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Emotion regulation deficits in generalized anxiety disorder, social anxiety disorder, and their co-occurrence

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ABSTRACT

Preliminary evidence supports the role of emotion-related deficits in generalized anxiety disorder (GAD), including heightened emotional intensity, poor understanding of emotion, negative cognitive reactivity to emotions, and maladaptive emotion management. However, questions remain concerning the specificity of these emotion-related deficits compared to highly comorbid conditions such as social anxiety disorder (SAD). In the current study, 113 undergraduate students were administered measures of GAD, SAD, and emotion-related factors in order to clarify relationships among these variables. In univariate analyses, presence of SAD did not significantly impact the association between GAD and the emotion-related measures. Further, a discriminant function analysis revealed that emotional intensity and impaired regulation strategies provided the greatest discrimination between groups and best predicted a diagnosis of GAD (regardless of SAD comorbidity). Although their discriminatory ability was weaker, poor emotional understanding best predicted a diagnosis of SAD (regardless of GAD comorbidity), and non-acceptance of emotions best predicted comorbid GAD and SAD.

Unlike other anxiety disorders, GAD lacks overt markers, such as behavioral avoidance of recurrent objects or situations as in specific or social phobias. Rather, symptoms occur primarily internally, thus making them difficult to observe. However, establishing worry as its central feature has improved both understanding and reliability of GAD (Mennin, Turk, & Heimberg, 2004). Delineation of the function of worry has shown the most promise in furthering our understanding of the disorder. Borkovec's *avoidance theory* (e.g., Borkovec, Alcaine, & Behar, 2004) posits that worry in GAD serves to decrease aversive imagery and physiological hyperarousal associated with negative emotion. Evidence for this conceptualization of worry in GAD has been found in a number of studies which demonstrate that worry is verbal–linguistic, as opposed to imagery-based, and during worry physiological arousal appears to be invariable on indices of heart rate and skin conductance. Borkovec and colleagues extrapolate from these findings that the worry process may become negatively reinforced by the reduction of autonomic arousal (Borkovec et al., 2004).

The avoidance function of worry may best be explained by the perceived aversive nature of emotional experience in GAD patients. When asked about reasons for worrying, individuals with GAD were distinguished from non-anxious controls by the greater likelihood to endorse that they engaged in worry to avoid thinking about more emotional topics (Borkovec & Roemer, 1995). An important question then is *why* individuals with GAD want to avoid emotional experience. One possibility may result from the nature of how emotions are generated and regulated (Gross, 1998). The relationship of emotional deficits to psychopathology has received increasing attention (e.g., Kring & Bachorowski, 1999). Much of this work has focused upon the functional role of emotions and has drawn considerably from findings in the basic affect sciences (Ekman & Davidson, 1994).

Mennin, Heimberg, Turk, and Fresco (2005) have proposed that GAD is characterized by significant deficits in emotional experience and regulation. In particular, they argue that individuals with GAD have difficulties in four components of emotion functioning. Specifically, individuals with GAD experience emotions with *heightened intensity* compared to persons without GAD. Second, individuals with GAD experience marked difficulties identifying, describing, and clarifying their emotional experiences (i.e., *poor understanding*). Third, they are prone to greater *negative cognitive reactivity* to emotions by holding catastrophic beliefs about the consequences of both negative and positive emotions and endorsing more difficulty attending to and allowing emotional experience to unfold. Fourth, individuals with GAD struggle to manage or soothe themselves when they experience negative emotions (i.e., *maladaptive management*). Within this model,

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maladaptive emotion management strategies in GAD can be classified either as difficulties modulating emotional experiences or as maladaptive attempts to control or suppress emotional experience.

Empirical evidence supports this theoretical perspective on GAD. An initial series of studies provided a test of this model (Mennin et al., 2005). In the first study, college students with and without GAD (assessed by self-report) were compared on their responses to a battery of measures assessing aspects of emotion. GAD participants reported increased intensity, poorer clarity, greater negative reactivity, and poorer management of emotions compared to control individuals (Study 1). In a follow-up study, these findings were replicated with a clinical sample of individuals who had received a primary diagnosis of GAD (Mennin et al., 2005; Study 2). In a third study, GAD students underwent a mood induction. Following a negative mood induction, control participants were able to report more acceptance of these emotions, greater clarity of feeling, and greater belief that they were able to change their mood state than participants with GAD (Mennin et al., 2005; Study 3). Evidence also suggests that specific regulation deficits, including diminished access to effective regulation strategies and poor ability to engage in goal-pursuit behavior when distressed, are associated with GAD and chronic worrying (Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006). In addition, Mennin et al. (2005; Studies 1 and 2) found that emotion-related deficits predicted a diagnosis of GAD above the effects of worry, anxiety and depression.

