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Perceived Emotion Control Moderates the Relationship between Neuroticism and Generalized Anxiety Disorder

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Abstract

The relationships between neuroticism, perceived emotion control, and generalized anxiety disorder (GAD) severity were examined in 293 individuals diagnosed with GAD at a specialty anxiety disorders clinic. Hierarchical regression analyses performed within a structural equation modeling framework revealed that (1) neuroticism and perceived emotion control both predicted a latent variable of GAD in the expected direction, and (2) perceived emotion control moderated the relationship between neuroticism and GAD severity, such that lower levels of perceived emotion control were associated with a stronger relationship between neuroticism and GAD severity. The other dimensions of perceived control (i.e., stress and threat control) did not moderate the effect of neuroticism on GAD severity. The findings are discussed with regard to their implications to conceptual models of the psychopathology of GAD, and theory-based differential relationships between dimensions of vulnerability, perceived control, and anxiety disorders.

Keywords

neuroticism; perceived control; generalized anxiety disorder; interaction

Generalized anxiety disorder (GAD) is characterized by persistent, excessive, and uncontrollable worry across multiple life domains (e.g., work, school, family, health, finances; American Psychological Association, 2013). This worry is accompanied more days than not by distressing symptoms of physiological tension and arousal. GAD is one of the most commonly reported mental disorders in primary care settings (Wittchen & Hoyer, 2001), and epidemiological studies suggest that roughly 5% of individuals will be affected by GAD at some point in their lives (Kessler et al., 2005). GAD exerts significant costs on society, which include decreased work productivity and attendance, over-utilization of healthcare resources, and the associated economic burden of medical and psychiatric treatment (Hoffman, Dukes, & Wittchen, 2008; Wittchen, 2002).

Several temperamental and personality factors have been linked to the onset and maintenance of GAD (Brown, 2007). Of these factors, neuroticism, or the tendency to experience negative emotions in response to stress, has garnered the most evidence as a

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temperamental vulnerability for emotional disorders (i.e., mood and anxiety disorders) (Brown, 2007). Specifically, neuroticism has demonstrated moderate to strong positive associations with all of the anxiety and mood disorders, with the strongest direct effect on GAD and depression (e.g., Brown, 2007; Brown & Naragon-Gainey, 2013; Kotov, Watson, Robles, & Schmidt, 2007). Although cross-sectional methodologies have typically been employed to demonstrate these relationships, a few longitudinal studies have yielded similar results. For example, one study of a large clinical sample assessed at intake and two-year follow-up revealed that higher initial levels of a neuroticism construct were associated with less improvement in GAD symptoms over time (Brown, 2007). Despite its robust role in the onset and maintenance of emotional disorders, conceptual models (e.g., Barlow, 2000, 2002; Chorpita & Barlow, 1998) suggest that neuroticism is not the sole risk factor for emotional disorders.

Barlow's (2000, 2002) triple vulnerability model posits that three vulnerabilities, broadly representing genetic/temperamental, psychological, and environmental influences, contribute to the development of emotional disorders. *General biological vulnerability* refers to relatively stable tendencies to experience positive and negative emotions. This disposition is theorized to be genetically based and to correspond to common temperamental constructs (e.g., neuroticism, extraversion). In addition to this temperamental vulnerability, individuals may also have a *general psychological vulnerability* that is the product of stressful childhood experiences, such as harsh or unpredictable early environments (e.g., authoritarian or inconsistent parenting styles). This general psychological vulnerability may manifest as a sense of unpredictability or lack of control over life events and emotions. In the context of situational stressors, individuals with both general vulnerabilities are more likely to develop emotional disorders (e.g., GAD, depression). Finally, a *disorder-specific psychological vulnerability* develops through learning experiences that emphasize the potential danger of certain situations, objects, or internal experiences (e.g., thoughts, memories, physical sensations). For example, some individuals may learn through parental modeling that unexplained somatic sensations are dangerous and may indicate illness (e.g., rapid heartbeat) or that specific situations or objects should be feared (e.g., mice). Others may learn through aversive experiences (e.g., being bitten by a dog) that certain situations, objects (e.g., dogs) or internal states demand their attention as likely sources of distress, regardless of the level of objective threat. Therefore, the specific psychological vulnerability, in the presence of the two general vulnerabilities, influences the expression of a particular anxiety and/or mood disorder. Although Barlow's model suggests specific psychological vulnerabilities for several mood and anxiety disorders (e.g., beliefs that physical sensations are dangerous in panic disorder, that certain thoughts or impulses are dangerous in obsessive-compulsive disorder, or that social evaluation is dangerous in social anxiety disorder), the model does not propose a specific vulnerability for GAD. In contrast, the model claims that the two general vulnerabilities alone may be sufficient for developing GAD.

