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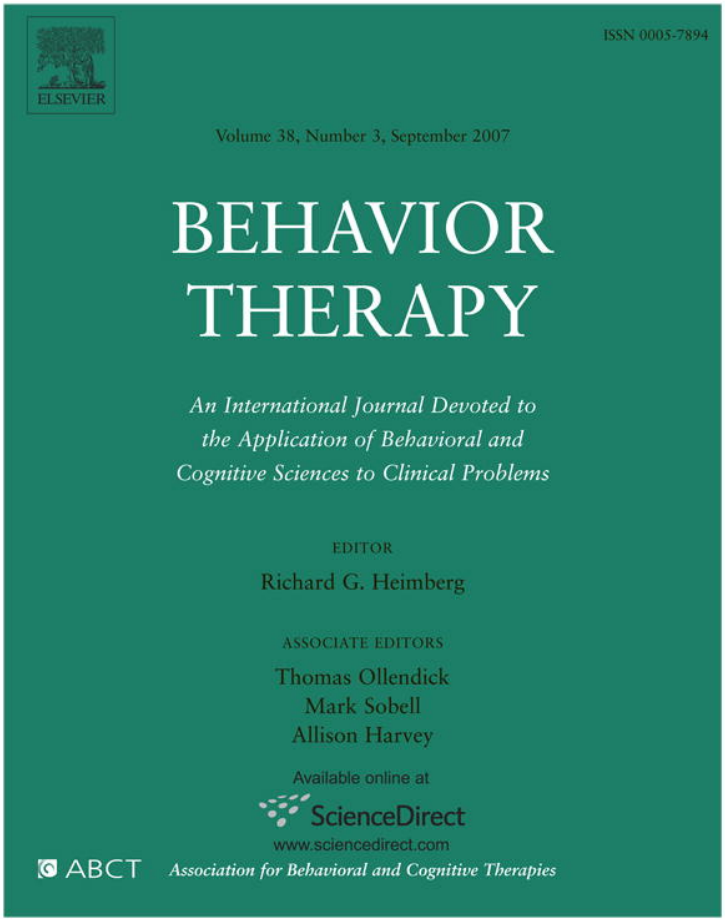

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Delineating Components of Emotion and its Dysregulation in Anxiety and Mood Psychopathology

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Two studies sought to elucidate the components of emotion and its dysregulation and examine their role in both the overlap and distinctness of the symptoms of 3 highly comorbid anxiety and mood disorders (i.e., generalized anxiety disorder, major depression, and social anxiety disorder). In Study 1, exploratory factor analyses demonstrated that 4 factors—heightened intensity of emotions, poor understanding of emotions, negative reactivity to emotions, and maladaptive management of emotions—best reflected the structure of 4 commonly used measures of emotion function and dysregulation. In Study 2, a separate sample provided support for this 4-factor model of emotion dysregulation. Poor understanding, negative reactivity, and maladaptive management were found to relate to a latent factor of emotion dysregulation. In contrast, heightened intensity of emotions was better characterized separately, suggesting it may relate more strongly to dispositional emotion generation or emotionality. Finally, the 4 components demonstrated both common and specific relationships to self-reported symptoms of generalized anxiety disorder, major depression, and social anxiety disorder.

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Approaches to understanding and treating anxiety and mood disorders have advanced considerably since the advent of *DSM-III* (American Psychiatric Association, 1980), at which time the overarching diagnostic syndromes of neuroses were first divided into discrete categories based on symptom content. For generalized anxiety disorder (GAD), major depressive disorder (MDD), and social anxiety disorder (SAD), which are the most impairing disorders outside of substance use (Kessler, Chiu, Demler, Merikangas, & Walters, 2005), the diagnostic movement toward greater specificity provided an opportunity for delineation of core elements of these conditions, including worry in GAD, anhedonia in MDD, and fear of evaluation in SAD. Increased precision in conceptual focus also led to greater success in treatments for these disorders (e.g., Borkovec & Costello, 1993; Heimberg et al., 1998; Jacobson et al., 1996).

Despite these advances, GAD, MDD, and SAD are characterized by high levels of comorbidity, particularly with one another. In fact, the high rate of comorbidity between GAD and MDD has led to calls to combine these disorders in *DSM-V* into a "distress disorder" category (e.g., Watson, 2005). SAD is the next most frequent comorbid condition for both GAD and MDD. Further, SAD can be a difficult differential diagnosis when these other disorders are present given its characteristics of social worry (Mennin, Heimberg, & Jack, 2000) and lack of positive affect (Brown, Chorpita, & Barlow,

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1998; Kashdan, 2004), components central to GAD and MDD, respectively. Comorbidity among these disorders has been associated with greater symptom severity and poorer functioning (e.g., Mennin et al., 2000; Stein & Heimberg, 2004). This high level of comorbidity also challenges the notion of these disorders as purely independent entities and suggests that delineation of *both* common and specific factors may provide further explanation of the nature of these conditions.

Studies of the anxiety and mood disorders, utilizing structural modeling, offer evidence for the importance of emotional processes common to these conditions (Brown et al., 1998; Shankman & Klein, 2003; Watson, Clark, & Carey, 1988; Zinbarg & Barlow, 1996). These investigations offer support for the *tripartite model* of emotional disorders, wherein a higher-order factor of negative affect or neuroticism accounted for much of the overlap among anxiety and mood disorders, particularly for the most strongly comorbid disorders, such as GAD and MDD (Mineka, Watson, & Clark, 1998). These findings suggest that emotional factors can aid in understanding the interplay of these disorders. In addition to being an index of commonality, however, affective features can also distinguish GAD, MDD, and SAD. The tripartite model demonstrates that anxious arousal appears to be more specific to fear-based disorders such as SAD and low positive affect appears more relevant for MDD and SAD (Watson, 2005). Also, each of these disorders is associated with a prominent, central, emotional element—fear in SAD, anxiety in GAD, and sadness in MDD—suggesting that although some emotional characteristics may be common to these disorders, others may help distinguish them. Delineating core emotional features may help clarify both the overlap and uniqueness among these disorders.

Emotion Function and Regulation

Contemporary theories define emotion as an adaptive, goal-defining aspect of experience that aids in decision-making, specifically, movement toward or away from particular actions or plans (e.g., Frijda, 1986). Knowledge of how typically functional emotional processes become dysfunctional and, in turn, become associated with psychopathology may improve our understanding of how these conditions interrelate and can be distinguished. Further, a greater understanding of emotion may also provide a broader framework for understanding how cognitive, behavioral, interpersonal, and biological factors are involved in the etiology and successful treatment of these conditions. Indeed, a number of investigators of psychopathology and clinical psychology (e.g., Barlow, 2002; Kring & Werner, 2004) have begun to draw from emotion theory and the contemporary study of emotion (see Davidson, Scherer, & Goldsmith 2003, for an introduction to this field of investigation).

Greater levels of negative emotions (and for MDD and SAD, diminished positive affect as well; cf. Watson, 2005) appear to be central to the symptomatology of GAD, MDD, and SAD. However, characteristic differences in emotionality may only be one way by which these disorders could be integrated and distinguished. In addition, an inability to respond effectively to one's intense emotional experiences may comprise another pathway for emotions to relate to psychopathology. As Frijda (1986) has commented, "people not only have emotions, they also handle them" (p. 401). The field of emotion regulation examines how individuals influence, manage, experience, and express their emotions (Gross, 1998). Regulating emotions to conform adaptively to a given context appears important to well-being (cf. Mayer, Salovey, & Caruso, 2004) and to the promotion of mental health (cf. Kring & Werner, 2004). Subsequently, in addition to emotionality or greater emotional intensity, the dysregulation of emotions may also be important to understanding GAD, MDD, and SAD.

Various factors may contribute to whether emotions are regulated effectively. Individuals who are able to recognize emotional experiences, understand their meaning, utilize their informational value, and manage their experience and expression of emotion in a context-appropriate manner appear most able to respond effectively to life's demands (see Mayer et al., 2004). This set of abilities is often referred to as *emotional intelligence*. Similarly, following the theoretical approaches of Thompson (1990) and Gross (1998), 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. As such, problems in initial generation of emotions, and subsequent difficulties in interpreting and utilizing these emotions, may be just as important to dysregulation as how emotions are managed.

