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Effects of Worry on Physiological and Subjective Reactivity to Emotional Stimuli in Generalized Anxiety Disorder and Nonanxious Control Participants

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The present study examined the effect of worry versus relaxation and neutral thought activity on both physiological and subjective responding to positive and negative emotional stimuli. Thirty-eight participants with generalized anxiety disorder (GAD) and 35 nonanxious control participants were randomly assigned to engage in worry, relaxation, or neutral inductions prior to sequential exposure to each of four emotion-inducing film clips. The clips were designed to elicit fear, sadness, happiness, and calm emotions. Self reported negative and positive affect was assessed following each induction and exposure, and vagal activity was measured throughout. Results indicate that worry (vs. relaxation) led to reduced vagal tone for the GAD group, as well as higher negative affect levels for both groups. Additionally, prior worry resulted in less physiological and subjective responding to the fearful film clip, and reduced negative affect in response to the sad clip. This suggests that worry may facilitate avoidance of processing negative emotions by way of preventing a negative emotional contrast. Implications for the role of worry in emotion avoidance are discussed.

Keywords: generalized anxiety disorder, worry, emotion avoidance, vagal tone

Theory and evidence suggest that whereas experiencing of most emotions is beneficial to psychological and physical health (Butler et al., 2003; Huang, 2004; Pennebaker & Beall, 1986; Pennebaker & Francis, 1996), avoidance of these emotions may be detrimental for such health (Butler et al., 2003; Huang, 2004; Solano et al., 2001; Wastell, 2002). Such emotional avoidance has been posited to be a factor in the maintenance of generalized anxiety disorder (GAD).

In particular, Borkovec and colleagues (Borkovec, Alcaine, & Behar, 2004) have suggested that worrisome thinking, the cardinal feature of GAD, may facilitate the avoidance of aspects of fearful responding. Indeed, several studies demonstrate that worrying (vs. relaxing) just prior to imaginal exposure to fear-related stimuli results in lower cardiovascular reaction to those stimuli (Borkovec & Hu, 1990; Peasley-Miklus & Vrana, 2000). Moreover, amount of time spent in worrisome thought (as opposed to imagery) during the worrying is negatively correlated with cardiac reactivity to subsequent fearful images, whereas the amount of time spent during relaxation is positively correlated with cardiac reactivity to the images (Borkovec, Lyonfields, Wiser, & Deihl, 1993). Taken together, these data suggest that it is the *worrisome* nature of thoughts that mutes cardiac responding to fearful images. According to Foa and Kozak (1986), reduced cardiovascular responding to fearful stimuli suggests that the fear structure has not been fully accessed, and thus emotional processing that would otherwise generate reductions in fearful associations has been precluded.

In addition to findings associating worrisome thinking with muted cardiac responding to fearful images, persons with GAD are more likely than nonanxious control participants to report using worry as a means to distract themselves from unwanted emotions (Borkovec & Roemer, 1995; Freeston, Rheaume, Letarte, Dugas, & Ladouceur, 1994). Additional studies have found that persons with GAD report greater fear of the negative consequences of a range of emotions than do nonanxious controls (Mennin, Heimberg, Turk, & Fresco, 2005; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). Mennin and colleagues (2005) tested these findings by inducing anxious, sad, and neutral states in both GAD and nonanxious groups, and found that persons with GAD endorsed greater subjective reactivity to the sad and anxious mood inductions than did controls. This is consistent with prior studies by Borkovec and colleagues showing greater subjective fear ratings during repeated exposures to fearful imagery, despite lowered cardiovascular reactivity (e.g., Borkovec et al., 1993). Taken together, these studies suggest that persons with GAD experience greater subjective distress associated with a range of emotions than do nonanxious controls.

Importantly, absent from the literature are experimental data regarding the impact of worry on cardiovascular responding to a range of emotions. Although worry has been shown to preclude physiological reactivity to fearful emotion inductions, it is unclear whether worry also inhibits psychophysiological responses to sad, calm, or happy emotions. Given that persons with GAD report subjective difficulty with a range of emotions, including the pos-

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itive realm (Mennin et al., 2005; Turk et al., 2005), it is possible that these individuals are motivated to avoid such emotions. In fact, one study showed that persons with GAD exhibited a defensive cardiac reaction to presentations of positively valenced images (Yamasaki, Behar, & Ray, 2002). Moreover, data have shown a pervasive lack of autonomic flexibility in chronic worriers (Brosschot, Van Dijk, & Thayer, 2007; Hoehn-Saric & McLeod, 1988; Hoehn-Saric, McLeod, & Zimmerli, 1989; Thayer, Friedman, & Borkovec, 1996). However, no study has tested whether worry leads to muted autonomic reactivity in response to sad, happy, or calm emotional stimuli.

The goal of the present study was to examine the causal effect of worry on physiological and subjective responding to fearful, sad, calm, and happy emotion stimuli. We predicted that worrying (vs. relaxing) prior to the presentation of each emotion stimulus would lead to the preclusion of autonomic and subjective reactivity in both GAD analogues and control participants.

Of note, in healthy controls each emotion is thought to lead to different patterns of sympathetic and parasympathetic reactivity. For example, positive emotions are associated with a shift toward higher parasympathetic (vagal) activity (McCraty, Atkinson, Tiller, Rein, & Watkins, 1995). Conversely, the fear response leads to increased sympathetic and decreased parasympathetic (vagal) activity, or vagal withdrawal (Kreibig, Wilhelm, Roth, & Gross, 2007; Rainville, Bechara, Naqvi, & Damasio, 2006). Moreover, in a number of studies, sadness was associated with increased parasympathetic activity (Gross, Fredrickson, & Levenson, 1994; Hendriks, Rottenberg, & Vingerhoets, 2007; Marsh, Beauchaine, & Williams, 2008; Rottenberg, Wilhelm, Gross, & Gotlib, 2003), and decreased sympathetic activity (Gross & Levenson, 1997; Kreibig et al., 2007; Tsai, Levenson, & Carstensen, 2000); although others have found HR acceleration to sad exposure (see Kreibig et al., 2007). Further, individuals with depression have evidenced parasympathetic decreases in response to sad stimuli (Rottenberg, Salomon, Gross, & Gotlib, 2005), which suggests some heterogeneity in terms of sad responding. Based on the avoidance theory of worry, as well as the majority of findings in healthy control participants, we predicted that worry would preclude vagal withdrawal in response to fearful stimuli, and preclude an increase in vagal activity in response to sad, calm, and happy stimuli.

Method

Overall Design

A 2 (group: GAD vs. nonanxious) × 3 (induction type: worry, relax, or neutral) block design was used to explore the differential effects of worry, relaxation, and neutral inductions on reactivity to four different emotional stimuli (fear, sadness, calm, and happiness).

Participants

Seventy-three participants (51 females; *M* age = 18.85 years, *SD* = .95 years) were recruited for this study from introductory psychology courses at a rural state university. Students were given class credit as compensation for their participation in this research. The ethnic distribution of participants was 87.7% Caucasian, 4.1% Asian, 2.7% African American, 2.7% Hispanic, 1.4% Middle Eastern, and 1.4% Native American.

Participants were selected based on their scores on the Generalized Anxiety Disorder Questionnaire—IV (GAD-Q-IV; Newman et al., 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 (n = 38) or nonanxious control group (n = 35). Individuals were included in our GAD group if they scored above the 5.7 cutoff on the GAD-Q-IV (*M* = 9.06, *SD* = 1.52), endorsed symptoms for at least 6 months, and answered affirmatively to each of the first four questions on the GAD-Q-IV representing the major diagnostic criteria for GAD (i.e., Do you experience excessive worry? Is your worry excessive in intensity, frequency, or amount of distress it causes? Do you find it difficult to control your worry [or stop worrying] once it starts? Do you worry excessively and uncontrollably about minor things such as being late for an appointment, minor repairs, homework, etc.?). We also required that individuals scored at least a standard deviation above the mean on the PSWQ (*M* = 70.45, *SD* = 5.70). Mean PSWQ scores were comparable to mean levels of those with clinically diagnosed GAD (see Startup & Erikson, 2006).

