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The contributory role of worry in emotion generation and dysregulation in generalized anxiety disorder

Katie A. McLaughlin, Douglas S. Mennin*, Frank J. Farach

Department of Psychology, Yale University, P.O. Box 208205, New Haven, CT 06520, USA

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Abstract

The role of worry in generalized anxiety disorder (GAD) has been posited to serve as an avoidance of emotional experience, and emotion regulation deficits in GAD have been found in several previous studies. It remains unclear whether those with GAD experience more dysregulated emotions during periods of euthymia and positive affect or whether these deficits occur only during periods of worry. Individuals with GAD (with and without co-occurring dysphoria) and non-anxious controls were randomly assigned to receive a worry, neutral, or relaxation induction. Following the induction, all participants viewed a film clip documented to elicit sadness. Intensity of emotions and emotion regulation were examined following the induction period and film clip. The results revealed that, regardless of co-occurring dysphoria, individuals with GAD in the worry condition experienced more intense depressed affect than GAD participants in the other conditions and controls participants. In contrast, presence of worry appeared to have less impact on indices of emotion dysregulation, which were greater in participants with GAD compared to controls, but largely insensitive to contextual effects of worry or of relaxation. Following film viewing, both GAD participants with and without dysphoria displayed poorer understanding, acceptance, and management of emotions than did controls. However, acceptance and management deficits were most pronounced in individuals with both GAD and co-occurring dysphoria. Implications for the role of emotions in conceptualization and treatment of GAD are discussed.

Keywords: Generalized anxiety disorder (GAD); Emotion; Emotion regulation; Worry

Introduction

Generalized anxiety disorder (GAD) is a common, chronic, and disabling condition. Nearly 5% of individuals in the United States will qualify for a diagnosis of GAD at some point in their lives (Kessler et al., 1994). Despite this comparatively high rate, it has received considerably less empirical and theoretical attention than other anxiety disorders (Dugas, 2000). Much of this lack of attention has come from misconceptions about the nature and treatment of GAD, even amongst mental health professionals. For example, a commonly held misconception about GAD is that the disorder is relatively innocuous and does not cause significant distress or impairment

*Corresponding author. Tel.: +1 203 432 2516; fax: +1 203 432 7172.
E-mail addresses: katie.mclaughlin@yale.edu (K.A. McLaughlin), doug.mennin@yale.edu (D.S. Mennin), francisco.farach@yale.edu (F.J. Farach).

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(Persons, Mennin, & Tucker, 2001). Actually, GAD is associated with high levels of disability and absenteeism from work (Olfson et al., 1997), increased health care utilization (Roy-Byrne & Katon, 1997), as well as marked impairments in role functioning (Wittchen, Zhao, Kessler, & Eaves, 1994). Further, individuals with GAD often experience significant symptoms of the disorder for as long as 20 years, and less than 40% of individuals experience remission after 5 years (Keller, 2002). Thus, its not surprising that GAD is one of the most difficult to treat anxiety disorders (Gould, Safren, Washington, & Otto, 2004).

Recent years have seen considerable advances in conceptualizing and treating GAD (cf. Borkovec, Alcaine, & Behar, 2004). Particularly instrumental in this progress has been the establishment of worry as the pathognomonic characteristic of GAD (American Psychiatric Association, 2000), and the empirical delineation of worry's avoidance function. Avoidance mechanisms in anxiety represent the hallmark of our understanding of anxiety disorders (Mowrer, 1947) and have led to improvements in treatment through the development of exposure-based therapies (e.g., Barlow, Craske, Cerny, & Klosko, 1989; Foa et al., 1999). However, whereas most anxiety disorders are characterized by overt behavioral avoidance, GAD typically does not display consistent behavioral markers and has not responded as well to traditional forms of exposure therapy as the other anxiety disorders (e.g., Gould et al., 2004). Increased focus on the avoidance function of worry and its centrality in GAD has underscored the cognitive aspects of this disorder. For example, in GAD, fear is organized around future threats or catastrophes that may occur as opposed to objects or people in the present environment.

Borkovec et al. (2004) posited that worry in GAD is associated with avoidance of emotional experience. Evidence for this conceptualization of worry in GAD has been found in a number of studies. The verbal-linguistic, as opposed to imagery-based, nature of worry provides initial evidence of worry's avoidant functions. During periods of relaxation, non-anxious participants experience a predominance of imagery-based mentation, whereas individuals with GAD experience equal amounts of thought and imagery. During subsequent worry, both control and GAD participants demonstrate increases in the experience of thought-based mentation (Borkovec & Inz, 1990). Subsequent investigations confirm that worry is experienced phenomenologically as primarily verbal-linguistic thought as opposed to imagery (Freeston, Dugas, & Ladouceur, 1996). The verbal-linguistic, as opposed to imaginal, nature of worry has important implications for understanding the avoidant function of worry.

It has been demonstrated that thinking about anxious material produces less cardiovascular response than does imagining the same anxious material (Vrana, Cuthbert, & Lang, 1986). Furthermore, worrying before repeated presentations of a phobic image precludes autonomic response to those images (Borkovec & Hu, 1990; Borkovec, Lyonfields, Wiser, & Deihl, 1993). The avoidance of anxiety-related imagery strengthens the worry process through negative reinforcement, because the aversive autonomic arousal associated with anxious images is decreased or eliminated during worry. In line with this conceptualization, decreased parasympathetic nervous system activity (vagal tone) has been found to occur in GAD and in the state of worry (Thayer, Friedman, & Borkovec, 1996). The assertion that worry in GAD is associated with avoidance of arousing fear-related imagery is thus well supported (see Borkovec et al., 2004 for a comprehensive review of evidence for this theory).

A key contributor to the avoidant function of worry may be the perceived aversive nature of the evaded emotional experience. When asked about reasons for worrying, individuals with GAD were distinguished from non-anxious controls by the greater likelihood to endorse that they engaged in worry to avoid thinking about more emotional topics (Borkovec & Roemer, 1995). An important question then is *why* individuals with GAD want to avoid emotional experience. One possibility may result from the nature of how emotions are generated and regulated (Gross, 2001). Mennin and colleagues (Mennin, Heimberg, Turk, & Fresco, 2002, 2005) argue that individuals with GAD have difficulties in four areas of emotion. Specifically, individuals with GAD reported experiencing emotions with *heightened intensity* compared to persons without GAD. Second, individuals with GAD experience marked difficulties identifying, describing, and clarifying their emotional experiences (i.e., *poor understanding*). Third, they are prone to greater *negative reactivity* to emotions by holding catastrophic beliefs about the consequences of both negative and positive emotions. Fourth, individuals with GAD struggle to manage or soothe themselves when they experience negative emotions (i.e., *maladaptive management*). Within this model, maladaptive emotion management strategies in GAD can be classified both as difficulties modulating or tempering emotional experiences (often due to their increased

emotional intensity) or as attempts to control or suppress emotional experience (often through the avoidant process of worry).

Mennin and colleagues have provided preliminary evidence for this emotion dysregulation model of GAD in a series of studies. Participants with self-reported GAD (Study 1) and treatment-seeking patients with clinician-assessed GAD (Study 2) have reported greater deficits in these four areas as compared to control participants (Mennin et al., 2005). Further, GAD participants, but not control participants, who underwent a negative mood induction showed increases in negative emotions as well as difficulties in their ability to regulate the negative mood, particularly with respect to their understanding of and willingness to accept the emotional experience (Study 3; Mennin et al., 2005). Also, individuals with self-reported GAD have reported greater emotional intensity and greater negative reactivity to depressive moods, than individuals with self-reported social anxiety disorder individuals and controls (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). Finally, a composite emotion regulation score derived from measures assessing each of the four branches of the emotion regulation model successfully predicted GAD after controlling for worry, anxiety, and depression (Mennin et al., 2005) and was more associated with a diagnosis of GAD than social anxiety disorder (Turk et al., 2005).

As such, intensity of emotional reactions to negative stimuli and inability to manage these reactions appears to characterize GAD. However, the contexts in which individuals with GAD may react and manage their emotions differently than those without the disorder remain unclear. Given the centrality of worry in the experience of GAD individuals, it is important to determine what role worry might play in emotion dysregulation. In the emotion dysregulation model of GAD, worry is viewed primarily as a maladaptive emotion management strategy; specifically, worry is conceptualized as a strategy used in attempts to control or suppress emotional experience (Mennin et al., 2002, 2005). Because worry is considered to be a response to dysregulated emotions, intense or under-regulated emotions are experienced prior to the occurrence of worry within this model. For example, an individual with GAD may experience a loss and feel sadness. Because the individual holds negative beliefs about the consequences of feeling sad, they engage in worry as an attempt to avoid feeling sad. However, it may be that worry also plays a role in the generation of dysregulated emotional experience in GAD.