A Model of Emotion and its Dysregulation in Anxiety and Mood Disorders

Given the possibility that dysfunction of emotional processes may occur at points of generation, understanding, reactivity, and regulation, overarching frameworks are necessary to help organize inquiry into the role of emotion factors in psychopathology

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(Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003; Kring & Werner, 2004). Conceptual frameworks advance our understanding of emotional dysregulation in these anxiety and mood disorders by operationalizing core emotional deficits and providing a common language for these deficits. Based on the notion that emotion dysregulation is multifaceted, Mennin and colleagues developed an emotion dysregulation model of anxiety and the mood disorders (for an introduction to this perspective, see Mennin, 2004; Mennin, Heimberg, Turk, & Fresco, 2005) that enumerates four components of emotion dysfunction: (a) heightened intensity of emotions; (b) poor understanding of emotions; (c) negative reactivity to one's emotional state (e.g., fear of the consequences of emotions); and (d) maladaptive emotional management responses. Accumulating evidence suggests that these deficits are related in either specific or common ways to GAD, MDD, and SAD.

Heightened intensity of emotions refers to frequently experiencing negative affect strongly and having emotional reactions that occur intensely, easily, and quickly. This construct is conceptualized as a characteristic of emotional generative processes and is likely related to overarching dispositions of emotionality (Watson et al., 1988). Heightened emotional intensity may not be pathological in and of itself (e.g., crying at weddings or sad movies; see Kring & Werner, 2004) but may make it more likely that one is unable to successfully manage emotions given the overall greater presence and strength of negative mood states (Eisenberg, Fabes, Guthrie, & Reiser, 2000; Linehan, 1993). Individuals with GAD demonstrate heightened intensity (Mennin et al., 2005) and do so to a greater degree than individuals with SAD (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005) or eating disorders (Fresco, Wolfson, Crowther, & Moore, 2005). *Poor understanding of emotions* involves inadequate understanding of one's emotions and has been found to negatively relate to active coping and positive attributions (Gohm & Clore, 2002). Deficits in understanding emotions have been related to GAD (e.g., Mennin et al., 2005; Turk et al., 2005) and SAD (e.g., Salovey, Stroud, Woolery, & Epel, 2002; Turk et al., 2005). Similar constructs such as alexithymia (Bagby, Parker, & Taylor, 1994) or a deficit in emotional clarity (e.g., Salovey et al., 2002) also show a relationship to depressive symptoms (e.g., Salovey et al., 2002; Wise, Mann, & Randell, 1995).

Rather than processing emotion information and utilizing its motivational or informational value, some individuals may, instead, evidence *negative reactivity to emotions*, which is characterized by holding negative beliefs, such as feared consequences, following emotion. This component involves a discomfort with the experience of emotions, which then leads to a strong cognitive reaction that these emotional responses are dangerous or harmful. Leahy (2002) found that depression and anxiety, assessed by self-report, were associated with viewing one's emotions as incomprehensible, uncontrollable, different than others' emotions, and characterized by guilt. However, whereas depression was more closely associated with expectations of long mood duration, anxiety was more likely to be associated with lack of acceptance of emotions. Chambless and colleagues found that individuals who feared emotions were more likely to be reactive to induced bodily sensations (Williams, Chambless, & Ahrens, 1997), even beyond the effects of state and trait anxiety (Berg, Shapiro, Chambless, & Ahrens, 1998). Individuals who suffer from GAD (Mennin et al., 2005, Studies 1 and 2; Roemer, Salters, Raffa, & Orsillo, 2005; Turk et al., 2005) and SAD (Turk et al.) have been found to report greater fear of anxiety, sadness, anger, and positive emotions than controls. Further, negative reactivity to one's emotions, measured 4 months after the terrorist attacks on September 11, 2001, mediated the relationship between analogue GAD (assessed September 10, 2001) and increases in anxiety and mood symptoms and functional impairment 12 months after the attacks in young adults directly exposed to the World Trade Center collapse (Farach, Mennin, Smith, & Mandelbaum, Submitted for publication).

Finally, there are numerous strategies for managing these aversively perceived emotional experiences, with some being more adaptive to a given situation and some inevitably leading to greater dysfunction. Individuals with GAD, MDD, and SAD may have difficulty knowing when or how to enhance or diminish their emotional experience in a manner that is appropriate to a particular environmental context (e.g., *maladaptive management of emotions)*. As reflected in both trait report and state responses to negative mood, individuals with GAD struggle to soothe themselves (Mennin et al., 2005). GAD and worry have been associated with deficits in the ability to engage in goal-directed behaviors when distressed, display impulse control, and access effective regulation strategies (Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, in press). Social anxiety and depressive symptoms are also associated with impaired ability to repair negative moods (Salovey et al., 2002; Turk et al., 2005).

The Present Study

Despite this preliminary evidence for the role of emotion factors in anxiety and mood psychopathology, methodological and design considera-

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tions qualify these findings, particularly for elucidating the overlap of GAD, MDD, and SAD symptoms. First, in our previous studies, we used subscales of a number of established measures, which we rationally derived to represent the four components of the model. Measures were assigned to components based on their content only, and no attempt was made to assign individual items to specific components. As a result, the components likely included aspects of constructs other than those intended and therefore contained a high degree of unwanted systematic variance. Therefore, it remains difficult to determine whether a cohesive set of items is represented in each of these components. For instance, negative reactivity to emotions has been measured using the Affective Control Scale developed by Chambless and colleagues (Berg et al., 1998; Williams et al., 1997). However, this measure combines elements of negative reactivity and maladaptive management in its items as many of the positively worded items contain actions associated with proper soothing and management of emotions. Further research is necessary to create factors at the individual item level that contain a minimum of variance not attributable to the target component of interest and inform theory-building with a greater degree of specificity than has been attempted in past research.

It will also be important to determine if these emotional elements are best represented by an overarching latent factor reflective of negative affect or neuroticism that incorporates both elements of excessive emotion generation and dysregulation. Indeed, there is disagreement over whether emotion generation and regulation should be considered separate or unified constructs (Campos, Frankel, & Camras, 2004; Cole, Martin, & Dennis, 2004). An alternative argument for this model would be that heightened intensity is a component of emotionality or dispositional emotion generation and thus distinct from, although correlated with, emotion dysregulation, which may involve dysfunctional aspects such as poor understanding, negative reactivity, and maladaptive management.

Most importantly, although heightened intensity, poor understanding, negative reactivity, and maladaptive management of emotions have been demonstrated in GAD, MDD, and SAD, it is unclear to what extent each of these components is common to these disorders and which may be unique to a particular disorder. Increased precision in delineating these emotion-related components may help elucidate high levels of comorbidity among GAD, MDD, and SAD. However, few studies have examined emotion deficits concurrently in these psychopathologies. It is important to determine how the combination of these components simultaneously relates to co-occurring symptoms of GAD, MDD, and SAD. In our previous investigations, a composite variable encompassing some of these emotion factors contributed to the prediction of GAD beyond the contributions of worry, anxiety, and depressive symptoms (Mennin et al., 2005), and a discriminant function analysis revealed that the combination of factors appeared to better detect a diagnosis of GAD than SAD (Turk et al., 2005). However, neither of these investigations examined all of these components concurrently in relation to GAD, MDD, and SAD. It will be important to determine the extent to which these components relate to symptoms of GAD, MDD and SAD, both independently of each other and when these disorders co-occur.

In two studies, we sought to demonstrate that these four components (a) are reflected in measures of emotion dysfunction and dysregulation previously used to denote relationships to psychopathology; (b) are independent and reliably indicated by sampling of items from these measures; (c) reflect a higher order latent factor of emotion dysregulation, with the exception of heightened intensity, which was expected to be an index of emotionality or characteristic differences in emotion generation; and (d) demonstrate both generalized and specific relationships to self-report indices of GAD, MDD, and SAD. College samples assessed by self-report measures were used in both studies. Although clinical samples with formal diagnoses would be ideal, we believed that it would be important to first establish a basis of relationship between emotion factors and concurrent symptoms of GAD, MDD, and SAD in a normative population before examining these relationships within patients. Further, the use of a college sample provides a greater range of scores in which to conduct analyses with these measures. A clinical sample would likely have a restricted range of extreme scores on these measures and, thus, might not be optimal for predicting levels of unshared variance. However, as a result, GAD, MDD, and SAD are self-reported in the present study, and, thus, reflect symptom presentations of these conditions rather than actual diagnoses.