Individuals were included in the control group if they answered negatively to the first 4 questions and scored below the cutoff on the GAD-Q-IV (*M* = 1.46, *SD* = 1.44), and within a standard deviation below the mean on the PSWQ (*M* = 42.40, *SD* = 7.22). Groups did not differ in terms of gender, ethnicity, or age, and these factors were also balanced across conditions.

In order to maximize the likelihood that our results would generalize to others with GAD, we chose not to restrict comorbid disorders as participants with GAD tend to have high rates of concurrent comorbidity (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Sanderson, Beck, & Beck, 1990). Nonetheless, because depression is associated with a diminished capacity to habituate to anxious states and may hinder emotional processing (Foa & Kozak, 1986), all participants were tested for comorbid depressed mood using the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) and completed this measure prior to the experiment. Measurement of depression allowed us to determine whether differential levels of depressed mood between those in differential induction conditions might explain any differences found between these two groups.

Measures

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). Internal consistency (Cronbach's alpha = .94) and 2-week retest reliability (92% of the sample) are strong. In addition, the measure has demonstrated convergent and discriminant validity, and kappa agreement of .67 with a structured interview. A cutoff of 5.7 leads to sensitivity of 83% and specificity of 89%. Students diagnosed with GAD by the GAD-Q-IV were not significantly different on this measure than a GAD community sample, but both groups had significantly higher scores than students identified as not meeting criteria for GAD, demonstrating clinical validity of the GAD-Q-IV (Newman et al., 2002).

The PSWQ (Meyer et al., 1990) 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. Factor analysis indicates that the PSWQ assesses a unidimensional construct with an internal consistency coefficient of .91 (Meyer et al., 1990). High test–retest reliability (ranging from .74–.93) was also demonstrated across periods ranging from 2–10 weeks (Molina & Borkovec, 1994). The PSWQ has also been shown to distinguish individuals with GAD from each of the other anxiety disorder groups (Brown, Antony, & Barlow, 1992). Correlations between the PSWQ and measures of anxiety, depression, and emotional control supported the convergent and discriminant validity of the measure (Brown et al., 1992).

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. 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 (Beck, Steer, & Garbin, 1988). 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 nonpsychiatric patients (Beck et al., 1988).

Emotion-eliciting stimuli. Film clips were thought to be the most appropriate stimuli for this study 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 stimuli such as photographs, film clips allow emotions to be experienced in a nuanced, gradually unfolding manner that may be more characteristic of emotional events experienced in participants' lives (Gross & Levenson, 1995; Rottenberg, Ray, & Gross, 2007).

Participants viewed four brief film clips (ranging in length from 120 to 165 seconds) representing negative (fear and sadness) and positive (calm and happiness) emotions. The film clips used in this study have been successful at eliciting the desired emotions in previous studies (e.g., Frederickson & Levenson, 1998; Gross & Levenson, 1995; Rottenberg, Kasch, Gross, & Gotlib, 2002; Rottenberg et al., 2005; Sloan, 2004). These clips include scenes of a plane crash (fear), a son grieving over his dying father (sadness), nature (calm), and slapstick comedy (happiness). The clips were presented in counterbalanced order to control for sequencing effects.

Self-report emotion measures. The Perceptions of Threat from Emotion Questionnaire (PTEQ; McCubbin & Sampson, 2006) is a 72 item measure of beliefs about the threat posed by seven basic emotions (including fear, sadness, happiness, and disgust) as well as "strong emotions in general." Items from 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 rate how they experience these emotions *most of the time* on a 5-point Likert scale ranging from "not at all" to "definitely." The PTEQ demonstrates good retest reliability ($r = .83$, $p < .001$), satisfactory internal consistency (apart from the "happiness" subscale Cronbach's alpha = .41) and divergent validity, as well as face and construct validity (McCubbin & Sampson, 2006).

The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 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 (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 et al., 1988).

Physiological measures. Heart-rate 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. In order to measure vagal activity, we observed differences in heart rate variability (HRV), a measure of physiological rhythms in the beat-to-beat interval of each cardiac signal. HRV was analyzed with Acq*Knowledge* 3.9 software using a template-matching approach in the frequency domain method. We first applied a bandpass 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 resampled to a continuous sampling rate in order to extract frequency information. Considering the low sampling rate of the MP30 system (200Hz), we used cubic-spline interpolation on the ECG signals to decrease error (Task Force of the European Society of Cardiology and North American Society of Pacing 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. We chose to examine power in the HF bands of the HRV output because research considers this to be a good estimate of vagal efferent effects on the heart (see Task Force, 1996).

For the purposes of this study, we used short-term recordings (2 minutes) to look at the HF components of the HRV output. Recordings were taken during the induction periods and during the film clips. Power spectral density values in the HF band are reported in milliseconds squared (ms^2).

Procedure

Research assistants followed a standardized protocol for all participants. Participants were informed that they would be tested individually in a study of how people respond to movie scenes. Upon arrival, they were randomly assigned to receive either the worry ($n = 25$), relaxation ($n = 24$), or neutral ($n = 24$) induction. After completing the consent form, participants were fitted with the heart-monitoring equipment, and subsequently completed the PTEQ and the BDI.

Next, participants were seated in a comfortable chair facing the computer monitor. They were given 10 minutes to acclimate to the

situation before beginning the experiment. A two minute resting baseline recording was taken during this time. Following this recording, participants were asked to practice their assigned induction. Instructions were given for worrying ("think about your most worrisome topic and worry about it as intensely as you can"), relaxing (instructions on slowed diaphragmatic breathing), or neutral activity ("think about what you did over the last weekend"), depending upon condition. Participants were informed that if at any point their mind wandered off task, they were to simply refocus their thoughts on the task. Our worry and relaxation inductions were congruent with those used in previous studies (e.g., Borkovec & Inz, 1990; Behar, Zuellig, & Borkovec, 2005; Oathes et al., 2008; Ray et al., 2009). However, there are few, if any, previous studies that included a neutral condition, which suggests that this is an important addition to the literature. In order to ensure that inductions were successful, participants were given a manipulation check following each induction consisting of two 5-point Likert scales assessing levels of worry and relaxation. The manipulation check measures were modeled after those used in other studies (e.g., Behar et al., 2005; Oathes et al., 2008).

Following practice, participants completed the PANAS then viewed a practice clip, followed by a postclip PANAS. Because this was a nonemotion clip and presented only to familiarize participants with the experimental paradigm, no physiological measures were obtained during this period.

Once 10 minutes had elapsed, individuals were asked to engage in their assigned induction for 2 minutes, complete the PANAS, view the first film clip, and complete the postclip PANAS. They were then asked to perform a distracter task (simple math problems) for 1 minute. To begin the next segment, participants were asked to reengage in the induction task (2 minutes) and repeat the same procedure until all four film clips had been viewed. At the end of the session, heart monitoring electrodes were removed and participants were fully debriefed. Care was taken that participants were not experiencing lingering and distressing negative emotions, and no participants terminated the study prematurely. As a follow-up, all participants were given contact information for university psychological services should they feel the need to talk with a counselor.