Individuals with GAD spend large amounts of time engaging in worry. Higher levels of worry are found in GAD than in any of the other anxiety disorders (Brown, Antony, & Barlow, 1992), and individuals with the disorder report that their worries are more pervasive (Roemer, Molina, & Borkovec, 1997) and less controllable (Borkovec, Shadick, & Hopkins, 1991; Craske, Rapee, Jackel, & Barlow, 1989) than individuals without the disorder. One study found that those with GAD reported worrying and feeling anxious 50% of the time (Sanderson & Barlow, 1990). Worry has been demonstrated to create both anxious and depressed affect (Andrews & Borkovec, 1988; Borkovec, Robinson, Pruzinsky, & DePree, 1983) and is associated with information processing biases that favor the processing of threatening information (e.g., MacLeod, Mathews, & Tata, 1986). Given that worry in GAD is pervasive, uncontrollable, and aversive, it seems likely that individuals with the disorder may experience greater difficulty managing their emotional reactions to events in the environment during or immediately following periods of worry. As such, the role of worry in emotion dysregulation in GAD may be bidirectional: worry may serve as a response to dysregulated emotional experience as an attempt to control or avoid such experience, but worry may also lead to increased intensity of emotional reactions to environmental events and a decreased ability to effectively manage those emotions. This latter conceptualization is consistent with the notion of experiential avoidance wherein control strategies such as worry beget more aversive emotional experiences (see Hayes, Strosahl, & Wilson, 2004).

The primary goal of the present study was to determine the role of worry in inducing negative emotional reactions and dysregulation in comparison to other contextual states such as relaxation or euthymia. The current investigation examined the impact of being in a worried, relaxed, or neutral state on emotion generation and regulation differences between individuals with GAD and non-anxious controls following a negative emotion induction. A previous experimental investigation of the emotion dysregulation model did not specifically examine the role of contexts such as worry in emotion regulation deficits in GAD (Mennin et al., 2005; Study 3). Also, in this prior study, participants with GAD did not undergo diagnostic interviews to confirm the diagnosis; rather, a self-report measure of GAD was used to identify an analog sample. In addition, music was used to induce mood rather than more evocative and well-studied film clips (e.g., Gross &

Levenson, 1995). Finally, the pre-induction state of participants was not a factor examined in the analyses. Differences in baseline worry, or relaxation, may have been confounded with participant group. The current investigation seeks to build upon the first experimental investigation of the emotion dysregulation model of GAD by addressing these limitations in study design. Participants were assigned to diagnostic groups using a structured diagnostic interview, a standardized film clip was used to induce mood, and the pre-induction state of participants was controlled using thought induction procedures (to ensure that GAD and control participants were equivalent prior to the emotion induction).

The target emotion elicited by our emotion induction was sadness. We chose to elicit sadness for a number of reasons. First, we wanted to examine the experience and regulation of a negative emotion, given that deficits in negative affect regulation are of theoretical and practical significance when examining emotional disorders. Secondly, sadness is a negative emotion that is an experience commonly experienced by individuals with GAD but is not as core to the diagnosis as anxiety, which supports the notion of a generalized emotion deficit in GAD, rather than one specific to anxiety (Mennin et al., 2005). Finally, sadness is a negative emotion that is experienced ubiquitously across cultures and individuals (e.g., Ekman, Sorenson, & Friesen, 1969), making it an emotion that virtually all individuals have experienced and had to manage in their everyday lives. Thus we focused on sad emotional reactions but also assessed anxiety reactions given their centrality to the disorder.

Our primary goals in this study are to examine experimentally differences in emotional intensity and emotion regulation between individuals with GAD and those without the disorder and to examine the role that worry plays in contributing to differences in emotional intensity and regulation. However, it is important to note that it is difficult in any study to examine the independent effects of GAD, given the high rates of comorbidity between GAD and depression and other anxiety disorders (Brown, Barlow, & Liebowitz, 1994; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Grant et al., 2005; Kessler, Dupont, Berglund, & Wittchen, 1999). Because GAD is highly comorbid with other emotional disorders, any differences found between GAD and control participants could be due, in part, to the effects of co-occurring disorders. This concern is particularly important to address with regard to depression, as depression has the highest rates of comorbidity with GAD with lifetime comorbidity rates ranging from 50–60% (Brown et al., 1994, 2001; Kessler et al., 1999) and given that recent conceptualizations of GAD have called into question the delineation of GAD from depression (Brown, Chorpita, & Barlow, 1998; Watson, 2005). The emotion regulation deficits of interest in this study have been found to occur in GAD but not in social phobia (Mennin, Holaway, Fresco, Moore, & Heimberg, in press), the anxiety disorder that is most frequently comorbid with GAD (Brown et al., 1994). However, the nature of the emotion regulation deficits of interest has not been thoroughly examined in depression. Given that we were particularly interested in the experience of sadness, we have included comparisons of subgroups of participants with GAD who did and did not have dysphoria. However, we did not anticipate that co-occurring dysphoric symptoms would impact emotional intensity or emotion regulation for individuals with GAD. Because previous research has documented that the emotion regulation deficits reviewed above are distinct (e.g., heightened intensity) or particularly elevated (e.g., maladaptive management) in GAD (Mennin et al., in press), we expected to find differences between GAD participants and controls regardless of the presence of dysphoria. Nonetheless, the role of co-occurring depressive symptoms was examined in all of our analyses to determine whether differences in dysphoria between the GAD and control group were responsible for any observed group differences in emotional intensity and regulation.

We hypothesized that GAD participants (regardless of co-occurring dysphoria) in the worry condition would: (1) report experiencing more intensely negative emotions (both sadness and anxiety) across time (i.e., from baseline to the worry period to post-film clip) than non-anxious controls and GAD participants in neutral and relaxation conditions; (2) would report experiencing less awareness, understanding, and acceptance of their emotions than controls and GAD participants in neutral and relaxation conditions; and (3) would report more difficulty in effectively managing their emotions than controls and GAD participants in neutral and relaxation conditions. Because we predicted that participants with GAD would only differ from control participants in their emotional reactions, understanding of emotions, and emotion management in their response to the sad mood clip occurring after the worry induction, we expected that no differences would emerge between GAD and control participants after the neutral and relaxation inductions.

Method

Participants

Students in an introductory psychology course completed the Generalized Anxiety Disorder Questionnaire-IV (GADQ-IV; Newman et al., 2002) as part of a larger packet of questionnaires completed at the beginning of the semester. Participants meeting criteria for GAD based on the cut-off described by Newman and colleagues (2002) and participants scoring in the non-anxious range (see below for criteria) were invited to participate in a structured diagnostic interview to confirm eligibility. Other college community participants were also recruited using flyers. All participants completed the GAD and social phobia (included for another study) sections of the Anxiety Disorders Interview Schedule-Revised (ADIS-IV; Di Nardo & Barlow, 1994). Participants meeting criteria for GAD were invited to participate in the study. Participants who did not meet criteria for GAD or social phobia and who scored below a 3 on clinical severity ratings of distress and impairment due to worry and social anxiety were invited to participate in the study as control participants.

Forty-nine participants met criteria for GAD (13 male, 36 female), and 44 participants were classified as controls (18 male, 26 female). Of the 49 participants in the GAD group, 17 (34.7%) also met criteria for social phobia. The mean age of the sample was 20.58 years (SD = 4.19). Participants' self-reported race/ethnicity was as follows: 59.1% Caucasian ($N = 55$), 12.9% African-American ($N = 12$), 8.6% Hispanic/Latino ($N = 8$), 12.9% Asian/Pacific Islander ($N = 12$), and 1.1% Middle Eastern descent ($N = 1$). 1.1% of the sample reported multiple race/ethnicity categories ($N = 1$), and 4.3% ($N = 4$) declined to report this information. Ethnic background was equally distributed in study groups (GAD, control) and across condition (worry, relaxation, neutral).

Stimulus film

We used a film clip from a set of standardized film stimuli used to induce emotion in a laboratory setting (Gross & Levenson, 1995). The approximately 3-min film clip shows a scene from *The Champ* in which "The Champ" dies with his friends and his distraught young son watching. This film clip has been documented to elicit self-reported sadness with little report of other emotions (Gross & Levenson, 1995).

Materials

Pre-session questionnaires

Participants completed a battery of self-report questionnaires prior to the experimental session including the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Garbin, 1996), the Affect Intensity Measure (AIM; Larson & Deiner, 1987), the Acceptance and Action Questionnaire (AAQ; Hayes et al., 2004), and the Difficulties with Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The BDI is a 21-item measure designed to assess the severity of depressive symptoms, including affective, cognitive, behavioral, somatic, and motivational symptoms. Items are rated on a 4-point scale and assess a 2-week time period. The BDI-II has excellent psychometric properties including internal consistency and convergent and divergent validity that have been demonstrated in numerous studies (e.g., Dozois, Dobson, & Ahnberg, 1998).

The AIM is a 40-item measure that assesses the intensity with which respondents typically experience positive and negative emotions. The 10-item subscale, AIM-N, designed to assess the intensity of negative emotional experiences was used in this study. Participants indicate how often they experience specific emotional reactions to situations using a 6-point scale, where 1 is *never*, 2 is *almost never*, 3 is *occasionally*, 4 is *usually*, 5 is *almost always*, and 6 is *always*. The AIM-N has adequate reliability and validity (Goldsmith & Walters, 1989; Larson & Deiner, 1987).