Study 1: Exploratory Factor Analyses

In this first study, we examined the structure of the measures we previously used to assess model components. In particular, four measures were submitted to a series of exploratory factor analyses to determine if they reflected the components of our model of emotion dysregulation in psychopathology and whether these factors could be reflected in cohesive, independent item sets. We expected that (a) heightened intensity of emotions would be reflected by

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items from both the impulse strength and expressivity subscales of the Berkeley Expressivity Questionnaire (Gross & John, 1995); (b) poor understanding would be reflected by items from the clarity subscale of the Trait Meta-Mood Scale (Salovey, Mayer, Goldman, Turvey, & Palfai, 1995) and the difficulty identifying and describing subscales of the Toronto Alexithymia Scale–20 (Bagby, Parker, et al., 1994; Bagby, Taylor, and Parker, 1994); (c) negative reactivity would be reflected by negatively worded items from all subscales of the Affective Control Scale (Berg et al., 1998; Williams et al., 1997) indicating beliefs of feared consequences of both negative and positive emotions; and (d) maladaptive management of emotions would be reflected inversely by the mood repair ability subscale of the Trait Meta-Mood Scale and the positively worded items of the Affective Control Scale, which include management and soothing actions in response to experienced emotions. We also sought to obtain, overall, a reduced set of items from these scales that would be most indicative of the model's components and which could be used to more precisely predict specific and nonspecific aspects of GAD, MDD, and SAD (see Study 2, below).

METHOD

Participants and procedure. Participants were 628 undergraduate students (71.2% female) who completed several measures of emotional characteristics and dysregulation, as well as additional measures not included in the current study. Participants were enrolled in an introductory psychology class at Temple University and received course credit for completion of questionnaires. The ethnic composition of this sample was 30.5% African-American, 3% Asian-American, 13.5% Hispanic, 41.9% Caucasian, with 11.1% reporting "other." The average age was 19.68 years ($SD = 3.82$). These demographic characteristics are representative of the introductory psychology class and the university overall.

Measures. The Affective Control Scale (ACS; Williams et al., 1997) is a 42-item self-report measure assessing fear of emotions and attempts to control emotional experience. The ACS has demonstrated high internal consistency ($\alpha = .94$ for the overall scale) and strong test-retest reliability over a 2-week period ($r = .78$; Williams et al., 1997). The present study demonstrated a similarly high level of internal consistency for items on the ACS ($\alpha = .94$). Further, the ACS total score is correlated with neuroticism and emotional control and minimally correlated with social desirability (Berg et al., 1998; Williams et al., 1997). Subscales include (a) fear of anxiety, (b) fear of depression, (c) fear of anger, and (d) fear of positive emotions. Items are scored on a 7-point Likert-type scale.

The Berkeley Expressivity Questionnaire (BEQ; Gross and John, 1995, 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. The BEQ has been shown to have acceptable indices of internal consistency ($\alpha = .86$; $\alpha = .82$, this sample) and retest reliability ($r = .86$ over 2 months; Gross and John, 1995). The BEQ has also been found to predict self- and peer-rated levels of expression (Gross and John, 1997). It is comprised of three subscales: (a) impulse strength; (b) negative expressivity; and (c) positive expressivity. Items are scored on a 7-point Likert-type scale.

The Toronto Alexithymia Scale—20 (TAS-20; Bagby, Parker, et al., 1994; Bagby, Taylor, & Parker, 1994) measures lack of emotional understanding and an inability to express emotions. The TAS-20 has evidenced high internal consistency in the literature ($\alpha = .81$; Bagby, Parker, et al., 1994) and in the current sample ($\alpha = .85$). In addition, the TAS-20 has demonstrated adequate test-retest reliability ($r = .77$), has correlated negatively with measures assessing access and openness to one's feelings, but demonstrated no relationship to agreeableness, conscientiousness, and extraversion (Bagby, Taylor, & Parker, 1994). The TAS-20 is comprised of three subscales: (a) difficulty identifying feelings, (b) difficulty describing feelings, and (c) externally oriented thinking. Items are scored on a 5-point Likert-type scale.

The Trait Meta-Mood Scale (TMMS; Salovey et al., 1995) is a 30-item self-report measure of emotional intelligence comprised of three subscales: (a) attention to emotion, (b) clarity of emotions, and (c) mood repair. Items are scored on a 1 to 5 Likert-type scale. These subscales are internally consistent (αs range from .82 to .88; Salovey et al.), with values similar to that found in the current sample (total $\alpha = .88$). In addition, the TMMS is related to other measures of emotion-related skills such as negative mood regulation, optimism, and the ability to express oneself without ambivalence (Salovey et al.).

RESULTS

Initial exploratory factor analysis. Given the large item set and our interest in greater precision in the delineation of relationships with psychopathology, a series of exploratory factor analyses were used to determine what constructs were represented by this item set. Although our hypothesis predicted the presence of a four-factor solution, there was insufficient prior research on the topic to follow expectations about which specific items would load

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onto the various factors, necessitating a more empirically guided approach. An initial common factor analysis[1] of the 108 items comprising the ACS, TAS-20, TMMS, and BEQ was conducted using the principal axis factoring method of extraction with oblique rotation. Oblique rotation was chosen given that we expected factors to be correlated reflecting hypothesized relationships among our model components (however, orthogonal rotation produced comparable results, a report of which is available from the first author). The Kaiser-Meyer-Olkin index of sampling adequacy revealed a high index of factorability with a value of .95 (the index ranges from 0 to 1). After extraction, examination of the scree plot revealed four relevant factors, retaining 61 items. The first factor had an eigenvalue of 17.96 and accounted for 17% of the variance. The second factor had an eigenvalue of 8.77 and accounted for 8% of the variance. The third factor had an eigenvalue of 5.01 and accounted for 5% of the variance. Finally, the fourth factor had an eigenvalue of 4.01 and accounted for 4% of the variance. The factors from this model were not interpreted.[2] Rather, this first-pass factor solution was submitted to a more precise method of exploratory factor analysis to determine if a four-factor model was the most appropriate model for these item correlations and to obtain more objective indices of an acceptable factor solution.

Follow-up exploratory factor analysis. Sixty-one items retained in the initial exploratory factor analysis were submitted to a follow-up common factor analysis using the Comprehensive Exploratory Factor Analysis program (CEFA 1.10; Browne, Cudeck, Tateneni, & Mehls, 2002). CEFA is especially well suited for determining the optimal number of factors in a covariance matrix because it employs maximum likelihood estimation that generates relative fit indices for comparison among models with different numbers of factors. Browne et al. (2002) also recommend the inclusion of five random variables to protect against underextraction of factors. The solution is underextracted when random items are forced to load on factors generated with actual data. Thus, these random items allow for a stopping rule for solutions with too few factors. Model fit to the data was assessed using several fit indices in addition to the standard χ^2 statistic. The CMIN/*df* statistic, a modification of the χ^2 statistic intended to reduce the tendency for χ^2 to be conflated by large sample sizes (Bollen, 1989), is calculated simply by dividing χ^2 by the degrees of freedom for the overall model. Values of CMIN/*df* lower than 3 to 4 indicate acceptable fit. In addition, root-mean squared error of approximation (RMSEA) values "close to 0.06" (Hu & Bentler, 1999; p. 1) represent adequate model fit. We tested three-, four-, and five-factor models using oblique rotation. The three-factor model had adequate fit indices ($\chi^2 = 6666.00$, $p < .001$; CMIN/*df* = 3.42; RMSEA = .06), as did the five-factor model ($\chi^2 = 4658.32$, $p < .001$; CMIN/*df* = 2.55; RMSEA = 0.05). However, every item on the fifth factor in the five-factor solution had unacceptably low item loadings or multiple factor loadings. In addition, items on this factor did not appear to have any conceptual cohesion but were, rather, a sampling of items from a number of the scales. In contrast, the four-factor solution demonstrated the best balance of fit indices ($\chi^2 = 5424.21$, $p < .001$; CMIN/*df* = 2.87; RMSEA = 0.06) and the greatest level of interpretability.

Individual items were considered to load on a factor if the factor loading exceeded .40 and if the difference in factor loadings between factors was greater than .20 based on prior studies examining the factor structure of some of these measures (e.g., Salovey et al., 1995). Using this rule, 51 of the original 61 items were retained. Rotated factor loadings for all items in the four-factor solution are shown in Table 1. Factor 1, with an eigenvalue of 13.0, consisted of 17 items and was labeled *Negative Reactivity to Emotions* (NR). Factor 2, with an eigenvalue of 6.0, consisted of 11 items and reflected *Heightened Intensity of Emotions* (HI). Factor 3, with an eigenvalue of 3.5, consisted of 11 items and was labeled *Poor Understanding of Emotions* (PU). Factor 4, with an eigenvalue of 2.9, consisted of 12 items and reflected effective management of emotions. However, to be consistent with the other factors that measured emotional dysfunction rather than ability, this factor was considered to be negatively related to its items and was labeled *Maladaptive Management of Emotions* (MM).

