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Preliminary evidence for an emotion dysregulation model of generalized anxiety disorder

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Abstract

Three studies provide preliminary support for an emotion dysregulation model of generalized anxiety disorder (GAD). In study 1, students with GAD reported heightened intensity of emotions, poorer understanding of emotions, greater negative reactivity to emotional experience, and less ability to self-soothe after negative emotions than controls. A composite emotion regulation score significantly predicted the presence of GAD, after controlling for worry, anxiety, and depressive symptoms. In study 2, these findings were largely replicated with a clinical sample. In study 3, students with GAD, but not controls, displayed greater increases in self-reported physiological symptoms after listening to emotion-inducing music than after neutral mood induction. Further, GAD participants had more difficulty managing their emotional reactions. Implications for GAD and psychopathology in general are discussed.

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Introduction

Approximately 5% of people will suffer from generalized anxiety disorder (GAD) at some point in their lives (Kessler et al., 1994). GAD is associated with significant role impairment (Wittchen, Zhao, Kessler, & Eaton, 1994), increased health care utilization (Blazer, Hughes, & George,

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1991), increased health care costs and decreased productivity (Greenberg et al., 1999). However, despite the prevalence of GAD and the suffering associated with it, GAD remains poorly understood relative to other anxiety disorders and, as a result, has been more difficult to treat. When compared to the other anxiety disorders, far fewer investigations have examined the psychopathological mechanisms involved in GAD (Dugas, 2000).

Recently, theorists have begun to expand our understanding of GAD through the development of models that highlight the importance of worry. One of the most comprehensive accounts of the role of worry in GAD is Borkovec's *avoidance theory* (e.g., Borkovec, Alcaine, & Behar, 2004). Borkovec and colleagues have presented convincing empirical support for the notion that worry is a perseverative, cognitive activity that serves an avoidance function for persons with GAD. More specifically, Borkovec and colleagues argue that worry allows individuals to approach emotional topics at an abstract, conceptual level and, consequently, to avoid aversive images, autonomic arousal, and intense negative emotions in the short-run (for a review of numerous studies in support of this view, see Borkovec et al., 2004). However, over the long term, the individual is repeatedly confronted with the emotional material, frequently has a more intense experience of anxiety, and engages in repetitive worry to "dull" this experience. In doing so, the person again fails to fully confront the distressing stimuli, and emotional processing of aversive experiences is inhibited.

As described above, from the perspective of the avoidance theory of worry, worry facilitates avoidance of the imagery and physiological arousal associated with negative emotion. However, the nature of the emotional experience that prompts individuals with GAD to engage in frequent avoidance strategies such as worry has not been directly addressed. Although individuals with GAD may use worry to avoid distressing emotional experience, the theory does not explain why this experience is so aversive that it would need to be avoided. To understand this, the characteristics of the emotional experience that may prompt avoidance need to be explored. Further, emotion may play a larger role in GAD than specifically in relation to worry. Emotion and its dysregulation may be integral, yet largely unexplored, factors in the psychopathology of GAD and thus may have important implications for treatment (see Samoilov & Goldfried, 2000).

Conceptualizations of GAD may benefit from attention to advances in the fields of emotion theory (e.g., Ekman & Davidson, 1994), emotion regulation (e.g., Gross, 1998), and affective neuroscience (e.g., LeDoux, 1996). Contemporary theories of emotion emphasize its adaptive value (e.g., Gross, 1998). Theorists have argued that emotions are cues for readiness for action or "action tendencies" that work to establish, maintain, or disrupt relationships with particular internal and external environments that signify importance to the person (see Barlow, 2002). Emotion serves an information function, notifying individuals of the relevance of their concerns, needs, or goals in a given moment. Attention to the adaptive value to emotions may account for the resurgence of interest in the role of emotion in psychopathology and psychotherapy (e.g., Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003; Greenberg, 2002; Kring & Bachorowski, 1999; Samoilov & Goldfried, 2000).

Emotion regulation, as a field of study, examines how individuals influence, control, experience, and express their emotions (Gross, 1998, p. 275). In discussing regulation of one's own emotions, Thompson (1990) stresses the importance of the restraint of emotion, as well as its maintenance and enhancement. Clearly, needs to diminish emotional arousal to work effectively or contain one's anger in a public setting are aspects of emotion regulation. However, investigators have

more recently argued for the importance of accentuating both positive and negative emotional experiences to gain a greater understanding of goal pursuit (see [Bonnano, 2001](#)). Adaptive regulation of emotions has been found to relate to positive functioning and emotional health. Salovey, Mayer, and colleagues (e.g., [Mayer, Salovey, Caruso, & Sitarenios, 2001, 2003](#)) theorize that people differ in their ability to attend to, process, and act as a result of their emotions, which they have called *emotional intelligence*. Research has begun to accumulate that supports the functional benefits of emotional intelligence (e.g., [Mayer et al., 2003](#)).

If greater ability to manage emotions is associated with productivity and positive emotional health, then low levels of emotion regulatory ability should be associated with psychopathology and difficulties in adjustment. Indeed, investigators have applied findings from emotion research to clinical phenomena such as unipolar depression (e.g., [Rottenberg, Kasch, Gross, & Gotlib, 2002](#)), eating disorders (e.g., [Westen & Harnden-Fischer, 2001](#)), and borderline personality disorder (e.g., [Wagner & Linehan, 1999](#)). Rottenberg and Gross (2003) caution that, when looking at the relationship between emotion dysregulation and psychopathology, investigators need to recognize that regulation occurs dynamically throughout different points in the emotion generative process. Using this perspective as a base, Mennin and colleagues (for an introduction to this perspective, see [Mennin, Heimberg, Turk, & Fresco, 2002](#); [Mennin, Heimberg, Turk, & Carmin, 2004](#)) have developed an emotion dysregulation model of GAD. Emotion may become dysfunctional in individuals with GAD through a rapid temporal process of dysregulation involving (1) heightened intensity of emotions; (2) poor understanding of emotions; (3) negative reactivity to one's emotional state (e.g., fear of emotion); and (4) maladaptive emotional management responses. Heightened emotional intensity coupled with an inadequate base of knowledge about emotions and discomfort with emotional experience may lead individuals with GAD to use maladaptive coping strategies to control and constrain their emotional experience to decrease this aversive state. Rather than processing an emotion through attention, understanding, and experiencing (Foa & Kozak, 1986), they may utilize worry and other intra- and interpersonal processes to avoid the distress associated with these emotions.

We have suggested that individuals with GAD have emotional reactions that occur more easily, quickly, and intensely than for most other people (i.e., heightened emotional intensity). They may frequently experience strong negative affect, which sometimes is elicited by situations that are not evocative to most other people. Consistent with the research showing that higher levels of emotional intensity are associated with more emotion expressivity ([Gross & John, 1997](#)), individuals with GAD may also express more of their emotions, especially negative emotions, than most other people. Being overly expressive of negative emotions on a regular basis may lead to criticism or rejection by others, which, in turn, may elicit high levels of negative affect in these emotionally sensitive individuals with GAD. Research suggests that individuals with GAD perceive their relationships with family, friends, and romantic partners as moderately to severely impaired ([Turk, Mennin, Fresco, & Heimberg, 2000](#)), and evidence is mounting that individuals with GAD may have interpersonal styles that contribute to the relationship problems that they perceive. [Pincus and Borkovec \(1994\)](#) and [Eng and Heimberg \(2004\)](#) found that the majority of individuals with GAD endorsed interpersonal styles that were best characterized as overly nurturant and intrusive.

Individuals with GAD may also have difficulty identifying primary emotions such as anger, sadness, fear, disgust, and joy and instead experience their emotions as undifferentiated,

confusing, and overwhelming (i.e., poor understanding of emotions). In this way, persons with GAD may be unable to access and utilize the adaptive information conveyed by their emotions. Difficulty identifying and describing emotions has been related to various forms of psychopathology (see Taylor, Bagby, Parker, & Alexander, 1997). Given strong emotional responses and a poor understanding of them, individuals with GAD may experience emotions as aversive and become anxious when they occur (i.e., negative reactivity to emotions). Associated reactions may include extreme hypervigilance for threatening information and activation of negative beliefs about emotions.

We also hypothesize that individuals with GAD have difficulty knowing when or how to enhance or diminish their emotional experience in a manner that is appropriate to the environmental context (i.e., maladaptive emotional management). Given the intensity of their emotion and both their lack of skills for utilizing emotions and their negative reactions to them, we suggest that individuals with GAD turn to a variety of management approaches that are maladaptive. Typically, dysregulation is viewed in terms of faulty control mechanisms. However, there may be other types of dysregulation that occurs in individuals with GAD. Cicchetti, Ackerman, and Izard (1995) suggest that problems of emotional regulation can be divided into two categories. The first involves difficulties in modulation of emotional experience and/or expression; the second involves frequent or automatic attempts to control or suppress emotional experience or expression. In the first scenario, the person experiences emotions with great intensity but is unable to adequately modulate the experience (e.g., self-soothe, inhibit emotional expression). In the second scenario, the person engages in control strategies that prevent emotion from being experienced. Berenbaum et al. (2003) propose a similar dichotomy in their discussion of two *emotional regulation disturbances*, which they term emotional hyperreactivity and hyporeactivity.