Results

Based on an a priori power analysis, it was determined that a sample size of at least 72 participants was required to observe a medium between-groups effect size (Cohen's $f = .30$) with an α-level of .05 and power of .80 (Faul, Erdfelder, Lang, & Buchner, 2007). In order to avoid chance effects, Bonferroni adjustments were used when comparing more than two groups in the same analysis.

Participant Parameters

Preliminary analyses showed no significant differences between GAD ($n = 38$, worry = 12, relax = 13, neutral = 13) and nonanxious participants ($n = 35$, worry = 13, relax = 11, neutral = 11) in age, gender, or ethnicity. Similarly, there were no demographic differences between those who were randomly assigned to the worry ($n = 25$), relaxation ($n = 24$), or neutral ($n = 24$) conditions. In addition, resting baseline measures of vagal activity showed no differences between individuals across induction conditions, $F(2, 70) = 0.28$, *ns*, nor were there any significant differences between the GAD and control groups during this time, $F(1, 71) = 0.06$, *ns*.

As expected, individuals with GAD had significantly higher BDI scores at baseline ($M = 9.66$, $SD = 5.93$) than did nonanxious participants ($M = 4.63$, $SD = 4.43$), $t(71) = 4.08$, $p < .001$. Although the means for each group were within the normal to low range of depressed mood, it should be noted that 10.5% of individuals in the GAD group had scores reflecting moderate-severe levels of depression ($M = 22.25$, $SD = 3.30$). More importantly, there were no significant differences in BDI scores across worry, relax and neutral induction conditions, $F(2, 70) = 1.01$, *ns*, and this was true within both the GAD, $F(2, 35) = 2.16$, *ns*, and control groups, $F(2, 32) = 0.20$, *ns*.

PTEQ

To determine whether participants with GAD reported more perceived threat from their emotional experiences than did nonanxious controls, *t* tests were conducted on PTEQ scores. Individuals with GAD reported greater perceived threat of emotion compared to controls on a range of PTEQ subscales, including sadness, guilt, fear, and strong emotions in general, as well as the total PTEQ score. (See Table 1 for mean scores and *t* values).

Manipulation Check

Because some manipulation check scores were significantly non-normal, we performed a log transformation on all manipulation check data. In order to assess the effectiveness of our inductions, a multivariate analysis of variance (MANOVA) was run using results of the manipulation check (collapsed across the four inductions) as the dependent variable, and group and induction type as the independent variables. Results indicated a main effect of group, $F(1, 71) = 7.97$, $p < .01$, induction type, $F(2, 70) = 12.66$, $p < .001$, and a group by induction interaction, $F(3, 68) = 2.90$, $p < .05$. There were no significant changes in manipulation check scores from the first to the last induction, suggesting that induction effects did not diminish over time.

Table 1
Means and Standard Deviations of PTEQ Scores for the GAD and Control Groups

	Group		
PTEQ subscale	GAD	Control	*t* values
Sadness	16.24 (5.53)	12.91 (3.16)	3.18**
Guilt	16.87 (6.31)	13.74 (4.77)	2.37*
Happiness	10.34 (2.61)	10.23 (2.57)	0.19
Anger	19.87 (6.20)	17.00 (5.40)	1.51
Fear	18.68 (5.38)	15.94 (4.72)	2.31*
Lust	16.08 (5.10)	14.57 (5.64)	1.12
Disgust	13.92 (6.27)	12.97 (5.46)	0.69
Strong emotions	18.45 (5.58)	15.43 (4.54)	2.52*
PTEQ total	112.0 (28.83)	98.17 (25.03)	2.18*

Note. PTEQ = Perceived Threat of Emotions Questionnaire.
* $p < .05$. ** $p < .01$.

For individuals with GAD, those in the worry condition reported significantly more worry than those in the neutral condition, whose scores were above those in the relaxation condition ($p < .001$ and $p < .01$, respectively). As for the control group, individuals in the worry condition again reported significantly more worry than those in the neutral and relaxation conditions ($p < .05$ and $p < .01$, respectively), whose scores remained statistically similar. We also found that individuals with GAD in the worry condition reported significantly more worry than controls in this condition, $t(23) = 3.89$, $p < .01$, and the same was true for the neutral condition, $t(14.8) = 2.52$, $p < .05$, but not for the relaxation condition.

As for reported levels of relaxation, there was a main effect of induction type, $F(2, 70) = 20.71$, $p < .001$, but no interaction with group. Individuals in the relaxation condition reported significantly higher levels of relaxation than those in the neutral condition ($p < .05$), whose scores were higher than those in the worry condition ($p < .01$). (See Table 2 for mean scores and standard deviation values.)

Physiological Measures

Induction period. Because some vagal activity scores were significantly non-normal, we performed a log transformation on all vagal activity data. A repeated-measures analysis of variance (ANOVA) with time as the within-subjects variable and GAD status and induction type as the between-subjects' variables showed no significant differences across the four induction periods on vagal activity, suggesting that induction effects were consistent in vagal activity across separate trials. However, there was a significant interaction of group by induction type, $F(3, 60) = 4.79$, $p < .05$.

Separate MANOVAs showed a significant effect of induction type for the GAD group, $F(2, 29) = 4.77$, $p < .05$. During the induction periods, worry led to significantly lower vagal activity ($M = 475.29$, $SD = 144.04$) than did relaxation ($M = 716.64$, $SD = 227.2$), $p < .05$, with the neutral condition falling nonsignificantly in between ($M = 541.32$, $SD = 106.3$) (See Figure 1). However, individuals in the control group had statistically similar vagal activity levels regardless of induction type. (HF power values are reported in nontransformed ms^2.)

Emotional exposure. Separate repeated measures ANOVAs were conducted for each film clip exposure (fear, sadness, calm, and happiness) to examine change from induction period to exposure. For the fear clip, there was a significant change in vagal activity over time, $F(1, 68) = 29.64$, $p < .001$, as well as a time by induction type interaction, $F(3, 65) = 7.1$, $p < .01$. Whereas prior relaxation led to the expected vagal withdrawal in response to the fear clip, $F(1, 20) = 25.89$, $p < .001$, partial $\eta^2 = .55$, prior worry did not lead to vagal activity change (see Figure 2). There was no difference between the prior neutral and prior worry inductions in vagal response to the fear clip, but there was a significant difference between the relaxation and neutral inductions, $F(2, 41) = 7.51$, $p < .01$, partial $\eta^2 = .15$, with relaxation leading to greater change in vagal activity than the neutral condition. However, when divided by group, the effect of the neutral induction fell nonsignificantly in between worry and relaxation. Specifically, whereas persons with GAD receiving the prior neutral induction did not experience a significant vagal response to the fear clip, controls in the neutral condition did, $F(1, 10) = 16.08$, $p < .01$, partial $\eta^2 = .62$.

In exploring the sad and calm clips, there was no interaction between time and induction, or time and induction by group. However, both the sad and calm clips led to a significant effect of time; $F(1, 68) = 313.53$, $p < .001$, partial $\eta^2 = .83$; $F(1, 67) = 116.89$, $p < .001$, partial $\eta^2 = .63$, respectively. In each case, vagal activity increased in response to the clip regardless of

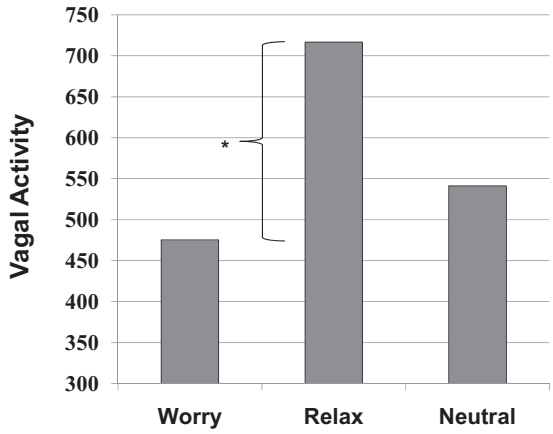

Figure 1. Vagal activity in the GAD group collapsed across induction periods. Considering that differences were statistically significant, the scale was enlarged to elucidate differences; PSD values are reported in nontransformed ms^2, $^* p < .05$.