SUMMARY OF FINDINGS

A four-factor model was found to best reflect these measures of emotion characteristics and dysregulation. The first derived factor corresponded wholly to negatively worded items from the ACS, including those from subscales related to fear of anxiety, fear of depression, fear of anger, and fear of positive emotion, suggesting that this factor reflects

[1] Based on the recommendations of Floyd and Widaman (1995), common factor analysis was chosen over principal components analysis. Common factor analysis is preferable when one wishes to understand the relationships among manifest variables to suspected latent variables. Further, estimates derived from common factor analysis tend to hold up better than estimates derived from principal components analysis to confirmatory replication with new data.

[2] Factor-item correlations for the initial exploratory factor analysis are available from the authors.

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Table 1
Exploratory and confirmatory factor loadings of items on four emotion measures

Item	Study 1				Study 2
	Factor 1	Factor 2	Factor 3	Factor 4	CFA
Factor 1 (Negative Reactivity)					
ACS36 Getting really ecstatic about something is a problem for me because sometimes being too happy clouds my judgment.	.73	−.05	.04	.01	.72
ACS42 I think my judgment suffers when I get really happy.	.73	−.06	.07	−.03	.72
ACS41 I am afraid that I'll do something dumb if I get carried away with happiness.	.72	−.03	.06	−.04	.76
ACS32 When I get really excited about something, I worry that my enthusiasm will get out of hand.	.72	−.03	.01	−.02	.78
ACS23 I worry about losing self-control when I am on cloud nine.	.66	−.10	.05	−.04	.74
ACS14 When I feel really happy, I go overboard, so I don't like getting overly ecstatic.	.62	−.06	.11	−.00	.74
ACS6 Being filled with joy sounds great, but I am concerned that I could lose control over my actions if I get too excited.	.62	−.06	.06	−.03	.68
ACS40 When I get nervous, I am afraid that I will act foolish.	.56	.15	.07	−.11	.64
ACS34 I get nervous about being angry because I am afraid I will go too far, and I'll regret it later.	.55	.06	.13	−.17	.66
ACS39 I am afraid that letting myself feel really angry about something could lead me into an unending rage.	.53	.00	.09	−.13	.65
ACS33 When I get nervous, I feel as if I am going to scream.	.52	.10	.12	−.15	.57
ACS15 When I get nervous, I think that I am going to go crazy.	.50	.15	.12	−.22	.64
ACS24 There is nothing I can do to stop anxiety once it has started.	.48	.13	.10	−.26	N/A
ACS11 If people were to find out how angry I sometimes feel, the consequences might be pretty bad.	.46	−.05	.19	−.06	.47
ACS2 I can get too carried away when I am really happy.	.46	.18	.16	.15	.44
ACS29 When I get "the blues," I worry that they will pull me down too far.	.44	.06	.18	−.23	N/A
ACS35 I am afraid that I will babble or talk funny when I am nervous.	.43	.20	.09	−.00	.53
Factor 2 (Heightened Intensity)					
BEQ15 I experience my emotions very strongly.	.03	.75	.07	−.05	.69
BEQ11 I have strong emotions.	−.07	.71	.01	−.00	.71
BEQ10 I am an emotionally expressive person.	−.02	.69	−.16	−.01	.69
BEQ12 I am sometimes unable to hide my feelings.	.02	.68	.02	−.06	.69
BEQ7 My body reacts very strongly to emotional situations.	.04	.61	.08	−.04	.65
BEQ2 I sometimes cry during sad movies.	−.11	.58	.07	.03	.49
BEQ6 When I'm happy, my feelings show.	−.09	.56	−.05	.23	.49
BEQ13 Whenever I feel negative emotions, people can easily see exactly what I am feeling.	.07	.53	−.00	−.07	.56
BEQ14 There have been times when I have not been able to stop crying even though I tried to stop.	.12	.52	.08	−.17	.42
BEQ4 I laugh out loud when someone tells me a joke that I think is funny.	−.07	.48	.03	.22	.36
BEQ1 Whenever I feel positive emotions, people can easily see exactly what I am feeling.	.02	.43	−.03	.17	.48
Factor 3 (Poor Understanding)					
TAS1 I am often confused about what emotion I am feeling.	.05	−.00	.74	.03	.70
TMMS16 I am usually confused about how I feel.	.01	.01	.71	−.03	N/A
TMMS22 I can't make sense out of my feelings.	.03	.05	.69	−.03	.59
TAS2 It is difficult for me to find the right words for my feelings.	.08	.10	.64	.05	.66
TMMS11 I can never tell how I feel.	.12	−.08	.61	.02	.52
TMMS25 I am usually very clear about my feelings.	.17	.11	−.58	.22	.55
TMMS5 Sometimes I can't tell what my feelings are.	.10	.09	.54	.08	.62
TMMS30 I almost always know exactly how I am feeling.	.11	.10	−.54	.26	.59
TAS9 I have feelings that I can't quite identify.	.14	.18	.52	−.03	.77
TAS17 It is difficult for me to reveal my innermost feelings, even to close friends.	.11	−.16	.46	.14	.37
TAS4 I am able to describe my feelings easily.	.03	.17	−.46	.14	.49

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Table 1 (*continued*)

Item	Study 1				Study 2
	Factor 1	Factor 2	Factor 3	Factor 4	CFA
Factor 4 (Maladaptive Management)					
ACS9 I feel comfortable that I can control my level of anxiety.	−.06	−.00	.02	.64	.64
ACS38 I don't really mind feeling nervous; I know it's just a passing thing.	−.05	.04	.06	.61	.55
ACS27 Being depressed is not so bad because I know it will soon pass.	−.04	.03	.05	.56	.52
ACS4 If I get depressed, I am quite sure that I'll bounce right back.	−.01	.00	−.05	.56	N/A
TMMS26 No matter how badly I feel, I try to think about pleasant things.	.15	−.01	−.24	.56	N/A
ACS17 I am able to prevent myself from being overly anxious.	−.10	−.09	−.03	.54	.71
TMMS8 Although I am sometimes sad, I have a mostly optimistic outlook.	.03	.06	−.16	.52	N/A
ACS21 Being nervous isn't pleasant, but I can handle it.	−.12	.02	.08	.52	.46
ACS31 Whether I am happy or not, my self-control stays about the same.	−.14	−.02	.15	.51	.47
TMMS1 I try to think good thoughts no matter how badly I feel.	.13	−.01	−.22	.48	.30
TMMS13 When I become upset I remind myself of all the pleasures in life.	.14	.04	−.19	.47	N/A
ACS18 No matter how happy I become, I keep my feet firmly on the ground.	−.12	−.02	.12	.44	.50

Note. "CFA" refers to confirmatory factory analytic results and represents the standardized loading each item had in Sample 2 on the factor on which it loaded the highest in Sample 1 (using exploratory factor analysis); "N/A" indicates an item that was dropped during CFA due to poor model fit. Factor 4 has been inversely labeled as "Maladaptive Management" to reflect its place in a model of deficits rather than abilities despite the positive loadings of items on the factor.

NR or negative beliefs about emotions. HI was reflected in the second factor, with items derived largely from the BEQ impulse strength subscale and a few items from the BEQ positive and negative expressivity subscales as well. The inclusion of these items in an intensity factor rather than a separate emotional expression factor is congruent with the nature of these items, which are less about strategic expression (e.g., "I expressed to my sister why I was angry with her") and more about unintentional expression as a result of strong feelings (e.g., "Whenever I feel negative emotions, people can easily see exactly what I am feeling"). Similarly, Gross and John (1997) reported that intensity and expressivity are distinct from more expressive regulatory behaviors (e.g., intentionally masking emotion displays). The third factor reflected PU and, as expected, was comprised of items from the TMMS clarity subscale and the TAS-20 subscales of difficulty identifying and describing emotions. Finally, the MM factor included items from the TMMS mood repair scale and positively worded items from the ACS, which include regulation-relevant soothing and optimism elements not present in the reactivity-focused negative items of this scale. Items from the external orientation subscale of the TAS-20 and the attention subscale of the TMMS did not load on any factor.

