, ranging from -.02 to -.18 (Watson, Clark et al. 1988).

The State Trait Anxiety Inventory-State Version (STAI-S: Spielberger, Gorsuch et al. 1983) is a 20 item scale used to measure current transitory feelings of anxiety. Items on this scale include "I feel secure", "I am tense", and "I feel over-excited and rattled." The STAI (state) scale was rated on a 4 point Likert scale from (1) "not at all" to (4) "very much so" to indicate how subjects "feel right now." Internal consistency reliability was shown to be high (in the .80's and .90's) and as
expected, test-retest reliability was much higher for the trait form (high 70's) than
the state form (from .27 to .54). Convergent and discriminant validity has also been
demonstrated for this questionnaire (Spielberger, Gorsuch et al. 1983).

Physiological Measures

Heart-rate (HR) was monitored throughout the experiment using a Biopac
MP30 one-channel isolated amplifier with wrist- and ankle-mounted electrodes, at a
sampling rate of 200Hz. We wanted to observe differences in heart rate variability
(HRV), a measure of physiological rhythms in the beat-to-beat interval of each
cardiac signal. Thus, HRV was analyzed with AcqKnowledge 3.9 software using a
template matching approach in the frequency domain method. We first applied a band
pass filter on the ECG data between 0.5 – 35Hz, using 1600 coefficients. We then
extracted R-R intervals using a modified Pan-Tomkins QRS detector. The R-R
intervals were then re-sampled to a continuous sampling rate in order to extract
frequency information. Considering the low sampling rate of the MP30 system, we
used cubic-spline interpolation on the ECG signals to decrease error
(Electrophysiology 1996). We then calculated HRV on the R-R intervals using power
spectrum analyses of the interpolated R-R tachograms, analyzing the power at the
high frequency (HF; 0.15 - 0.4Hz) bands of the power spectrum density.

For the purposes of this study, we used short-term recordings (1-2 minutes) to
look at the HF components of the HRV output. We chose to examine power in the HF
bands because research considers this to be a good estimate of vagal efferent
(parasympathetic) activity on the heart. Recordings were taken at baseline, during the
induction (worry vs. relaxation) periods, and during the film clips. Induction period
recordings were sampled at both the full 2 minutes, as well as two 1-minute epochs in order to detect more subtle changes. Film clip recordings were likewise sampled at both the full 2-3 minutes, as well as either 2 or 3 one-minute epochs, depending on the length of the clip. Power spectral density values in the HF band will be reported in milliseconds$^2$.

**Procedure**

Once participants were identified, they were contacted via email and assigned a date and time to complete the procedure. Participants were informed that they would be tested individually in a study of how people respond to movie scenes. Upon arrival, participants were randomly assigned to receive either the worried, relaxed, or neutral induction. After completing the consent form, the PTEQ, and the BDI, participants were fitted with the Biopac heart-monitoring equipment, consisting of three electrodes with wrist and ankle mount. Wrists and ankles were prepared by cleaning the skin with an exfoliant and swabbing with alcohol before placing electrodes. Participants were informed that this device would monitor their heart rate. They were also informed that they would be engaging in a period of either worry, relaxation, or neutral thought activity prior to viewing each of a series of film clips.

Once fitted with the heart rate monitor, participants were seated in a comfortable chair facing the computer monitor. Subjects were given a total of 10 minutes to acclimate to the situation before beginning the experiment. During this time, participants were asked to practice their assigned induction. This was either worrying (“think about your most worrisome topic and worry about it as intensely as
you can”), relaxing (instructions on diaphragmatic breathing), or focusing on a neutral activity (“think about what you did over the last weekend”). If at any point their mind wandered off track, they were instructed to simply refocus their thoughts on the task. In order to ensure that each induction was successful, participants were given a manipulation check. This consisted of three 5-point Likert scales assessing levels of anxiety, worry, and relaxation, and participants were asked to rate the extent to which they were currently experiencing each state. Once participants were comfortable engaging in the induction on command, the experimenter had them fill out a pre-clip set of self-report emotion measures (PANAS and STAI), and ran a practice clip (duck pond). To assess for change in emotion following the clip, participants then completed the post-clip self-report emotion ratings (PANAS and STAI).

Although there was no true physiological baseline (e.g., subjects were listening to instructions, practicing inductions, and so forth), the first 2 minutes of this acclimation period were analyzed for baseline differences in vagal tone.

Once 10 minutes had elapsed, participants were instructed to engage in either the worry, relaxation, or neutral induction for 2 minutes, complete pre-clip emotion ratings, view the neutral film clip, and complete the post-clip emotion ratings. They were then asked to perform a distracter task (math problems) for 1 minute. To begin the next segment, participants were asked to re-engage in the induction period (2 minutes) and repeat the same procedure until all 5 film clips had been viewed. At the end of the session, the heart monitoring electrodes were removed and participants
were fully debriefed. Care was taken that participants were not experiencing lingering and distressing negative emotions, and counselors were available if necessary.

Results

Participant Parameters

Demographics

To examine whether analogue GAD participants and non-anxious controls differed in age, gender, or ethnicity, we used univariate analyses of variance (ANOVA) for each demographic factor. There were no significant differences between the groups on age, $F(1, 71) = 0.178$, $p > .05$, gender, $F(1, 71) = 0.539$, $p > .05$, or ethnicity, $F(1, 71) = 2.327$, $p > .05$.