Mennin et al. (2002, 2004) argue that both types of deficits characterize GAD. For example, intense emotions that are misunderstood and misinterpreted as dangerous may contribute to the problems that individuals with GAD have in modulating their emotional experience and expression. Furthermore, Mennin and colleagues propose that individuals with GAD over-engage control mechanisms, decreasing emotional experience through avoidance and blunting. This latter form of dysregulation has been discussed extensively by Hayes and colleagues (see Hayes, Strosahl, & Wilson, 1999) in their examination of experiential avoidance. Experiential avoidance refers to one's difficulty engaging internal experiences such as emotions, thoughts, images, and sensations. Often this unwillingness stems from efforts to control and diminish the experience of pain associated with these internal events. However, paradoxically, these efforts to control often lead to further and stronger levels of discomfort, and may be an integral component of many forms of psychopathology (Hayes et al., 1999). Borkovec's avoidance model of worry can be viewed within such an emotion regulatory framework. Worry can be viewed as a cognitive control strategy employed in attempts to "fix" the regulatory problems associated with subjectively jarring emotional experience.

The following three investigations aim to provide preliminary support for this emotion dysregulation perspective on GAD. The first study is an initial investigation of difficulties in regulating emotions among college students with and without GAD. The ability of emotion dysregulation to predict the presence of GAD was also assessed. The second study was a replication of the first, comparing treatment-seeking patients with GAD to community controls.

Finally, an experimental study is presented that examined whether self-reported physiological responses to negative mood induction, regardless of subjective awareness of emotions, are greater in individuals with GAD than control participants. Further, participants' abilities to manage this induced mood experience were examined.

Study 1: preliminary examination in an undergraduate sample

This initial study examined the relationships among components of emotion, its dysregulation and GAD. We hypothesized that individuals with GAD, in comparison to control participants, would demonstrate heightened intensity of emotions (i.e., greater emotion impulse strength, greater emotional expressivity), poorer understanding of emotions (i.e., less clarity of emotions, greater difficulty identifying and describing emotions), more negative reactivity to emotions (i.e., greater attention to emotions, greater endorsement of fear-laden beliefs activated by various emotions), and more maladaptive management of emotions (i.e., greater difficulty self-soothing and returning to baseline following an aversive emotional experience). Further, we hypothesized that indices of emotion dysregulation would predict the presence of GAD, contributing unique variance to the equation beyond that accounted for by the negative affective characteristics typically associated with GAD (i.e., trait anxiety, worry, or depressive symptoms).

Method

Participants

A sample of 538 unselected undergraduate students in an introductory psychology class (73% women, 27% men) participated in the present study. Mean age of the sample was 19.52 ($SD = 3.32$). The ethnicity of the sample was diverse: 40% Caucasian, 33% African American, 13% Asian-American, 4% Latino, and 10% students who endorsed a "mixed" ethnic heritage. Participants were predominantly single and never married (97%).

Measures

The *Generalized Anxiety Disorder Questionnaire—IV* (GAD-Q-IV; Newman et al., 2002) is a 9-item self-report questionnaire that reflects the criteria for GAD as delineated in the *Diagnostic and Statistical Manual for Mental Disorders, 4th Edition* (DSM-IV; American Psychiatric Association, 1994). Items are phrased according to DSM-IV criteria. Most items are dichotomous and measure the excessive and uncontrollable nature of worry as experienced by persons with GAD (e.g., "During the last six months, have you been bothered by excessive worries more days than not?") and related physical symptoms including restlessness, sleep difficulties, difficulty concentrating, irritability, fatigue, and muscle tension. One item is open-ended and asks for a listing of the most frequent worry topics. Two items are rated on a scale of zero ("None") to eight ("Very Severe") and measure functional impairment and subjective distress.

The GAD-Q-IV was originally scored by comparing individual items to specific DSM-IV criteria for GAD and determining whether a diagnosis was present (Newman, Zuellig, Kachin, Constantino, & Cashman, 2001). However, Newman et al. (2002) later recommended using a dimensional scoring system (range 0–13) with cut-off scores to determine presence or absence of

GAD. Thus, an individual may fail to endorse an item on the GAD-Q-IV required by DSM-IV (e.g., excessive worries more days than not during the past 6 months) but still receive a diagnosis of GAD. The former method, being slightly more conservative, was utilized in this investigation. The GAD-Q-IV has been found to have high concordance with a diagnosis of GAD as determined by the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Brown, DiNardo, & Barlow, 1994), a semi-structured diagnostic interview. It is also related to measures of excessive worry such as the Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990) and uncorrelated with conceptually unrelated measures (Newman et al., 2002).

The *Affective Control Scale* (ACS; Williams, Chambless, & Ahrens, 1997) is a 42-item self-report measure assessing fear of emotions and attempts to control emotional experience. Subscales include (1) fear of anxiety (e.g., “It scares me when I am nervous”); (2) fear of depression (e.g., “When I get “the blues”, I worry that they will pull me down too far”); (3) fear of anger (e.g., “I am afraid that letting myself feel really angry about something could lead me into an unending rage”); and (4) fear of positive emotions (e.g., “I worry about losing self-control when I am on cloud nine”). Items are scored on a 7-point likert-type scale (i.e., “Strongly Agree” to “Strongly Disagree”). The subscales have demonstrated satisfactory internal consistency (Berg, Shapiro, Chambless, & Ahrens, 1998; Williams et al., 1997). The ACS total score is highly correlated with neuroticism and emotional control and minimally correlated with social desirability (Berg et al., 1998; Williams et al., 1997).

The *Toronto Alexithymia Scale-20* (TAS; Bagby, Parker, & Taylor, 1994a; Bagby, Taylor, & Parker, 1994b) is a 20-item self-report measure that generates three factor-analytically derived subscales including (1) difficulty identifying feelings (e.g., “When I am upset, I don’t know if I am sad, frightened, or angry”); (2) difficulty describing feelings (e.g., “It is difficult for me to find the right words for my feelings”), and (3) externally oriented thinking. The externally oriented thinking subscale was not used in this study, as it does not solely assess understanding of emotion. Items are scored on a 5-point Likert-type scale. The factor scores have evidenced acceptable internal consistency (Bagby et al., 1994a). As expected, TAS-20 factor scores correlate negatively with measures assessing access to one’s feelings and openness to feelings (Bagby et al., 1994b).

The *Trait Meta-Mood Scale* (TMMS; Salovey, Mayer, Goldman, Turvey, & Palfai, 1995) is a 30-item self-report measure of emotional intelligence that is comprised of three factor-analytically derived subscales: (1) attention to emotion (e.g., “I pay a lot of attention to how I feel”); (2) clarity of emotions (e.g., “I almost always know exactly how I am feeling”); and (3) mood repair (e.g., “The best way for me to handle my feelings is to experience them to the fullest”). Items are scored on a 1–5 Likert-type scale. These subscales are internally consistent and related to other measures of mood and mood management (Salovey et al., 1995).

The *Berkeley Expressivity Questionnaire* (BEQ; Gross & John, 1997) is a 16-item self-report measure that assesses both the strength of emotional response tendencies and the degree to which these emotional impulses are expressed overtly. It is comprised of three factor-analytically derived subscales: (1) impulse strength (e.g., “I have strong emotions”); (2) negative expressivity (e.g., “Whenever I feel negative emotions, people can easily see exactly what I am feeling”); and (3) positive expressivity (e.g., “I laugh out loud when someone tells me a joke that I think is funny”). Items are scored on a 7-point Likert-type scale. The BEQ subscales have been shown to have adequate internal consistency and retest reliability (Gross & John, 1997).

The *Penn State Worry Questionnaire* (PSWQ; Meyer et al., 1990) is a 16-item self-report inventory designed to assess trait worry and to capture the generality, excessiveness, and uncontrollability characteristic of pathological worry. Items include “Once I start worrying, I can’t stop” and “When I am under pressure, I worry a lot”. Items on the PSWQ are scored on a 5-point scale from “Not at all typical” to “Very typical”. In samples of college undergraduates, the PSWQ has repeatedly demonstrated good internal consistency and good test–retest reliability over intervals as long as 8–10 weeks (Meyer et al., 1990). Patients with GAD have been found to score higher on the PSWQ than community controls and patients with panic disorder, panic disorder with agoraphobia, social anxiety disorder, simple phobia, or obsessive compulsive disorder (Brown, Antony, & Barlow, 1992).

The *State-Trait Anxiety Inventory* (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) is a 20-item self-report measure of chronic and current levels of anxiety. For this study, we used the Trait version of the scale, which measures characteristic tendencies to be anxious. Items (e.g., “I get in a state of tension or turmoil as I think over recent concerns and interests” and “I feel nervous and restless”) are rated on a 4-point Likert-type scale. The STAI Trait scale has been found to have excellent internal consistency (coefficient alphas range from .86 to .92) and levels of test–retest stability that would be expected for a trait measure (e.g., test–retest correlations across 1–4 months range from .73 to .86). In addition, the STAI has also demonstrated both convergent and discriminant validity (Spielberger et al., 1983).