Perceived control functions as an important component of the *general psychological vulnerability* that may increase the risk of developing GAD in those with elevated levels of neuroticism (Barlow, 2002). Individuals who have negative emotional reactions to stressful internal and external events and who also view these reactions as uncontrollable may

develop worry as a strategy to avoid highly uncomfortable emotions. Indeed, evidence suggests that individuals with GAD report intense subjective experiences of emotions, have difficulty identifying and understanding their emotions, and perceive these emotions as aversive (Mennin, Heimberg, Turk, & Fresco, 2005). Worry has been conceptualized as a primarily verbal thought process used to limit the occurrence and duration of more emotionally evocative mental imagery (Borkovec & Inz, 1990), which would in turn limit the experience of strong emotions. In one experimental manipulation, worrying before imagining giving a speech resulted in decreased physiological reactivity (i.e., heart rate) when exposed to speech-related images, as compared to engaging in relaxed or neutral thoughts (Borkovec & Hu, 1990). In other words, individuals with GAD may engage in the worry process to suppress autonomic arousal triggered by negative mental imagery (Borkovec & Hu, 1990). Other researchers have extended this theory to suggest that worry serves the purpose of limiting reactivity to abrupt changes in emotional states, such as going from a neutral or positive emotion to a negative emotion (Contrast Avoidance model; Newman & Llera, 2011). For example, Llera and Newman (2010) found that individuals with GAD who were randomly assigned to a worry induction before being exposed to “fearful” film clips demonstrated lower heart-rate variability and self-reported negative affect than those who were assigned to a relaxation induction. While worry led to an initial increase in physiological arousal, that arousal was maintained by individuals with GAD possibly to avoid experiencing a negative emotional state that contrasts with the previously experienced emotional state (i.e., relaxed, euthymic, or even positive). Hence worry may be used as a method to control the experience of sharp increases in negative arousal that might occur in the future. Central to these theories and experimental findings is the importance of regulating or controlling emotional responses for individuals with GAD, especially emotions that are perceived as unwanted.

Low levels of perceived control over anxiety-related events, such as internal experiences associated with anxiety or external events that trigger fear or anxiety (as measured by the Anxiety Control Questionnaire, ACQ; Rapee et al., 1996) have demonstrated significant positive associations with both worry and GAD (Chapman, Kertz, & Woodruff-Borden, 2009; Gould & Edelstein, 2010; Stapinski, Abbott, & Rapee, 2010). Furthermore, individuals who received psychotherapeutic treatment for GAD have shown increases in perceived control (e.g., Treanor, Erisman, Salters-Pedneault, Roemer, & Orsillo, 2011). Extending these findings, one recent study of adolescents demonstrated that perceived control predicted worry and GAD severity above and beyond the effects of age, gender, and negative affect (Frala, Leen-Feldner, Blumenthal, & Barreto, 2010). Likewise, Brown and Naragon-Gainey (2013) demonstrated that both perceived control and neuroticism predicted GAD in a large clinical sample (700 outpatients with anxiety and mood disorders). Specifically, perceived control exerted significant negative direct effects on the latent variables of GAD and obsessive-compulsive disorder (but not depression or social phobia) when controlling for neuroticism and extraversion.

The 30-item Anxiety Control Questionnaire (ACQ; Rapee et al., 1996) was used in these studies to measure perceived control over anxiety-related events and emotions (i.e., external threats and internal emotions). Although the original evaluation of the ACQ suggested a two-factor structure (Rapee et al., 1996), a subsequent, more extensive psychometric

analysis revealed a latent structure comprised of a single higher-order factor of Perceived Control and three lower-order factors: Threat Control (i.e., control over the occurrence of and escape from externally threatening events), Emotion Control (i.e., control over internal emotional experiences of anxiety and distress), and Stress Control, (i.e., control over one's ability to cope with and regulate emotional reactions to stressful events) (Brown, White, Forsyth, & Barlow, 2004). A limited body of research suggests that these lower-order factors of perceived control may exhibit differential effects on anxiety outcomes and may have relevance to the prediction of these outcomes as moderating variables. For example, low perceived control over threatening events in the environment (Threat Control) has been shown to strengthen the relationship between anxiety sensitivity and agoraphobia in individuals with panic disorder (White, Brown, Somers, & Barlow, 2006); i.e., the influence of anxiety sensitivity on agoraphobia severity was stronger in individuals with lower perceived control over threatening events. Emotion and Stress Control, on the other hand, did not moderate this relationship. While a moderational effect of perceived control on GAD outcomes has not yet been tested, Brown and Naragon-Gainey's (2013) analyses provided evidence for the specificity of Emotion Control to GAD severity. Specifically, Emotion Control was found to be more strongly predictive of GAD in a negative direction (holding neuroticism and extraversion constant) than either Threat or Stress Control (i.e., higher levels of perceived emotion control were associated with lower GAD severity).