The AAQ is a 9-item measure designed to assess experiential avoidance. Participants indicate how much each statement applies to them on a 7-point scale ranging from 1, *never true*, to 7, *always true*. The AAQ has demonstrated adequate psychometric properties across both clinical and non-clinical samples (see Hayes et al., 2004).

The DERS is a 39-item measure that assesses dispositional tendencies for emotion dysregulation along a number of dimensions, including awareness and understanding of emotions, clarity of emotional experience, acceptance of emotions, ability to engage in goal-directed behavior and refrain from impulsive behavior when experiencing negative emotions, and accessibility of effective emotion regulation strategies. Four of the six subscales (related to study hypotheses) were administered (a total of 25 items): acceptance of emotion, awareness of emotion, ability to access effective emotion regulation strategies, and understanding of emotion. Participants indicate how much each item applies to them on a 5-point scale ranging from 1, *not at all*, to 5, *completely*. The DERS has been shown to have high internal consistency ($\alpha = 0.93$), and has demonstrated good construct and predictive validity as a whole as well as within the individual subscales (Gratz & Roemer, 2004).

Dependent measures

We assessed the quality and intensity of participants' emotional reactions to the stimulus film using the depression and anxiety scales from the Multiple Adjective Affect Checklist-Revised (MAACL-R; Zuckerman & Lubin, 1985). These subscales demonstrate high internal consistency ($\alpha = 0.86$–0.88) and have well-documented convergent, discriminant, and predictive validity (e.g., Lubin, Van Whitlock, Reddy, & Petren, 2001; Lubin & Zuckerman, 1999).

To assess emotion regulation strategies, we modified the awareness, clarity, acceptance, and regulation scales to measure state levels of emotion dysregulation as opposed to dispositional emotion regulation. Participants indicated how much each item applied to them *right now*, rather than in general, on a 5-point scale where 1 was *not at all*, 2 was *somewhat*, 3 was *moderately*, 4 was *very much*, and 5 was *completely*. Our state version of the DERS (DERS-S) demonstrated good internal consistency ($\alpha = 0.81$).

Procedure

Participants were randomly assigned to conditions (worry, neutral, or relaxation). Participants were first asked to create a 0–100 worry visual-analog scale (WVAS) using anchors from their own lives. Within this scale, participants anchored 0 with a topic they were not worried about at all, 25 with a topic they were mildly worried about, 50 with a topic they were moderately worried about, 75 with a topic they were very worried about, and 100 with a topic they were most worried about in their lives at the time of the experiment. Following completion of anchoring the scale, participants completed the battery of self-report questionnaires.

Participants then engaged in a 5-min induction procedure (McLaughlin, Borkovec, & Sibrava, in press). Prior to the induction, all participants recorded their current level of worry using the worry visual analog scale. Participants in the worry condition were asked to write down the three topics about which they worried the most. They then received the following instructions:

> During this period, we would like you to create a worrisome state. Please refer to your list of worrisome topics. When the experimenter asks you to begin, please close your eyes and worry about your most worrisome topic, in the way you usually worry about it but as intensely as you can, until the experimenter asks you to stop and to open your eyes. If you normally worry about only one topic at a time, please try to do the same during this period. However, if your thoughts change to another worry topic during this period feel free to allow these thoughts to continue. It is all right to change topics during this period if the changes occur naturally during the worry process.

Participants in the neutral condition were asked to write down three things that they did last weekend. They then received the following instructions:

> During this period, we would like you to take a few minutes to think about what you did this past weekend. When the experimenter asks you to begin, please close your eyes and think about what you did last weekend, until the experimenter asks you to stop and to open your eyes. It may help to start by thinking about the three things that you listed above. Please close your eyes and begin thinking.

Participants in the relaxation condition were asked to write down three of the most pleasant events that had happened in their life. They then received the following instructions:

> During this period, we would like you to create a relaxed state. Please refer to you list of pleasant events. When the experimenter asks you to begin, please close your eyes and merely allow your body and mind to relax. A good way to deepen the relaxation is to breathe from your diaphragm, rather than from your chest, and to slow the rate of your breathing down to a comfortable pace. While you are relaxing, imagine some of the most pleasant things that have happened to you in your life. It may help to start by thinking about the pleasant events that you listed above. Please try to become as relaxed as you possibly can during this period.

Participants were stopped 3 times during the 5-min induction and immediately after the induction period to record their current level of worry. The mean of these 3 worry assessments was used as a manipulation check to ensure the induction was successful at creating a worried state.

Following the induction, participants completed the dependent measures. They then engaged in a 1-min re-induction period (either worry, neutral or relaxation, consistent with their first induction). Immediately following this re-induction period, participants recorded their current level of worry and then viewed the film clip. Participants recorded their level of worry after the film clip and completed the dependent measures again.

Analysis strategy

The purpose of the current study was to evaluate the effect of being in a worried, neutral, or relaxed state on emotion intensity and regulation in GAD participants vs. controls. However, the high rates of comorbidity between GAD and depression (Brown et al., 2001; Kessler et al., 1999) lead to difficulty in concluding that group differences are truly due to the effect of GAD and not depression. As such, we attempted to disentangle the effects of GAD and of depression on emotion intensity and regulation by splitting our GAD sample into high- and low-dysphoria groups based on baseline score on the BDI-II. Participants scoring below 12 on the BDI-II ($N = 25$) were classified as low-dysphoria and participants scoring 12 and higher on the BDI-II ($N = 23$) were classified as high-dysphoria.[1] One individual with GAD scored a 50 on the BDI-II and was, thus, not included in the study. Further, 2 control participants scored above 12 on the BDI-II and were also not included in the study. In all analyses, a 3-level grouping variable was used (control, GAD with low dysphoria, GAD with high dysphoria). Omnibus tests revealing group differences between GAD and control participants were followed by a series of contrasts to determine whether group differences were being driven by differences in depression between the GAD and control groups. The first contrast compared GAD participants high in dysphoria to GAD participants low in dysphoria. If this contrast revealed significant differences between the two groups, each group was then compared to controls separately. If this contrast did not reveal significant differences between GAD participants high and low in dysphoria, the two GAD groups were collapsed and compared to controls.

To examine hypotheses related to emotional intensity, we examined group and condition differences in trends of anxiety and depression across the study (from baseline to post-induction period to post-film) using repeated-measures ANOVAs. Significant time by group by condition interactions were followed-up by examining group by time interactions within each study condition. This allowed us to compare GAD participants (high and low in dysphoria) to controls in the observed trends of anxiety and depression scores within each study condition (worry, relaxation, and neutral). To examine hypotheses related to emotion regulation, the effects of group and condition on emotional awareness, non-acceptance of emotion, clarity of emotional experience, and emotion regulation strategies were examined after the induction period and after the film using univariate ANOVAs. Significant group effects were followed up using contrasts, as outlined previously. Significant interactions were followed up using simple effects analyses examining differences in GAD participants (high and low in dysphoria) vs. control participants within each study condition.

[1] We used a cutoff of 12 rather than 14 as suggested by Beck et al. (1996) based on subsequent psychometric evaluations that found a lower cutoff score yielded higher sensitivity and specificity (see Dozois et al., 1998 Dozois, Dobson, & Ahnberg, 1998).

We chose a post-only strategy to assess emotion dysregulation for a number of reasons. First, we wanted to assess affect and regulation in ways that were consistent with previous literature. While it is important to assess mood in a way that accounts for baseline differences between participants, emotion regulation is not typically assessed in the same way (see Mayer & Stevens, 1994; Mennin et al, 2005, Study 3; Salovey, Mayer, Goldman, Turvey & Palfai, 1995). Rather, state emotion regulation is typically measured at one point in time. Convention is this way because while mood is not necessarily linked to a specific event or trigger, emotion regulation is conceptualized as a response for managing specific emotions. In this case, the specific emotions are those elicited by the induction and film. We would not expect baseline levels of emotion regulation to necessarily relate to strategies following either the induction or the film. Thus, we opted to utilize the conventional approach for post-induction and post-film assessments of emotion regulation rather than examining state changes.

Results

Pre-session questionnaires

To ensure that no condition differences existed prior to the thought induction, univariate ANOVAs were conducted on baseline BDI-II, AAQ, AIM-N, and dispositional DERS as well as on baseline anxiety and depression scales of the MAACL. No significant differences emerged between study conditions (all p values >0.34), indicating that random assignment was successful at creating equivalent groups at baseline.