The *Beck Depression Inventory* (BDI; Beck, Rush, Shaw, & Emery, 1979) is a 21-item instrument that broadly assesses the symptoms of depression, including the affective, cognitive, behavioral, somatic, and motivational components of depression as well as suicidal wishes. Items (e.g., “Sadness”) are rated on a 4-point scale (e.g., ranging from “I do not feel sad” to “I am so sad or unhappy that I can’t stand it”) and reflect a 1-week time period. Beck, Steer, and Garbin (1988b) conducted a meta-analytic study of the BDI and reported the mean coefficient alpha across 25 years of studies to be .86 in psychiatric populations and .81 in non-psychiatric populations.

Procedure

Students were given questionnaires to complete and return, including measures not related to the present study. Approximately 75% of students returned the questionnaires and were awarded partial course credit and then debriefed. Forty-seven participants met criteria for GAD according to the GAD-Q-IV and were subsequently included in the GAD analogue group (“GAD”). The remaining 491 participants served as controls.

Data analyses

Sets of *t*-tests (group: GAD, control) were conducted separately for measures of emotion regulation. Measures of emotion regulation were grouped according to the components of the emotion dysregulation model of GAD. Heightened intensity of emotions was measured by impulse strength and the other subscales of the BEQ. The Clarity subscale of the TMMS and both the Difficulty Identifying and Difficulty Describing Emotion subscales of the TAS-20 measured the poor understanding of emotions component. Negative reactivity to emotions was measured by the Attention subscale of the TMMS and the Fear of Anxiety, Depression, Anger, and Positive Emotion subscales of the ACS. Maladaptive Management of Emotions was measured by the

Mood Repair subscale of the TMMS. It was hypothesized that, compared to the Control group, the GAD group would report higher scores on all indices of heightened intensity of emotions, negative reactivity to emotions and poor understanding of emotions (with the exception of TMMS Clarity, on which they were expected to achieve lower scores), and lower scores on maladaptive management of emotions. Bonferroni correction was applied to the measures in each domain to control for alpha inflation (type I error). Cohen's (1988) effect sizes (d) were included for hypothesis-related univariate analyses to ensure that meaningful differences were not ignored due to sample size (type II error). A discriminant function analysis (DFA) was performed in which the measures of emotion were used to predict the presence of GAD. Emotion subscales that strongly predicted (r 's > .50) GAD in the DFA were converted to z -scores and summated. This composite emotion dysregulation variable was entered into a logistic regression to predict a diagnosis of GAD while simultaneously controlling for the contributions of worry (PSWQ), trait anxiety (STAI), and depression (BDI).

Results

Preliminary analyses

The GAD and control groups did not differ in mean age [$t(526) = -1.94, ns$], gender [$\chi^2(1, N = 538) = 1.42, ns$], ethnicity [$\chi^2(5, N = 538) = 4.62, ns$], or marital status [$\chi^2(4, N = 538) = 3.62, ns$]. Expectedly, the GAD group ($M = 54.05, SD = 11.43$) reported higher scores on the PSWQ than the control group [$M = 39.53, SD = 11.43; t(537) = 8.32, p < .001$]. Similarly, the GAD group ($M = 52.20, SD = 9.27$) scored significantly higher than the controls ($M = 40.32, SD = 10.06$) on the STAI [$t(527) = 7.71, p < .001$]. The GAD group ($M = 9.92, SD = 9.97$) also scored higher on the BDI than the controls ($M = 6.10, SD = 6.77; t(536) = 3.54, p < .001$). Table 1 displays correlations among the dependent measures used in the study.

Heightened intensity of emotions

Table 2 displays the mean differences between groups on the subscales pertaining to heightened intensity of emotions. Specifically, individuals in the GAD group reported higher levels of emotional intensity than the control group, as indicated by greater scores on the BEQ impulse strength subscale. They also reported significantly higher levels of BEQ negative emotional expressivity than the control group. These findings remained significant after Bonferroni correction ($p = .05/3 = .016$). The groups did not differ on the BEQ positive expressivity scale.

Poor understanding of emotions

Individuals in the GAD group were found to have significantly more difficulty identifying and describing emotions than did the control individuals, as reflected in scores on the TAS, and lesser skill in understanding their emotional experience, as evidenced by lower scores on the TMMS Clarity subscale (Table 2). These effects remained significant following Bonferroni correction ($p = .05/3 = .016$).

Negative reactivity to emotions

Negative reactivity to emotions was assessed by differences in attention towards emotions and the presence of negative beliefs about emotions. No differences were found on attention paid to

Table 1
Correlations among emotion and symptom measures in study 1

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
<i>Heightened intensity of emotions</i>															
1. BEQ impulse strength	—														
2. BEQ negative expressivity	.30**	—													
3. BEQ positive expressivity	.59**	.15**	—												
<i>Poor understanding of emotions</i>															
4. TMMS Clarity	-.10*	-.18**	.23**	—											
5. TAS difficulty identifying emotions	.22**	.28**	-.11**	-.63**	—										
6. TAS difficulty describing emotions	.05	.28**	-.21**	-.58**	.62**	—									
<i>Negative reactivity to emotions</i>															
7. TMMS attention to emotions	.40**	-.16**	.41**	.32**	-.23**	-.28**	—								
8. ACS fear of anxiety	.24**	.30**	-.06	-.49**	.59**	.39**	-.15**	—							
9. ACS fear of depression	.26**	.24**	-.18**	-.48**	.58**	.40**	-.23**	.67**	—						
10. ACS fear of anger	.15	.29**	-.13**	-.41**	.53**	.42**	-.26**	.56**	.64**	—					
11. ACS fear of positive emotion	.01	.20**	-.24**	-.48**	.50**	.41**	-.38**	.64**	.63**	.66**	—				
<i>Maladaptive management of emotions</i>															
12. TMMS mood repair	-.09*	-.15**	.26**	.55**	-.47**	-.34**	.33**	-.41**	-.55**	-.42**	-.39**	—			
<i>Symptom measures</i>															
13. PSWQ	.34**	.26**	.10*	-.32**	.37**	.27**	.02	.48**	.39**	.26**	.25**	-.32**	—		
14. STAI	.25**	.25**	-.16**	-.54**	.57**	.42**	-.21**	.61**	.68**	.50**	.54**	-.63**	.54**	—	
15. BDI	.23**	.22**	-.07	-.35**	.41**	.32**	-.12**	.36**	.45**	.29**	.35**	-.44**	.35**	.54**	—

Note: BEQ=Berkeley Expressivity Questionnaire; TMMS=Trait Meta Mood Scale; TAS=Toronto Alexithymia Scale-20 item version; ACS=Affective Control Scale; PSWQ=Penn State Worry Questionnaire; STAI=State-Trait Anxiety Inventory; BDI=Beck Depression Inventory. * $p < .05$. ** $p < .01$.

Table 2

Means (and standard deviations) of emotion measures in students with and without analogue GAD (study 1)

	Group		<i>t</i>	Cohen's <i>d</i>
	GAD, mean (<i>SD</i>)	Control, mean (<i>SD</i>)		
<i>Heightened sensitivity to emotions</i>				
BEQ impulse strength	5.17 (1.02)	4.56 (.96)	4.16**	.36
BEQ negative expressivity	4.34 (.59)	3.99 (.62)	3.66**	.32
BEQ positive expressivity	4.98 (.94)	4.89 (.95)	0.63	.05
<i>Poor understanding of emotions</i>				
TMMS clarity of emotions	2.85 (.57)	3.31 (.60)	−5.13**	.44
TAS difficulty identifying emotions	3.07 (.73)	2.34 (.74)	6.53**	.56
TAS difficulty describing emotions	3.17 (.84)	2.72 (.79)	3.69**	.32
<i>Negative reactivity to emotions</i>				
TMMS attention to emotions	3.65 (.63)	3.71 (.55)	−0.63	.05
ACS fear of anxiety	4.17 (.91)	3.36 (.83)	6.31**	.54
ACS fear of depression	4.12 (1.00)	3.13 (1.05)	6.21**	.54
ACS fear of anger	3.95 (.83)	3.52 (.85)	3.29**	.28
ACS fear of positive emotions	3.50 (.65)	3.11 (.77)	3.41**	.29
<i>Maladaptive management of emotions</i>				
TMMS mood repair	2.95 (.73)	3.42 (.69)	−4.46**	.38

Note: BEQ=Berkeley Expressivity Questionnaire; TMMS=Trait Meta Mood Scale; TAS=Toronto Alexithymia Scale-20 item version; ACS=Affective Control Scale. * $p < .05$, ** $p < .01$. *d* refers to Cohen's effect size; small effect = .20; medium effect = .50; large effect = .80.

emotions between GAD and control participants. Individuals with GAD endorsed a greater level of negative beliefs concerning activated emotions, including fears about the consequences of emotional experiences and the necessity of attempts to control these experiences. This was the case not only for anxiety but also for depression, anger, and positive emotions. These effects remained significant following Bonferroni correction ($p = .05/5 = .01$).

Maladaptive management of emotions

Control participants had significantly higher TMMS Mood Repair scores than the GAD participants, suggesting a relative deficit in the latter group's ability to return to a baseline emotional state once a negative mood has been experienced.