Although Barlow's triple vulnerability model does not specify the precise nature of the effect of perceived emotion control on GAD, some evidence for an additive model has recently emerged (e.g., Brown & Naragon-Gainey, 2013). The results of this study, however, may have underestimated the role of perceived emotion control in GAD due to a failure to examine its moderating influence on the relationship between neuroticism and GAD (i.e., the effects of neuroticism on GAD are strongest in individuals with low perceived emotion control). Furthermore, the triple vulnerability model asserts that low perceived control over negative emotional states intensifies and/or prolongs uncomfortable affective states or reactions to stressful events. Individuals with GAD then cope with this augmented negative emotional experience by entering a state of preparation for negative outcomes, or anxious apprehension, which is often futile (Barlow, 2002). Therefore, perceived emotion control may not merely contribute additively to the prediction of GAD severity beyond the effect of neuroticism, but instead may influence the very nature of the relationship between the two variables, such that the strength of the neuroticism's direct effect on GAD severity increases as perceived emotion control decreases. While studies to date have examined the additive effect of perceived emotion control on GAD severity in adolescent and adult samples (e.g., Brown & Naragon-Gainey, 2013; Frala et al., 2010), no study has explored this potential moderating effect.

Current Study

Taken together, these results suggest that further research is needed to elucidate the nature of the effect of perceived control on GAD severity (i.e., additive, moderational). Although not explicitly posited by conceptual models (e.g., Barlow, 2002), the potential moderating role of perceived control on GAD severity warrants further investigation. Moreover, there is some evidence that the different subdimensions of perceived control have differential effects

on emotional disorders; for example, perceived threat control was found to be particularly relevant to severity of agoraphobia in individuals with panic disorder (White et al., 2006). Worry is a primarily verbal cognitive process believed to help individuals avoid negative mental imagery that would otherwise activate strong negative emotions (Borkovec & Inz, 1990). Therefore, perceived emotion control (rather than perceived threat or stress control) may be specifically germane to GAD, a disorder characterized by worry and emotion dysregulation.

The present study seeks to examine the nature of the relationship between neuroticism, perceived emotion control, and GAD severity in a clinical sample. Specifically, it is hypothesized that (1) both higher levels of neuroticism and lower levels of perceived emotion control are associated with more severe GAD, (2) the relationship between neuroticism and GAD severity is moderated by perceived emotion control, such that the relationship between neuroticism and GAD severity is strongest at lower levels of perceived emotion control, and (3) the direct and moderating effects will be specific to perceived emotion control (i.e., will not be significant for perceived threat or stress control). From a conceptual standpoint, perceived emotion control was selected to moderate neuroticism's effects on GAD severity (as opposed to consideration of a neuroticism as the moderator) based on prior theory (i.e., in the Barlow, 2002, model, neuroticism is a genetically based temperamental construct that precedes early psychological experiences that lead to low perceived control), and the fact that neuroticism is firmly entrenched in the empirical and conceptual literature as a risk factor for GAD and the emotional disorders in general (thus, a more psychologically based variable such as perceived control may moderate this well-established relationship). In sum, the proposed interaction effect is based on the substantive premise that emotional reactivity to stress (i.e., neuroticism) will exert stronger effects on GAD severity in individuals who feel they have difficulty controlling their emotions.

Method

Participants

The sample consisted of 293 consecutive outpatients with a current principal or additional diagnosis of GAD (56% principal) who presented for psychological evaluation and treatment at the Center for Anxiety and Related Disorders at Boston University (CARD). In those with an additional diagnosis of GAD, the most common principal diagnoses were: social phobia (13%), panic disorder with agoraphobia (11.3%), OCD (4.4%), and MDD (3.4%). The most common comorbid clinical diagnoses, collapsing across those with GAD as a principal or additional diagnosis, were: social phobia (46.8%), MDD (27%), OCD (13%), posttraumatic stress disorder (3.8%), and dysthymia (1.7%). Data were collected as part of a larger study of the classification of anxiety and mood disorders at CARD. The majority of the sample was female (65.9%), and the average age was 32.5 ($SD = 12.3$, range = 18 to 67). Most of the sample identified their race as Caucasian (89.4%), and a smaller proportion identified as Asian (5.8%) and African-American (4.8%). Potential participants were excluded from this study if they reported two or more hospitalizations for severe psychopathology (e.g., psychosis) within the previous five years, organic brain syndrome

within the past six months, or current suicidality or homicidality that would warrant crisis intervention.