As expected, significant differences among the 3 study groups (GAD with high dysphoria, GAD with low dysphoria, control) existed prior to the induction period. Group differences were found at baseline on the BDI-II, $F(1, 87) = 87.24$, $p<0.001$, $\eta^2 = 0.67$, AAQ, $F(1, 87) = 33.97$, $p<0.001$, $\eta^2 = 0.44$, and on the AIM-N, $F(1, 87) = 23.58$, $p<0.001$, $\eta^2 = 0.35$. As per our group definition, GAD participants high in dysphoria had higher BDI-II scores than GAD participants low in dysphoria, $L = 11.12$, $p<0.001$, CI: (8.591, 13.653). GAD participants high in dysphoria had higher BDI-II scores than control participants, $L = 15.022$, $p<0.001$, CI: (12.749, 17.294), and GAD participants low in dysphoria also reported more depressive symptoms on the BDI-II than controls, $L = 3.90$, $p<0.001$, CI: (1.687, 6.113). GAD participants high in dysphoria reported less acceptance of their emotions on the AAQ than GAD participants low in dysphoria, $L = -0.445$, $p<0.024$, CI: (-0.831, -0.059). GAD participants high in dysphoria, $L = -1.357$, $p<0.001$, CI: (-1.704, -1.010), and low in dysphoria, $L = -0.912$, $p<0.001$, CI: (-1.250, -0.574), each reported less emotional acceptance than control participants. Finally, GAD participants high in dysphoria did not report greater negative emotional intensity on the AIM-N than GAD participants low in dysphoria, $L = 0.189$, $p<0.307$, CI: (-0.177, 0.554). The GAD group was collapsed and compared to the control group, revealing that GAD participants reported greater negative emotional intensity than controls, $L = 0.917$, $p<0.001$, CI: (0.650, 1.184).

Group differences were also found at baseline on the MAACL-R anxiety, $F(1, 87) = 18.30$, $p<0.001$, $\eta^2 = 0.30$, and depression scales, $F(1, 87) = 16.49$, $p<0.001$, $\eta^2 = 0.28$. GAD participants high in dysphoria reported higher state anxiety on the MAACL-R than GAD participants low in dysphoria, $L = 3.003$, $p<0.032$, CI: (0.262, 5.745). GAD participants high in dysphoria, $L = 7.258$, $p<0.001$, CI: (4.796, 9.719), and low in dysphoria, $L = 4.254$, $p<0.001$, CI: (1.857, 6.651), each reported higher state anxiety at baseline than control participants. GAD participants high in dysphoria also reported higher state depression on the MAACL-R than GAD participants low in dysphoria, $L = 4.489$, $p<0.003$, CI: (1.540, 7.437). GAD participants high in dysphoria, $L = 7.633$, $p<0.001$, CI: (4.986, 10.279), and low in dysphoria, $L = 3.144$, $p<0.001$, CI: (0.566, 5.721), each reported higher state depression at baseline than control participants.

Finally, group differences in trait emotion regulation were found at baseline on 3 of the 4 subscales of the DERS. Baseline group differences were found for acceptance of emotion, $F(1, 90) = 6.94$ $p<0.001$, $\eta^2 = 0.14$, clarity of emotional experience, $F(1, 87) = 4.88$, $p<0.010$, $\eta^2 = 0.10$, and access to effective emotion regulation strategies, $F(1, 87) = 17.87$, $p<0.001$, $\eta^2 = 0.29$, but not in awareness of emotional experience, $F(1, 87) = 0.59$, $p<.442$, $\eta^2 = 0.02$. GAD participants high in dysphoria did not differ from GAD participants low in dysphoria on acceptance of emotion, $L = 0.294$, $p<0.263$, CI: (-0.225, 0.813), or clarity of emotional experience, $L = 0.036$, $p<0.858$, CI: (-0.438, 0.365). GAD participants as a group reported greater emotion

Table 1
Means and standard deviations on baseline measures for GAD and control participants

Measure	GAD		Control
	High dysphoria	Low dysphoria	
BDI-II	18.52(7.15)	7.40(2.69)	3.50(3.13)
AIM-N	4.02(0.57)	3.83(0.57)	3.01(0.70)
AAQ	3.66(0.71)	4.10(0.77)	5.01(0.59)
DERS			
Awareness	2.36(0.94)	2.39(0.61)	2.17(0.75)
Clarity	2.35(0.86)	2.38(0.62)	1.91(0.64)
Acceptance	2.64(1.00)	2.35(0.94)	1.81(0.82)
Strategies	2.55(0.69)	2.17(0.72)	1.57(0.59)
MAACL-R			
Anxiety	20.04(6.93)	17.04(4.94)	12.79(2.88)
Depression	21.61(8.59)	17.12(4.07)	13.98(2.59)

Note. GAD = participants with generalized anxiety disorder, Control = control participants, High dysphoria = participants scoring 12 or greater on the BDI-II; Low dysphoria = participants scoring below 12 on the BDI-II. Higher scores on the DERS reflect greater emotion dysregulation.

dysregulation than controls on both acceptance of emotion, $L = 0.684$, $p < 0.001$, CI: (0.304, 1.064), and emotional clarity, $L = 0.460$, $p < 0.003$, CI: (0.166, 0.754). GAD participants high in dysphoria reported decreased ability to access effective emotion regulation strategies compared to GAD participants low in dysphoria, $L = 0.384$, $p < 0.045$, CI: (0.008, 0.760). GAD participants both high in dysphoria, $L = 0.977$, $p < 0.045$, CI: (0.640, 1.315), and low in dysphoria, $L = 0.594$, $p < 0.001$, CI: (0.265, 0.922), reported greater emotion dysregulation than control participants. See Table 1 for all means and standard deviations of baseline measures for GAD participants (high and low in dysphoria) and control participants.

Manipulation check

Participants reported their current level of worry numerous times throughout the experiment using the WVAS. To ensure that participants in the worry condition experienced an increase in worry during the induction period, and to ensure that participants in the neutral and relaxation conditions did not experience an increase in worry during the induction period, changes in WVAS scores from baseline to the induction period to the film were examined. A repeated-measures ANOVA involving time as a within-subjects factor and group and condition as between-subjects factors was conducted on WVAS scores. This analysis revealed a main effect of time, $F(2, 156) = 9.50$, $p < 0.001$, $\eta^2 = 0.11$, which was qualified by a time by condition interaction, $F(4, 156) = 29.52$, $p < 0.001$, $\eta^2 = 0.43$. Within the interaction, a significant quadratic trend emerged, $F(1, 84) = 15.55$, $p < 0.001$, $\eta^2 = 0.27$. Participants in the worry condition reported an increase in WVAS scores from baseline ($M = 23.94$) to the induction period ($M = 45.87$) which then decreased following the film ($M = 41.21$). In contrast, participants in the neutral condition reported no change in WVAS scores from baseline ($M = 26.46$) to the induction period ($M = 26.93$) followed by an increase following the film ($M = 33.29$), and participants in the relaxation condition reported a decrease in WVAS scores from baseline ($M = 21.81$) to the induction period ($M = 15.41$) followed by an increase following the film ($M = 24.88$). Importantly, the interaction between time and group status was not significant, $F(1, 156) = 1.13$, $p = 0.357$, $\eta^2 = 0.03$, indicating that the inductions did not have different effects in GAD and control participants or in the two GAD groups. Additionally, the time by condition by group status was not significant, $F(8, 156) = 1.02$, $p = 0.424$, $\eta^2 = 0.03$, demonstrating that the worry induction was not differentially effective at creating a worried state in GAD participants vs. controls.

Emotional intensity

Intensity of emotional responses to the induction and film clip, based on the MAACL-R anxiety and depression subscales, were examined using repeated-measures ANOVAs with time (baseline to induction to film clip) as a within-subjects factor, and with condition and group as between-subjects factors. This analysis revealed a main effect of time, $F(2, 160) = 7.61$, $p < 0.001$, and a time by condition interaction for MAACL depression scores, $F(2, 160) = 3.83$, $p < 0.005$, which were qualified by a 3-way time by condition by group interaction, $F(2, 160) = 2.08$, $p < 0.040$, $\eta^2 = 0.094$. To determine which condition(s) were driving this interaction, 3 repeated-measures ANOVAs were conducted, one for each condition, with time as a within-subjects factor and group as a between-subjects factor.

These analyses revealed a significant time by group interaction in the worry condition, $F(2, 56) = 2.74$, $p < 0.037$, $\eta^2 = 0.16$, but not in the neutral condition, $F(2, 48) = 0.87$, $p < 0.489$, $\eta^2 = 0.07$, or relaxation condition, $F(2, 56) = 1.81$, $p < .141$, $\eta^2 = 0.11$. In the worry condition, a significant linear trend was associated with the time by group interaction, $F(1, 28) = 3.73$, $p < 0.037$, $\eta^2 = 0.21$. Contrasts revealed that GAD participants high in dysphoria did not differ from GAD participants low in dysphoria on depression changes from baseline to the induction to the film, $L = 0.329$, $p < 0.899$, CI: (−5.597, 4.939). Thus, the GAD groups were collapsed and compared to controls. Within those assigned to the worry condition, GAD participants experienced a systematic linear increase in MAACL depression scores from baseline ($M = 19.12$, SD = 5.77) to the induction ($M = 21.88$, SD = 7.61) to the film ($M = 24.65$, SD = 8.15), whereas control participants did not experience this increase from baseline ($M = 14.29$, SD = 2.87) to the induction ($M = 15.21$, SD = 2.83) to the film ($M = 14.93$, SD = 3.89), $L = 7.063$, $p < 0.001$, CI: (3.147, 10.979). See Table 2 for means and standard deviations of MAACL depression scores at baseline, following the induction period, and following the film clip across conditions and groups.