We also examined whether there were any demographic differences between participants who were randomly assigned to the worry, relaxation, or neutral condition. Again, there were no significant differences between these groups on age, $F(2, 70) = 0.113$, $p > .05$, gender, $F(2, 70) = 0.473$, $p > .05$, or ethnicity $F(2, 70) = 1.818$, $p > .05$.

Depression

As predicted, there were significantly higher scores on the BDI for individuals with GAD ($M = 9.658$, $SE = .963$) than for non-anxious controls ($M = 4.629$, $SE = .749$), $t(71) = 4.075$, $p < .001$. Both scores were within the normal to low range of depressed mood, and as such, neither group met criteria for clinical levels of depression. However, in order to determine whether levels of depression were equalized across conditions, an ANOVA was run on just the participants in the GAD group, using
induction type (worry, relax, or neutral) as the between-subjects independent variable. There were no significant differences between conditions, $F(2,35)=2.16$, $p>.05$.

**Manipulation Check**

To determine the effectiveness of our inductions (worry vs. relax), we used a manipulation check to test for differences in self-reported worry, anxiety, and relaxation following each of the 5 inductions. We then collapsed this data across induction periods to create aggregated scores. Results confirmed that our inductions were indeed effective, with individuals in the worry condition reporting significantly higher levels of worry ($M=14.0$, $SE=1.06$) than those in the relaxation condition ($M=6.21$, $SE=.45$), $t(32.14)=6.75$, $p<.001$, $r=.77$. This was also true for self-reported anxiety levels ($M=12.76$, $SE=.89$; $M=7.17$, $SE=.61$, respectively), $t(47)=5.15$, $p<.001$, $r=.60$. Likewise, individuals in the relaxation condition reported feeling significantly more relaxed ($M=18.92$, $SE=.97$) than did those in the worry condition ($M=10.96$, $SE=.86$), $t(47)=6.15$, $p<.001$, $r=.67$.

**Physiological Measures**

**Baseline**

To examine whether analogue GAD participants and non-anxious controls differed in levels of vagal tone at baseline, a $t$-test was conducted on the mean HF values collected during the first 2 minutes of the overall 10 minute acclimation period. These groups did not differ significantly from each other during this baseline
period \((p > .05)\). However, because this was not a true baseline period (e.g., subjects were listening to instructions, practicing inductions, etc.), these results may not be interpretable.

**Induction Period**

We then examined the effect of group (GAD vs. Control) and induction type (worry, relax, or neutral) on levels of vagal tone during the induction period. We achieved this by collapsing across the five induction periods. The collapsed scores for the total induction period, as well as the first and second minute of each induction, were significantly non-normal, \(D(352) = .08, p < .001; D(352) = .07, p < .05;\) and \(D(352) = .07, p < .001\), respectively. Therefore, we performed a log transformation on this data. A multivariate analysis of variance (MANOVA) was then run on the log-transformed collapsed induction data, using GAD status and induction type as the between-subjects’ variables, and mean HF power as the dependent variable. We calculated this for the total induction period (2 minutes) as well as the first and second minute of the induction period separately. Results showed a significant main effect for group, \(F(1,350) = 2.84, p < .05\), as well as for induction, \(F(2,349) = 4.72, p < .001\), and a significant interaction, \(F(3,347) = 7.52, p < .001\). Univariate analyses of variance (ANOVA)s on the individual inductions showed a main effect of induction type for the total induction period, \(F(2,349) = 10.036, p < .001\), as well as the first, \(F(2,349) = 8.789, p < .001\), and second minute of the induction period, \(F(2,349) = 6.681, p < .05\). There was also a significant interaction between group and induction for the total induction period, \(F(3,347) = 21.471, p < .001\), as well as the first, \(F(3,347) = 13.586, p < .001\), and second minute of the induction period, \(F(3,347) = 
19.714, \( p < .001 \). However, there was no longer a main effect for group, \( F(1,350)=1.8, \ p > .05 \), at this level of analysis. Therefore we will not report this factor for the remainder of this section. Specific effects for induction type and the interaction of group and induction were then tested in the following analyses.

To further explore the effect of induction type (worry vs. relaxation) on power in the HF variable, we used Mann-Whitney tests on the untransformed induction data, collapsing across groups (GAD and control). On average, participants had significantly lower power in the HF band during the total worry period \( (Mdn=533.24) \), compared to the total relax period \( (Mdn=599.72) \), \( U=5831.0, \ p = .012 \). However, the effect size for this analysis was small \( (r=-.15) \) and should thus be interpreted with caution.

To explore the interaction, we then tested the effects of induction type by group, using Mann-Whitney tests on the untransformed induction data. For participants with GAD only, worry led to significantly lower HF power \( (Mdn=445.6) \) than did relaxation \( (Mdn=696.55) \) in the total induction period, \( U=856, \ p < .001 \), with a medium effect size \( (r=-.44) \). This effect was also evident for the first and second minute of the induction period separately \( (U=1009.5, \ p < .001; \ U=946.0, \ p < .001, \ \text{respectively}) \). However, examination of the control group showed that, contrary to our hypotheses, worry led to significantly higher HF power \( (Mdn=565.94) \) than did relaxation \( (Mdn=532.57) \) in this group during the total induction period, \( U=1359.0, \ p = .033 \). (See Figure 1.) It should be noted that this analysis also had a small effect size \( (r=-.20) \), and should again be interpreted with caution. This effect was evident for the second minute of the induction period as well \( (U=1211.0, \ p = .005) \).
Emotional Exposure

To determine the amount of change in the HF variable between the induction and emotion stimulation (film clips: neutral, sad, fear, calm, and happy) periods, the HF values from each induction period were subtracted from the subsequent film clip period. This provided an estimate of change in parasympathetic activity from the induction to the emotional exposure period. Greater numbers, either positive or negative, indicated greater flexibility in vagal tone in response to the emotion stimulation.