Study 2: Confirmatory Factor Analysis and Relationship to Anxiety and Mood Measures

In Study 2, we sought to replicate the factor solution from Study 1 via confirmatory factor analysis in a separate sample utilizing the reduced item set from Study 1. Another aim was to demonstrate, within a structural equation model (SEM), that these factors are, in part, reflected by a higher order latent factor of emotion dysregulation, which would be indicated by PU, NR, and MM. In contrast, HI was expected to be an independent factor more reflective of characteristic generation of emotion or dispositional emotionality and, thus, was not expected to load on this higher-order latent factor (although we did expect it to correlate with this latent factor given past demonstrated relationships, such as the significant association of the ACS with neuroticism; Williams et al., 1997). Further, using SEM, we sought to determine, first, the generalized relationships of both the lower- and higher-order emotion factors to symptoms of GAD, MDD, and SAD and, second, these relationships controlling for the overlapping co-occurrence of these psychopathologies in order to determine whether any of these components would demonstrate specificity. It was expected that, when overlap was not considered, all disorders would reflect each of these components, except HI, which would not

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be relevant for SAD given past research findings (see Turk et al., 2005). However, when examining these components in unison, some of the emotion dysregulation components were expected to show specific relationships as well. In particular, PU would be particularly relevant for depressive symptoms given its established relationship with alexithymic characteristics (e.g., Wise et al., 1995), NR would be a nonspecific component given the presence of meta-emotions or negative beliefs about emotion present in many psychopathologies (e.g., Leahy, 2002), and MM would be most indicative of GAD given this disorder's heightened intensity and subsequent greater occasion for management need. HI was expected to remain uniquely related to GAD even when comorbidity was addressed.

METHOD

Participants and procedure. Participants were 869 undergraduate students (69.6% female) who completed measures of anxiety and depression in addition to the measures of emotion administered in Study 1. The ethnic composition of this sample was 29.9% African-American, 2.9% Asian-American, 8.2% Hispanic, 44.1% Caucasian, 5.8% of mixed heritage, with 9% reporting "other." The average age was 19.43 years ($SD = 3.95$). As with Study 1, participants were enrolled in an introductory psychology class at Temple University and received course credit for their participation. Further, these demographic characteristics were, again, reflective of both the class and the ethnicity of the greater university student composition.

Measures. The ACS, TAS, TMMS, and BEQ were administered as in Study 1 (internal consistencies remained high, with αs ranging from 0.82 to 0.93).

The Beck Depression Inventory–II (BDI-II; Beck, Steer, and Brown, 1996) is a 21-item self-report measure that asesses the severity of depressive symptoms, including the affective, cognitive, behavioral, somatic, and motivational components of depression as well as suicidal wishes. Items are rated on a 0-to-3 scale and reflect a 2-week time period. The BDI-II has strong internal consistency in both student and clinical samples (Beck et al., 1996) and excellent test-retest reliability (Sprinkle et al., 2002).

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 *DSM-IV-TR* (American Psychiatric Association, 2000). Most items are dichotomous and measure the excessive and uncontrollable nature of worry as experienced by persons with GAD and related physical symptoms. The GAD-Q-IV demonstrates high concordance with a diagnosis of GAD yet is uncorrelated with conceptually unrelated measures (Newman et al., 2002). For the purpose of this investigation, a dimensional total score was utilized without any diagnostic cutoff.

The Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998) was used as an index of social anxiety symptoms. The SIAS is a 20-item self-report measure that assesses anxiety experienced in dyadic and group interactions. Individuals rate how well items describing anxiety in social interactions characterize them, ranging from 0 to 4. The SIAS has been frequently used and has demonstrated good reliability and validity (for a review, see Hart, Jack, Turk, & Heimberg, 1999).

RESULTS

Confirmatory factor analysis (CFA) was undertaken using structural equation modeling (SEM). Attempts to achieve adequate fit failed in an initial CFA using the 51-item solution that emerged in the Study 1 CEFA analysis. Due to the large number of parameters being estimated, we consequently used a two-step modeling approach (Anderson & Gerbing, 1988; Bollen & Biesanz, 2002).[3] The model was decomposed into the four individual factors, which were run separately with the variance of the highest loading item on each factor (as defined by the Study 1 CEFA) fixed and all other parameters freely estimated. These analyses indicated that 7 items evidenced poor fit on all four factors (standardized factor loadings $<|0.40|$). None of these items were judged to represent key facets of their respective factors and so were subsequently deleted. Model fit was substantially improved upon deletion of these 7 items.

In the second step, we fit a model in which the factor scores were used as observed variables and allowed to intercorrelate. For the four individual factors, maximum likelihood (ML) estimation indicated generally acceptable fit.[4] However, Mardia's statistic (average value of 49.59 in the model tested) indicated significant violations of the assumption of normally distributed data in SEM (Satorra & Bentler, 1994). The structural models were therefore

[3] In two-step modeling, a hybrid model (with both measurement and path aspects) is decomposed and the measurement model is run in CFA separately from the path model. The reasoning behind this approach is that if poor model fit is detected, it may be due to misspecification between indicators loading onto latent variables (the measurement model) or path coefficients between the latent factors and/or observed variables (the path model). In using two-step modeling in the current application, poor fit can be precisely pinpointed as residing either in any of the four factors individually, or in the relationships proposed between them, and the computational difficulty in fitting such a complex model to such a large dataset can be overcome.

[4] Fit indices using ML estimation are available from the authors upon request.

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re-fit using robust variances to obtain Satorra-Bentler Scaled statistics, corrected fit indices used to more accurately calculate the significance of a model employing nonnormal data (Satorra & Bentler, 1994). All four models converged in five iterations (with the exception of poor understanding, which converged in seven iterations). The values of the scaled χ^2 statistics were significant in all four models [NR: scaled χ^2 (83) = 321.47, $p < .00001$; HI: scaled χ^2 (39) = 178.03, $p < .00001$; PU: scaled χ^2 (32) = 118.55, $p < .00001$; MM: scaled χ^2 (18) = 33.84, $p = .01$]. However, given the influence of sample size on the χ^2 statistic, the comparative fit index (CFI) and RMSEA were utilized as additional fit indices. Hu and Bentler (1999) suggest the use of these two indices and recommend a cutoff on the CFI of at least .95 as well as an RMSEA "close to .06" (p. 1) to signify a good fit. Although PU, NR, and MM met these criteria, HI did not (see Table 1 for item-factor loadings; negative reactivity: scaled CFI = .95, scaled RMSEA = .06; HI: scaled CFI = .93, scaled RMSEA = .07; PU: scaled CFI = .96, scaled RMSEA = .06; MM: scaled CFI = .98, scaled RMSEA = .03). Therefore, all of the factors, with the exception of HI, can be said to fit the data well, whereas the results for HI were more suggestive of adequate fit (Kline, 2005).

We compared this four-factor emotion dysregulation model to the three- and five-factor models identified by CEFA in Study 1. As with the four-factor model, items were discarded if their highest standardized loading onto a factor was less than .40 and the difference in factor loadings between the highest two factors was greater than .20. Using this approach, 47 items were retained in the three-factor model and 48 items in the five-factor model. CFA using ML estimation was again employed, and scaled fit indices were again used to control for nonnormality in the data. Only one of the three factors in the three-factor model evidenced adequate fit [Factor 1: scaled χ^2 (145) = 790.99, $p < .00001$, scaled CFI = .89, scaled RMSEA = .07; Factor 2: scaled χ^2 (114) = 711.94, $p < .00001$, scaled CFI = .77, scaled RMSEA = .08; Factor 3: scaled χ^2 (40) = 205.82, $p < .00001$, scaled CFI = .92, scaled RMSEA = .07]. Four of the five factors in the five-factor model illustrated good fit [Factor 1: scaled χ^2 (50) = 203.06, $p < .00001$, scaled CFI = .96, scaled RMSEA = .06; Factor 2: scaled χ^2 (25) = 104.35, $p < .00001$, scaled CFI = .95, scaled RMSEA = .06; Factor 3: scaled χ^2 (41) = 176.28, $p < .00001$, scaled CFI = .94, scaled RMSEA = .06; Factor 5: scaled χ^2 (14) = 48.87, $p < .00001$, scaled CFI = .97, scaled RMSEA = .05], however one factor did not [Factor 4: scaled χ^2 (25) = 284.50, $p < .00001$, scaled CFI = .78, scaled RMSEA = .11]. Therefore in practice, the five-factor model collapsed into a four-factor model. These findings confirm the CEFA results in Study 1, which attested to the superiority of a four- versus three- or five-factor emotion dysregulation model.

Next, we examined, simultaneously, the relationship of these emotion factors with a higher-order latent factor of emotion dysregulation, as well as to self-report indices of GAD, MDD, and SAD. Specifically, we estimated three models using the GAD-Q-IV, BDI-II, SIAS, and the index scores of the four factors as observed variables. Index scores were calculated by converting all item-level raw scores to *z*-scores and then taking the average of all of the items retained in CFA to represent the given factor. Prior research indicates that the use of index scores of this type, when used to determine the relationships among constructs (and not the structure of a set of items), produces less biased results (Bandalos, 2002; Little, Cunningham, Shahar, & Widaman, 2002). Using SEM, the relationship between the emotion dysregulation latent variable, the four factors that compose it, and the three aforementioned measures of symptoms of psychopathology were tested. In addition, the proposed interrelations among the four factors were also examined. We predicted that a model wherein HI did not serve as an indicator of, but was correlated with, an emotion dysregulation latent factor would produce the best fit of the data. This finding would be consistent with research suggesting that generation and regulation of emotions may be distinct processes (Rottenberg & Gross, 2003), as well as prior research that indicated that HI correlated poorly with the other three emotion dysregulation factors (Mennin et al., 2005). To test this assumption, we compared this model to another that incorporated all four factors as indicators of a latent factor of emotion dysregulation.