Using MAACL anxiety as the dependent measure, a 3-way repeated-measures ANOVA revealed a time by condition interaction, $F(2, 160) = 7.87$, $p < 0.001$, $\eta^2 = 0.16$, and a 3-way time by condition by group interaction, $F(2, 160) = 2.30$, $p < 0.023$, $\eta^2 = 0.10$. To determine which condition(s) were driving the time by condition by group interaction predicting MAACL anxiety scores, 3 repeated-measures ANOVAs were conducted, 1 for each condition, with time as a within-subjects factor and group as a between-subjects factor. These analyses revealed a significant time by group interaction in the relaxation condition, $F(2, 58) = 2.84$, $p < 0.033$, $\eta^2 = 0.17$, but not in the worry condition, $F(2, 56) = 1.45$, $p < 0.23$, $\eta^2 = 0.09$, or neutral condition, $F(2, 48) = 1.85$, $p < 0.13$, $\eta^2 = 0.13$.

In the relaxation condition, GAD participants high in dysphoria did not differ from GAD participants low in dysphoria in changes in anxiety from baseline to the induction to the film, $L = 2.444$, $p < 0.135$, CI: (−.808, 5.697). The GAD groups were then collapsed and compared to controls. Within those assigned to the relaxation condition, GAD participants experienced a decrease in MAACL anxiety scores from baseline ($M = 18.77$; SD = 4.85) to the induction ($M = 15.06$, SD = 4.72) that was maintained after viewing the film ($M = 16.77$, SD = 2.55) such that their anxiety level following the film was lower than their anxiety level at baseline. In contrast, control participants experienced a less pronounced decrease in MAACL anxiety scores from baseline ($M = 12.27$, SD = 2.71) to the induction ($M = 11.73$, SD = 2.28) and an increase in anxiety

Table 2
Means and standard deviations of depressed affect at baseline and following induction period and film viewing, Study 2

Condition	GAD						Control		
	High dysphoria			Low dysphoria			Baseline	Induction	Film
	Baseline	Induction	Film	Baseline	Induction	Film			
Worry	20.00(6.08)	21.44(6.42)	24.67(7.57)	18.13(5.62)	22.38(9.21)	24.63(9.29)	14.29(2.97)	15.21(2.83)	14.93(3.89)
Neutral	21.75(9.69)	21.63(9.15)	20.88(10.33)	15.33(3.39)	14.00(2.28)	17.50(2.81)	14.62(2.90)	13.77(2.68)	15.38(2.79)
Relaxation	23.83(11.09)	22.50(17.71)	22.17(10.40)	17.36(3.01)	13.64(2.29)	17.91(4.41)	13.07(1.77)	13.00(2.66)	15.14(2.63)

Note. GAD = participants with generalized anxiety disorder, Control = control participants, High dysphoria = participants scoring 12 or greater on the BDI-II; Low dysphoria = participants scoring below 12 on the BDI-II.

Table 3
Means and standard deviations of anxious affect at baseline and following induction period and film viewing, Study 2

Condition	GAD						Control		
	High dysphoria			Low dysphoria			Baseline	Induction	Film
	Baseline	Induction	Film	Baseline	Induction	Film			
Worry	18.33(7.21)	22.00(9.01)	20.33(9.89)	16.50(4.96)	22.88(8.39)	20.25(8.03)	13.21(3.70)	15.93(3.97)	12.79(2.75)
Neutral	21.50(8.75)	24.13(12.11)	19.00(10.28)	16.50(4.89)	15.17(2.14)	17.50(2.59)	13.31(2.69)	12.00(1.78)	12.54(2.50)
Relaxation	20.67(3.44)	17.50(6.80)	17.17(4.49)	17.73(5.33)	13.73(2.65)	16.55(5.20)	11.79(2.04)	11.43(2.03)	12.93(2.27)

Note. GAD = participants with generalized anxiety disorder, Control = control participants, High dysphoria = participants scoring 12 or greater on the BDI-II; Low dysphoria = participants scoring below 12 on the BDI-II.

after viewing the film ($M = 13.27$, SD = 4.83) such that their anxiety level following the film was higher than their anxiety level at baseline, $L = 5.175$, $p < 0.001$, CI: (2.813, 7.537). See Table 3 for means and standard deviations of MAACL anxiety scores at baseline, following the induction period, and following the film clip across conditions and groups.

Emotion regulation

Participants reported the emotion regulation strategies that they engaged in following the induction and the film clip on the DERS-S. Univariate ANOVAs were conducted on each of the four DERS-S subscales following the induction and the film clip with condition and group as between-subjects factors. No significant effects were found for awareness of emotion following the induction period or following the film clip.

When DERS clarity of emotional experience, a measure of emotional understanding, was examined following the induction, a main effect of group emerged, $F(1, 80) = 5.09$, $p < 0.008$, $\eta^2 = 0.11$. Planned contrasts revealed that GAD participants high in dysphoria did not differ from GAD participants low in dysphoria on emotional clarity following the induction period, $L = 0.425$, $p < 0.056$, CI: (0.12, 0.863). The GAD groups were then collapsed and compared to the control group. GAD participants as a whole reported less understanding of their emotions ($M = 2.37$, SD = 0.88) than control participants ($M = 1.91$, SD = 0.73), $L = 0.414$, $p < 0.012$, CI: (0.093, 0.735).

Following the film clip, a main effect of group was also found for DERS clarity of emotional experience, $F(1, 80) = 7.69$, $p < 0.001$, $\eta^2 = 0.16$. Planned contrasts revealed that GAD participants high in dysphoria did not differ from GAD participants low in dysphoria on emotional clarity following the film clip, $L = 0.221$, $p < 0.220$, CI: (−0.135, 0.577). When the GAD groups were collapsed and compared to the control group, GAD participants as a whole reported less emotional clarity ($M = 2.37$, SD = 0.76) than control participants ($M = 1.82$, SD = 0.67), $L = 0.482$, $p < 0.001$, CI: (0.223, 0.741).

When DERS non-acceptance of emotion following the induction period was examined, a main effect of group emerged, $F(1, 80) = 8.56$, $p < 0.001$, $\eta^2 = 0.18$. Planned contrasts revealed that GAD participants high in dysphoria ($M = 1.78$, SD = 0.93) differed from GAD participants low in dysphoria ($M = 1.43$, SD = 0.49) on non-acceptance of emotion following the induction period, $L = 0.348$, $p < 0.042$, CI: (0.012, 0.684). Each GAD group was then compared to controls separately. GAD participants high in dysphoria reported more non-acceptance of their emotions following the induction than controls ($M = 1.15$, SD = 0.33), $L = 0.622$, $p < 0.001$, CI: (0.321, 0.922) while GAD participants low in dysphoria did not significantly differ from controls, $L = 0.274$, $p < 0.069$, CI: (−0.021, 0.569).

A main effect of group also was found for DERS non-acceptance of emotion following the film clip, $F(1, 80) = 10.83$, $p < 0.001$, $\eta^2 = 0.21$. GAD participants high in dysphoria ($M = 1.66$, SD = 0.69) reported more non-acceptance of their emotions than GAD participants low in dysphoria ($M = 1.37$, SD = 0.43), $L = 0.285$, $p < 0.036$, CI: (0.019, 0.551). Each GAD group was then compared to controls separately. GAD participants high in dysphoria reported less acceptance of their emotions following the film than controls

($M = 1.11$, SD $= 0.23$), $L = 0.549$, $p < 0.001$, CI: (0.312, 0.787), and GAD participants low in dysphoria also reported less acceptance of their emotions than controls, $L = 0.264$, $p < 0.027$, CI: (0.031, 0.498).

When these analyses were performed on the strategies subscale of the DERS, which captures participants' ability to access and use effective emotion regulation strategies, a main effect of group, $F(1, 80) = 15.55$, $p < 0.001$, $\eta^2 = 0.28$, was qualified by a significant group by condition interaction following the induction period, $F(4, 80) = 3.89$, $p < 0.006$, $\eta^2 = 0.16$. Simple effects analyses revealed that after engaging in worry, the 3 groups differed in their ability to use effective emotion regulation strategies, $F(1, 28) = 12.20$, $p < 0.001$, $\eta^2 = 0.47$. Using contrasts, GAD participants high in dysphoria did not differ from GAD participants low in dysphoria in emotion dysregulation, $L = 0.026$, $p < 0.912$, CI: (−0.451, 0.503). The aggregate GAD groups were then compared to controls using contrast analyses. GAD participants as a whole reported a decreased ability to use effective emotion regulation strategies ($M = 2.10$, SD $= 0.56$) compared to control participants ($M = 1.24$, SD $= 0.33$), $L = 0.855$, $p < 0.001$, CI: (0.501, 1.210) within those who were assigned to the worry condition. Following the neutral induction, a significant difference in the ability to effectively manage emotions was also found between the three groups, $F(1, 28) = 8.51$, $p < 0.002$, $\eta^2 = 0.43$. Contrast analyses revealed that GAD participants high in dysphoria ($M = 2.38$, SD $= 0.95$) reported more emotion dysregulation than GAD participants low in dysphoria ($M = 1.48$, SD $= 0.24$), $L = 0.896$, $p < 0.008$, CI: (0.253, 1.539). Thus, the GAD groups were each compared separately to the control group. GAD participants high in dysphoria reported more emotion dysregulation than controls ($M = 1.32$, SD $= 0.30$), $L = 1.052$, $p < 0.001$, CI: (0.509, 1.596), while no differences in emotion dysregulation were found between GAD participants low in dysphoria and controls, $L = 0.156$, $p < 0.592$, CI: (−0.439, 0.752). No differences were found among the groups after engaging in relaxation, $F(1, 30) = 1.23$, $p < 0.308$.