A multivariate analysis of variance (MANOVA) was run on the change scores for all emotional exposures, using group and induction type as the between-subjects’ variables, and change in HF power as the dependent variable. Results showed a significant main effect for induction type, $F(2,70)= 2.749, p<.05$, but not for group, $F(1,71)= 1.38, p>.05$, nor an interaction, $F(3,68)= 1.49, p>.05$. Univariate analyses of variance (ANOVAs) on the individual inductions revealed a significant main effect of induction type for the fear clip, $F(2,70)= 7.99, p=.001, \omega^2=.44$, the calm clip, $F(2,70)= 4.65, p<.05, \omega^2=.21$, and the happy clip, $F(2,70)= 3.37, p<.05, \omega^2=.28$. However, there was no effect of induction on the neutral or sad clips. The specific effects of the different inductions were tested in the following analyses.

Planned contrasts showed that worry led to significantly less change in vagal tone than did relaxation during the fear exposure, $F(2,57)= 7.99, p=.001, r=.35$ (see Table 1 for all exposure change values). Both values dropped, indicating a decrease in parasympathetic responding in reaction to the fearful clip, which would allow for a corresponding increase in sympathetic responding. However, there was significantly
less of a drop in parasympathetic activity following the worry induction compared to the relaxation induction. We then ran a repeated measures ANOVA on the induction and fear clip periods, using induction type as the between subjects factor, to see if these increases were significant. Results showed a significant effect of time, $F(1,45)=20.22, p<.001, r=.56$, for increase in HF power from induction to the fear clip, as well as a significant interaction between time and induction type, $F(1,45)=11.56, p<.001, r=.45$. We therefore ran separate repeated measures ANOVAs for worry and relaxation to see if each induction led to a significant change in vagal tone. Results showed that there was in fact no significant change in vagal tone from the worry induction to fearful exposure, regardless of GAD status, $F(1,24)=1.83, p>.05, r=.27$ (see Table 2 for all exposures). However, there was a significant decrease in vagal tone from the relaxation induction to the fear clip, $F(1,21)=16.826, p=.001, r=.67$, suggesting a significant increase in physiological arousal. Taken together, these results suggest that, as predicted, worrying prior to a fearful emotional exposure precluded the ability to respond to that emotion with appropriate physiological arousal as compared to relaxation, and this was true for both individuals with GAD as well as non-anxious controls.

As for the calm clip, planned contrasts from the MANOVA test revealed that worry led to significantly more change in vagal tone than did relaxation, from induction to clip, $F(2,57)= 4.65, p<.05, r=.27$. We then ran a repeated measures ANOVA on the induction and calm clip periods, using induction type as the between subjects factor, to see if these increases were significant, as well as to determine whether worry led to more of an increase than relaxation. Results showed a
significant effect of time, $F(1,44)=52.94, p<.001, r=.74$, for increase in HF power from induction to the calm clip, but no interaction between time and induction type, $F(1,44)=2.95, p>.05$. These results suggest that both worry and relaxation allow for significant increases in vagal tone during a calm exposure.

For the happy clip, planned contrasts from the MANOVA test revealed that worry also led to significantly more change in vagal tone than did relaxation, from induction to the happy clip, $F(2,57)= 3.37, p<.05, r=.24$. We then ran a repeated measures ANOVA on the induction and happy clip periods, using induction type as the between subjects factor, to see if these increases were significant. Results showed a significant effect of time, $F(1,41)=9.46, p<.001, r=.43$, for increase in HF power from induction to the happy clip, as well as a significant interaction between time and induction type, $F(1,41)=6.44, p<.05, r=.37$. We therefore ran separate repeated measures ANOVAs for worry and relaxation to see if each induction led to a significant change in vagal tone. Results showed that while worry led to a significant increase in vagal tone from the induction to the happy clip, $F(1,21)=23.44, p<.001, r=.73$, relaxation did not, $F(1,20)=.11, p>.05$. This may indicate a ceiling effect for individuals in the relaxation condition. What this also demonstrated is that, again, worry did not preclude positive physiological responding to the happy clip.

Finally, we performed a repeated measures ANOVA on the sad clip, using induction type as the between subjects factor. Results showed a significant effect of time, $F(1,45)=98.60, p<.001, r=.83$, for increase in HF power from induction to the sad clip, but no interaction between time and induction type, $F(1,41)=.52, p>.05$. Though worry and relaxation did not vary as to their effects on the physiological
response to sadness, it is interesting to note that both led to an increase in vagal activity.

**Self-report Measures**

**PTEQ**

To determine whether participants with GAD reported more perceived threat from their emotional experiences than did non-anxious controls, *t*-tests were conducted on the PTEQ scores. Results showed significantly greater perceived threat of emotion for individuals with GAD compared to controls on a range of PTEQ subscales, including sadness (*t*[59.66]=3.182, *p*=.002), guilt (*t*[71]=2.37, *p*=.02), fear (*t*[71]=2.305, *p*=.024), and strong emotions in general (*t*[71]=2.523, *p*=.014), as well as the total PTEQ score (*t*[71]=2.18, *p*=.033). (See Table 3 for mean scores.) These results suggest that participants with GAD reported experiencing sadness, guilt, fear, and any strong emotion as more threatening than did non-anxious controls.