Table 2
Means and Standard Deviations of Manipulation Check Scores by Group and Induction Type

Manipulation scale by group	Induction type		
	Worry	Neutral	Relax
Worry			
GAD	17.42 (4.17)	10.31 (5.69)	5.69 (0.85)
Control	10.85 (4.28)	6.09 (1.81)	6.82 (3.06)
Relaxation			
Collapsed	10.96 (4.30)	15.42 (5.87)	18.92 (4.76)

Note. Manipulation check scores are reported in nontransformed values.

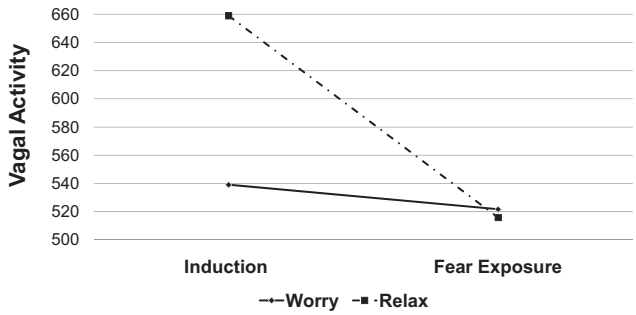

Figure 2. Vagal activity changes in response to the fear exposure. HF power values are reported in nontransformed ms^2.

induction type, suggesting no differences between worry, relaxation, and neutral inductions on physiological responses to sad and calm exposures.

The happy clip led to change over time in vagal activity, $F(1, 64) = 29.32$, $p < .001$, as well as a significant interaction between time and induction type, $F(3, 61) = 3.32$, $p < .05$, but no time by induction by group interaction. When comparing inductions, there was a difference between worry and relaxation in response to the happy clip, $F(2, 39) = 5.44$, $p < .05$, partial $\eta^2 = .12$, with worry leading to a greater increase in vagal activity than relaxation, and neutral falling nonsignificantly in between. In fact, whereas worry led to a significant change in vagal activity over time, $F(1, 20) = 32.88$, $p < .001$, partial $\eta^2 = .62$, relaxation did not.

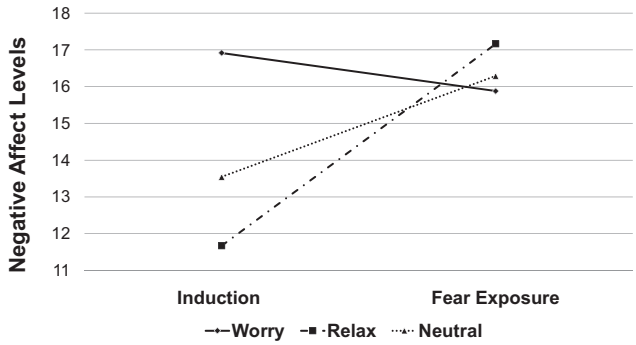

Figure 3. Change in negative affect levels in response to the fear exposure. (Negative Affect scores are reported in nontransformed values.)

Self-Report Measures

Induction period. Because some PANAS scores were significantly non-normal, we performed a log transformation on all PANAS data. Comparing postinduction NA levels averaged across trials, we found a significant effect of group, $F(1, 71) = 24.84$, $p < .001$, induction type, $F(2, 70) = 19.92$, $p < .001$, as well as a group by induction interaction, $F(3, 68) = 5.0$, $p < .01$. For participants with and without GAD, worry led to significantly higher NA levels than neutral and relaxation conditions ($p < .05$ for both), but neutral and relaxation conditions did not differ significantly from each other. Furthermore, individuals with GAD endorsed greater NA levels than did controls during the worry inductions, $F(1, 23) = 21.71$, $p < .001$, partial $\eta^2 = .49$ ($M = 21.98$, $SD = 5.84$; $M = 13.78$, $SD = 3.10$, respectively), as well as the neutral inductions, $F(1, 22) = 6.86$, $p < .05$, partial $\eta^2 = .24$ ($M = 16.02$, $SD = 6.85$; $M = 11.04$, $SD = 1.12$, respectively), but not during the relaxation inductions ($M = 11.62$, $SD = 1.44$; $M = 11.11$, $SD = 1.81$, respectively). When comparing PA, however, all inductions led to statistically similar preclip levels, and there were no group differences. (All NA scores are reported in nontransformed values.)

Emotional exposure. In exploring subjective reactions to the nonemotional practice clip, we found no increased responding (either NA or PA) regardless of group or condition. However, there was a significant effect of time on NA scores from the induction to the fear clip, $F(1, 71) = 22.56$, $p < .001$, as well as a significant interaction between time and induction type, $F(3, 68) = 13.39$, $p < .001$, but no interaction of time and induction type by group. Whereas relaxation led to a significant increase in NA in response to the fear clip, $F(1, 22) = 36.96$, $p < .001$, partial $\eta^2 = .62$, worry did not (see Figure 3). For the sad film clip, there was a also an effect of time, $F(1, 71) = 4.88$, $p < .05$, as well as an interaction between time and induction type, $F(3, 68) = 12.81$, $p < .001$. Again, there was no interaction of time, induction type, and group. As with the fear exposure, relaxation facilitated a significant increase in NA in response to the sad clip, $F(1, 23) = 20.44$, $p < .001$, partial $\eta^2 = .47$. However, worry actually led to a significant *decrease* in NA levels in response to the sad clip, $F(1, 24) = 5.86$, $p < .05$, partial $\eta^2 = .20$ (see Figure 4).

Although there was a significant difference between the worry and neutral inductions in amount of NA responding to both the fearful, $F(2, 45) = 10.83$, $p < .05$, partial $\eta^2 = .19$, and sad clips, $F(2, 45) = 12.17$, $p = .001$, partial $\eta^2 = .21$, there was no difference between relaxation and neutral inductions for either clip.

Next we explored the positively valenced clips. For the calm clip, there was no change in PA levels from the induction to the clip, nor was there an interaction between time and induction type, or time and induction type by group. Results from the happy clip revealed a significant effect of time, $F(1, 71) = 105.36$, $p < .001$, partial $\eta^2 = .61$, but no effect of time by induction type nor a time by group by induction type interaction, suggesting that the happy clip elicited a significant increase in PA regardless of group or induction type. As for differences in overall PA levels, all groups and induction types reached statistically similar levels during each positive clip.

Discussion

The avoidance theory of worry (Borkovec et al., 2004) states that prior worry may mitigate the impact of an emotional exposure through reduced autonomic responding. Before the current study, this theory had been tested experimentally, but only in the context of fear avoidance. Our inclusion of sadness, calm, and happy emotion inductions in addition to fear allowed us to examine whether this theory applied more broadly to negative and/or positive emotions. We hypothesized that worrying (vs. relaxing) prior to fearful, sad, calm, and happy emotion inductions would lead to the preclusion of autonomic and subjective reactivity in both GAD analogues and control participants. Our results partially supported the hypotheses, demonstrating different patterns across each emotion.