Measures

GAD severity—A latent variable of GAD severity was formed using the clinical dimensional rating of the controllability of participants' worry (ranging from 0 [*never/no difficulty*] to 8 [*constantly/extreme difficulty*]) from the Anxiety Disorders Interview Schedule for *DSM-IV*: Lifetime version (Di Nardo, Brown, & Barlow, 1994), as well as two self-report indicators: the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and the Generalized Anxiety Disorder Screener (GAD-7; Spitzer, Kroenke, Williams, & Löwe, 2006). These measures are described below.

Anxiety Disorders Interview Schedule for DSM-IV: Lifetime version (ADIS-IV-L):

Diagnoses were established using the ADIS-IV-L. The ADIS-IV-L is a semi-structured interview that comprehensively assesses current and lifetime *DSM-IV* anxiety, mood, and substance use disorders, as well as selected somatoform disorders (e.g., hypochondriasis); it also screens for the presence of other disorders (e.g., psychosis). For each diagnosis, clinicians assign a clinical severity rating (CSR) that captures the degree of distress and interference in functioning associated with each disorder, which ranges from 0 (*none*) to 8 (*very disturbing/disabling*). Disorders that meet all formal *DSM-IV* criteria are rated at a CSR of 4 (*definitely disturbing/disabling*) or higher. When two or more diagnoses are assigned, the principal diagnosis is the one receiving the highest CSR. The ADIS-IV-L has demonstrated good to excellent interrater reliability for diagnosing current and lifetime anxiety and mood disorders (range of *ks* for current principal diagnoses = .67 to .86, except dysthymia *k* = .22; Brown, Di Nardo, Lehman, & Campbell, 2001). In addition, good interrater agreement was obtained for GAD collapsing principal or additional diagnoses (i.e., any current clinical diagnosis of GAD; κ = .65) as well as for dimensional interviewer ratings of uncontrollability of worry (r = .78) (Brown et al., 2001).

Penn State Worry Questionnaire: The PSWQ is a 16-item self-report measure of worry (e.g., “Many situations make me worry”; Meyer et al., 1990). Items are rated on a 5-point Likert scale ranging from 1 (*not at all typical of me*) to 5 (*very typical of me*) and were summed in the present study (with reverse-scoring as appropriate) to calculate the PSWQ total score. The PSWQ has demonstrated a unidimensional structure, high internal consistency, good test-retest reliability, and convergent and discriminant validity in clinical (Brown, 2003; Brown, Antony, & Barlow, 1992) and nonclinical (Meyer et al., 1990) samples. In the current sample, the PSWQ demonstrated good internal consistency (α = .86).

Generalized Anxiety Disorder Screener: The GAD-7 is a 7-item self-report measure designed to identify clinical cases of *DSM-IV* GAD (Spitzer et al., 2006). Participants rated the degree to which they have been bothered by worry (e.g., “Not being able to stop or control worrying”) and its associated symptoms (e.g., “Trouble relaxing,” “Becoming easily annoyed or irritable,” etc.) in the last two weeks using a 4-point Likert scale ranging from 0 (*not at all*) to 3 (*nearly every day*). Item scores were summed to calculate the GAD-7 indicator. The GAD-7 has exhibited a unidimensional factor structure, high internal

consistency, good test-retest reliability and construct validity in primary care (Spitzer et al., 2006) and community (Löwe et al., 2008) samples. The GAD-7 demonstrated good internal consistency in the current sample ($\alpha = .87$).

Neuroticism—Neuroticism was assessed by the NEO Five-Factor Inventory (NFFI; Costa & McCrae, 1992), a 60-item self-report measure of the five-factor model of personality (e.g. neuroticism, extraversion, openness, etc.). Items consisted of self-descriptive statements rated on a 5-point Likert scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*). This study calculated the Neuroticism scale (NFFI-N) by summing the 12 NFFI items that assess patients' tendency to experience negative emotional states (e.g. "When I'm under a great deal of stress, sometimes I feel like I'm going to pieces"). Each of the five scale domains has demonstrated adequate internal consistency (specifically, Neuroticism $\alpha = .86$; Costa & McCrae, 1992), and the posited latent structure of the NFFI has been supported in clinical samples (e.g., Rosellini & Brown, 2011). The NFFI-N demonstrated good internal consistency in the current sample ($\alpha = .75$).