Emotion dysregulation and the prediction of GAD

To determine whether emotion dysregulation was an independent factor involved in GAD, a DFA was conducted. All of the subscales of the BEQ, TMMS, TAS, and the ACS were used to predict a GAD-Q-IV diagnosis in the full sample of 538 participants. The discriminant function was significant [Wilk's $\Lambda = .88$, $\chi^2(12, N = 538) = 66.64$, $p < .001$]. The linear combination of the emotion indices accurately classified 34 of 47 (72.3%) individuals with GAD. Similarly, 374 of 491 (76.2%) control individuals were correctly classified. TAS difficulty identifying emotions ($r = .77$), ACS Fear of Anxiety ($r = .74$), ACS Fear of Depression ($r = .73$), TMMS Clarity of Emotion ($r = -.60$), and TMMS Mood Repair ($r = -.52$) subscales were found to most strongly correlate

with the emotion dysregulation function (determined by r 's > .50), suggesting they may be particularly important in predicting GAD.

The DFA demonstrates that emotion indices can predict a diagnosis of GAD, but it does not parse the relative contribution of emotion dysregulation when compared to known correlates of GAD. In service of this type of analysis, we created z -scores for the subscales that most strongly predicted GAD in the DFA (r 's > .50, as above) and summated these z -scores into a composite emotion dysregulation variable. This variable was entered simultaneously with the PSWQ, STAI, and BDI into a logistic regression analysis predicting a GAD-Q-IV diagnosis. The overall model was found to be significant [$\chi^2(4, N = 538) = 71.36, p < .001$]. Specifically, the emotion dysregulation composite remained significant in the model ($B = .163, Wald(1) = 5.57, p < .02$), controlling for the PSWQ ($B = .049, Wald(1) = 9.04, p < .01$), the STAI ($B = .072, Wald(1) = 8.27, p < .01$), and the BDI ($B = -.013, Wald(1) = 1.25, ns$).

Summary and limitations

These results provide initial support for the association of emotion dysregulation to GAD. Individuals with self-reported GAD reported experiencing more intense moods and expressing negative, but not positive, moods more often. Individuals with GAD also reported greater deficits in their ability to understand their emotional experience including identifying emotions, describing them, differentiating them, and clarifying what motivational information emotions may convey. Further, although they did not report paying greater attention to their emotions, individuals with GAD reported greater negative reactivity to their emotions in that they feared the negative consequences of these experiences. This finding may be expected for anxiety given the wealth of research on anxiety sensitivity or fear of fear (see Taylor, 1999). However, individuals with GAD also had difficulty experiencing other emotions including depressed mood, anger and even positive emotions such as elation. Finally, individuals with GAD reported more difficulty believing that they would be able to soothe themselves following a negative mood than controls.

Emotion dysregulation was found to effectively predict GAD. More than three quarters of individuals with GAD were correctly identified. This finding is impressive given that no item on any of the emotion regulation scales measured symptoms associated with GAD. Further, this relationship remained significant after known correlates of GAD were controlled. Emotion dysregulation may have a unique relationship with GAD beyond trait anxiety, worry, or depression and may represent a psychopathological component beyond emotionality that is more representative of deficits in the generation, utilization, and management of emotional experience.

Although this study provides a positive first step to understanding the role of emotion dysregulation in GAD, some limitations preclude definitive conclusions. First, participants were college students, limiting generalizability to the greater community setting due to age, education, and other restrictions of range inherent in this type of sample. Further, they were diagnosed via self-report rather than by semi-structured diagnostic interview, the "gold-standard" for determining the presence of psychiatric diagnosis. Finally, the role of other psychiatric disorders was not directly examined. Comorbidity at the syndromal and symptomatic level are common in the emotional disorders (see Maser & Cloninger, 1990), and it may be expected that individuals who met self-reported criteria for GAD may also have met criteria for other emotional difficulties, which could also be responsible for a relationship with emotion regulation deficits. These

difficulties are addressed in the following study, which sought to demonstrate whether these patterns would emerge in a comparison between a clinical sample of patients with GAD and a community control sample.

Study 2: application to a clinical sample

A sample of treatment-seeking GAD patients was compared to a sample of individuals from the local community on the same indices of emotion dysregulation. However, the BEQ was not administered as part of the battery at the time of this study, so it was not included in this replication. As in study 1, it was hypothesized that individuals with GAD, compared to the control group, would have greater difficulty on the three dimensions of emotion dysregulation assessed. Further, it was hypothesized that the emotion dysregulation would again significantly predict the presence of GAD.

Method

Participants

The clinical sample consisted of 42 individuals (29 women; 69.5%) who presented for treatment of chronic worry and its associated difficulties at the Adult Anxiety Clinic of Temple University (AACT) and who met DSM-IV (APA, 1994) criteria for a principal diagnosis of GAD. Since we had an interest in determining the role of co-occurring disorders, comorbidity was allowed. The most common comorbid diagnoses included social anxiety disorder (36%), specific phobia (17%), and unipolar mood disorder (36%; including major depression, dysthymia, and mood disorder not otherwise specified). The control sample was comprised of 55 individuals (42% women) who were recruited through flyers and advertisements in local newspapers soliciting the paid participation of individuals who did not experience anxiety or depression. They could not meet criteria for any current or past year Axis I diagnosis, although 2 participants with mild specific phobias were included. Diagnoses were made with the Anxiety Disorders Interview Schedule for DSM-IV: Lifetime Version (ADIS-IV-L; DiNardo, Brown, & Barlow, 1994). In a sample of 362 patients, Brown, DiNardo, Lehmann, and Campbell (2001) found good inter-rater reliability for anxiety and mood disorders including GAD, which achieved a kappa of .67. Interviewers were trained according to the criteria specified by Brown et al. (2001) and highly experienced. Inter-rater agreement was assessed in the subset of the patient sample that later participated in a study evaluating the efficacy of cognitive-behavioral treatment for GAD. These 32 patients were also interviewed by an independent assessor who completed the GAD module of the ADIS-IV-L and other measures not included here. The diagnostic interviewer and independent assessor agreed on 100% of occasions on the presence of GAD. Scores on the ADIS-IV-L Clinician's Severity Rating, which assesses the severity of social anxiety symptoms and the degree of associated impairment, were also recorded, with 100% agreement within one rating point (range from 0 to 8).

Measures

The ACS, TAS, TMMS, PSWQ, BDI, and the *Beck Anxiety Inventory* (BAI; Beck, Epstein, Brown, & Steer, 1988a) were administered. The BAI assesses the presence and severity of anxiety

symptoms. It was specifically developed to focus on those symptoms that are distinct from depressive symptoms (Steer, Ranieri, Beck, & Clark, 1993) and emphasizes somatic symptoms and cognitions associated with anxiety and panic rather than emotional components of anxiety (e.g., feeling like a failure) that might overlap with depression. Its 21 items are rated on a 4-point scale and inquire about a 1-week period. The BAI has good internal consistency ($\alpha = .94$; Fydrich, Dowdall, & Chambless, 1992) and adequate concurrent validity with other measures of anxiety (Steer et al., 1993).

Procedure and data analyses

Individuals in the GAD group were assessed during their pre-treatment evaluation. Self-report measures and clinician-administered assessments not used in the present investigation were also administered at this time. Control individuals were brought into the AACT solely for the purpose of these assessments. The ADIS was administered prior to completion of the self-report battery. Data analyses were as in study 1.

Results

Preliminary analyses

GAD patients ($M = 33.88$, $SD = 12.86$) did not differ significantly from control participants ($M = 30.05$, $SD = 10.38$) in age [$t(95) = 1.62$, *ns*]. Both the GAD (80.0%) and control (73.2%) samples consisted primarily of Caucasian individuals [$\chi^2(4, N = 101) = 4.76$, *ns*]. Both GAD patients (73.8%) and control participants (75.9%) were predominantly single and never married [$\chi^2(4, N = 97) = 2.26$, *ns*]. However, the GAD group included a significantly higher percentage of women than the control group [$\chi^2(1, N = 97) = 7.10$, $p < .01$]. Further, gender was significantly associated with ACS fear of anxiety ($r = -.24$, $p < .05$), ACS fear of depression ($r = -.23$, $p < .05$) and TMMS Repair ($r = .24$, $p < .05$). As a result, analyses were conducted while controlling for gender. Analyses also controlled for comorbid diagnoses (given the high rate of comorbid diagnoses in the GAD group). In the comorbidity analyses, control subjects were included but were assigned automatically to the “no comorbidity” group. However, as results were similar in both gender and comorbidity analyses, the simpler uncontrolled analyses are presented here.

As expected, individuals with GAD ($M = 69.14$, $SD = 7.19$) scored significantly higher on the PSWQ than controls [$M = 32.22$, $SD = 8.84$; $t(95) = 22.16$, $p < .001$]. They ($M = 19.44$, $SD = 10.17$) also had higher BAI scores than controls [$M = 3.14$, $SD = 3.82$; $t(84) = 8.47$, $p < .001$] and higher BDI scores ($M = 14.56$, $SD = 8.19$) than controls [$M = 2.31$, $SD = 2.60$; $t(94) = 9.99$, $p < .001$]. Table 3 presents the correlations among all study measures.