When this analysis was repeated on DERS emotion regulation strategies following the film clip, a main effect of group emerged, $F(1, 80) = 16.57$, $p < 0.001$, $\eta^2 = 0.29$. Regardless of condition, GAD participants high in dysphoria ($M = 1.95$, SD $= 0.71$) reported more emotion dysregulation than GAD participants low in dysphoria ($M = 1.55$, SD $= 0.46$), $L = 0.405$, $p < 0.005$, CI: (0.126, 0.683). However, both GAD participants high in dysphoria, $L = 0.715$, $p < 0.001$, CI: (0.467, 0.963), and low in dysphoria, $L = 0.310$, $p < 0.013$, CI: (0.066, 0.554), reported a lower ability to use effective emotion regulation strategies compared to controls ($M = 1.24$, SD $= 0.28$).

Discussion

The purpose of this study was to experimentally examine the emotion dysregulation model of GAD and to determine under what conditions individuals with GAD experience greater emotion intensity and dysregulation than non-anxious controls. We hypothesized that after engaging in worry, participants with GAD (regardless of their level of dysphoria) would experience more intensely negative emotions, report less awareness, understanding, and acceptance of their emotions, and experience more difficulty in effectively managing their emotions than controls. Our results suggested that worry has a negative impact on the intensity of emotional reactions to a negative stimulus for individuals with GAD. In contrast, worry was largely not required for the presence of emotion dysregulation in participants with GAD. Further, comorbid dysphoria was not found to account for changes in emotional intensity but did appear to play a role in some indices of emotion regulation. Whereas comorbid dysphoria had no effect on deficits in emotional clarity, it did impact non-acceptance and poor utilization of emotion regulation strategies.

We predicted that participants with GAD would experience more intense sadness than controls after engaging in worry. This hypothesis was supported. GAD participants who engaged in worry reported a systematic increase in sadness from baseline to the worry induction to the film. This systematic increase in sad affect was not evident for participants with GAD in either the neutral or relaxation conditions or for control participants in any of the three study conditions. This finding suggests that when individuals with GAD experience negative events in their environment or have negative interpersonal interactions while in a worried state, they are likely to respond with more intensely negative emotions to those events than they would while in a non-worried state. Consistent with prior investigations (Mennin et al., 2005), this result also supports the notion of a generalized emotion deficit in GAD, rather than one specific to anxiety given that GAD participants experienced increased intensity of sad, as opposed to anxious, reactions to the film. It is important

to note that GAD participants high in dysphoria exhibited the same pattern of sadness across the study as GAD participants low in dysphoria in each of the 3 conditions, demonstrating that the increased sadness experienced by GAD participants following the worry induction was not the result of co-occurring dysphoria.

While we found a linear increase in sadness across the duration of the study for GAD participants who engaged in worry, a different pattern of anxiety findings emerged. Differences between participants with GAD and controls were observed in the intensity of anxious affect only following the relaxation induction. Participants with GAD, regardless of dysphoria, reported a greater decrease in anxiety from baseline to the relaxation induction and continued to report this lower level of anxiety following the film. Control participants, in contrast, experienced a small decrease in anxiety from baseline to the relaxation induction and subsequently experienced an increase in anxiety following the film. This pattern of findings indicates that the relaxation induction was more helpful in decreasing anxiety for the participants with GAD than for controls, and that the decrease in anxiety experienced following relaxation was maintained for GAD participants even after the negative emotion induction. These findings provide evidence for the benefits of relaxation for individuals with GAD and are in accordance with a large body of work that has found relaxation to be a beneficial treatment for individuals with GAD (e.g., Borkovec & Costello, 1993; Borkovec, Newman, Pincus, & Lytle, 2002; Roemer & Orsillo, 2005).

It is important to note that observed increases in depression, but not anxiety, as a result of worry are consistent with the avoidance theory of worry (Borkovec et al., 2004). The avoidance theory posits that worry functions as a cognitive avoidance of anxious affect and is supported by evidence indicating suppression of both autonomic activity and anxiety-laden imaginal thought during worry. Importantly, the avoidance theory does not claim that non-anxious emotions occurring during or immediately after episodes of worry will be associated with suppressed affective responses. Suppression of subsequent affective response has been documented only for anxious material (e.g., Borkovec & Hu, 1990; Hazlett-Stevens, & Borkovec, 2001). In fact, recent evidence indicates that worry leads to amplification of depressed affect during subsequent ruminative processing (McLaughlin et al., in press), which is consistent with our sadness findings.

We also hypothesized that participants with GAD would experience less awareness, understanding, and acceptance of their emotions than controls after engaging in worry. However, a pattern of findings emerged such that GAD participants exhibited greater emotion dysregulation compared to control participants regardless of whether they engaged in worry. Thus, it appears that several aspects of emotion dysregulation are largely independent of the presence of a worried state. Rather, individuals with GAD appeared to display emotion regulation deficits across a number of contexts. In particular, participants with GAD reported less clarity and understanding of their emotional experience than control participants in all study conditions after both the induction period and the film clip. Clarity of emotional experience did not differ between the GAD groups high and low in dysphoria at either time point. This suggests that individuals with GAD have difficulty identifying and understanding their emotions regardless of whether they are in a worried state or have co-occurring depressive symptoms.

Induction of worry also did not impact the extent to which individuals with GAD had difficulty accepting their emotions. Rather, GAD participants appeared to have poorer emotional acceptance than controls, regardless of condition. Further, those GAD individuals with co-occurring dysphoria appeared to have the lowest levels of emotional acceptance. GAD participants without co-occurring dysphoria did not differ from controls in emotional acceptance following the inductions but were significantly lower in emotional acceptance by the end of film viewing. This suggests that, regardless of worry, when individuals with GAD are exposed to sad emotions, they may react in a non-accepting manner. This type of response appears to be particularly true for those individuals with comorbid dysphoria.

Although worry did not impact emotional understanding and acceptance of emotions, a differential effect was found for worry and neutral conditions in the utilization of emotion regulation strategies, which refers to the ability to rapidly access effective techniques for managing moods. GAD participants who received a worry induction, regardless of co-occurring dysphoria, reported greater emotion dysregulation than controls immediately following the induction. Additionally, GAD participants high in dysphoria reported greater emotion dysregulation than controls and GAD participants low in dysphoria immediately following the neutral induction. However, by the end of film viewing, no conditional effect remained, suggesting that exposure to sadness resulted in difficulty utilizing regulation strategies in individuals with GAD regardless of

the presence of a worried state. Therefore, worry may exacerbate difficulty in utilizing effective strategies but is not a necessary component of emotion dysregulation in GAD. Further, individuals with GAD and co-occurring depressive symptoms appear to experience greater difficulty in utilizing effective regulatory strategies than individuals with no comorbid dysphoria, even when they are in a relatively neutral affective state. However, this deficit in utilization of effective regulatory strategies was not limited to those with co-occurring depressive symptoms, indicating that the relationship between GAD and poor access to emotion regulation strategies is not solely due to co-occurring dysphoria.

Unlike the other emotion regulation indices, no differences were found between GAD and control participants in reported awareness of emotional experience, regardless of condition. The items on the DERS-S that comprise the awareness scale primarily assess attention to emotions (e.g., "I am attending to my feelings"). Prior findings regarding attention to emotion deficits have largely not supported attention to emotion differences between individuals with GAD and controls (Mennin et al., 2005; Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006; Turk et al., 2005). It remains unclear whether attention to emotion is adaptive or maladaptive. On one hand, acceptance and flexible awareness of emotions is likely to be adaptive and facilitate functioning by allowing individuals to use the information their emotions provide (Roemer & Orsillo, 2005). On the other hand, non-accepting awareness of emotions or attending to emotions without concurrent emotional understanding is likely to be problematic. This type of attention to emotions likely has similar consequences as self-focused attention, which is associated with negative affect (Mor & Winquist, 2002), anxiety, and depression (e.g., Ingram, 1990). While differences in awareness of emotion were not found between GAD and control participants in this study, it remains unclear whether this finding reflects a true lack of difference in awareness of emotional experience or reflects a difference in the *type* of awareness given that attention to emotions can be adaptive or maladaptive. For example, control participants may have attended to their emotions in an accepting and non-judgmental way, whereas GAD participants may have attended to their emotions in a critical and non-accepting way. Future studies will need to further examine emotional awareness and attention to emotions in ways that allow the type of attention to be discerned.