Consistent with the avoidance of emotional processing theory, prior worry (vs. relaxation) resulted in less vagal withdrawal and less subjective reactivity for all participants in response to a fearful exposure. As for the effects of the neutral induction, however, it appears that this condition may have led to the participants' natural state (i.e., participants with GAD may have been somewhat worried and nonanxious participants may have been calm). Specifically, those with GAD in the prior neutral induction did not evidence vagal withdrawal upon exposure to the fear clip, whereas nonanxious participants did. However, compared to prior worry, all participants receiving the neutral induction reported greater subjective responding to the fear exposure (with no differences between reactivity in neutral and relaxation conditions). Overall, this provides some support for the idea that this effect was driven

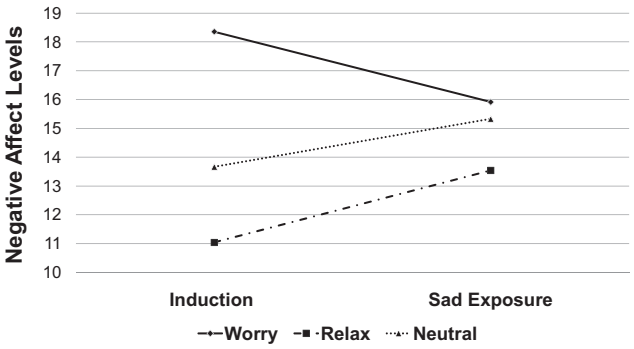

Figure 4. Change in negative affect levels in response to the sad exposure. (Negative Affect scores are reported in nontransformed values.)

by worry precluding reactivity as opposed to relaxation enhancing reactivity. Further, these results represent a conceptual replication of prior research that used imaginal emotion inductions (rather than standardized film clips) as well as changes in heart rate (rather than changes in vagal activity) to examine fear reactivity (Borkovec & Hu, 1990; Borkovec et al., 1993; Peasley-Miklus & Vrana, 2000).[1]

Results for sadness partially supported our hypotheses. Whereas subjective reactivity was inhibited by prior worry, vagal reactivity was not. Specifically, all prior inductions led to increased vagal activity, but whereas relaxation and neutral inductions led to increased subjective negative affect, prior worry actually led to reduced negative affect in response to the sad clip. Together, these results suggest that although worry does not interfere with the natural physiological response to sadness, it nonetheless inhibits increases in subjective negative emotion.

The subjective emotion findings for our sad exposure are similar to a series of recent studies demonstrating that worry can: a) mute anxious responding during a subsequent rumination task, as well as reduce depressed mood when following rumination (McLaughlin, Borkovec, & Sibrava, 2007), and b) reduce subjective anxiety during subsequent trauma-recall tasks (Behar et al., 2005). Together, these results relate well to the finding that on a survey of the perceived functions of worry, distraction from more emotional topics was the only item that significantly distinguished individuals with GAD from both subclinical as well as nonanxious controls (Borkovec & Roemer, 1995).

In terms of positive emotional exposures, however, results differed from what we expected. Due to recent findings that persons with GAD report difficulty with positive emotions (Mennin et al., 2005; Turk et al., 2005), we had predicted that the avoidance theory of worry would extend to positive exposures as well. In fact, we found no inhibiting effects of worry, either physiologically or subjectively, for our calm and happy clips. In response to the calm clip, individuals in all conditions exhibited vagal increases, but no increases in positive affect.[2] Interestingly, we found that worry led to increased vagal activity in response to the happy clip, whereas relaxation did not. The latter result may reflect a ceiling effect for individuals in the relaxation condition. Moreover, the happy clip elicited positive subjective responding regardless of prior induction type. Overall, these results demonstrate that worry did not inhibit autonomic or subjective emotional responding to the positively valenced clips, which suggests that any effect of worry on avoidance of emotional reactivity may be limited to negative emotions.

The fact that we demonstrated physiological changes following worry in response to the sadness, happiness and calm exposures, even for our GAD group, is somewhat surprising considering that extant research posits chronic worry to be associated with decreased autonomic flexibility (Brosschot et al., 2007; Hoehn-Saric & McLeod, 1988; Hoehn-Saric et al., 1989; Thayer et al., 1996) and the preclusion of any physiological changes in response to emotional stimuli (Borkovec & Hu, 1990; Borkovec et al., 1993; Peasley-Miklus & Vrana, 2000). However, previous studies observing emotional avoidance have focused solely on fearful exposures, which are associated with vagal withdrawal, and have not observed emotional responses associated with vagal increases (e.g., sadness and positive emotions). Considering that worry (vs. relaxation) led to reduced vagal activity levels in our GAD group, perhaps the previously observed inflexibility is the result of a floor effect, in that no further vagal withdrawal is possible, as opposed to overall vagal inflexibility. Our findings parallel this conclusion, such that worry muted vagal changes only in response to the fear exposure but allowed for vagal increases in response to remaining exposures.

Exploring Effects of Worry, Relaxation, and Neutral Inductions

The worry and relaxation manipulations had the intended subjective impact such that all participants reported feeling more worried during the worry induction and more relaxed during the relaxation induction as compared to other inductions. Further, the inductions had the same impact throughout the experiment and did not diminish over time. However, as mentioned above, levels of worry during the neutral induction appeared to be different for those with and without GAD. Whereas those with GAD in the neutral induction reported worry levels falling in between the other two conditions, control participants in the neutral induction reported low worry levels similar to those in the relaxation induction.

Physiologically, worrying impacted those with and without GAD in different ways. Participants with GAD had lower vagal activity during worry than during relaxation, with the neutral induction falling nonsignificantly in between. Thayer, Friedman, and Borkovec (1996) found similar differences during worry, relaxed, and neutral inductions for individuals with GAD. For participants without GAD, we found no overall vagal activity differences between inductions. This result is consistent with Borkovec and colleagues (Borkovec & Hu, 1990; Borkovec et al., 1993), who found no physiological differences during similar inductions using participants with public speaking anxiety.

The fact that we replicated studies by Borkovec and colleagues (1990, 1993) with our non-GAD group could be due to similarities

[1] Of note, prior studies did not examine change in levels of subjective emotion from inductions to emotion exposure, and instead examined absolute levels of subjective emotionality during emotion exposures.

[2] The failure to evidence any change in PA in response to the calm clip is similar to findings of Sloan (2004), who also used the PANAS to measure change in response to this clip.

in methodology. Specifically, Borkovec and colleagues induced brief worry and relaxation periods (30 seconds) in a series of repeated trials (10) with subsequent fear exposures, which is more similar to our methodology than Thayer et al. (1996) who employed a single, longer induction (10.5 minutes) with no intermittent exposures. Our replication of Thayer et al.'s findings in our GAD group despite methodological differences may reflect the habitual response of chronic worriers to even relatively brief worry inductions. Also, as noted above, despite the fact that for control participants there appeared to be no differential physiological effects during inductions, there were differential impacts of inductions (e.g., worry vs. relaxation) on subsequent responding to negative emotion exposures.

Exploring the Emotional Avoidance Model

Examination of participants' absolute levels of subjective and physiological responses during worry and emotional exposure may help elucidate the mechanism behind the emotional effects of worry. Those with GAD exhibited lower vagal activity during worry than during relaxation. Moreover, all participants reported greater negative affect during worry than during relaxed or neutral states. Such findings of heightened negative emotionality during worry are consistent with data showing accelerated heart rate, increased skin conductance, and lower vagal tone during worry when compared to neutral tasks, relaxation, or baseline levels (Andor, Gerlach, & Rist, 2008; Hofmann et al., 2005; Lyonfields, Borkovec, & Thayer, 1995; Peasley-Miklus & Vrana, 2000; Thayer et al., 1996; Vrana, Cuthbert, & Lang, 1989; Vrana & Lang, 1990).