Perceived Control—Perceived control was measured using the Anxiety Control Questionnaire – Revised, a 15-item self-report measure of perceived control (ACQ-R; Brown et al., 2004). Participants rated the extent to which they feel that they have control over anxiety and anxiety-related events using a 6-point Likert scale ranging from 0 (*strongly disagree*) to 5 (*strongly agree*), with higher scores indicating greater perceived control. Brown and colleagues (2004) found a three-factor solution (Emotion, Threat, and Stress Control), as well as a single higher-order factor representing a broader dimension of Perceived Control. The Emotion Control subscale consists of 5 items that measure perceived ability to control emotional reactions related to anxiety and distress (e.g., "I am able to control my level of anxiety"). The Threat Control subscale contains 6 items that measure the belief that the occurrence of and escape from threatening events is outside of one's control (e.g., "There is little I can do to change frightening events" [reverse-coded item]). The Stress Control subscale consists of 4 items that reflect perceived ability to cope with and regulate emotional reactions to stressful situations (e.g., "When I am put under stress, I am likely to lose control" [reverse-coded item]). All three lower-order factors demonstrated acceptable internal consistency in Brown and colleagues' (2004) sample ($\rho s = .71$ to $.73$; in the current sample, $\alpha s = .71$ to $.78$) and contributed uniquely to the prediction of autonomic anxiety and depression. Further, the measurement properties of the ACQ-R have been found to be invariant between sexes and between clinical and nonclinical samples.

Procedure

Participants completed a brief initial screen via telephone as part of routine CARD procedures. Eligible participants then provided informed consent and completed the ADIS-IV-L and a battery of self-report questionnaires. The ADIS-IV-L was administered by trained licensed psychologists, post-doctoral fellows, and doctoral-level clinical psychology graduate students. Details of the ADIS-IV-L certification process are described in Brown et al. (2001). The institutional review board of Boston University approved all study procedures.

Data Analysis

The raw data were analyzed using a latent variable software program and maximum-likelihood minimization functions (Mplus 6.0; Muthén & Muthén, 1998-2011). Missing data and non-normality were accommodated in all analyses using robust maximum likelihood estimation (Raykov, 2005). Goodness of fit of the model was evaluated using the root-mean-square error of approximation (RMSEA) and its 90% confidence interval (CI) and test of close fit (CFit), the Tucker-Lewis index (TLI), the comparative fit index (CFI), and the standardized root-mean-square residual (SRMR). Multiple goodness-of-fit indices were evaluated to examine various aspects of model fit (i.e., absolute fit, parsimonious fit, fit relative to the null model; cf. Brown, 2015). Acceptable model fit was defined in part using Hu and Bentler's (1999) criteria: RMSEA values close to 0.06 or below (with a nonsignificant CFit), TLI and CFI values close to .95 or above, and SRMR values close to .08 or below. Model acceptability was further evaluated by the presence or absence of salient localized areas of strain in the solution (i.e., modification indices), and the strength and interpretability of the parameter estimates.

Results

Means, standard deviations, and correlations for all observed variables are presented in Table 1. A confirmatory factor analysis (CFA) was conducted prior to specifying the structural model of interest to ensure an acceptable measurement model. The CFA examined a measurement model of two observed variables (i.e., neuroticism and perceived emotion control), and one latent variable (i.e., GAD severity) defined by three indicators. This measurement model provided a good fit to the data, $\chi^2(6) = 8.07, p = 0.23$, SRMR = .07, RMSEA = 0.03 (CFit $p = .62$), TLI = 0.96, CFI = .98. Inspection of modification indices and standardized residuals revealed no localized areas of strain in the model. Factor loadings of the three GAD indicators onto the latent variable of GAD severity were all statistically significant (λ s ranged from .44 to .72; $ps < .001$). CFAs examining measurement models respecified with the Threat and Stress Control indicators also revealed a good fit to the data, $\chi^2(6) = 9.03, p = 0.06$, $\chi^2(6) = 5.37, p = 0.25$, respectively.

As predicted, the Emotion Control subscale of the ACQ-R was negatively associated with neuroticism and the GAD factor ($r_s = -.30$ and $-.45$, respectively, $ps < .05$). At the zero-order level, the GAD – Emotion Control relationship was significantly stronger than the GAD – Threat Control ($r = .00, p = \text{n.s.}$) and GAD – Stress Control ($r = -.14, p = \text{n.s.}$) relationships, as confirmed by inferential tests of the differential magnitude of dependent correlations (Steiger's $z_s = -6.46$ and -5.13 , respectively, $ps < .01$; Steiger, 1980).

A hierarchical two-step analysis was conducted within the structural equation modeling framework to examine (1) the main effects of neuroticism and perceived emotion control on the latent variable of GAD, and (2) the moderating role of perceived emotion control on the relationship between neuroticism and GAD severity. Results of this analysis are reported in Table 2. The continuous predictor variable (neuroticism) and moderator variable (perceived emotion control) were mean-centered for the interaction effect analysis to decrease potential problems with multicollinearity that could impede maximum likelihood estimation, as well as to facilitate interpretation of the results. These mean-centered variables were then

multiplied to create an interaction term. After the main effects model was run, a second analysis was conducted to examine the interaction term's unique contribution to the prediction in GAD severity.