Poor understanding of emotions

Table 4 displays scores on the three components of emotion regulation used in this study. Individuals in the GAD group, compared to controls, demonstrated elevated scores on the TAS Difficulty Identifying and Describing Emotions subscales. They also achieved lower scores on the

Table 3
Correlations among emotion and symptom measures in study 2

	1	2	3	4	5	6	7	8	9	10	11	12
<i>Poor understanding of emotions</i>												
1. TMMS clarity	—											
2. TAS difficulty identifying emotions	-.82**	—										
3. TAS difficulty describing emotions	-.63**	.72**	—									
<i>Negative reactivity to emotions</i>												
4. TMMS attention to emotions	.11	-.08	-.18	—								
5. ACS fear of anxiety	-.65**	.74**	.56**	.08	—							
6. ACS fear of depression	-.59**	.66**	.54**	.03	.75**	—						
7. ACS Fear of Anger	-.53**	.66**	.50**	-.06	.72**	.59**	—					
8. ACS fear of positive emotion	-.49**	.60**	.44**	-.15	.64**	.61**	.68**	—				
<i>Maladaptive management of emotions</i>												
9. TMMS mood repair	.59**	-.61**	-.49	.00	-.61**	-.69**	-.47**	-.45**	—			
<i>Symptom measures</i>												
10. PSWQ	-.56**	.59**	.43**	.22*	.80**	.65**	.57**	.48**	-.61**	—		
11. BAI	-.52**	.58**	.42**	.14	.76**	.61**	.54**	.44**	-.57**	.73**	—	
12. BDI	-.55**	.60**	.41**	.16	.67**	.62**	.53**	.42**	-.68**	.75**	.76**	—

Note: BEQ=Berkeley Expressivity Questionnaire; TMMS=Trait Meta Mood Scale; TAS=Toronto Alexithymia Scale-20 item version; ACS=Affective Control Scale; PSWQ=Penn State Worry Questionnaire; BAI=Beck Anxiety Inventory; BDI=Beck Depression Inventory. * $p < .05$. ** $p < .01$.

Table 4

Means (and standard deviations) of emotion measures in patients with GAD and control participants (study 2)

	Group		<i>t</i>	Cohen's <i>d</i>
	GAD, mean (<i>SD</i>)	Control, mean (<i>SD</i>)		
<i>Poor understanding of emotions</i>				
TMMS clarity of emotions	2.85 (.57)	3.31 (.60)	−6.30**	1.29
TAS difficulty identifying emotions	2.81 (.96)	1.70 (.65)	6.45**	1.56
TAS difficulty describing emotions	2.67 (.93)	2.01 (.70)	3.81**	0.89
<i>Negative reactivity to emotions</i>				
TMMS attention to emotions	4.00 (.50)	3.70 (.64)	2.42*	.50
ACS fear of anxiety	4.52 (.96)	2.39 (.91)	11.20**	2.30
ACS fear of depression	4.11 (1.22)	2.36 (1.02)	7.70**	1.58
ACS fear of anger	4.08 (1.11)	2.85 (.90)	6.04**	1.24
ACS fear of positive emotions	3.27 (.98)	2.37 (.85)	4.84**	0.99
<i>Maladaptive management of emotions</i>				
TMMS mood repair	2.95 (.91)	4.07 (.66)	−6.76**	1.60

Note: TMMS = Trait Meta Mood Scale; TAS = Toronto Alexithymia Scale-20 item version; ACS = Affective Control Scale. * $p < .05$, ** $p < .01$. *d* refers to Cohen's effect size; small effect = .20; medium effect = .50; large effect = .80.

TMMS Clarity of Emotions subscale than controls. These results remained significant after Bonferroni correction ($p = .05/3 = .016$).

Negative reactivity to emotions

GAD patients also reported more negative beliefs about emotional experiences than controls, as reflected in elevated scores on all subscales of the ACS (all large effects). GAD patients also reported greater awareness of their emotions on the TMMS Attention subscale (demonstrating a moderate effect size). However, unlike the findings for the ACS subscales, the finding for TMMS Attention did not remain significant after Bonferroni correction ($p = .05/5 = .01$).

Maladaptive management of emotions

Finally, patients with GAD also reported less ability to recover from a negative mood than controls on the TMMS Mood Repair subscale (large effect size).

Emotion dysregulation and the prediction of GAD

The TMMS, TAS, and ACS subscales were utilized to generate a discriminant function. This linear combination of predictors significantly predicted the presence of GAD [Wilk's $\Lambda = .36$, $\chi^2(9, N = 101) = 92.58$, $p < .001$]. Using the emotion dysregulation function, 92.9% of individuals with GAD were correctly classified. Similarly, 89.1% of controls were classified correctly by this function. ACS Fear of Anxiety ($r = .86$), ACS Fear of Depression ($r = .59$), TMMS Mood Repair ($r = -.54$), and TAS Difficulty Identifying Emotions ($r = .52$) emerged as the subscales that had

the greatest correlations with the discriminant function (determined by $r^2s > .50$). Given high correlations among the emotion and psychopathology variables (all $r^2s > .60$; see Table 3), we were unable to perform a logistic regression analysis to examine the unique contribution of emotion to GAD beyond other indices of psychopathology (i.e., BDI, BAI, PSWQ). Multicollinearity among predictors is a violation of the assumptions of logistic regression (Tabachnick & Fidell, 2001).

Summary and limitations

Similar to study 1, patients with GAD had greater difficulty understanding, reacting to, and managing their emotional experiences than individuals from the community. In particular, patients with GAD reported poorer ability to identify, describe, and comprehend the motivational value of their emotions than did control participants. Further, as in study 1, individuals with GAD, compared to controls, reported greater fear of the perceived negative consequences of anxiety, depression, anger, and positive emotions. They also had greater difficulties returning to a euthymic state following a negative mood when compared to controls, suggesting poor management of emotional responses. After Bonferroni correction, however, patients with GAD (like the students with GAD in study 1) did not report greater attention to emotions than controls. Thus, attention to emotions may not be an integral variable in the emotion dysregulation model of GAD. Alternatively, it may be that attentional allocation to one's emotional state may not be consistent. Pulling attention away from one's emotions may at times be adaptive. Other situations may require closer attention to emotions. As such, contextual factors that may determine the adaptability of attention to emotions need to be further studied. Indices of emotion dysregulation remained significantly related to GAD after controlling for the effects of gender and comorbid disorders, suggesting that these findings are not an artifact of the higher percentage of women in the study or the presence of other disorders in the GAD group. Indeed, GAD patients have often been found to have high levels of comorbidity (Wittchen et al., 1994). However, recent epidemiological investigations have demonstrated that GAD has unique associated dysfunction that is not accounted for by this comorbidity (Kessler, DuPont, Berglund, & Wittchen, 1999). Finally, emotion dysregulation was found to strongly differentiate the GAD patient sample from the community control group. However, given violations to the assumptions of logistic regression, we were unable to examine the unique contributions of emotion dysfunction beyond the PSWQ, BDI, and the BAI. Multicollinearity is likely a result of a bimodal sample where participants were either "psychopathological" or "normal".

One major difficulty with Studies 1 and 2 is the reliance on beliefs about one's own characteristic behaviors. It is difficult to determine the extent to which beliefs about one's ability to regulate emotions correlate with the actual ability to do so (Petrides & Furnham, 2000; Roberts, Zeidner, & Matthews, 2001). It is important to examine reactions to an induced mood to better understand the nature of emotion dysregulation in individuals with GAD. Further, in testing our model of GAD, it is also important to begin to delineate the mechanisms involved in this process. In particular, it would be important to determine if individuals with GAD do indeed have more aversive emotional experiences in response to a given negative mood than other individuals. study 3 addresses these concerns.

Study 3: emotion dysregulation in response to induced negative mood

This study sought to experimentally investigate whether elicitation of emotion would lead to increases in reported somatic distress and subsequent difficulties in understanding, accepting, and managing this emotional experience in individuals with GAD compared to controls. The purpose of this study was twofold. First, by experimentally inducing negative emotions, negative reactions and dysregulatory responses would concern a specific episode of mood rather than asking individuals to reflect on the myriad of emotional episodes they encounter. Second, the study was a preliminary investigation of the relationship between components of the model. In particular, a goal was to determine whether heightened emotional intensity indeed leads to high levels of negative reactivity and poor understanding and management of emotions. To test whether the experience of emotion elicits these regulatory deficits, musical mood induction procedures were utilized. Music has been described as a strong elicitor of emotions (Gaver & Mandler, 1987; Staats & Eifert, 1990). In addition, musical mood induction procedures have been utilized extensively to elicit both negative and positive moods (for a review, see Martin, 1990). As shown above, the experience of many emotions may be distressing for individuals with GAD. Further, physiological differences between emotions are often difficult to determine (Levenson, 1992). Therefore, both anxiety-inducing and sadness-inducing musical pieces were utilized and compared with neutral music. Prior to mood induction, measures of subjective mood, somatic anxiety, and depression were administered. These measures were re-administered along with a state measure of emotional intelligence that focuses on the ability to understand, recognize, accept and manage a given mood following the mood induction (Mayer & Stevens, 1994).

It was hypothesized that (1) for GAD participants, no differences between anxious and sad mood inductions would occur in changes in self-reported physiological anxiety symptoms (because of the non-specific negative reactivity of individuals with GAD demonstrated in studies 1 and 2); (2) GAD participants who underwent a negative mood (i.e., anxious or sad) induction would report greater increases in physiological symptoms of anxiety than GAD participants who underwent a neutral mood induction; (3) GAD participants would report a greater increase in physiological symptoms of anxiety in response to the negative mood induction (compared to the neutral mood induction) than control participants; and (4) GAD participants would report less adaptive emotion understanding, tolerance, and management than control participants following negative mood induction (i.e., anxious or sad).