Thus, during the worry induction, participants were already experiencing heightened negative emotionality when compared to other inductions. Following this, we failed to demonstrate further increases in negative affect or vagal withdrawal in response to the fear exposure (which is necessary for habituation). However, despite the avoidance of *increased* negative emotionality via prior worry, negative emotion during the fear exposure was not avoided. In fact, there were no differences between induction conditions in absolute levels of negative emotionality during the fear clip. Instead, the heightened negative emotionality created by worry was *sustained*. Thus, the only benefit of worry insofar as emotional avoidance is concerned was to prevent a negative contrast (or further increase in negative emotionality) from the worry period to the experience of the fearful film clip. It is unlikely that this represents a ceiling effect because, during worry, control participants had lower NA levels than GAD participants but still did not experience an increase in these levels in response to the fear exposure.

Similarly, although there was a preclusion of initial subjective reactivity to the sad clip as a result of worry, the experience of sadness itself was not avoided. Specifically, whereas participants experienced a decrease in sad mood following worry, those in both relaxation and neutral inductions experienced increased sadness upon exposure to the film clip. However, absolute levels of negative affect during the sad clip were not significantly lower following worry as compared to other conditions. Thus, as with fear, prior worry may not function to avoid sadness, but only the experience of a negative contrast or surprise when experiencing sad emotions.

These findings are synchronous with Gray's (1982) neurophysiological theory of anxiety, which states that one trigger for anxious responding is the detection of a mismatch between expected and encountered stimuli in the environment. As such, if individuals use worry to prepare for the worst on an emotional level, then they are reducing the likelihood of an increase in negative emotion if indeed they do experience a negative event (see Borkovec, 1994). Moreover, this is consistent with the finding that persons with GAD report using worry to anticipate and prepare for future negative events (Borkovec & Roemer, 1995). However, these data also suggest that individuals who chronically employ worry to keep themselves braced for the worst are in fact maintaining chronically heightened levels of negative affectivity.

This is similar to what we tell clients who believe that worry helps them to prepare for negative events. When they worry, their body reacts as though the worst thing has already happened. This is the case even though the vast majority of what they have worried about will never happen (Borkovec, Hazlett-Stevens, & Diaz, 1999). Thus, in terms of the impact of worry, rather than preventing negative outcomes, it actually creates an unnecessary negative impact on the body. This idea is in line with theories and data by Brosschot and colleagues (Brosschot, Pieper, & Thayer, 2005; Brosschot, Gerin, & Thayer, 2006; Brosschot et al., 2007; Pieper & Brosschot, 2005) who have suggested that worry may be the primary mechanism by which a person prolongs a stressor's cognitive representation, along with its physiological effects, including its link to cardiovascular risk.

As mentioned previously, a wave of new studies (Mennin et al., 2005; Turk et al., 2005) suggest that persons with GAD report fear of both positive and negative emotions, as well as difficulty understanding and recovering from emotion. Nonetheless, baseline self-report data from our study found no differences between individuals with GAD and nonanxious control participants in perceived threat of positive emotions (i.e., happiness and lust). GAD participants in our study only reported significantly greater perceived threat of sadness, guilt, fear, and strong emotions in general, compared to nonanxious controls. This is consistent with data showing that worry is more strongly associated with fear of anxiety and depression than with fear of positive emotions (Roemer, Salters, Raffa, & Orsillo, 2005). Further, given our findings indicating that worry did not preclude processing of positive emotions, this suggests that chronic worriers are more likely to be motivated toward avoidance of a negative change in their emotional state than toward a positive change. It may be the case that congruent with the James-Lange theory of emotion (James, 1890), repeated attempts to use worry to avoid physiological and subjective changes in response to negative emotions may perpetuate the belief that such change is threatening (e.g., if I avoid something, it must be dangerous). Furthermore, according to Foa and Kozak (1986), this suggests that individuals who are chronically worried may obviate the capacity to learn from their emotional reactions and may thus maintain anxious emotional associations.

Interestingly, we found no resting baseline vagal tone differences between our GAD and nonanxious control groups. However, whereas some prior research has found resting baseline differences between these groups (Lyonfields et al., 1995; Thayer et al., 1996), other research has found no differences (Hoehn-Saric, McLeod, & Zimmerli, 1989; Kollai & Kollai, 1992), suggesting some hetero-

geneity in this domain. Possibly this reflects the fact that worry levels are not controlled during resting baseline, and as such, some individuals may be in a worried state whereas others may not.

Our data also replicate and extend the cause-effect relationship between worry and reactivity to subsequent sadness exposure. Because levels of depression were equalized across induction groups, our findings for differential effects of worry compared to relaxed and neutral states were not due to differential levels of baseline depression. Further, although we found baseline differences between GAD and control participants in level of depression, there were no differences between these groups in the impact of inductions on reactivity to emotion exposure. This provides additional evidence that depression did not play a role in our results. As for additional anxiety disorders, we did not assess for these in either sample, and given general rates of comorbidity it is likely that many individuals in the GAD group met criteria for additional anxiety disorders. However, in most of our findings, worry had the same impact for both GAD and nonanxious control participants who were asked to worry. Given that the impact of worry was the same for those with and without any anxiety disorder, this rules out the possibility that it was comorbid anxiety or the interaction between GAD and comorbid anxiety that led to our results. Instead, our findings provide further support for the causal effects of worry.

Limitations

A number of limitations of this study should be addressed. First, our neutral induction appeared to have a different impact on participants with and without GAD. In particular, participants with GAD reported worrying more during the neutral induction than they did during the relaxation induction, whereas nonanxious participants did not. Consistent with these data, participants with GAD who received the neutral induction demonstrated a similar level of physiological nonreactivity to the fear clip as did participants with GAD who received the worry induction. In contrast, GAD participants in the neutral induction reported worrying less than GAD participants in the worry induction and they demonstrated greater subjective reactivity to the fear clip than those in the worry induction. This suggests that although the neutral induction had the intended subjective impact on GAD participants, it did not have the intended physiological impact. Because participants in the neutral condition were instructed to reflect on events from the previous weekend, it is possible that this led to an idiosyncratic, rather than the intended neutral, state of mind. Thus, in future investigations, a more uniform neutral condition would be recommended. For instance, rather than reflect on idiosyncratic events that may be prone to varying emotional valence, participants could instead read a list of sentences on neutral topics (e.g., Borkovec & Hu, 1990).

In addition, the results from this study are based on an analogue GAD population and did not include a treatment-seeking group. It is therefore possible that effects specific to the GAD group may not generalize to a treatment-seeking population. Another limitation of our sample is that it was not very ethnically diverse. As such, results should be replicated with a more diverse sample before they can be generalized to the population at large. Also, because the current study was not designed to determine whether those with both GAD and comorbid depression had differential responses to emotion inductions compared to those with GAD without comorbid depression, depression levels were balanced across the different inductions. Moreover, only 10.5% of our sample was in the range of participants with clinical depression. As such, effects specific to the GAD group may not generalize to samples with comorbid GAD and depression. Future research could usefully explore the effects of comorbid depression on emotional responding for individuals with GAD.

Perhaps because the rate of clinically severe depression within our sample (10.5%) is much lower than rates of comorbid depression reported elsewhere (Noyes et al., 1992; Roy-Byrne, 1996), this suggests that our sample may not be fully representative of the clinical population. Nonetheless, data shows that GAD is generally more independent of MDD in community samples than in specialty treatment samples, and that the high comorbidity of GAD with major depression in specialty samples is an artifact of selective help-seeking based on comorbidity (Kessler, 2000; Wittchen, Zhao, Kessler, & Eaton, 1994; Wittchen et al., 2002). Moreover, a review of psychotherapy studies for GAD suggest that our 10.5% rate of comorbid depression is within the range of rates found within many randomized controlled trial samples for which the participants were not ruled out for comorbid depression (Borkovec, Newman, Pincus, & Lytle, 2002; Durham et al., 2004; Ladouceur et al., 2000; Wetherell, Gatz, & Craske, 2003; Zinbarg, Lee, & Yoon, 2007), as well as rates of major depression within a treatment seeking university-based clinic (see Brown & Barlow, 1992).