Our experimental findings are consistent with previous research that has documented greater emotional intensity and deficits in understanding, acceptance, and managing emotions in individuals with GAD (e.g., Mennin et al., 2005; Salters-Pedneault et al., 2006; Turk et al., 2005). This study extends prior findings by demonstrating intensity in state response to induced mood states as well as addressing the role of context in instigated emotion dysfunction. In addition, the results from the pre-session questionnaires in the current study replicate findings in these previous studies of trait differences in emotional intensity, acceptance of emotional experience, and ability to effectively manage emotions between individuals with GAD and non-anxious controls. The present study extends these findings by demonstrating that levels of emotion intensity and dysregulation are not due solely to co-occurring depressive symptoms as demonstrated by baseline trait characteristics and the majority of experimental findings. Further, it appears that emotion regulation deficits are present not only during episodes of worry, but also during periods when individuals are experiencing neutral and non-anxious affective states, particularly for individuals with GAD and co-occurring dysphoria.

A number of limitations present in the current study require that these findings be interpreted with caution. The first limitation involves the use of a primarily student sample of participants. Student samples, even those meeting diagnostic criteria for a mental disorder, tend to be higher functioning than clinical samples. As such, these findings may not be generalizable to clinical samples of individuals with more severe GAD. Despite the limitations inherent with student samples, all participants completed a well-validated structured diagnostic interview to verify diagnosis. This represents a clear improvement on past studies examining emotion dysregulation in GAD in student populations (e.g., Turk et al., 2005). Nevertheless, future studies should examine the conditions under which individuals with GAD experience increased emotion dysregulation using clinical samples. Additionally, the present study used a self-report measure of depression to classify GAD participants into groups high and low in dysphoria rather than a diagnostic interview. Due to time constraints, participants did not complete the entire ADIS-IV; rather, they only completed the GAD and social phobia sections but not the major depressive disorder module. Although the BDI-II is a good analogue measure of major depression (Beck, Steer, & Garbin, 1988; Kumar, Steer, Teitelman, & Villacis, 2002) and is the most commonly used self-report measure of depressive symptoms (Nezu, Ronan, Meadows, & McClure, 2000), to fully explore the separate influence of GAD pathology and depression on emotion dysregulation, future

studies must be conducted using GAD samples both with and without comorbid depression. Finally, because the entire ADIS-IV was not completed, our assessment of disorders other than GAD, social phobia, and depression was limited. Future studies examining emotion regulation in GAD should assess the full range of psychopathology to fully account for the effects of comorbidity with other disorders.

Given the exclusive use of self-report measures of emotion, our assessment of emotion was therefore limited to the subjective aspect of emotional experience and did not examine physiological or expressive dimensions. Future studies must clearly examine physiological and expressive dimensions of emotion in individuals with GAD relative to controls. Another limitation of the current study involves small sample size. Comparisons between the GAD groups represent a particular concern in regards to sample size and power to detect group differences. It is possible that the small number of GAD participants in each of these groups made it difficult to detect differences in emotional intensity and dysregulation between GAD groups. Future studies should aim to include larger representative samples of individuals with GAD, both with and without comorbid depression.

Elevated depression in GAD participants was expected, given the high levels of comorbidity among GAD and major depression (e.g., Kessler et al., 1999). However, because participants with GAD were also more depressed than controls, we must recognize the possibility that the differences between these study groups in emotional intensity and regulation were a function of co-occurring depression rather than GAD or a function of both. For most outcome measures, both GAD individuals with and without dysphoria displayed greater emotion-related deficits than controls. However, for emotional acceptance and utilization of emotion regulation strategies, dysphoric GAD participants clearly reported greater deficits than their non-dysphoric counterparts and controls suggesting that, in part, some of our results could be due to the presence of depression. Further, given the use of a generally high functioning college sample, ''pure'' cases of depression (which often present as severe and impaired) were hard to locate and recruit. Thus, the lack of a clinical comparison group limits our ability to conclude that our findings are specific to GAD. Future research should aim to examine the specificity of these findings by including clinical comparison groups of participants meeting criteria for depression and other anxiety disorders.

The similar patterns of results found between GAD individuals with and without dysphoria might suggest that emotion-related deficits are a common factor shared by a number of emotional disorders. Indeed, a number of studies have shown that generalized anxiety disorder and depression are related to a latent negative affectivity or neuroticism factor (e.g., Brown et al., 1998), leading to recent calls to unify GAD and depression (e.g., Watson, 2005). It may be that the increased intensity and dysregulation seen in the present study are common characteristics shared by a number of emotional disorders. Clearly, emotion-related deficits are not specific to any one disorder (Kring & Werner, 2004). Thus, a possible interpretation of the current findings is that they represent the core dysfunction of any ''neurotic'' disorder and had another anxiety or mood diagnostic comparison group been assessed, the results would be indistinguishable from the GAD group.

Although we might expect emotionality and dysregulation to be a characteristic in many disorders related to neuroticism, it is possible that patterns of specificity are also present. Although the current study cannot adequately address specificity, prior findings of emotion-related deficits in GAD and depression show both non-specific and specific patterns. For instance, levels of positive affectivity (see Watson, 2005) and expectancies concerning positive events (Miranda & Mennin, in press) have distinguished GAD and depression. Further, recent studies have shown that depression is characterized by a *decreased* emotional sensitivity that is less responsive to differing contexts, possibly as a function of motivational resource conservation (see Rottenberg, 2005). In contrast, heightened intensity of emotions, which could be considered to be a central aspect of emotional distress, has been shown to be characteristic of GAD but not depression or social anxiety (e.g., Mennin et al., in press). These findings of unique patterns of emotional dysfunction among different disorders suggest the importance of more complex models of emotional factors than could be explained solely by a global distress model of neuroticism. Likely, there will be emotion factors that are common to all neurotic disorders and others that may demonstrate specificity, particular when present in combination or in interactions with contexts particularly relevant to a given disorder (e.g., disgust sensitivity in spider phobia). This may represent where emotion regulation models will have their greatest impact, as they can help produce more fine grained analyses of emotional processes and the ways in which individuals with emotional disorders respond to and regulate their emotions. However, carefully designed studies utilizing numerous clinical comparison groups are necessary to address these assertions.

The results of this study indicate that individuals with GAD experience more intense emotional reactions to negative events following periods of worry, even for emotions not central to the phenomenology of the condition such as sadness. Additionally, individuals with GAD, regardless of their level of depressive symptoms, experience poorer understanding of their emotions and greater difficulty managing their emotions following a negative emotion induction when compared to non-anxious individuals, regardless of the presence of a worried state. Moreover, individuals with GAD and high levels of depressive symptoms reported decreased acceptance of their emotions and less access to effective emotion regulation strategies than non-anxious individuals and those with GAD and low levels of depressive symptoms prior to the negative emotion induction, indicating that co-occurring depressive symptoms may exacerbate certain emotion regulation deficits in individuals with GAD. In contrast, individuals with GAD did not experience dysregulated emotions in response to events that occur while they are in a state of relaxation. Future investigations of the psychopathology and treatment of GAD may benefit from an understanding of emotion-related deficits during both worried and non-worried states.

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References

American Psychiatric Association. (2000). *Diagnostic and statistical manual for psychiatric disorders-fourth edition-text revision (DSM-IV-TR)*. Washington, DC: Author.

Andrews, V. H., & Borkovec, T. D. (1988). The differential effects of inductions of worry, somatic anxiety, and depression on emotional experience. *Journal of Behavior Therapy and Experimental Psychiatry*, *19*, 16–21.

Barlow, D. H., Craske, M. G., Cerny, J. A., & Klosko, J. S. (1989). Behavioral treatment of panic disorder. *Behavior Therapy*, *20*, 261–282.

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.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1996). *Beck depression inventory manual* (2nd ed.). San Antonio, TX: Psychological Corporation.

Borkovec, T. D., Alcaine, O., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 77–108). New York: Guilford Press.

Borkovec, T. D., & Costello, E. (1993). Efficacy of applied relaxation and cognitive-behavioral therapy in the treatment of generalized anxiety disorder. *Journal of Consulting and Clinical Psychology*, *61*, 611–619.

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*, *31*, 321–324.

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., Robinson, E., Pruzinsky, T., & DePree, J. A. (1983). Preliminary exploration of worry: Some characteristics and processes. *Behaviour Research and Therapy*, *21*, 9–16.

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.

Borkovec, T. D., Shadick, R. M., & Hopkins, M. (1991). The nature of normal and pathological worry. In R. M. Rapee, & D. H. Barlow (Eds.), *Chronic anxiety: Generalized anxiety disorder and mixed anxiety-depression*. New York: Guilford Press.