Method

Overview of design

The study was conducted in a 2 (Group: GAD Analogue, Control) \times 3 (Mood Induction: Anxious, Sad, Neutral) \times 2 (Time: Pre-Induction, Post-Induction) mixed factorial design with repeated measurement on the third factor. Participants were assigned to groups based on prior screening, randomly assigned to mood induction condition, and assessed on measures of subjective mood and somatic anxiety before and after the experimental induction. Further reactions to induced mood were assessed following the mood induction.

Participants

Eighty-eight participants were selected from introductory psychology subject pool at Temple University ($N = 865$). In addition, 34 participants were recruited from upper level psychology classes. Participants were selected on the basis of scores on the GAD-Q-IV (Newman et al., 2001, 2002). However, recent evidence suggests that individuals with high levels of worry (but not GAD) may have their own distinct psychopathological presentation, albeit to a less severe degree than individuals with GAD (Ruscio, 2002). Therefore, more stringent criteria for inclusion were used. To be considered for inclusion in the “GAD group”, students also had to score above the median on the PSWQ. Similarly, individuals in the control group were required to score below the PSWQ median to ensure that they would not be extreme worriers who actually had the disorder but were not detected by the GAD-Q-IV. Sixty-one students who met criteria for GAD on the GAD-Q-IV and who scored above the median of 46 on the PSWQ were invited to participate as the “GAD analogue” group. Sixty-one students did not meet GAD criteria and scored below the median of 46 on the PSWQ and were invited to participate as the normal control group. Eligible students were scheduled for participation by a research assistant who did not serve as an experimenter during the formal experiment. Further, experimenters were not informed about a participant’s group classification.

Materials

Mood induction. Three pieces of music were recorded on separate compact discs and played for participants on a Sony Portable Compact Disc Player with Koss headphones. Participants were told what emotion they would be expected to feel and were instructed to imagine themselves in that mood. Hence, all mood induction conditions contained an instructed imaginal component (see Martin, 1990, for a discussion of this approach). Priming the participant to experience the desired mood is a standard practice in the mood induction literature (e.g., Segal, Gemar, & Williams, 1999).

Anxious mood: Participants in the “anxious mood” condition listened to an 8-min clip of the 28-min “Erwartung [Anticipation]” composed by Arnold Schoenberg (1909/1996). Previous research has suggested that this piece can induce anxious mood in undergraduates (Blagden & Craske, 1996; Slyker & McNally, 1991). An effect size (Cohen’s $f = .56$) based on these previous studies suggests that it can effectively induce anxious mood.

Sad mood: Participants in the “sad mood” condition listened to “Russia under the Mongolian Yolk”, composed by Sergei Prokofiev (1934/1987). This piece was remastered at half-speed and runs for 8 min. This piece, played at half-speed, has been shown to be very effective in inducing a negative or depressed mood (e.g., Segal et al., 1999). A meta-analysis of musical mood induction procedures found a good effect size (Cohen’s $f = .56$) for sad mood inductions including, but not limited to, this Prokofiev piece played at half-speed (Westermann, Spies, Stahl, & Hesse, 1996).

Neutral mood: Tchaikovsky’s “Waltz of the Flowers” (7 min) from *The Nutcracker Suite* (Tchaikovsky, 1892/1997) was used to provide a neutral mood condition. All study participants heard a 3-min clip of this piece before listening to their randomly assigned mood induction music. However, participants in the neutral mood condition heard the selection in its entirety following the 3-min clip. This piece of music had not been tested for neutrality of mood in previous research. Therefore, a pilot study was conducted with 10 undergraduate and graduate students at Temple University. There was no significant effect for either anxious [$t(9) = 2.04$, *ns*] or depressive

[$t(9) = 2.04, ns$] mood as measured before and after mood induction by the Multiple Affect Adjective Checklist—Revised (MAACL-R; Zuckerman & Lubin, 1985).

Measures. *Beck Depression Inventory:* The BDI was administered in the beginning of the study to screen for clinically significant dysphoria. Two participants demonstrated severe depressive symptomatology (as evidenced by a total score of 25 or above on the BDI; Beck et al., 1988a,b) and were excluded.

MAACL-R (Zuckerman & Lubin, 1985): The MAACL-R is a 132-item adjective checklist of current moods that is commonly used as a manipulation check in mood induction studies. The MAACL-R consists of five unipolar scales—Anxiety, Depression, Hostility, Positive Affect, and Sensation Seeking. It has been shown to have high internal consistency and good convergent and discriminant validity (Lubin, Whitlock, & Zuckerman, 1998). A number of studies have detected change in response to musical mood induction procedures using the MAACL-R (e.g., Blagden & Craske, 1996; Segal et al., 1999). The Anxiety (10 words) and Depression (12 words) subscales of the “Today” form of the MAACL-R were used in the current study to rate levels of state affect. For the purposes of this study, the MAACL-R was modified (from a checked item procedure) to be rated on a 5-point scale (from “right now I feel very much like this” [1] to “right now I feel not at all like this” [5]). This alteration was meant to increase the sensitivity of the MAACL-R to changes in state affect. Further, it has been suggested that checklists are less reliable than dimensionally rated scales due to the tendency for raters to “over-check” items (Herron, 1969).

Mood and Anxiety Symptom Questionnaire: The Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991) is a 77-item measure assessing symptoms that commonly occur in the mood and anxiety disorders. The MASQ consists of five subscales: General Distress: Anxious Symptoms (GDA), General Distress: Depressive Symptoms, General Distress: Mixed Symptoms (GDM), Anxious Arousal, and Anhedonic Depression. Since GAD is most typically characterized by generalized anxiety and mood symptoms rather than panic-related anxious arousal symptoms, the GDA and GDM subscales were used to assess changes in physiological symptoms. The GDA subscale includes 11 items that are indicators of anxious mood (e.g., “Had an upset stomach”). The GDM subscale contains 15 items that are common in both mood and anxiety disorder symptom criteria (e.g., “Felt restless”). For the purposes of this study, a 15-item version of the scale (MASQ-GD) that reflects physiological symptoms related to GAD, comprised of the GDA subscale and four items from the GDM subscale (those most relevant to GAD: fatigue, difficulty concentrating, insomnia, and irritability) was administered to assess change in reported physiological symptoms in response to the mood induction. Items were reworded to reflect state change in arousal. For example, “Felt Faint” became “Feel Faint”. Each item is rated on a 1 (“not at all”) to 5 (“extremely”) scale. Watson et al. (1995) reported high levels of internal consistency for the GDA and GDM subscales in student (all α 's greater than .78), adult (all α 's greater than .85), and patient (all α 's greater than .86) samples.

State Meta-Mood Scale: The State Meta-Mood Scale (SMMS; Mayer & Gaschke, 1988; Mayer & Stevens, 1994) is a state measure of degree of monitoring, evaluating, and regulating of one's current mood. It is comprised of two scales: the Meta-Evaluation Scale (MES; 24 items) and the Meta-Regulation Scale (15 items; not administered in this study). The MES includes four factor-analytically derived subscales including belief about the control and influence of the mood (“This mood will never change” [reverse-worded]), its clarity (“I know exactly how I'm feeling”),

acceptability (“There’s nothing wrong with it”), and typicality (“I feel this mood often”). The SMMS is scored on a 5-point scale ranging from “Definitely does not describe my mood” (1) to “Definitely does describe my mood” (5). Internal consistency is good (.75–.87), and correlations among subscales are low (all r ’s = .05–.25; Mayer & Stevens, 1994). Further, Mayer and Stevens (1994) demonstrated convergent validity of the MES with other measures of mood evaluation.

Procedure

All participants were run through the study procedures individually. Upon arrival at the experimental setting, participants were given a brief introduction to the study and a consent form. On signing the consent form, they completed the BDI and the MASQ-GD. Participants then listened to the 3-min neutral mood clip to neutralize any negative mood-inducing effects from the completion of questionnaires. Thereafter, participants completed the MAACL-R. Participants were randomly assigned to sad, anxious, or neutral mood induction conditions. Participants were asked to imagine themselves in the proper mood and then listened to the appropriate musical piece. Following the musical mood induction procedure, the participant completed a questionnaire battery that contained the MAACL-R, MASQ-GD and the SMMS.

Data analysis

t - and χ^2 tests were utilized for 1-factor independent-sample comparisons (for preliminary analyses and hypothesis 4). For multiple factors, mixed univariate analyses of variance (ANOVAs) were conducted (hypotheses 2–4). Given a priori directional hypotheses, differences between induction conditions (i.e., neutral, sad, anxious) in the GAD or control participants were examined using planned contrasts. For hypothesis 1 (anxious and sad mood inductions would not differ in the GAD group), planned orthogonal contrasts were used to test differences in dependent measures between participants who received the anxious versus the sad inductions across time (and within groups). If differences were not found (as predicted), these groups (GAD participants in the sad and anxious mood conditions) were pooled in a planned orthogonal contrast comparing this composite negative mood induction GAD group to the neutral induction GAD group on the dependent measures across time (hypothesis 2). In addition, the comparison between negative and neutral mood inductions was also conducted in the control group. Finally, planned contrasts were utilized to compare the difference between negative mood and neutral mood induction in the GAD group to that observed in the control group (hypothesis 3). For multiple comparisons utilizing subscales of the same measure, Bonferroni corrections were applied. For hypothesis-related analyses, Cohen’s (1988) effect sizes are presented. Cohen’s d was utilized for t -tests (small effect = .20, medium effect = .50, large effect = .80) and Cohen’s f was utilized for F -tests (small effect = .10, medium effect = .25, large effect = .40).