Lastly, in order to create a more complete observation of the psychophysiological effects of worry, it would be useful to monitor both sympathetic and parasympathetic activity. Although the observation of vagal withdrawal infers the possibility of parallel increases in sympathetic arousal, it would be more accurate to determine sympathetic activity by monitoring it directly, such as with skin conductance measures. Additionally, respiration rate was not recorded as part of our psychophysiological battery. Measurement of respiration rate in future research could facilitate a more comprehensive picture of physiological changes in response to worry.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Andor, T., Gerlach, A. L., & Rist, F. (2008). Superior perception of phasic physiological arousal and the detrimental consequences of the conviction to be aroused on worrying and metacognitions in GAD. *Journal of Abnormal Psychology, 117,* 193–205.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

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

Behar, E., Zuellig, A. R., & Borkovec, T. D. (2005). Thought and imaginal activity during worry and trauma recall. *Behavior Therapy, 36,* 157–168.

Borkovec, T. D. (1994). The nature, functions, and origins of worry. In G. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment, and treatment* (pp. 5–33). Sussex, England: Wiley.

Borkovec, T. D., Alcaine, O., & Behar, E. S. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. Heimberg, D. Mennin & C. Turk (Eds.), *The Nature and Treatment of Generalized Anxiety Disorder* (pp. 77–108). New York: Guilford Press Publication Inc.

Borkovec, T. D., Hazlett-Stevens, H., & Diaz, M. L. (1999). The role of positive beliefs about worry in generalized anxiety disorder and its treatment. *Clinical Psychology and Psychotherapy, 6,* 126–138.

Borkovec, T. D., & Hu, S. (1990). The effect of worry on cardiovascular response to phobic imagery. *Behaviour Research and Therapy, 28,* 69–73.

Borkovec, T. D., & Inz, J. (1990). The nature of worry in generalized anxiety disorder: A predominance of thought activity. *Behaviour Research and Therapy, 28,* 153–158.

Borkovec, T. D., Lyonfields, J. D., Wiser, S. L., & Deihl, L. (1993). The role of worrisome thinking in the suppression of cardiovascular response to phobic imagery. *Behaviour Research and Therapy, 31,* 321–324.

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

Borkovec, T. D., & Roemer, L. (1995). Perceived functions of worry among generalized anxiety disorder subjects: Distraction from more emotionally distressing topics? *Journal of Behavior Therapy and Experimental Psychiatry, 26,* 25–30.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60,* 113–124.

Brosschot, J. F., Pieper, S., & Thayer, J. F. (2005). Expanding stress theory: Prolonged activation and perseverative cognition. *Psychoneuroendocrinology, 30,* 1043–1049.

Brosschot, J. F., Van Dijk, E., & Thayer, J. F. (2007). Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. *International Journal of Psychophysiology, 63,* 39–47.

Brown, T. A., Antony, M. M., & Barlow, D. H. (1992). Psychometric properties of the Penn State Worry Questionnaire in a clinical anxiety disorders sample. *Behaviour Research and Therapy, 30,* 33–38.

Brown, T. A., & Barlow, D. H. (1992). Comorbidity among anxiety disorders: Implications for treatment and *DSM–IV*. *Journal of Consulting and Clinical Psychology, 60,* 835–844.

Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the *DSM–IV* anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology, 110,* 585–599.

Butler, E. A., Egloff, B., Wlhelm, F. H., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion, 3,* 48–67.

Durham, R. C., Fisher, P. L., Dow, M. G. T., Sharp, D., Power, K. G., Swan, J. S., . . . Morton, R. V. (2004). Cognitive behaviour therapy for good and poor prognosis generalized anxiety disorder: A clinical effectiveness study. *Clinical Psychology & Psychotherapy, 11,* 145–157 .

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39,* 175–191.

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

Fredrickson, B. L., & Levenson, R. W. (1998). Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cognition & Emotion, 12,* 191–220.

Freeston, M. H., Rheaume, J., Letarte, H., Dugas, M. J., & Ladouceur, R. (1994). Why do people worry? *Personality and Individual Differences, 17,* 791–802.

Gray, J. A. (1982). Precis of The neuropsychology of anxiety (with commentaries). *The Behavioral and Brainsciences, 5,* 469–534.

Gross, J. J., Fredrickson, B. L., & Levenson, R. W. (1994). The psychophysiology of crying. *Psychophysiology, 31,* 460–468.

Gross, J. J., & Levenson, R. W. (1995). Emotion elicitation using films. *Cognition and Emotion, 9,* 87.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106,* 95–103.

Hendriks, M. C. P., Rottenberg, J., & Vingerhoets, Ad, J. J. M. (2007). Can the distress-signal and arousal-reduction views of crying be reconciled? Evidence from the cardiovascular system. *Emotion, 7,* 458–463.

Hoehn-Saric, R., & McLeod, D. R. (1988). The peripheral sympathetic nervous system: Its role in normal and pathologic anxiety. *Psychiatric Clinics of North America, 11,* 375–386.

Hoehn-Saric, R., McLeod, D. R., & Zimmerli, W. D. (1989). Somatic manifestations in women with generalized anxiety disorder. Psychophysiological responses to psychological stress. *Archives of General Psychiatry, 46,* 1113–1119.

Hofmann, S. G., Moscovitch, D. A., Litz, B. T., Kim, H.-J., Davis, L. L., & Pizzagalli, D. A. (2005). The worried mind: Autonomic and prefrontal activation during worrying. *Emotion, 5,* 464–475.

Huang, S.-t. T. (2004). The effects of emotion suppression and reappraisal on dyadic interaction. *Chinese Journal of Psychology, 46,* 267–282.

James, W. W. (1890). *The principles of psychology, Vol I.* New York: Holt.

Kessler, R. C. (2000). The epidemiology of pure and comorbid generalized anxiety disorder: A review and evaluation of recent research. *Acta Psychiatrica Scandinavica, 102,* 7–13.

Kollai, M., & Kollai, B. (1992). Cardiac vagal tone in generalised anxiety disorder. *British Journal of Psychiatry, 161,* 831–835.

Kreibig, S. D., Wilhelm, F. H., Roth, W. T., & Gross, J. J. (2007). Cardiovascular, electrodermal, and respiratory response patterns to fear- and sadness-inducing films. *Psychophysiology, 44,* 787–806.

Ladouceur, R., Dugas, M. J., Freeston, M. H., Léger, E., Gagnon, F., & Thibodeau, N. (2000). Efficacy of a cognitive–behavioral treatment for generalized anxiety disorder: Evaluation in a controlled clinical trial. *Journal of Consulting and Clinical Psychology, 68,* 957–964.

Lyonfields, J. D., Borkovec, T. D., & Thayer, J. F. (1995). Vagal tone in generalized anxiety disorder and the effects of aversive imagery and worrisome thinking. *Behavior Therapy, 26,* 457–466.

Marsh, P., Beauchaine, T. P., & Williams, B. (2008). Dissociation of sad facial expressions and autonomic nervous system responding in boys with disruptive behavior disorders. *Psychophysiology, 45,* 100–110.

McCraty, R., Atkinson, M., Tiller, W. A., Rein, G., & Watkins, A. D. (1995). The effects of emotions on short-term power spectrum analysis of heart rate variability. *The American Journal of Cardiology, 76,* 1089–1093.