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., & Liebowitz, M. R. (1994). The empirical basis of generalized anxiety disorder. *American Journal of Psychiatry*, *151*, 1272–1280.

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.

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology*, *107*, 179–192.

Craske, M. G., Rapee, R. M., Jackel, L., & Barlow, D. H. (1989). Qualitative dimensions of worry in DSM-III-R generalized anxiety disorder subjects and nonanxious controls. *Behaviour Research and Therapy*, *27*, 397–402.

Di Nardo, P. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV (ADIS-IV)*. San Antonio, TX: Psychological Corporation.

Dozois, D. J. A., Dobson, K. S., & Ahnberg, J. L. (1998). A psychometric evaluation of the Beck Depression Inventory-II. *Psychological Assessment*, *10*, 83–89.

Dugas, M. J. (2000). Generalized anxiety disorder publications: Where do we stand? *Journal of Anxiety Disorders*, *14*, 31–40.

Ekman, P., Sorensen, E. R., & Friesen, W. (1969). Pan-cultural elements in facial displays of emotion. *Science*, *164*, 86–88.

Foa, E. B., Dancu, C., Hembree, E., Jaycox, L., Meadows, E. A., & Street, G. P. (1999). A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. *Journal of Consulting and Clinical Psychology*, *67*, 194–200.

Freeston, M. H., Dugas, M. J., & Ladouceur, R. (1996). Thoughts, images, worry, and anxiety. *Cognitive Therapy & Research*, *20*, 265–273.

Goldsmith, R. E., & Walters, H. (1989). A validity study of the Affect Intensity Measure. *Journal of Social Behavior and Personality*, *4*, 133–140.

Gould, R. A., Safren, S. A., Washington, D. O., & Otto, M. W. (2004). A meta-analytic review of cognitive-behavioral treatments. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice*. New York: The Guilford Press.

Grant, B. H., Stinson, F., Dawson, D., Ruan, W., Goldstein, R., Smith, S., et al. (2005). Prevalence, correlates, comorbidity, and comparative disability of DSM-IV generalized anxiety disorder in the USA: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Psychological Medicine*, *35*, 1747–1759.

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and validation of the Difficulties with Emotion Regulation Scale. *Journal of Psychopathology and Behavioral Assessment*, *26*, 41–54.

Gross, J. J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science*, *10*, 214–219.

Gross, J. J., & Levenson, R. W. (1995). Emotion elicitation using films. *Cognition and Emotion*, *9*, 87–108.

Hayes, S. C., Strosahl, K., Wilson, K. G., Bissett, R. T., Pistorello, J., Toarmino, D., et al. (2004). Measuring experiential avoidance: A preliminary test of a working model. *The Psychological Record*, *54*, 553–578.

Hazlett-Stevens, H., & Borkovec, T. D. (2001). Effects of worry and progressive relaxation on the reduction of fear in speech phobia: An investigation of situational exposure. *Behavior Therapy*, *32*, 503–517.

Ingram, R. E. (1990). Self-focused attention in clinical disorders: Review and a conceptual model. *Psychological Bulletin*, *107*, 156–176.

Keller, M. B. (2002). The long-term clinical course of generalized anxiety disorder. *Journal of Clinical Psychiatry*, *63*(Suppl. 8), 11–16.

Kessler, R. C., Dupont, R. L., Berglund, P., & Wittchen, H.-U. (1999). Impairment in pure and comorbid generalized anxiety disorder and major depression at 12 months in 2 national surveys. *American Journal of Psychiatry*, *156*, 1915–1923.

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the National Comorbidity Survey. *Archives of General Psychiatry*, *51*, 8–19.

Kring, A. M., & Werner, K. H. (2004). Emotion regulation and psychopathology. In P. Philippot, & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 359–385). Mahwah, NJ: Erlbaum.

Kumar, G., Steer, R. A., Teitelman, K. B., & Villacis, L. (2002). Effectiveness of Beck Depression Inventory-II subscales in screening for major depressive disorders in adolescent psychiatric inpatients. *Assessment*, *9*, 164–170.

Larson, R. J., & Deiner, E. (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality*, *21*, 1–39.

Lubin, B., Van Whitlock, R., Reddy, D., & Petren, S. (2001). A comparison of the short and long forms of the Multiple Adjective Affect Checklist-Revised (MAACL-R). *Journal of Clinical Psychology*, *57*, 411–416.

Lubin, B., & Zuckerman, M. (1999). *The Multiple Adjective Affect Checklist-Revised* (3rd ed.). San Diego, CA: Education and Industrial Testing Service.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, *95*, 15–20.

Mayer, J. D., & Stevens, A. A. (1994). An emerging understanding of the reflective (meta-) experience of mood. *Journal of Research in Personality*, *28*, 351–373.

McLaughlin, K. A., Borkovec, T. D., & Sibrava, N. J. (in press). The effect of worry and rumination on affect states and cognitive activity. *Behavior Therapy*.

Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2002). Applying an emotion regulation framework to integrative approaches to generalized anxiety disorder. *Clinical Psychology: Science and Practice*, *9*, 85–90.

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

Mennin, D. S., Holaway, R., Fresco, D. M., Moore, M. T., & Heimberg, R. G. (in press). Delineating components of emotion dysregulation in anxiety and mood psychopathology. *Behavior Therapy*.

Miranda, R., & Mennin, D. S. (in press). Depression, generalized anxiety disorder, and certainty in pessimistic predictions about the future. *Cognitive Therapy and Research*.

Mor, N., & Winquist, J. (2002). Self-focused attention and negative affect: A meta-analysis. *Psychological Bulletin*, *128*, 638–662.

Mowrer, O. H. (1947). On the dual nature of learning: A re-interpretation of "conditioning" and "problem-solving". *Harvard Educational Review*, *17*, 102–148.

Newman, M. G., Zuellig, A. R., Kachin, K. E., Constantino, M. E., Przeworski, A., Thane, E., et al. (2002). Preliminary reliability and validity of the Generalized Anxiety Disorder Questionnaire-IV: A revised self-report diagnostic measure of generalized anxiety disorder. *Behavior Therapy*, *33*, 215–233.

Nezu, A. M., Ronan, G. F., Meadows, E. A., & McClure, K. S. (2000). *Practicioner's guide to empirically based measures of depression*. Dordrecht, Netherlands: Kluwer Academic Publishers.

Olfson, M., Fireman, B., Weissman, M. M., Leon, A. C., Sheehan, D. V., Kathol, R. G., et al. (1997). Mental disorders and disability among patients in a primary care group practice. *American Journal of Psychiatry*, *154*, 1734–1740.

Persons, J. B., Mennin, D. S., & Tucker, D. E. (2001). Common misconceptions about the nature and treatment of generalized anxiety disorder. *Psychiatric Annals*, *31*, 501–508.

Roemer, L., Molina, S., & Borkovec, T. D. (1997). An investigation of worry content among generally anxious individuals. *Journal of Nervous and Mental Disease*, *185*, 314–319.

Roemer, L., & Orsillo, S. M. (2005). An acceptance-based behavior therapy for generalized anxiety disorder. In S. M. Orsillo, & L. Roemer (Eds.), *Acceptance and mindfulness-based approaches to anxiety: Conceptualization and treatment* (pp. 213–240). New York, NY: Springer.

Rottenberg, J. (2005). Mood and emotion in major depression. *Current Directions in Psychological Science*, *14*, 167–170.

Roy-Byrne, P. P., & Katon, W. (1997). Generalized anxiety disorder in primary care: The precursor/modifier pathway to increased health care utilization. *Journal of Clinical Psychiatry*, *58*(Suppl. 3), 34–38.

Salovey, P., Mayer, J. D., Goldman, S. L., Turvey, C., & Palfai, T. P. (1995). Emotional attention, clarity, and repair: Exploring emotional intelligence using the Trait Meta-Mood Scale. In J. W. Pennebaker (Ed.), *Emotion, disclosure, and health* (pp. 125–154). Washington, DC: American Psychological Association.

Salters-Pedneault, K., Roemer, L., Tull, M. T., Rucker, L., & Mennin, D. S. (2006). Evidence of broad deficits in emotion regulation associated with chronic worry and generalized anxiety disorder. *Cognitive Therapy and Research*, *30*, 469–480.

Sanderson, W. C., & Barlow, D. H. (1990). A description of patients diagnosed with DSM-III-R generalized anxiety disorder. *Journal of Nervous and Mental Disease*, *178*, 588–591.

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

Turk, C. L., Heimberg, R. G., Luterek, J. A., Mennin, D. S., & Fresco, D. M. (2005). Delineating emotion regulation deficits in generalized anxiety disorder: A comparison with social anxiety disorder. *Cognitive Therapy and Research*, *29*, 89–106.

Vrana, S. R., Cuthbert, B. N., & Lang, P. J. (1986). Fear imagery and text processing. *Psychophysiology*, *23*, 247–253.

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, *114*, 522–536.

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

Zuckerman, M., & Lubin, B. (1985). *Manual for the Multiple Affect Adjective Checklist-R*. San Diego, CA: Educational & Industrial Testing Service.