A model reflecting HI correlated with the emotion dysregulation latent variable fit the data equally well to a model where HI served as an indicator of this latent variable (both models converged in 6 iterations, scaled CFI = 1.00, scaled RMSEA = .05). Correlations between the four emotion dysregulation factors, the emotion dysregulation latent variable, and measures of psychopathology are presented in Table 2. Examination of the bivariate correlations between HI and the other factors indicated weak correlations with one another. Further, there was a small, nonsignificant correlation between HI and the emotion dysregulation latent variable ($r = -.01$). Given these findings, it was felt that emotion dysregulation was best represented by the three facets of PU, NR, and MM with HI as a separate, relatively uncorrelated construct. Subsequent

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Table 2
Intercorrelations among emotion factors and indices of psychopathology in study 2

Measure	1	2	3	4	5	6	7	8
1. Intensity	–	−.01	−.11**	.02	−.00	.27**	.10**	−.07*
2. ED		–	.67**	.91**	.58**	.27**	.29**	.27**
3. Understanding			–	.17**	.20**	.17**	.29**	.32**
4. Reactivity				–	.31**	.31**	.33**	.37**
5. Management					–	.28**	.25**	.17**
6. GAD						–	.56**	.33**
7. BDI–II							–	.37**
8. SAD								–

Note. Intensity = Heightened Intensity of Emotions (Factor 2); ED = Latent Emotion Dysregulation Factor; Understanding = Poor Understanding of Emotions (Factor 3); Reactivity = Negative Reactivity to Emotions (Factor 1); Management = Maladaptive Management of Emotions (Factor 4); GAD = Generalized Anxiety Disorder Questionnaire-IV; BDI-II = Beck Depression Inventory-II; SAD = Social Interaction Anxiety Scale; *p<.05. **p<.01.

modeling utilized this approach. Correlations also demonstrated significant zero-order positive relationships of all the emotion factors with the GAD-Q-IV, BDI-II, and the SIAS. However, whereas HI was positively related to GAD-Q-IV and BDI-II scores, it was weakly and inversely correlated with the SIAS.

Within the model with HI as a separate factor, we examined the direct association of the four factors, as well as the emotion dysregulation latent variable itself, with the indices of psychopathology. In particular, we examined the standardized regression weights (β) of the emotion factors predicting scores on the GAD-Q-IV, BDI-II, and SIAS. Symptoms of GAD and MDD were predicted equally well by all four factors and symptoms of SAD were best predicted by PU and NR (see Table 3). In addition, the emotion dysregulation latent variable served as a significant predictor of all three indexes of psychopathology and was approximately equally related to each.[5]

Next, we were interested in examining the relationship between the four emotion factors and the three indices of psychopathology to address the high levels of comorbidity often found among GAD, MDD, and SAD (Kessler et al., 2005). We tested a third model wherein PU, NR, and MM served as indicators of a higher-order emotion dysregulation latent variable, which was correlated with HI, identical to the model mentioned above. Also identical to the above model, all four emotion dysregulation factors, as well as the latent variable itself, served as predictors. In this model, however, residualized psychopathology variables were created and used as criterion variables to determine the unique relationships between the emotion dysregulation variables and purer measures of each psychopathology, to control for the substantial overlap among these measures. To create these residualized variables, the standardized residual of one of the three psychopathology variables was saved after variance from the other two measures was removed. This procedure was repeated for each index of psychopathology. This model converged in 6 iterations and fit the data well (scaled CFI = 1.00, scaled RMSEA = 0.00). The standardized regression weights (β) were examined and are shown in Table 3. Results indicated that high levels of HI were uniquely associated with residualized GAD; whereas *low* levels were uniquely associated with residualized SAD. PU and NR were common to both residualized MDD and SAD, but not to residualized GAD. However, MM only demonstrated a relationship with residualized GAD. The emotion dysregulation latent variable again served as a significant predictor of all three indexes of psychopathology and was again approximately equally related to each[5].

SUMMARY OF FINDINGS

Although modeling of all four emotion dysregulation factors using item-level indicators in CFA was not possible, modeling of the four factors in isolation allowed for a more subtle and nuanced method for determining the locus of model fit or misspecification, if it was detected. This approach indicated that three of the four factors of emotion dysregulation identified in Study 1 (PU, NR, and MM) confirmed the factor solution. In contrast, HI demonstrated weaker but still adequate support. Further, as in Study 1, these four factors were found to better reflect the data than three- or five-factor models. A latent factor of emotion dysregulation was found, which was separate from HI, but indicated by PU, NR, and MM. This model fit the data better than a

[5] A model with the emotion dysregulation latent variable predicting the various indices of psychopathology was run separately from the model where its four indicators were used as predictors. Both relationships examined simultaneously would address the incremental validity of the emotion dysregulation latent variable to predict scores on the BDI-II, GAD-Q-IV, and SIAS above its individual indicators, which is not the question that we are attempting to address here. However, both models fit the data well (CFI > 0.95 and RMSEA ≤ 0.05).

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Table 3
Prediction of symptoms of psychopathology by indices of emotion dysregulation

Criterion	Predictor	SEM psychopathology models							
		Direct relationship model				Residual variable model			
		β	*b* (SE)	*z*	*p*-value	β	*b* (SE)	*z*	*p*-value
GAD-Q-IV									
	ED Latent Factor	.54	.14 (.46)	8.66	<.0001	.20	.81 (.20)	4.07	<.0001
	Heightened Intensity	.27	1.32 (.15)	8.87	<.0001	.27	.43 (.05)	8.26	<.0001
	Poor Understanding	.12	.98 (.26)	3.93	.0001	−.03	−.08 (.09)	−.89	.37
	Negative Reactivity	.22	.98 (15)	6.87	<.0001	.06	.09 (.05)	1.69	.09
	Maladaptive Management	.18	1.00 (.19)	5.50	<.0001	.15	.27 (.07)	4.13	<.0001
BDI–II									
	ED Latent Factor	.59	2.65 (1.32)	9.13	<.0001	.25	1.04 (.20)	5.29	<.0001
	Heightened Intensity	.11	1.54 (.42)	3.69	.0002	−.01	−.01 (.05)	−.23	.82
	Poor Understanding	.23	4.66 (.72)	7.11	<.0001	.14	.38 (.09)	4.08	<.0001
	Negative Reactivity	.26	2.71 (.41)	7.84	<.0001	.10	.15 (.05)	2.80	.0051
	Maladaptive Management	.12	.99 (.53)	3.52	.0004	.03	.06 (.07)	.86	.39
SIAS									
	ED Latent Factor	.58	8.22 (1.98)	9.03	<.0001	.35	1.42 (.19)	7.29	<.0001
	Heightened Intensity	−.05	.97 (.63)	−1.56	.12	−.14	−.22 (.05)	−4.34	<.0001
	Poor Understanding	.24	6.92 (1.08)	7.62	<.0001	.17	.45 (.09)	5.11	<.0001
	Negative Reactivity	.32	4.50 (.62)	9.76	<.0001	.23	.33 (.05)	6.57	<.0001
	Maladaptive Management	.02	2.20 (.80)	.69	.49	−.05	−.09 (.07)	−1.34	.18

Note. GAD-Q-IV = Generalized Anxiety Disorder Questionnaire-IV; SIAS = Social Interaction Anxiety Scale; BDI-II = Beck Depression Inventory-II; ED = Emotion Dysregulation.

model wherein HI was included as an indicator of this latent factor. Within this model, a significant relationship was found between all four factors and symptoms of GAD and MDD. In contrast, only PU and NR were related to symptoms of SAD. As predicted, HI did not predict symptoms of SAD but did predict symptoms of MDD when overlap was not considered. The latent emotion dysregulation factor demonstrated significant relationships with all psychopathology variables.