Despite this preliminary support for the emotion regulation perspective on GAD, Turk, Heimberg, Luterek, Mennin, and Fresco (2005) found that a number of these deficits were not specific to GAD. Individuals with GAD reported greater emotion intensity and negative reactivity to sad emotions than individuals with SAD and non-anxious controls; however, individuals with SAD indicated being less expressive of positive emotions, paying less attention to their emotions, and having more difficulty describing their emotions than individuals with GAD or non-anxious controls. Thus, emotion deficits may not be entirely specific to this disorder, and may characterize other forms of psychopathology such as social anxiety disorder (SAD). Similar to these findings, other investigators (Salovey, Stroud, Woolery, & Epel, 2002) have also shown a link between SAD and deficiencies in emotion regulatory ability.

The present study sought to extend the findings reported in previous work examining emotion-related deficits in GAD and SAD (Turk et al., 2005). A number of limitations regarding the Turk et al. (2005) paper suggest further investigation is warranted. First, participants did not undergo diagnostic interviews to confirm the diagnosis; rather, a self-report measure was used to identify an analogue sample. In this study, we used a structured clinical interview to ascertain diagnoses. Second, GAD and SAD commonly co-occur (12-month prevalence tetrachoric correlation = .47; Kessler, Chiu, Demler, & Walters, 2005). Thus, it is important to delineate emotion-related deficits in a comorbid group compared to non-comorbid GAD and SAD groups (as well as non-anxious individuals). Third, given that the measures of emotion intensity and management used in the Turk et al. study were not designed to denote dysfunction, the inclusion of instruments that have previously been utilized in clinical populations may provide more generalizable results.

The primary goals of this study were to examine reported differences in emotion intensity, understanding, attendance and acceptance, and regulation strategies among individuals with generalized anxiety disorder (GAD), social anxiety disorder (SAD), their co-occurrence, or neither condition. We hypothesized that GAD participants (regardless of co-occurring SAD), when compared to individuals with SAD (without GAD) or control participants, would report experiencing: (1) greater emotion intensity; (2) poorer understanding of their emotions; (3) greater negative cognitive reactivity as indexed by more difficulty attending to (i.e., awareness) and accepting emotions, and (4) maladaptive management of emotions as indexed by a difficulty in accessing effective regulatory strategies. We also hypothesized that emotion-related deficits, particularly emotional intensity, would better predict a diagnosis of GAD (with or without SAD) than SAD, in a discriminant function analysis.

1. Method

1.1. Participants

Undergraduate students at a northeastern university responded to recruitment efforts in an introductory psychology course or through campus-posted flyers and participated for payment. All participants were administered the GAD and SAD sections of the Anxiety Disorders Interview Schedule-IV (ADIS-IV; Di Nardo & Barlow, 1994) to confirm eligibility. ADIS-IV interviewers were upper-level graduate students who had been trained specifically in a year-long psychopathological diagnostic assessment seminar taught by the first author. Participants meeting criteria for GAD or SAD were invited to participate in the study. Participants who did not meet criteria for GAD or SAD and who scored below a 3 on a Clinician's Severity Rating of distress and impairment (CSR; range of 0–8 with a 4 denoting clinically significant symptom severity; Di Nardo & Barlow, 1994) due to worry or social anxiety were invited to participate in the study as control participants.

Thirty-one participants met criteria for GAD without SAD (9 male, 22 female), 18 participants met criteria for GAD and SAD (6 male, 12 female), 20 participants met criteria for SAD alone (6 male, 14 female) and 44 participants were classified as controls (18 male, 26 female). All diagnoses used to form groups were primary (CSR $\geq$ 4). Further, secondary diagnoses of GAD and SAD were not allowed in the SAD and GAD only groups, respectively. However, other disorders were allowed as secondary diagnoses as long as they were deemed to be of lesser severity (at least 1 CSR point below primary disorder). The mean age of the sample was 21.28 years (SD = 4.01) and was evenly distributed across the four groups without significant differences. Participants' self-reported race/ethnicity was as follows: 54.86% Caucasian (N = 62), 16.81% African-American (N = 19), 7.96% Hispanic/Latino (N = 9), 13.27% Asian/Pacific Islander (N = 13), and 2.65% Middle Eastern descent (N = 2) and 4.42% (N = 5) declined to report this information. Ethnic background was equally distributed in study groups.

1.2. Procedure and measures

Participants who met criteria consented to participate in the study received the ADIS interview and then completed the Affect Intensity Measure (AIM; Larson & Deiner, 1987) and the Difficulties with Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). Participants were then debriefed and paid $15 for participation in the study.

The AIM (Larson & Deiner, 1987) is a 40-item measure that assesses the intensity and reactivity in which respondents typically experience positive and negative emotions. The 10-item subscale, AIM-N, designed to assess the intensity of negative emotional experiences was used in this study. Participants indicate how often they experience specific emotional reactions to situations using a 6-point scale, where 1 is *never*, 2 is *almost never*, 3 is *occasionally*, 4 is *usually*, 5 is *almost always*, and 6 is *always*. Larson and Deiner (1987) report a test–retest reliability of .81 for the AIM after a 3-month interval. Internal consistency is high for the AIM ($.90 \leq \alpha \leq .94$) and construct validity has been