**Negative Affect**

To determine the impact of film clips on negative affect, we subtracted the NA scores taken before the clip from those taken after. We were interested in change in negative affect in the neutral, fear, and sad clips. Due to non-normality of NA change scores (*D*(73)=.22, *p*<.001; *D*(73)=.17, *p*<.001; and *D*(73)=.11, *p*<.05, respectively), a log transformation was performed on this data. A multivariate analysis of variance (MANOVA), using group and induction type as the between-subjects’ variables, and change in NA scores as the dependent variable, showed a significant main effect for induction type, *F*(2,70)= 6.869, *p*<.001, and interaction,
$F(3,68)= 2.24, p<.05$, but not for group, $F(1,71)= 1.0, p>.05$. Univariate analyses of variance (ANOVAs) on the individual inductions revealed a significant main effect of induction type for the neutral clip, $F(2,70)= 20.566, p<.001, \omega=.47$, the fear clip, $F(2,70)= 13.385, p<.001, \omega=.53$, and the sad clip, $F(2,70)= 12.812, p<.001, \omega=.47$. There was also a significant interaction for the neutral clip, $F(3,68)= 5.982, p<.01$. (See Table 4 for NA, PA, and STAI changes scores for all exposures.)

Planned contrasts showed that worry led to significantly less change in negative affect ($M=-.13$) than did relaxation ($M=.032$) during the neutral exposure, $p<.001$. However, when taking group status into account, result indicated that individuals with GAD in the worry condition experienced a much greater drop in NA following the neutral clip ($M=-.21$) than did controls ($M=-.05$). On the contrary, participants with GAD in the relax condition reported a greater increase in NA following the neutral clip ($M=.04$) than did controls ($M=.02$). Taken together, this suggests that individuals with GAD reported more exaggerated changes in negative affect in response to the neutral clip, either increase or decrease depending upon condition, than did controls.

Planned contrasts for induction type in the fear and sad clips showed that worry led to significantly less change in negative affect than did relaxation for each clip ($p<.001$ for both exposures). We then tested to see whether these changes in NA were significant over time. Due to nonnormality of NA scores, the non-parametric Wilcoxon signed-rank test was used on the untransformed scores from before and after the clips. Results showed that the worry induction did not lead to significant change in NA from before to after the fearful exposure, $z=-1.3, p>.05, r=-.18$. By
contrast, the relaxation induction was followed by significant increases in NA from before to after the film clip, \( z = 4.022, p < .001, r = .59 \) (see Table 5 for NA, PA, and STAI scores for all exposures). These results suggest that worrying prior to a fearful exposure may have precluded the ability to respond subjectively to the negative emotional stimulus, but that relaxation facilitated this process.

We then examined the sad film clip. In this case, worry was actually followed by a small but significant decrease in NA from before to after the sad clip, \( z = -2.34, p < .05, r = -.33 \). Conversely, relaxation lead to a significant increase in NA from before to after the film clip, \( z = 3.517, p < .001, r = .51 \). Again, this pattern suggests that worry prior to a sad exposure precludes the incorporation of negative emotional information, and that relaxation may facilitate successful incorporation of this material.

We also examined whether worrying would lead to greater NA than relaxation, and whether or not this would be sustained across the clip. Although the worry induction led to significantly higher NA than the relaxation induction \( (U = 130.5, p = .001, r = -.49) \), these conditions ended with statistically similar NA levels following the fear exposure \( (U = 237.5, p = ns, r = -.18) \). This pattern was also true for the sad exposure, in that worry led to significantly higher NA than did relaxation \( (U = 71.0, p = .001, r = -.67) \), but again these conditions ended up with statistically similar NA following the sad exposure \( (U = 229.5, p = ns, r = -.20) \). This suggests that worry did lead to greater negative affect than relaxation, but may have resulted in a ceiling effect, reducing the emotional impact of subsequent exposure to fearful and sad stimuli. On the other hand, relaxation resulted in lower NA post-induction, with
significant increases in NA following these exposures, suggesting an appropriate emotional response to the fear and sad clips.

*Positive Affect*

To determine the impact of film clips on positive affect, we subtracted the PA scores taken before the clip from those taken after. We were interested in change in negative affect in the neutral, calm, and happy clips. Considering that these change scores were also significantly non-normal \((D(73)=1.9, p<.001; D(73)=1.9, p<.05; \text{ and } D(73)=1.4, p<.01, \text{ respectively})\), we performed a log transformation on this data. A multivariate analysis of variance (MANOVA), using group and induction type as the between-subjects’ variables, and change in PA scores as the dependent variable, showed no main effect for group \(F(1,69)=1.43, p>.05\), induction type, \(F(2,68)=.111, p>.05\), or interaction, \(F(3,66)=.04, p>.05\), suggesting that there were no significant effects for either group membership or induction type on positive responding to the clips.