In the first analysis, significant main effects were obtained for both neuroticism and perceived emotion control on GAD severity in the expected directions, such that these variables collectively accounted for 30.4% of the variance in GAD severity ($f^2 = .43$) (see Table 2). In the second analysis, the interaction term was statistically significant ($p < .01$) and uniquely explained 7% of the variance in GAD severity ($f^2 = .11$).

Next, main and interaction effects models were tested using the Stress and Threat Control variables to examine the role of other subdimensions of perceived control in the relationship between neuroticism and GAD severity. In the Stress Control main effects model, both neuroticism and perceived stress control exerted significant direct effects on GAD severity ($ps < .01$). Collectively, these variables accounted for 19.6% of the variance in GAD severity ($f^2 = .25$). In the Threat Control main effects model, perceived threat control did not exert a significant effect on GAD severity ($\beta = -.10, p = .25$). Moreover, the interaction effect analyses examining the moderating influence of perceived threat and stress control were not significant, and the associated effect sizes were zero or close to zero ($f^2 = .00$ and .01 for Threat and Stress Control interaction terms, respectively).

To more fully convey the nature of the neuroticism $\times$ perceived emotion control interaction effect, model-implied estimates of GAD severity at high and low levels of neuroticism and perceived emotional control (mean $\pm$ one standard deviation) were plotted. As depicted in Figure 1, the strength of the relationship between neuroticism and GAD severity decreased as the level of perceived emotion control increased.

Discussion

This study examined the nature of the relationship between neuroticism, perceived emotion control, and GAD severity in a clinical sample. Results indicating direct effects of neuroticism and perceived emotion control on GAD severity are consistent with prior studies investigating the role of neuroticism and perceived control in predicting GAD (e.g., Brown 2007; Brown & Naragon-Gainey, 2013). Barlow's (2002) triple vulnerability model states that the presence of neuroticism and low perceived control places individuals at greater risk of developing anxiety or depression in the context of stressful events. Although this theory does not specify the nature of the relationship between neuroticism, perceived control, and emotional disorders (i.e., additive, moderational), structural equation modeling results from the present study extended the findings of Brown and Naragon-Gainey's (2013) examination of the triple vulnerability model; not only does perceived emotion control exert a direct effect on GAD severity when holding neuroticism constant, but it also moderates the relationship between neuroticism and GAD severity.

This study further hypothesized that the subdomain of perceived emotion control, rather than perceived stress or threat control, would be particularly relevant to the relationship between neuroticism and GAD severity, because GAD is inherently a disorder of emotion

dysregulation (Barlow, 2002). As previously discussed, individuals with GAD may attempt to regulate their emotional experiences or avoid emotionally evocative mental imagery of aversive or catastrophic events by engaging in the primarily verbal process of worry (Borkovec & Inz, 1990). Although Threat and Stress Control were related to GAD, the magnitude of these relationships was significantly weaker than for Emotion Control, as evidenced by inferential tests of differential magnitude of these associations. Nevertheless, perceived stress control demonstrated a significant direct effect on GAD severity, such that lower levels of perceived stress control were associated with higher GAD severity. In other words, individuals who believed that they have trouble managing their emotions specifically when they are “under stress” or in “difficult situations” were, in this sample, likely to have higher levels of GAD severity. Furthermore, neither of these two other dimensions moderated the neuroticism – GAD relationship. In this sample, perceived control over basic emotions themselves influenced neuroticism's predictive power for GAD, rather than perceived control over dangerous external events (Threat Control) or one's reaction to such threatening situations (Stress Control). This finding bolsters recent analyses (i.e., Brown & Naragon-Gainey, 2013) that demonstrated perceived emotion control's unique influence on the severity of GAD in individuals with high levels of negative affect. Hence, perceived emotion control has proven particularly germane to GAD and the strength of the neuroticism – GAD relationship. On the other hand, White and colleagues' (2006) study provided evidence that perceived threat control (and not perceived emotion or stress control) uniquely moderated the relationship between anxiety sensitivity and agoraphobia in patients with panic disorder. Collectively, these results support the notion that the various dimensions of perceived control have differential relationships with emotional disorders.

Given that worry has been implicated in the avoidance of emotionally evocative mental imagery (Borkovec & Inz, 1990) and the suppression of autonomic stress responses (Borkovec & Hu, 1990; Fisher & Newman, 2013), control of negative emotional responses seems particularly relevant to GAD. Indeed, one survey study examining potential functions of worry found that using worry as “distraction from more emotional topics” (p. 25) best discriminated those with GAD from both anxious and nonanxious comparison groups (Borkovec & Roemer, 1995). It may be that maladaptive beliefs about the consequences of experiencing strong negative emotions (e.g., “I will not be able to cope”) may cause people with GAD to be more sensitive to feeling negative emotions and to desire high levels of control over strong emotions. They may then engage in the worry process as an attempt to avoid (and thus control) future negative emotional experiences or external events that they predict will cause unmanageable emotional reactions.