McCubbin, R. A., & Sampson, M. J. (2006). The relationship between obsessive-compulsive symptoms and appraisals of emotional states. *Journal of Anxiety Disorders, 20,* 42.

McLaughlin, K. A., Borkovec, T. D., & Sibrava, N. J. (2007). The effects of worry and rumination on affect states and cognitive activity. *Behavior Therapy, 38,* 23–38.

Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2005). Preliminary evidence for an emotion regulation deficit model of generalized anxiety disorder. *Behaviour Research and Therapy, 43,* 1281–1310.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28,* 487–495.

Molina, S., & Borkovec, T. D. (1994). The Penn State Worry Questionnaire: Psychometric properties and associated characteristics. In G. C. L. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment and treatment* (pp. 265–283). Oxford, England: Wiley.

Newman, M. G., Zuellig, A. R., Kachin, K. E., Constantino, M. J., Przeworski, A., Erickson, T., & Cashman-McGrath, L. (2002). Preliminary reliability and validity of the Generalized Anxiety Disorder Ques-

tionnaire-IV: A revised self-report diagnostic measure of generalized anxiety disorder. *Behavior Therapy, 33,* 215–233.

Noyes, R., Woodman, C., Garvey, M. J., Cook, B. L., Suelzer, M., Chancy, J., & Anderson, D. J. (1992). Generalized anxiety disorder vs. panic disorder: Distinguishing characteristics and patterns of comorbidity. *Journal of Nervous & Mental Disease, 180,* 369–379.

Oathes, D. J., Ray, W. J., Yamasaki, A. S., Borkovec, T. D., Castonguay, L. G., Newman, M. G., & Nitschke, J. (2008). Worry, generalized anxiety disorder, and emotion: Evidence from the EEG gamma band. *Biological Psychology, 79,* 165–190.

Peasley Miklus, C., & Vrana, S. R. (2000). Effect of worrisome and relaxing thinking on fearful emotional processing. *Behaviour Research and Therapy, 38,* 129–144.

Pennebaker, J. W., & Beall, S. K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology, 95,* 274–281.

Pennebaker, J. W., & Francis, M. (1996). Cognitive, emotional, and language processes in disclosure. *Cognition and Emotion, 10,* 601.

Pieper, S., & Brosschot, J. F. (2005). Prolonged stress-related cardiovascular activation: Is there any? *Annals of Behavioral Medicine, 30,* 91–103.

Rainville, P., Bechara, A., Naqvi, N., & Damasio, A. R. (2006). Basic emotions are associated with distinct patterns of cardiorespiratory activity. *International Journal of Psychophysiology, 61,* 5–18.

Ray, W. J., Molnar, C., Aikens, D., Yamasaki, A., Newman, M. G., Castonguay, L. G., & Borkovec, T. D. (2009). Startle response in generalized anxiety disorder. *Depression and Anxiety, 26,* 147–154.

Roemer, L., Salters, K., Raffa, S. D., & Orsillo, S. M. (2005). Fear and avoidance of internal experiences in GAD: Preliminary tests of a conceptual model. *Cognitive Therapy and Research, 29,* 71–88.

Rottenberg, J., Kasch, K. L., Gross, J. J., & Gotlib, I. H. (2002). Sadness and amusement reactivity differentially predict concurrent and prospective functioning in major depressive disorder. *Emotion, 2,* 135–146.

Rottenberg, J., Ray, R. D., & Gross, J. J. (2007). Emotion elicitation using films. In J. A. Coan & J. B. Allen (Eds.), *Handbook of emotion elicitation and assessment. Series in affective science* (pp. 9–28). New York: Oxford University Press.

Rottenberg, J., Salomon, K., Gross, J. J., & Gotlib, I. H. (2005). Vagal withdrawal to a sad film predicts subsequent recovery from depression. *Psychophysiology, 42,* 277–281.

Rottenberg, J., Wilhelm, F. H., Gross, J. J., & Gotlib, I. H. (2003). Vagal rebound during resolution of tearful crying among depressed and nondepressed individuals. *Psychophysiology, 40,* 1–6.

Roy-Byrne, P. P. (1996). Generalized anxiety and mixed anxiety-depression: Association with disability and health care utilization. *Journal of Clinical Psychiatry, 57,* 86–91.

Sanderson, W. C., Beck, A. T., & Beck, J. (1990). Syndrome comorbidity in patients with major depression or dysthymia: Prevalence and temporal relationships. *The American Journal of Psychiatry, 147,* 1025–1028.

Sloan, D. M. (2004). Emotion regulation in action: Emotional reactivity in experiential avoidance. *Behaviour Research and Therapy, 42,* 1257.

Solano, L. L., Zoppi, L., Barnaba, L., Fabbrizi, S., Zani, R., Murgia, F., Nicotra, M., et al. (2001). Health consequences of differences in emotional processing and reactivity following the 1997 earthquake in Central Italy. *Psychology, Health & Medicine, 6,* 267.

Startup, H. M., & Erickson, T. M. (2006). The Penn State Worry Questionnaire (PSWQ). In G. C. L. Davey & A. Wells (Eds.), *Worry and its psychological disorders: Theory, assessment and treatment.* (pp. 101–119). Hoboken, NJ: Wiley.

Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *European Heart Journal, 17,* 354–381.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry, 39,* 255–266.

Tsai, J. L., Levenson, R. W., & Carstensen, L. L. (2000). Autonomic, subjective, and expressive responses to emotional films in older and younger Chinese Americans and European Americans. *Psychology and Aging, 15,* 684–693.

Turk, C. L., Heimberg, R. G., Luterek, J. A., Mennin, D. S., & Fresco, D. M. (2005). Emotion dysregulation in generalized anxiety disorder: A comparison with social anxiety disorder. *Cognitive Therapy and Research, 5,* 89–106.

Vrana, S. R., Cuthbert, B. N., & Lang, P. J. (1989). Processing fearful and neutral sentences: Memory and heart rate change. *Cognition and Emotion, 3,* 179–195.

Vrana, S. R., & Lang, P. J. (1990). Fear imagery and the startle-probe reflex. *Journal of Abnormal Psychology, 99,* 189–197.

Wastell, C. A. (2002). Exposure to trauma: The long-term effects of suppressing emotional reactions. *Journal of Nervous and Mental Disease, 190,* 839–845.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 1063–1070.

Wetherell, J. L., Gatz, M., & Craske, M. G. (2003). Treatment of generalized anxiety disorder in older adults. *Journal of Consulting and Clinical Psychology, 71,* 31–40.

Wittchen, H. U., Kessler, R. C., Beesdo, K., Krause, P., Hoefler, M., & Hoyer, J. (2002). Generalized anxiety and depression in primary care: Prevalence, recognition, and management. *Journal of Clinical Psychiatry, 63,* 24–34.

Wittchen, H. U., Zhao, S., Kessler, R. C., & Eaton, W. W. (1994). *DSM–III–R* generalized anxiety disorder in the National Comorbidity Survey. *Archives of General Psychiatry, 51,* 355–364.

Yamasaki, A. S., Behar, E., & Ray, W. J. (2002, November). *Is there a failure to process general emotionality, regardless of valence in generalized anxiety disorder?* Paper presented at the 36th Annual Meeting of the Association for Advancement of Behavior Therapy, Reno, NV.

Zinbarg, R. E., Lee, J. E., & Yoon, K. L. (2007). Dyadic predictors of outcome in a cognitive-behavioral program for patients with generalized anxiety disorder in committed relationships: A "spoonful of sugar" and a dose of non-hostile criticism may help. *Behaviour Research and Therapy, 45,* 699–713.

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