We then tested to see whether these changes, though not different from each other, were significant over time. Due to nonnormality of PA scores, the non-parametric Wilcoxon signed-rank test was used on the untransformed scores from before and after the clips. In this case, there were no significant changes in PA following the neutral or calm clips. However, we found that both worry and relaxation lead to a significant increase in PA from before to after the happy clip \((z=4.239, p<.001, r=.60; \text{ and } z=3.902, p<.001, r=.56, \text{ respectively})\). These results suggest that worry did not preclude subjective emotional responding to the happy exposure.
Anxiety Levels

Finally, we examined changes in the STAI-S scores from before to after viewing the film clips to assess for differences in anxiety between group and induction type. We created change scores using the methods detailed above, and a multivariate analysis of variance (MANOVA) was then run on the change scores for all films, using GAD status and induction type as the between-subjects’ variables, and change in STAI scores as the dependent variable. Results showed a significant main effect for group, $F(1,68)=3.564, p<.01$, as well as for induction, $F(2,67)=4.56, p<.001$, and a significant interaction, $F(3,65)=2.41, p<.05$. Univariate analyses of variance (ANOVAs) showed a main effect of group for the calm, $F(1,68)=13.73, p<.001$, and happy clips, $F(1,68)=13.97, p<.001$, as well as a main effect for induction type in all clips (neutral: $F(2,67)=10.08, p<.001$; fear: $F(2,67)=9.53, p<.001$; sad: $F(2,67)=13.10, p<.001$; calm: $F(2,67)=12.39, p<.001$; and happy: $F(2,67)=22.23, p<.001$). There was also a significant interaction between group and induction for the neutral, $F(3,65)=3.70, p<.05$, and happy clips, $F(3,65)=7.90, p=.001$. Specific effects for group, induction type, and the interaction were then tested in the following analyses.

We then explored group differences on the neutral and happy clips by collapsing across condition. Results indicate that individuals with GAD reported a greater decrease in anxiety from before to after the happy clip than did controls, $t(49.22)=-2.56, p<.05$, but this was not true for the neutral clip.

To further explore the effect of induction type (worry vs. relaxation) on changes in anxiety levels, planned contrasts were used on all the clips. On average,
participants viewing the neutral and negative (sad and fear) clips reported decreases in anxiety from the worry induction to the clips, as compared to increases in anxiety from the relaxation induction to the clips (\( p \leq .001 \) for each contrast). These results suggest that worry helps to reduce subjective anxiety in response to a negative stimulus. Considering that the neutral clip was neither positively nor negatively valenced, the decrease in anxiety from worry to the clip, and increase from relaxation to the clip, may suggest a return to more neutral levels of anxiety.

Alternatively, participants viewing the positive (calm and happy) clips reported decreases in anxiety from both the worry and relaxation inductions to the clips (worry: \( M = -13.37, M = -15.84 \); relax: \( M = -3.8, M = -2.3 \), respectively), though the inductions were significantly different from each other (\( p < .001 \) for all contrasts). This may suggest that individuals in the relaxation condition experienced a floor effect.

To examine whether worry and relaxation led to significant changes in anxiety levels over time, STAI scores were then run in separate repeated measures ANOVAs. First, we analyzed the effect of worry on changes in STAI from before to after the fear clip. Similar to the pattern with NA scores, worry precluded change in anxiety levels during the fear clip, \( F(1,23) = 1.546, p > .05, r = .25 \). However, relaxation facilitated a strong increase in self-reported anxiety from before to after the fear clip, \( F(1,23) = 18.938, p < .001, r = .67 \).

For the sad clip, worry lead to a small but significant decrease in STAI scores from before to after the film clip, \( F(1,23) = 4.84, p < .05, r = .42 \), whereas relaxation lead to a significant increase in self-reported anxiety scores from before to after the clip,
Taken together, these results suggest that worry helps to reduce subjective anxiety in response to both fearful and sad emotional exposures.

As before, we looked to see if worry would lead to greater anxiety than relaxation, and whether or not this would be sustained across the clips. Though the worry induction led to significantly higher STAI scores than the relaxation induction (see means listed in Table 5), $t(37.14)=3.71, p=.001$, these conditions ended up with statistically similar anxiety levels following the fear clip, $t(46)=.21, p>.05$. This pattern was similar for the sad clip, with the worry condition starting out significantly higher, $t(38.74)=5.27, p<.001$, but both conditions ending up statistically similar, $t(46)=1.57, p>.05$. Again, these results suggest a ceiling effect of anxiety for individuals in the worry condition.

Finally, we looked at changes in STAI scores from before to after the calm and happy clips. Again, we found that both the worry and relax conditions lead to a decrease in anxious responding to the calm clip, $F(1,24)=31.39, p<.001, r=.57$; $F(1,22)=9.89, p<.05, r=.31$, respectively. This pattern was repeated in the happy clips as well, $F(1,23)=33.63, p<.001, r=.59$; $F(1,22)=4.77, p<.05, r=.17$, respectively. However, the small effect size for the relaxation condition suggests that these individuals may have experienced a floor effect.
Discussion

Results from the manipulation check confirmed that our worry and relaxation inductions were indeed effective, with the worry conditions leading to significantly more self-reported worry and anxiety than the relaxation conditions, and the relaxation conditions leading to significantly more self-reported relaxation than the worry conditions. This suggests that the following results may be interpreted as reflecting the influence of the intended inductions.

Physiological Effects

We failed to replicate prior research that has found baseline differences in vagal tone between individuals with GAD and non-anxious controls. However, this may simply reflect the fact that we did not use a true baseline time period. Instead, baseline measures were taken from the 10 minute acclimation period, during which time participants were still adjusting to the heart-monitoring device, listening to instructions, and even possibly beginning to practice the assigned induction. Therefore, this was not a true physiological baseline.