In a separate SEM examining overlap of self-report indices of GAD, MDD, and SAD, both common and specific patterns of relationships were found. The latent factor of emotion dysregulation continued to be related to all residual indices of psychopathology. It was predicted that when accounting for psychopathology overlap, high levels of HI and MM would be specific to GAD, PU would be specific to MDD, and NR would remain a common factor in all three psychopathologies. Indeed, HI and MM were only positively related to the residualized GAD variable. However, both PU and NR remained significantly related to both residualized MDD and SAD, but not GAD. Interestingly, HI demonstrated a negative relationship with residualized SAD.

General Discussion

The purpose of these studies was to elucidate components of emotion and its dysregulation and to determine their role in both the overlap and distinctness of anxiety and mood psychopathology. Factor analyses revealed a four-factor model of emotion and its dysregulation and demonstrated the relationship of its components to symptoms of GAD, MDD, and SAD. In Study 1, exploratory factor analyses revealed that four factors—heightened intensity, poor understanding, negative reactivity, and maladaptive management of emotions—best reflected the structure of four measures of emotion function and dysregulation. In Study 2, a separate sample was examined using SEM to replicate the four-factor structure of these measures. Poor understanding, negative reactivity, and maladaptive management were confirmed and were found to relate to a latent factor of emotion dysregulation. In contrast, heightened intensity of emotions demonstrated adequate fit of the data and was better characterized separately, suggesting it may relate more strongly to dispositional emotion generation or emotionality. Finally, the four components demonstrated both common and specific relationships to self-reported GAD, MDD, and SAD.

THE STRUCTURE OF EMOTION AND ITS DYSREGULATION

The emotion dysregulation model (Mennin, 2005; Mennin et al., 2005) defines dysregulation broadly as represented by maladaptive emotional responsiveness reflected in dysfunctional understanding,

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reactivity, and management. This formulation is congruent with those of others who define regulation both in terms of processes related to managing emotions and processes involved in evaluating and responding to emotions (e.g., Cole et al., 2004; Thompson, 1990). In contrast, other definitions of emotion regulation focus solely on processes meant to effect change in emotional states and are, thus, more specifically related to the management of emotions (e.g., Gross, 1998; Mayer et al., 2004). Theorists posit, however, that emotions are not only regulated by other processes such as cognition or behavior, but they are also frequent regulators of these processes (e.g., Cole et al.). If emotion can be both a cause and recipient of regulatory processes, then emotion dysregulation might not only reflect poor ability to manage emotions but also emotion-mediated, altered cognitive states regarding one's experience or lack of ability to properly evaluate one's emotional state (e.g., negative reactivity). This assertion was supported by the presence of a higher-order emotion dysregulation factor that was reflected by these three components of the model.

We expected that heightened intensity of emotions would be reflective of dispositional tendencies to generate emotions (i.e., "emotionality") and that this would be separate from components of emotion dysregulation, which are better characterized as poor responsiveness to emotions. Unlike the other model components, heightened intensity of emotions was not strongly related to the indices of emotion dysregulation or to their latent higher-order factor. This finding suggests that intensity of generated emotions should not be characterized as an emotion regulation deficit. Although emotion generation and regulation may be quite difficult to differentiate, Brackett and Mayer (2003) have distinguished between emotional intelligence competencies and measures of intensity or emotionality. In addition, experimental evidence has demonstrated the independence of generative and regulatory processes of emotion (Jackson et al., 2003). Kring and Werner (2004) point out that intensity alone may not be pathological (e.g., someone who reacts strongly at weddings with tears of joy or screams loudly at a horror movie). It may take the presence of emotion regulation deficits for intense emotions to be problematic. Indeed, regulation strategies have been found to mediate the detrimental effect of intensity on negative clinical outcomes (e.g., Lynch, Robins, Morse, & MorKrause, 2001). Further research is clearly necessary to address the distinctness of emotion generative and regulatory processes. Also, it will be important to gain a better understanding of the emotional characteristics of heightened intensity such as whether this factor is mostly reflective of arousal, valence, or both.

SPECIFICITY OF EMOTION FACTORS IN PREDICTING CONCURRENT ANXIETY AND MOOD SYMPTOMS

A main goal of the present investigation was to determine if components of the emotion dysregulation model demonstrate specific and nonspecific relationships to GAD, MDD, and SAD. To accomplish this, simultaneous contributions of the four emotion factors were examined both independently and concurrently in relation to self-reported measures of GAD, MDD, and SAD. Not surprisingly given past research, zero-order correlations of factors with each measure of psychopathology revealed a majority of nonspecific positive relationships. Further, the latent factor of emotion dysregulation displayed significant relationships with GAD, MDD, and SAD, even when the overlap among these psychopathologies was constrained. These findings support the presence of an overarching, nonspecific emotion factor in these anxiety and mood disorders, a common finding of other CFA studies (e.g., Brown et al., 1998; Watson et al., 1988; Zinbarg & Barlow, 1996). This suggests that these emotion dysregulatory factors may be seen in some form in these anxiety and mood disorders and, thus, may be an important area for conceptualization and incorporation into treatment. Indeed, aspects of emotion dysregulation, including poor understanding, negative reactivity, and maladaptive management, have been treatment targets for GAD (e.g., Mennin, 2004; Roemer & Orsillo, 2005) and MDD (e.g., A. Hayes et al., in press), and, based on previous findings, have been suggested as points of focus in SAD intervention as well (e.g., Kashdan, 2004; Turk et al., 2005).

When factors were examined concurrently, however, patterns of specificity also emerged. As predicted, heightened intensity remained a particularly strong predictor of GAD. The central role for heightened intensity in predicting GAD suggests that, for this disorder, the presence of intense emotional reactions may represent a high level of activation when emotions are generated and may reflect dispositionally high levels of emotionality as argued by others (e.g., Watson, 2005). In contrast, although heightened intensity demonstrated a relationship with symptoms of MDD, once overlap with symptoms of GAD was controlled, this relationship disappeared. This finding suggests that heightened intensity may be more related to generalized anxiety than depression and, thus, might be better characterized by arousal than valence (e.g., Watson et al., 1988). Rottenberg (2005) has argued that MDD is associated with insensitivity to emotional stimuli. Rot-

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tenberg surmises that, although moods may remain generally negative, emotion is constricted in individuals with MDD such that both positive *and negative* stimuli are responded to in a similarly inhibited manner. Given the high degree of comorbidity between MDD and GAD, examining levels of intensity may help determine how individuals move through periods characterized by greater levels of depressive or worried moods.

Unexpectedly, heightened intensity of emotions *negatively* predicted social anxiety both in zero-order relationships and when overlap with GAD and MDD symptoms was considered. This finding suggests that social anxiety, particularly when not in the presence of comorbid GAD or depression, is associated with decreased emotionality. It may be that purer forms of social anxiety are more strongly related to temperamental variables such as shyness or introversion, which might be characterized by weaker impulses of emotion. However, emotional intensity has been shown to be a predictor of perceived intensity of nonclinical panic beyond the effects of anxiety sensitivity, negative affect, anticipatory anxiety, and gender (Vujanovic et al., 2006), suggesting that other fear-related disorders (e.g., panic disorder) not examined within this study may involve heightened intensity. Future research examining generative processes of emotion in these disorders will be helpful to determine when intensity may play a role in fear and anxiety. In addition, further studies are necessary to determine specific mechanisms in subjective intensity and whether it indicates dysfunction or simply heightened emotionality. Specifically, it will be important to clarify the extent to which heightened intensity is characterized by physiological activation or more cognitively mediated distress (or both). As well, it will be important to determine what motivational components may be involved in this emotion generation (cf. Gray & McNaughton, 2000).

Another factor that demonstrated specificity was maladaptive management, which did not significantly predict symptoms of SAD regardless of whether overlapping variance with GAD and MDD was constrained. Maladaptive management also did not demonstrate a relationship to symptoms of MDD after overlap was considered. In contrast to SAD and MDD, GAD remained significantly predicted by maladaptive management after accounting for overlap. This finding suggests that maladaptive management may be particularly important for individuals with GAD. Although a number of anxiety and mood disorders are likely characterized by some degree of poor emotion management, individuals with GAD may have the greatest difficulty managing their emotional responses given their high levels of intensity. Being confronted with greater emotional responses might create a greater need to regulate these emotions. This link between intensity and management may provide an explanation for why symptoms of SAD were not associated with maladaptive management of emotions. Given the decreased levels of intensity in SAD, these individuals may not be as likely to have occasion to need to manage emotions. Further, intense emotional experiences may be more bound to circumscribed social situations in individuals with noncomorbid SAD. And, since these individuals are more likely to be behaviorally avoidant, they may have a decreased need to invoke strategies for soothing negative emotional experiences, given decreased contact with negative emotion-eliciting stimuli. Despite this possibility, zero-order correlations between heightened intensity and maladaptive management were quite low in the current study, which challenges this hypothesized relationship. Experimental investigations that can isolate generative and regulatory elements of a state-level emotional response will need to be utilized to truly test the relationship of these components within GAD, MDD, and SAD.