The specific relevance of uncontrollability over negative emotional experiences in individuals with GAD also has bearings on the newer emotion dysregulation model of GAD (Mennin, Turk, Heimberg, & Carmin, 2004). This model's four basic components are that individuals with GAD experience heightened negative and positive emotionality, poorly understand their emotions, react negatively to their emotions, and employ maladaptive emotion regulation strategies in response to distressing emotions. Consistent with this theory, our study found higher neuroticism and lower perceived emotion control predicted higher GAD severity. The simultaneous experience of heightened negative emotionality and a perceived inability to self-soothe may set in motion a chain of negative reactions about

initial emotions (e.g., sadness, anger, anxiety). Individuals with GAD may then invoke the worry process to avoid these escalated and threatening emotions (cf. Borkovec, Alcaine, & Behar, 2004).

The current findings foster our understanding of the relationships between neuroticism, perceived emotion control, and GAD severity. While neuroticism and low perceived control both exert direct effects on GAD severity, in those with high levels of neuroticism, high perceptions of control over one's emotions may serve as a mitigating factor in the development of excessive worry and associated physical tension. The clinical significance of this study's results, however, should be interpreted with caution due to the small-to-medium effect size of the interaction effect ($f^2 = .11$), which was considerably smaller than neuroticism's and perceived emotion control's main effects on GAD (cf. Table 2). It is possible that the use of a clinical sample restricted the range of the study measures, which would attenuate the observed main and interaction effects. The small-to-medium interaction effect may thus represent a conservative estimate of the population effect relative to estimates that would be obtained in broader samples.

Moreover, some researchers have questioned the discriminant validity of neuroticism and worry. To differentiate these constructs at the conceptual level, neuroticism has been defined as a trait characteristic of heightened emotionality, whereas worry reflects a cognitive process of negative, self-referential thought that occurs in response to this elevated emotionality (Mennin & Fresco, 2013). Some individuals with high levels of neuroticism likely use worry as a cognitive regulation strategy; they may engage in repetitive worry to suppress uncomfortable emotional arousal (Borkovec et al., 2004) or to avoid experiencing sharp changes in emotional states (Newman & Llera, 2011). From an empirical standpoint, elaborate latent measurement and structural models have routinely found neuroticism to be factorially distinct from GAD (e.g., Brown & Naragon-Gainey, 2013; Rosellini & Brown, 2011), and have found neuroticism to predict the temporal course of GAD holding initial levels of GAD constant (Brown, 2007). Indeed, a meta-analytic examination of rumination, another form of negative self-referential processing (NSRP), found NSRP to be distinct from neuroticism (Olatunji et al., 2013), such that NSRP mediated the relationship between neuroticism and depression in a community youth sample followed from birth to adolescence (Mezulis, Priess, & Hyde, 2011). The results of this study indicated that low perceived controllability of anxiety-related emotions strengthened the association between high emotionality (i.e., neuroticism) and the tendency to engage in worry, potentially as a cognitive regulation strategy.

This study produced important evidence for the specific relevance of subdimensions of perceived control in understanding the relationship between temperamental vulnerability factors and various emotional disorders. A growing body of evidence supporting the unique role of perceived emotion control in the development and maintenance of GAD (Borkovec, Alcaine, & Behar, 2004; Brown & Naragon-Gainey, 2013; Mennin et al., 2005; Newman & Llera, 2011) suggests that treatment interventions for GAD may be improved by aiming to (1) increase the individual's understanding of emotions, (2) reduce negative attitudes about emotional experiences, and (3) provide effective emotion regulation strategies, such as acceptance, or allowing oneself to fully experience the natural course of negative emotions

without attempting to change the process in any way (Roemer & Orsillo, 2012). Treatment protocols that target emotional experiences and regulation strategies may improve our ability to effectively treat GAD. Recent developments such as acceptance-based behavioral therapy for GAD (ABBT; Roemer & Orsillo, 2012), emotion regulation therapy for GAD (ERT; Fresco, Mennin, Heimberg, & Ritter, 2013), and the Unified Protocol for Transdiagnostic Treatment of Emotional Disorders (Barlow et al., 2011), all of which are cognitive-behaviorally based approaches, represent promising steps in the direction of targeting emotion recognition and regulation in GAD.