Results

Preliminary analyses

No differences in age or years in college were found between groups or mood induction conditions (all F ’s < 2.85). No differences in race were found between GAD and control groups [χ^2 (3, N = 93) = 1.72, *ns*] or as a function of mood induction condition [χ^2 (6, N = 93) = 8.89, *ns*]. The

GAD and the control groups differed in gender distribution, with more females in the GAD group (69%) than the control group [41%; $\chi^2(1, N=108)=5.87, p<.05$]. Although gender distinguished the groups, it was not related to the dependent measures and was therefore not controlled in the following analyses. No gender difference in mood induction conditions was found [$\chi^2(2, N=108)=3.47, ns$]. Finally, the GAD group ($M=12.38, SD=7.91$) had higher BDI scores than did the normal control group [$M=6.78, SD=4.53; t(119)=-4.76, p<.001$]. However, mood induction conditions [$F(2, 114)=-1.48, ns$] did not differ on the BDI.

Paired *t*-tests with Bonferroni corrections ($p = .05/6 = .008$) were used to determine if the index negative mood induction procedure truly raised the targeted negative emotion, as well as the non-targeted negative emotion, in each mood induction (manipulation check). Both MAACL-R anxiety [$t(37)=-7.08, p<.001, d=2.33$] and depression [$t(37)=-2.96, p<.008, d=.97$] scores were significantly raised by the anxious mood induction. Similarly, both MAACL-R anxiety [$t(37)=-4.82, p<.001, d=1.59$] and depression [$t(37)=-4.71, p<.001, d=1.55$] scores were raised as a result of the sad mood induction. However, neither MAACL-R anxiety [$t(37)=-1.84, ns, d=.61$] nor depression [$t(37)=-1.29, ns, d=.42$] scores were raised as a result of the neutral mood induction.

Pre-post mood induction analyses

According to a planned orthogonal contrast, GAD participants who received the anxious mood induction did not differ from those who received the sad mood induction on changes in pre- to post-induction MASQ-GD scores [$F(1, 110)=.00, ns, f=.00$] (hypothesis 1). Subsequently, GAD participants in the anxious and sad mood inductions were pooled into a negative mood induction condition and compared to participants in the neutral mood induction condition by planned orthogonal contrast (hypothesis 2). GAD participants who received a negative mood induction demonstrated significantly greater increases in pre- ($M=28.24, SD=1.46$) to post-induction ($M=32.29, SD=1.62$) MASQ-GD scores than GAD participants who received the neutral mood induction [pre-induction $M=27.94, SD=2.14$; post-induction $M=22.94, SD=2.37$; $F(1, 110)=15.18, p<.001, f=.74$].

Within the control group, participants who received the negative mood induction [pre-induction $M=21.77, SD=1.44$; post-induction $M=23.15, SD=1.60$] did not differ from participants who received the neutral mood induction on pre- to post-induction changes in MASQ-GD scores [pre-induction $M=22.48, SD=1.98$; post-induction $M=21.67, SD=2.20$; $F(1, 110)=.67, ns, f=.16$].

The degree of change on the MASQ-GD from before to after the negative versus neutral mood induction for the GAD and normal control groups was then examined (hypothesis 3). The difference in MASQ-GD scores in the negative mood induction versus the neutral mood induction was greater in the GAD group than the normal control group [$F(1, 110)=4.56, p<.05, f=.41$].

Emotion dysregulation following induction

A series of *t*-tests was used to examine responses to the negative mood induction (pooled anxious and sad inductions) between groups on the SMMS-MES subscales (hypothesis 4). As displayed in Table 5, the GAD group scored lower than the control group following negative mood induction on the SMMS-MES acceptance and influence subscales. A non-significant trend was also demonstrated for SMMS-MES clarity. However, this effect was not demonstrated for

Table 5

Means (and standard deviations) of state-level emotion measures following negative mood inductions (anxious + sad) in individuals with and without Analogue GAD (study 3)

	Group		<i>T</i>	Cohen's <i>d</i>
	GAD, mean (<i>SD</i>)	Control (<i>SD</i>)		
SMMS acceptance of emotions	18.87 (4.38)	21.56 (4.91)	−2.52*	.59
SMMS clarity of emotions	20.42 (5.39)	22.54 (5.29)	−1.73 [†]	.40
SMMS influence on emotions	17.34 (6.43)	21.26 (7.07)	−2.58*	.60
SMMS typicality of emotions	21.84 (3.61)	21.28 (2.90)	0.79	.19

Note: SMMS = State Meta Mood Scale. * $p < .05$, [†] $p < .10$. *d* refers to Cohen's effect size; small effect = .20; medium effect = .50; large effect = .80.

SMMS-MES typicality. When applying a Bonferroni correction ($p = .05/4 = .0125$), differences in SMMS-MES acceptance and influence remained significant.

Summary and limitations

A major aim of study 3 was to demonstrate that elicited negative emotion would significantly impact subsequent levels of self-reported physiological symptoms among persons with (analogue) GAD compared to normal control participants who would not demonstrate this effect. Indeed, GAD participants were found to have greater levels of self-reported physiological anxiety symptoms in reaction to the negative mood inductions (either anxious or sad) to a greater degree than did the normal control participants. It was also hypothesized that, in addition to an increase in self-reported physiological anxiety symptoms, GAD participants would have more difficulty evaluating and managing their induced negative emotions than would control participants. This hypothesis was supported. Specifically, GAD participants, following a negative mood induction, had more difficulty accepting their emotions and believed that they had less influence over their course than did control participants. Although a non-significant trend (but moderate effect size), GAD participants were also found to have slightly more difficulty understanding (emotional clarity) their emotional state than did control participants. However, neither group reported the resulting mood as more typical than the other, suggesting the prior significant effects were not simply due to differences in familiarity with negative mood but rather individuals with GAD react to emotions in a maladaptive manner that may function to maintain this negative state.

The notion that individuals with GAD would have difficulty in managing specific emotional states is consistent with the findings of Studies 1 and 2 that individuals with GAD had more difficulty understanding, reacting to, and managing their emotions when compared to control individuals. Miranda, Gross, Persons, and Hahn (1998) found that participants with a history of depression reported increases in dysfunctional attitudes following a commonly used sad mood-inducing film (Gross & Levenson, 1995). However, in that study, participants without a history of depression who reported induced negative mood were found to have *decreases* in dysfunctional attitudes. The authors interpreted these findings in terms of ability to regulate emotions. They suggested that participants without a history of depression may have more adaptive responses or compensatory skills at their disposal that allow them to overcome their negative mood. These

skills may be absent in participants with a history of depression. Similarly, findings from study 3 suggest that individuals with GAD find it difficult to manage their self-reported physiological reactions to negative emotions. This aversive reaction to a negative mood may also motivate individuals with GAD to invoke the worry process as a means to escape this distressing state.

A number of limitations in the design of the present study need to be considered when interpreting the mood induction findings. When examining constructs such as emotions, physiological symptoms, and emotion regulation, it becomes difficult to determine if participants are able to identify and differentiate these processes (Petrides & Furnham, 2000). As such, it is unclear whether the reported increases in physiological symptoms indeed represent physiological amplification, elevated attention to such changes, or just a perceived change without actual physical change. More objective assessment would be preferable. For instance, in addition to self-report, physiological activity may need to be directly measured (McLeod, Hoehn-Saric, & Stefan, 1986; Turpin, 1991). Further, emotion dysregulation may be better assessed by observer-coded measures (see Westen, Muderrisoglu, Fowler, Shedler, & Koren, 1997) or ability-based tests (see Mayer et al., 2001).

Also problematic was the active nature of the neutral mood induction (i.e., Tschaikovsky's "Waltz of the Flowers"), which had not been used before this study. Examination of mean changes for self-reported physiological anxiety symptoms revealed *decreases* from pre- to post-induction for the GAD participants. Indeed, anecdotally, many participants reported to the experimenters that this musical piece "calmed [them]." Thus, the neutral mood induction may have been an active condition (inducing positive mood) rather than merely a control condition. A control task that does not have active effects on mood would have been preferable. Nonetheless, only the GAD group had an elated response to the neutral mood induction, supporting the notion that these individuals are more emotionally reactive.

Both targeted and non-targeted emotions were raised by the sad mood induction and, to a lesser extent, the anxious mood induction. This suggests that pure states of emotion may be difficult to induce in experimental settings. However, emotions rarely occur in pure forms, and efforts to obtain such a pure induction may have poor ecological validity. Finally, participants were sampled from an undergraduate population. Individuals with GAD met this diagnosis according to self-report. It will be important to examine mood effects in a clinical sample of individuals who meet structured diagnostic interview criteria for GAD compared to a community control sample.