Induction Period

Consistent with our hypotheses we found that, overall, worry led to significantly lower vagal activity than did relaxation during the induction period. However, contrary to our predictions, further analysis revealed an interaction of group and induction type in that, while the GAD group showed the predicted physiological pattern for induction type, the control group paradoxically showed the
opposite pattern. As for the worry induction, perhaps this effect can be explained by the fact that participants with GAD were so practiced at the act of worrying that they were able to engage successfully in this task in a short period of time, leading to the predicted physiological results. Controls, on the other hand, may have had difficulty either achieving or maintaining intense worry during the induction period, resulting in less vagal impact of this task. It is less clear, however, when considering why the relaxation condition led to lower vagal activity for the non-anxious controls than the GAD group. One possibility may be that, perhaps in attempting to focus on the breathing exercise, the control group could have actually reduced their vagal tone as a function of concentration during the relaxation task. Unfortunately, there is not enough data to explore this hypothesis thoroughly. However, it should be taken into consideration that the effect size for this interaction was relatively small ($r=.20$), and as such must be interpreted with caution.

**Emotional Exposures**

The avoidance theory of worry suggests that worrying prior to an emotional exposure may suspend the impact of autonomic emotional responding via cognitive distraction. Consistent with our predictions, we found that worrying prior to a fearful exposure precluded appropriate vagal withdrawal, whereas relaxation facilitated it. Specifically, worry did not lead to significant change in vagal activity from the induction to the fear clip, suggesting muted sympathetic activation in response to a fearful exposure. By contrast, relaxation led to a significant decrease in vagal activity during the fear clip, facilitating an appropriate sympathetic response. These results
supplement and extend prior research which has used changes in heart rate as an indicator of fear avoidance (Borkovec & Hu, 1990; Borkovec et al., 1993; Peasley-Miklus & Vrana, 2000).

It is important to note that this pattern of physiological avoidance was not replicated for the sad exposure. Results indicate that neither worry nor relaxation precluded changes in vagal activity during the sad clip. Additionally, we found that both inductions actually led to increases in vagal activity in response to the sad clip. This is, in fact, consistent with extant research findings showing sadness to be associated with higher levels of vagal activity - the opposite of the pattern in fear (Kreibig, Wilhelm et al. 2007). Altogether, these results suggest that fear and sadness may have divergent patterns of physiological responding. Whereas fear is clearly sympathetically driven, sadness appears to be parasympathetically driven. Nonetheless, results from our study indicate that worry does not interfere with this response pattern, suggesting that the physiological avoidance role of worry may not generalize to other negative emotions, but may instead remain unique to the avoidance of fear.

Results from the positive emotional exposures lend further support to this hypothesis. Both the worry and relaxation inductions led to a significant increase in vagal activity for the calm clip, indicating no differential effect of worry on physiological responding. For the happy clip however, worry again led to an increase in vagal activity, but in this case relaxation was followed by no significant vagal changes during the clip. This may reflect a ceiling effect of vagal activity for individuals in the relaxation condition. Regardless of this effect, results demonstrate
that worry did not inhibit appropriate vagal responding to the positively valenced clips, again supporting the idea that the physiological avoidance effects of worry may remain unique to fear.

**Self-Report Emotional Responding**

An additional aim of this study was to test the hypothesis that worrying prior to emotional exposure would lead to greater subjective intensity of emotional experience and anxiety. What we found when examining the fear and sad clips, however, was that worry actually led to the *preclusion* of negative responding to these emotions. For instance, following the fear exposure, subjects in the worry condition reported no change in their negative affect and anxiety levels from before to after the film clip. In the sad exposure, subjects actually reported reductions in these levels following the clip. This is in contrast with the relaxation condition, in which subjects reported significant increases in their negative affect and anxiety levels following both clips. Overall, these results imply that worrying forestalled the subjective impact of subsequent negative emotional exposures.

Though this finding is inconsistent with our hypothesis that worry would increase subjective emotional responding, it is similar to a series of recent studies which have demonstrated that worry can: a) mute anxious responding during a subsequent rumination task, as well as reduce depressed mood when following a rumination (McLaughlin, Borkovec et al. 2007), and b) reduce subjective anxiety during subsequent trauma-recall tasks (Behar, Zuellig, & Borkovec, 2005).
To synthesize results from the examination of both subjective and physiological data, we conclude that worry in fact led to the preclusion of both subjective and physiological responding to an emotional stimulus, but this phenomenon was unique to the fear exposure. Additionally, although worry did not preclude the physiological response to sadness, it did reduce the negative affect and subjective anxiety associated with it. Taken together, these results suggest that worry does help to avoid the emotional impact of negatively-valenced emotional exposures. Results also map onto the finding that individuals with GAD rank distraction from more emotional topics as one of their top motivations for worrying (Borkovec & Roemer, 1995).

Given the results from this study, we can attempt to examine and elucidate the mechanism behind the process of emotional avoidance. We propose two different potential mechanisms by which worry may impede the processing of negative emotional material: either a) worry mutes overall levels of negative affect and anxiety and precludes change during a stressful emotional exposure, or b) worry raises negative affect and anxiety in anticipation of possible distressing emotional exposures. Findings from our study point to the second hypothesis. Worry inductions did in fact lead to greater levels of negative affect and anxiety and lower vagal activity for individuals with GAD, and these levels were sustained across the exposure (for fear) or somewhat alleviated (for sadness). This suggests that individuals with GAD may use worry to anticipate and prepare for future negative events. This is again in synchrony with the finding that persons with GAD report using worry to help them prepare for the worst (Borkovec & Roemer, 1995).
Though we did not find that worry led to a desynchrony between physiological and subjective emotional responding for individuals in the worry condition, the aforementioned results indicate that these individuals were already at high levels of negative affect and anxiety which were sustained across emotion exposures. As such, it is possible that individuals who chronically worry tend to associate heightened negative affect and anxiety with their emotions, and thus report them as subjectively more intense.