To our knowledge, this was the first study to examine the moderating effects of perceived control on the relationship between neuroticism and GAD severity. In addition, a strength of this study was the use of a latent GAD outcome variable that was dimensional in nature and theoretically free of measurement error. Future studies could further benefit from employing dimensional latent variables for both neuroticism and perceived emotion control to provide a more balanced measurement of these constructs, perhaps by also utilizing measures of emotion regulation. Understanding the potentiating role of perceived emotion control on the relationship between neuroticism and GAD severity enriches our knowledge of how putative vulnerability factors interact to exacerbate GAD symptoms. However, given that these analyses were correlational in nature, neither causality nor directionality can be inferred from the results. Although the cross-sectional nature of the study was appropriate for a moderation model, future studies could strengthen these findings by using longitudinal methods to examine temperamentally vulnerable samples (i.e., high neuroticism). Such studies could help clarify temporal relationships among variables that have been widely implicated in the development of GAD (i.e., neuroticism, perceived emotion control; Barlow, 2002; Brown, 2007). Indeed, future longitudinal investigations may reveal that those with high neuroticism go on to experience GAD only after developing a sense of uncontrollability over their emotions. Recent evidence suggests that increases in perceived control are associated with better long-term outcomes in anxiety disorder symptoms (Gallagher, Naragon-Gainey, & Brown, 2014). Therefore, future experimental interventions may seek to examine the potentially beneficial effect of strengthening perceived control, and particularly perceived emotion control, on preventing or reducing pathological worry.

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Figure 1.

Nature of the interaction between neuroticism and perceived emotion control on GAD severity. NFFI = NEO Five-Factor Inventory; Emotion Control = Anxiety Control Questionnaire – Revised, Emotion Control subscale. Possible range of scores for GAD severity (y-axis) = 16 to 80. The Penn State Worry Questionnaire was the marker indicator for the GAD factor.

Table 1
Means, Standard Deviations, and Zero-order Correlations among Study Variables

	PSWQ	GAD7	GAD Rating	Neuroticism	Emotion Control	Threat Control	Stress Control	Mean	Standard Deviation
PSWQ	—							70.26	7.97
GAD7	.46	—						14.42	4.88
GAD Rating	.28	.50	—					5.01	0.91
NFFI – Neuroticism	.30	.03	.14	—				33.80	6.73
ACQ-R									
Emotion Control	-.35	-.24	-.24	-.30	—			4.45	3.40
Threat Control	-.14	-.19	-.12	-.30	.21	—		17.32	5.71
Stress Control	-.28	-.14	-.13	-.43	.39	.44	—	8.22	4.01

Note. PSWQ = Penn State Worry Questionnaire; GAD-7 = Generalized Anxiety Disorder Screener; GAD Rating = the *ADIS-IV-L* clinician rating of worry uncontrollability; NFFI = NEO Five-Factor Inventory; ACQ-R = Anxiety Control Questionnaire – Revised.

Values above/below |.11| are significant at the $p < .05$ level. $N = 293$.

Summary of Structural Regression Analyses for the Main and Interaction Effects of Neuroticism and Perceived Control on GAD Severity

Table 2

Model and predictor variable	GAD severity				
	B	SE	β	R^2	f^2
<i>EC – Main Effect</i>				.30**	.43
Neuroticism	.22**	.07	.26**		
Emotion Control	-.69**	.14	-.41**		
GAD Intercept	65.94**	2.45			
<i>EC – Interaction Effect</i>				.37**	.11
Neuroticism (N)	.23**	.06	.28**		
Emotion Control (EC)	-.76**	.13	-.47**		
N $\times$ EC	-.06**	.02	-.27**		
GAD Intercept	69.86**	.46			
<i>TC – Main Effect</i>				.16**	.19
Neuroticism	.30**	.08	.35**		
Threat Control	-.10	.08	-.10		
GAD Intercept	61.86**	3.68			
<i>TC – Interaction Effect</i>				.16**	.00
Neuroticism (N)	.03**	.01	.36**		
Threat Control (TC)	-.01	.01	-.09		
N $\times$ TC	-.00	.00	-.06		
GAD Intercept	70.15	.05			
<i>SC – Main Effect</i>				.20**	.25
Neuroticism	.24**	.07	.27**		
Stress Control	-.37**	.13	-.25**		
GAD Intercept	65.15**	2.93			
<i>SC – Interaction Effect</i>				.21**	.01

GAD severity						
Model and predictor variable	B	SE	B	β	R^2	f^2
Neuroticism (N)	.02**	.01		.28**		
Stress Control (SC)	-.04**	.01		-.26**		
N $\times$ SC	-.00	.00		-.12		
GAD Intercept	70.00**			.05		

Note. GAD = Generalized Anxiety Disorder; EC = Emotion Control subscale of the Anxiety Control Questionnaire – Revised (ACQ-R); TC = Threat Control subscale of the ACQ-R; SC = Stress Control subscale of ACQ-R.

* $p < .05$.
** $p < .01$.