General discussion

This series of studies provides initial evidence for the presence of emotion dysregulation in individuals with GAD. Specifically, individuals who met criteria for GAD reported greater intensity of emotional experience and a greater tendency to express negative emotions than control individuals. Further, they also displayed marked difficulties in their ability to identify, describe, and clarify their emotional experiences. Individuals with GAD also displayed greater negative reactivity than control individuals, as demonstrated in greater endorsement of catastrophic beliefs concerning the consequences of emotions (including anxiety, sadness, anger, and positive emotions). Ability to soothe one's self when experiencing negative emotions was also found to be impaired in individuals with GAD, compared to the control group. Furthermore, a

composite emotion dysregulation score significantly predicted the presence of GAD when controlling for worry, trait anxiety and depression. These findings were largely replicated in a clinical sample of persons with DSM-IV GAD and community controls. Furthermore, persons who met criteria for GAD by self-report responded differently to a negative mood induction than persons without GAD. Specifically, after listening to sadness- or anxiety-inducing music, GAD participants, but not controls, displayed greater increases in self-reported physiological symptoms of anxiety than was evident after a neutral mood induction. Further, in response to the mood induction, GAD participants had more difficulty accepting, influencing, and, to a lesser degree, understanding their emotional reactions when compared to normal control individuals.

A small number of other studies have also examined emotion dysregulation in individuals with GAD and have demonstrated similar findings. As in Studies 1 and 2, Borkovec and colleagues examined responses to the TAS (Abel, 1994; Yamas, Hazlett-Stevens, & Borkovec, 1997). College students with self-reported GAD endorsed greater difficulty identifying and describing emotions than control participants. A recent study by Roemer, Salters, Raffa, and Orsillo (in press) examined the ability of the ACS and the Action and Acceptance Questionnaire (Hayes et al., 2002), a measure of experiential avoidance to predict the severity of GAD (while controlling for worry). Using both college and clinical samples, Roemer et al. (in press) found that both experiential avoidance and the fear of emotional experiences predicted the severity of GAD above and beyond worry. Fear of anxiety emerged as the strongest predictor of severity when compared to fear of depression, anger, and positive emotions. These results provide convergent evidence for the role of emotion dysregulation in GAD.

Although emotion dysregulation was found to be highly (and uniquely) sensitive in predicting a diagnosis of GAD, its specificity in distinguishing this condition from others was not examined. Indeed, a number of investigations of other types of psychopathology have also found characteristic deficits in the regulation of emotion. Other anxiety disorders (e.g., panic disorder, social anxiety disorder) have been shown to be associated with “alexithymic” characteristics (Cox, Swinson, Shulman, & Bourdeau, 1995; Parker, Taylor, Bagby, & Acklin, 1993) and fear of emotions (Berg et al., 1998). A number of studies have emerged that demonstrate constricted and avoidant emotional responses in individuals with PTSD (e.g., Roemer, Litz, Orsillo, & Wagner, 2001). Further, other disorders have also been found to be characterized by emotion dysregulation such as depression (Flett, Blankstein, & Obertynski, 1996; Rottenberg et al., 2002) eating disorders (Westen & Harnden-Fischer, 2001) and borderline personality disorder (Wagner & Linehan, 1999).

These findings suggest that emotion dysregulation may play a role in a number of disorders. It may be that these broad categorizations apply to many forms of dysfunction but that more specific characteristics of emotion dysregulation will be more related to specific disorders (Kring, 2001). For instance, a recent investigation suggests that certain aspects of the first component of our model, heightened intensity of emotions, differentiate GAD from social anxiety disorder (Turk, Heimberg, Luterek, Mennin, & Fresco, in press). Clearly, further studies are needed to determine the relationship between emotion dysregulation and specific disorders. In particular, studies that compare emotion regulation deficits in GAD to those in other anxiety disorders are vital to determine which aspects of this dysregulation are specific to GAD and which may be more nonspecific factors of all anxious psychopathology. It may also be that, as mentioned above, our current instruments for the assessment of emotion regulation deficits are not refined enough to

detect differences between different forms of psychopathology. In the present investigations, all measurements utilized a self-report format. As discussed above, self-report methodology is problematic when assessing emotional factors. Participant's self-report responses often may reflect their inability to judge their own emotional functioning and may thus be subject to inflations in scoring due to a general level of self-doubt rather than a specific deficit in emotion regulation ability. Owing to these problems inherent in self-report measurement of emotion, observational and skill-based assessments (e.g., MSCEIT; Mayer, Salovey, Caruso, & Sitarenios, 2003) may be better able to distinguish between different forms and components of emotion regulation deficits such that their relationship to different disorders could be delineated.

It will be vital to examine the relationship among the individual components of the emotion dysregulation model. Although the present investigation provided evidence for the presence of all four components of the model (i.e., heightened intensity of emotions, poor understanding of emotions, negative reactivity to emotions, and maladaptive management of emotions), it did not explicitly examine the causal links among them. As stated above, a rapid, temporal, causal chain is hypothesized to link these components, it will be important to determine if there is a linear relationship or whether there may be multidirectional connections between components. For instance, could maladaptive management of emotions directly raise negative reactivity to emotions without increasing emotional intensity? It is likely that a feedback loop exists wherein increasingly maladaptive emotional management leads back to heightened intensity. However, as Ochsner and Feldman-Barrett (2001) note, emotional regulatory acts typically lead to the generation of new responses. Further, changes in labeling emotions would also be expected to change the course of emotional events. This is consistent with the theoretical approach of Hayes et al. (1999), who argue that emotional avoidance not only results from emotional intensity but can cause it as well.

Worry is the central component of GAD. The present investigations were conducted as a preliminary investigation of our model of GAD that builds upon Borkovec's avoidance theory of worry. Indeed, we did find emotion dysregulation to be characteristic of GAD. However, the present investigations did not directly examine the relationship between emotion dysregulation and the worry process. In study 3, it was suggested that the strong physiological reactions of individuals with GAD might have made it more difficult for them to withdraw from a negative mood. We did not directly examine the role that increased physiological symptoms may play in the worry cycle. There is substantial evidence (see Borkovec et al., 2004) to suggest that worry functions to constrict symptoms of autonomic arousal. However, it may also be the case that the experience of this hyperarousal is what cues worry itself. This may suggest a cyclical process involving both physiological arousal and worry. Further, although speculative, increases in self-reported physiological symptoms and difficulties in emotion regulation in the GAD participants following the induction may be evidence for a possible mediational model. It may be that individuals with GAD have a generalized sensitivity to emotional experience in that they find these experiences aversively arousing. They may react to arousal-inducing emotional experiences with anxiety (as evidenced by greater physiological activation). Further, they may find these anxious reactions difficult to manage and, possibly in conjunction with the belief that worry is an effective tool for escaping the aversive emotional state (see Startup & Davey, 2001), begin to use worry as a method to escape the threatening emotional experience, which may lead to constriction of physiological hyperreactivity. Such mediational approaches would more directly test the

relationship between components of the model. Preexisting traits (such as heightened emotional intensity) may predispose one to a given mood state, which in turn would influence emotion processing tendencies (including regulation strategies; [Rusting, 1998](#)).

Further studies are clearly necessary to delineate and expand upon the components of this emotion dysregulation perspective. As mentioned above, studies utilizing more objective approaches that examine the relationship among components of the model in a number of disorders would further our understanding of an emotion dysregulation perspective on psychopathology. It will also be important to determine whether emotion regulation deficits have predictive value for outcome related to GAD. For example, a recent investigation demonstrated that emotion dysregulation mediated the effect of pre-existing GAD on psychological distress in the year following the terrorist attacks of 9/11 in a sample of New York University students directly affected by the event ([Mennin, Alliger, Smith, & Mandelbaum, 2002](#)). Predictive validity can be further established through the ability of emotion dysregulation to predict outcome of treatment for GAD. Findings of all 3 current studies may suggest that an emotion dysregulation perspective may have implications for the treatment of GAD. The authors are currently developing and testing an integrated approach to treatment (for an introduction to this treatment approach and a case study, see [Mennin, 2004](#)). Although novel, this treatment approach builds upon the efficacy of currently available cognitive-behavioral treatments ([Gould, Safren, O'Neill Washington, & Otto, 2004](#)). Other investigators have also developed such integrative treatments that target related components of GAD such as interpersonal deficits and non-acceptance ([Newman, Castonguay, Borkovec, & Molnar, 2004](#); [Roemer & Orsillo, 2002](#)).

On a more general level, results from the present investigation provide preliminary support for the importance of emotion variables in both psychopathological processes and treatment. However, by stressing the importance of emotion variables, it is not suggested that other variables such as cognition, behavior, or interpersonal relations are any less vital to the understanding or treatment of GAD or other forms of psychopathology. Rather, emotions and their regulation may be the “tie that binds” these phenomena together. An emotion regulation perspective may accomplish this goal by integrating seemingly incompatible theories of psychopathology that stress these different factors ([Gross & Muñoz, 1995](#); [Westen, 1994](#)). Further research is clearly necessary to determine the relationship between emotion and other cognitive, behavioral, biological, and interpersonal phenomena. However, there appears to be good reason to suggest that this integrative goal would further our understanding of psychopathology and its treatment.

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