Furthermore, repeated avoidance of negative emotions may perpetuate the belief that emotions are threatening, and additional results from our study support this conclusion. Specifically we found that, prior to the experimental manipulations, individuals with GAD reported significantly greater perceived threat of a range of emotions, including sadness, guilt, fear, and strong emotions in general, than did non-anxious controls. This parallels extant research showing that individuals with GAD report difficulty understanding and controlling their emotions, as well as a fear of the consequences of experiencing emotion (Mennin et al., 2005; Roemer, Salters et al. 2005; Turk et al., 2005). We suggest that, though using worry as emotional avoidance may feel useful in the short-term, over time it reinforces the belief that emotions are dangerous and overwhelming, and hence adds to the motivation to continue avoidance in the future.

Positive Emotions

It is important to note, however, that we did not find worry to impede either physiological or subjective emotional responding to the positive exposures. In fact, worry actually led to greater increases in positive affect and reductions in anxiety
during these exposures than did relaxation. Additionally, individuals with GAD reported no more perceived threat of their positive emotions than did non-anxious controls. Thus these findings lend further support to our conclusion that the avoidance theory of worry may apply only to negative emotions.

These results, however, are in contrast with prior research findings showing that individuals with GAD do report fear of their positive as well as negative emotions (Mennin et al., 2005; Turk et al., 2005). It is possible that this is a vestige of the fact that our sample did not include individuals with clinical levels of GAD. Additionally, we used film clips to induce emotion. It is possible that more idiosyncratic, personally relevant emotional exposures could more likely be perceived as threatening to individuals with GAD. Therefore future research could usefully address these questions by using clinical samples and diversifying the types of emotional exposures used to tease out this effect.

Limitations

As stated above, the population for this study included analogue GADs. It is possible that these effects may not generalize to a population of individuals with clinical levels of GAD. An important step for future research would be to further explore these effects using a clinical population.

Another limitation to this study was the absence of a true baseline. We did not find baseline differences in vagal tone between our GAD and control samples, which may have resulted from additional tasks being performed during the baseline period. In future studies, a pure baseline period should be recorded after the participants have
acclimated to the heart monitor and before starting the experiment. This would be more accurate for detecting baseline differences in vagal tone between individuals with GAD and non-anxious controls.

Additionally, we found that our control group exhibited a counter-indicated pattern of physiological responding to the worry and relaxation inductions. It is possible that this result could have been influenced by an insufficient amount of time to rehearse these inductions. Future studies could address this concern by allowing ample time for all participants to practice the worry and relaxation tasks before beginning the experiment. Perhaps this would assist a non-anxious group in maintaining sufficient levels of worry and relaxation during the induction periods, thereby allowing for the full physiological effects both of worry and relaxation.

Finally, as noted above, our emotional exposures (film clips) were systematic rather than idiosyncratic or personally relevant. Our justification for this was to ensure that all participants had consistent exposures, rather than relying on individuals to conjure their own emotion stimulations. However, this may have altered the pattern of responding in a way that differs from real-life emotional experiences. Future research could usefully address this question through the use of idiosyncratic material for emotional exposures, such as memories of an emotionally salient event, or exposure to personal memorabilia (e.g., family photos).
Figure 1.

Interaction of Induction Type and Group on HF Power During Total Induction Period.

* significant at the <.05 level
** significant at the <.01 level

Note. Power spectral density values in the HF band represent non-transformed data, and are reported in milliseconds^2.

GAD
Control

worry relax
Table 1.

Means and Standard Errors of Change Scores of Power in the HF Variable from Induction to Emotional Exposure.

<table>
<thead>
<tr>
<th>Emotional Exposure Values</th>
<th>Induction Type</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Worry</td>
<td>Relaxation</td>
</tr>
<tr>
<td>Fear</td>
<td>-17.42(12.87)</td>
<td>-125.33**(30.55)</td>
</tr>
<tr>
<td>Sad</td>
<td>253.94**(27.24)</td>
<td>219.53**(39.57)</td>
</tr>
<tr>
<td>Calm</td>
<td>176.53**(24.72)</td>
<td>91.06**(26.31)</td>
</tr>
<tr>
<td>Happy</td>
<td>109.9**(22.7)</td>
<td>10.56(32.27)</td>
</tr>
</tbody>
</table>

*Note.* Power spectral density values in the HF band are reported in milliseconds\(^2\).

* change over time is significant at the <.05 level

** change over time is significant at the <.01 level
Table 2.

Means and Standard Errors of Power in the HF Variable from Induction to Emotional Exposure.