We predicted that poor understanding would be related to all forms of psychopathology when considered independently and that this emotion component would only remain relevant for symptoms of MDD when the overlap of GAD and SAD symptoms was constrained. In partial support of our prediction, *both* MDD and SAD remained significantly associated with poor understanding after overlap was considered. It is not surprising that the MDD symptoms remained strongly related to poor understanding given that the relationship of "alexithymia," a construct similar to poor understanding of emotions, and depression has been well documented in prior studies (e.g., Salovey et al., 1995, 2002; Wise et al., 1995). Although the strength of association was not predicted in this study, symptoms of SAD have previously demonstrated a strong relationship to poor understanding, as well. Turk et al. (2005) found that individuals with SAD symptoms, compared to both individuals with GAD symptoms and controls, reported more difficulty describing emotional experiences, a component of poor understanding.

Contrary to our predictions, negative reactivity to emotions was found to be particularly relevant to symptoms of MDD and SAD, but not GAD. We had predicted that this component would not demonstrate specificity given that negative beliefs concerning emotions are common to a number of disorders. Indeed, similar constructs related to negative beliefs about emotion, such as meta-emotions (e.g., Leahy, 2002), have been shown to be important to a

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number of anxiety and mood disorders. The lack of effect for GAD stands in contrast with past studies that have shown a strong relationship between indicators of negative reactivity and GAD (Fresco et al., 2005; Mennin et al., 2005; Roemer et al., 2005; Turk et al., 2005). However, these studies did not explicitly measure co-occurring psychopathology. It may be that this negative reactivity found in GAD could better be explained by the occurrence of other disorders in this often highly comorbid disorder. Given the broadness of the negative reactivity construct, it will be important for future studies to determine what aspects of this reactivity may be particularly relevant for SAD or MDD versus GAD.

These analyses demonstrate the importance of examining both common and specific relationships between components of emotion and its dysregulation with psychopathology. However, it will also be important to understand how these factors dynamically interact with each other in their prediction of anxiety and mood disorders. A number of investigations have demonstrated relationships among these emotion components in predicting functionality and disorder. For instance, firefighter trainees who reported greater understanding of their emotions were more able to effectively manage a series of live-fire exercises (evidenced by clearer thinking and fewer instances of "blanking out") than those with lower levels of understanding (Gohm, Baumann, & Sniezek, 2001). Also, individuals were more likely to effectively manage their intense emotional experiences when they could differentiate the emotions being experienced (Feldman Barrett, Gross, Conner Christensen, & Benvenuto, 2001). Cognitions regarding induced moods (i.e., negative reactivity) have also been found to mediate the effects of distraction (i.e., maladaptive management) on mood (Siemer, 2005). Further, the effects of intensity on negative outcomes such as depression have been found to be fully mediated by management variables such as avoidance or poor coping (Lynch et al., 2001).

Also important to understanding the interrelationships among emotion dysfunction components will be their temporal delineation in predicting psychopathology (Cole et al., 2004; Gross, 1998; Kring & Werner, 2004). Gross (1998) has argued for the importance of identifying dysregulation patterns along the emotion generative process and has demonstrated distinctions in regulation strategies that occur prior to the elicitation of emotion (i.e., antecedent-focused regulation strategies) and those that occur once an emotion has been evoked (i.e., response-focused regulation strategies). The emotion dysreguation model of Mennin and colleagues (e.g., Mennin et al., 2005) argues that, for GAD, the interaction of heightened intensity and poor understanding of emotions may instigate negative reactivity regarding an emotional state. This negative reactivity would then beget maladaptive management. However, other temporal relationships are possible as well. For instance, individuals with GAD who actively avoid emotional stimuli through worrying, which, given its avoidant properties, may be seen as a form of maladaptive management, may experience increased intensity when next in contact with emotional stimuli (see Borkovec, Alcaine, & Behar, 2004). It is also likely, given the present specificity findings, that these factors have distinct patterns of relationships in predicting different disorders.

Limitations and future directions. The present investigation suffers from a number of methodological limitations. Most notably, the results are tentative because the proportions of variance accounted for by the components, particularly poor understanding and maladaptive management, are modest. This indicates that the measurement of these constructs could be improved. Indeed, these measures were largely ones of convenience given their common usage to assess emotional characteristics in clinical studies. Future studies should utilize more precise measures of these constructs. For instance, heightened intensity may be better captured by the Affect Intensity Measure (AIM; Larson & Diener, 1987) and maladaptive management might be better assessed by the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), which assesses a number of characteristics of emotional management difficulties. Also, the adoption of scales with fewer items would permit analysis of the hierarchical structure of this full item set. The CFA model in Study 2 was unable to be run with item-level data, which made it difficult to determine whether a higher-order model provided any further explanation than a lower-order model. Despite the adoption of more precise scales to assess components, the exclusive reliance on self-report measures remains problematic. More objective assessments will need to be utilized to measure emotional dysfunction independent of the reporter's biased opinion about his or her own ability. For instance, performance-based tests of emotional intelligence, such as the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), have been shown to be independent of rater bias and demonstrate stronger relationships with functional outcomes (Mayer et al., 2004). These instruments instruct individuals to complete tasks relevant to emotion-related skills rather than asking directly about perceived ability.

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If temporal relationships are to be investigated, more controlled studies will also be needed to effectively delineate momentary changes in emotion process (Cole et al., 2004). These investigations will require measurement of multiple channels of emotional responding, including subjective report as well as physiological and expressive-behavioral components (Lang, Cuthbert, & Bradley, 1998). Further, the focus on only four measures likely omits a number of important variables germane to the understanding of emotion dysfunction in psychopathology. Future investigations will be needed to assess other emotion variables not included in these analyses, such as perception of others' emotional displays (e.g., Pollak, Cicchetti, Hornung, & Reed, 2000). Finally, the use of college samples limits generalizability and thus necessitates replication in clinical samples, including diagnostic groups not included here.

The present investigation demonstrates that variables related to emotion and its dysregulation can be reliably distinguished and show both generalized and specific relationships to symptoms of GAD, MDD, and SAD. This research suggests that, as Watson (2005) and others (e.g., Brown et al., 1998) have argued, common emotional elements may account for the high levels of comorbidity in disorders such as GAD and MDD. However, specificity findings also emerged. GAD demonstrated unique relationships with factors of emotionality and emotion dysregulation compared to MDD, which displayed similar emotion-characteristic correlates with SAD. These specificity findings suggest that grouping disorders together (e.g., GAD and MDD) based on higher-order allegiance alone may discount specific relationships among lower-order emotion components. However, these findings are clearly tentative and future research into the role of emotional factors in anxiety and mood psychopathology is necessary.

It will also be important to determine the differential role of emotion variables compared with other related constructs that have shown to be important in predicting anxiety and mood disorders. Experiential avoidance has been defined as an unwillingness to remain in contact with internal experiences (Hayes, Strosahl, & Wilson, 1999), particularly those characterized by emotional intensity or valence (cf. Mennin, 2005). Individuals with GAD have reported state (Mennin et al., 2005; study 3) and trait (Salters-Pedneault et al., in press) levels of difficulty accepting experienced emotions. In addition, Roemer et al. (2005) found that experiential avoidance was closely related to fear of negative emotions (i.e., negative reactivity) and demonstrated similar relationships to GAD. Further, preliminary investigations have demonstrated unique relationships between emotion-related deficits and poor mindfulness (e.g., Salters-Pedneault, Roemer, & Mennin, 2005) as well as cognitive inflexibility (e.g., Fresco, Mennin, Heimberg, & Hambrick, Submitted for publication) in predicting anxiety and mood disorders. In addition to increasing our understanding of these psychopathologies, the delineation of emotion dysregulation components may shed light on treatment-resistant anxiety and mood disorders (Samoilov & Goldfried, 2000). Indeed, treatments that stress emotion factors (Linehan, 1993) and the allowance and acceptance of emotional experiences (Hayes et al., 1999; Segal, Williams, & Teasdale, 2002) have begun to gain prominence. Approaches utilizing a functional emotions perspective have, more recently, been applied to treatment-resistant anxiety (e.g., Mennin, 2004; Roemer & Orsillo, 2005) and mood (e.g., Greenberg & Watson, 2005; Hayes et al., in press) disorders, as well. Although quite a bit more work is necessary, the study of emotion and its dysregulation in the psychopathology and treatment of anxiety and mood disorders has the potential to strengthen our approaches to these complex and debilitating disorders.

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