<table>
<thead>
<tr>
<th>Emotional Exposure Score</th>
<th>HF Scores Worry</th>
<th>HF Scores Relax</th>
<th>Interaction Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Induction</td>
<td>Exposure</td>
<td>Induction</td>
</tr>
<tr>
<td>Fear</td>
<td>539.1(42)</td>
<td>521.7(37.9)</td>
<td>641.1(44.7)</td>
</tr>
<tr>
<td>Sad</td>
<td>580.6(55.8)</td>
<td>834.5**(67.7)</td>
<td>660.6(57)</td>
</tr>
<tr>
<td>Calm</td>
<td>534.3(37.8)</td>
<td>710.9**(49.3)</td>
<td>565(39.5)</td>
</tr>
<tr>
<td>Happy</td>
<td>549.7(51.5)</td>
<td>659.6**(51.2)</td>
<td>660.3(52.7)</td>
</tr>
</tbody>
</table>

*Note.* Power spectral density values in the HF band are reported in milliseconds\(^2\).

* change over time is significant at the <.05 level

** change over time is significant at the <.01 level
Table 3.

Means and Standard Errors of PTEQ Scores for the GAD and Control Groups.

<table>
<thead>
<tr>
<th>PTEQ Subscale</th>
<th>Group</th>
<th>Significance Values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GAD</td>
<td>Control</td>
</tr>
<tr>
<td>Sadness</td>
<td>16.24(.9)</td>
<td>12.9(.53)</td>
</tr>
<tr>
<td>Guilt</td>
<td>16.87(1.02)</td>
<td>13.74(.81)</td>
</tr>
<tr>
<td>Fear</td>
<td>18.68(.87)</td>
<td>15.94(.80)</td>
</tr>
<tr>
<td>Strong Emotions</td>
<td>18.45(.91)</td>
<td>15.43(.77)</td>
</tr>
<tr>
<td>PTEQ Total</td>
<td>112.0(4.68)</td>
<td>98.17(4.23)</td>
</tr>
</tbody>
</table>

Note. PTEQ = Perceived Threat of Emotions Questionnaire.
Table 4.

Means and Standard Errors of Change Scores in NA, PA, and Anxiety Levels from Induction to Emotional Exposure.

<table>
<thead>
<tr>
<th>Clip</th>
<th>NA Worry</th>
<th>NA Relax</th>
<th>PA Worry</th>
<th>PA Relax</th>
<th>NA/PA Sig.</th>
<th>STAI-S Worry</th>
<th>STAI-S Relax</th>
<th>STAI Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fear</td>
<td>-1.04(9)</td>
<td>5.5**(1.3)</td>
<td></td>
<td></td>
<td>ρ&lt;.01</td>
<td>-2.05(2.0)</td>
<td>8.95**(2.1)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Sad</td>
<td>-2.44*1.1</td>
<td>2.5**(.6)</td>
<td></td>
<td></td>
<td>ρ&lt;.01</td>
<td>-3.0*(1.6)</td>
<td>7.36**(.6)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Calm</td>
<td></td>
<td></td>
<td>1.08(1.1)</td>
<td>-.17(.8)</td>
<td>ns</td>
<td>-13.37**(.6)</td>
<td>-3.8*(1.6)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Happy</td>
<td></td>
<td></td>
<td>5.12**(.9)</td>
<td>5.83**(1.1)</td>
<td>ns</td>
<td>-15.84**(.5)</td>
<td>-2.3*(1.6)</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

Note. NA = Negative Affect, PA = Positive Affect, STAI-S = Anxiety, NA and PA scores are reported as non-transformed values.

* change over time is significant at the <.05 level

** change over time is significant at the <.01 level
Table 5.

Means and Standard Errors of NA, PA, and STAI-S Scores from Induction to Emotional Exposure.

<table>
<thead>
<tr>
<th>Emotional Exposure</th>
<th>Self-Report Measure</th>
<th>Worry Pre-Clip</th>
<th>Worry Post-Clip</th>
<th>Relax Pre-Clip</th>
<th>Relax Post-Clip</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fear</td>
<td>NA</td>
<td>16.92(1.25)</td>
<td>15.9(1.25)</td>
<td>11.67(.36)</td>
<td>16.9**(1.36)</td>
</tr>
<tr>
<td></td>
<td>STAI-S</td>
<td>48.21(2.88)</td>
<td>46.21(2.4)</td>
<td>36.83(1.56)</td>
<td>45.5**(1.98)</td>
</tr>
<tr>
<td>Sad</td>
<td>NA</td>
<td>18.36(1.4)</td>
<td>15.9*(1.13)</td>
<td>11.04(.33)</td>
<td>13.54**(.57)</td>
</tr>
<tr>
<td></td>
<td>STAI-S</td>
<td>49.21(2.48)</td>
<td>46.33**(2.2)</td>
<td>34.83(1.44)</td>
<td>42.13**(1.55)</td>
</tr>
<tr>
<td>Calm</td>
<td>PA</td>
<td>15.6(.81)</td>
<td>16.68(.98)</td>
<td>16.33(.79)</td>
<td>16.17(.92)</td>
</tr>
<tr>
<td></td>
<td>STAI-S</td>
<td>50.2(2.32)</td>
<td>36.56**(1.85)</td>
<td>38.04(1.88)</td>
<td>33.83**(1.7)</td>
</tr>
<tr>
<td></td>
<td>STAI-S</td>
<td>49.33(2.79)</td>
<td>34.17**(1.63)</td>
<td>34.71(1.73)</td>
<td>32.54*(1.4)</td>
</tr>
</tbody>
</table>

Note. NA = Negative Affect, PA = Positive Affect, STAI-S = Anxiety.

* change over time is significant at the <.05 level

** change over time is significant at the <.01 level
References


