The Interaction Between Positive and Negative Temperament in Anxiety and Depression: Examples From the Tripartite, Big Five, and Behavioral Inhibition/Activation Models

Master’s Thesis

Presented in Partial Fulfillment of the Requirements for the Degree Master of Arts in the Graduate School of The Ohio State University

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Abstract

Research has demonstrated that risk for psychological disorders does not lie in single factors or main effects, but rather in the interaction and co-action of multiple factors. Consistent with this, the Tripartite, Big Five, and Reinforcement Sensitivity models all posit the dual influence of positive and negative emotionality in risk for depression. Specifically, the Tripartite Model posits the dual influence of positive and negative affectivity (PA and NA, respectively), the Big Five Model posits the dual influence of extraversion and neuroticism (E and N, respectively), and the Reinforcement Sensitivity Model posits the dual influence of behavioral activation and inhibition (BAS and BIS, respectively). Research involving each of these theories suggests that positive and negative emotionality interact to predict depressive, and to some extent, anxious symptoms. To further understand the nature and reliability of this interaction, the present study provided a cross-sectional test of the interactions between the positive and negative emotionality dimensions of the Tripartite, Big Five, and Reinforcement Sensitivity models in a large sample of undergraduates (N = 1242). It was hypothesized that, for each set of temperament dimensions, at low positive emotionality, high negative emotionality would be more strongly associated with depressive and mixed anxious-depressive symptoms and that at high negative emotionality, low positive emotionality would be more strongly associated with anhedonic symptoms. Results indicated that the interactions between all three sets of
temperament variables are indeed associated with such symptoms, but that the exact nature of the association depends on the set of temperament constructs and the type of symptoms considered. Specifically, results indicated that the NA x PA interaction predicted all three types of symptoms such that high NA was more strongly associated with depressive and mixed anxious-depressive symptoms when PA was low and low PA was more strongly associated with anhedonic depressive symptoms when NA was high; that the N x E interaction predicted depressive and mixed anxious-depressive symptoms such that high N was more strongly associated with symptoms when E was low; and that while the BIS x BAS interaction also predicted depressive and mixed anxious-depressive symptoms, the form of this interaction was unexpected. Specifically, high BIS was more strongly associated with symptoms than low BIS when BAS was high. However, the expected pattern was found for anhedonic symptoms, when the interaction between BIS and the Reward Responsiveness facet of BAS was considered. That is, anhedonic symptoms were more strongly associated with low levels of Reward Responsiveness at high versus low levels of BIS. Implications for theory, prevention, and treatment are discussed.
Acknowledgements

I wish to thank my advisor, Dr. Michael Vasey, for his encouragement and direction throughout this process and for sharing his knowledge and expertise with me. I wish to thank my committee members, Dr. Jennifer Cheavens and Dr. Daniel Strunk, for their recommendations and guidance on this project.

I am very appreciative of Adam Buffington, for his willingness to share study participants with me and for his patient assistant with data analysis. Also, I would like to acknowledge the undergraduate research assistants who contributed to data collection.
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Chapter 1: Introduction

The past two decades of research have brought about an increasing recognition of the importance of examining psychological risk factors in the context of other risk factors (e.g., Hotard, McFatter, McWhirter, & Stegall, 1989; McFatter, 1994; Claridge & Davis, 2001; Kraemer, Stice, Kazdin, Offord, and Kupfer, 2001; Shahar, Gallagher, Blatt, Kuperminc, & Leadbeater, 2004; Compas, Conner-Smith, & Jaser, 2004; Kashdan, Zvolensky, & McLeish, 2008). For instance, Kraemer, et al. (2001) pointed out that no single risk factor can be fully understood in isolation and, thus, while studies investigating single risk factors are important, they only initiate the process of elucidating the causes of most disorders. Further, it is important not only to determine which factors combine to influence which disorders, but also how those factors combine and what that means for particular disorders. For example, the implications of a model in which risk factors interact are quite distinct from those of a model in which risk factors contribute only independent additive effects. While the field as a whole is moving towards the study of risk factors in combination, the issue of exactly how those factors work together has yet to be considered by many researchers (Quilty, et al., 2008), particularly within the depression and anxiety literatures. Shahar, et al. (2004) pointed out that within the literature on personality vulnerability to depression, researchers have largely assumed an additive main effects approach at the expense of
investigating the synergistic effects of personality dimensions. Shahar, et al. (2004) elaborate on the meaning of “synergistic effects” in the following passage:

“…this predominant emphasis on main effects overlooks the possibility of a dynamic interplay between various personality dimensions [emphasis original]; namely, the possibility that the vulnerability status of a certain personality dimension is contingent upon its interaction with other dimensions. For example, a vulnerability dimension might interact with another vulnerability dimension, resulting in a greater level of depression as compared to that brought about by the independent effect of both dimensions. Alternatively, a resilience dimension might buffer the adverse effect of one or both vulnerability dimensions, rendering them relatively benign” (p. 606).

While the study reported by Shahar et al. (2004) examined the interaction between dependency and self-criticism in depression, their comments invite inquiry into how potential interactions between other vulnerability factors influence depression. Clark and Watson’s (1991) influential tripartite model of depression and anxiety offers a two such factors that are prime candidates for this type of inquiry. The tripartite model asserts that depression is best characterized by the combination of high negative affectivity (NA) and low positive affectivity (PA) and that anxiety is best characterized by the combination of high NA and high physiological hyperarousal (PH). The temperamental-vulnerability extension (Clark, Watson, & Mineka, 1994; Mineka, Clark, & Watson, 1998) that follows from this originally descriptive model holds that the trait level counterparts of state NA and PA can act as temperamental vulnerabilities to depression. Because PH is less clearly related to personality and temperament than NA and PA, it is most often not included in the temperamental-vulnerability extension of the model (Clark et al., 1994; Brown, Chorpita, & Barlow, 1998). While this central tenant of dual influences of NA and PA on depression is indeed an example of
considering multiple risk factors in combination, the tripartite model does not explicitly describe the relations between NA and PA or what those relations mean for depression. In other words, the model does not explicitly articulate how high NA and low PA combine to influence depression. Do they combine additively as separate main effects or interactively such that they lead to greater vulnerability in combination than can be accounted for by their separate main effects? Empirical tests and discussion of the tripartite model within psychopathology research are almost exclusively additive, suggesting that most investigators have yet to consider the latter possibility.

In sum, an important way in which the tripartite vulnerability model of depression can be extended is by examining how its proposed risk factors work together to influence depression. As will be discussed below, a clear understanding of the type of relation between these factors is beneficial, as the implications of a model in which they interact are different from those of a model in which they combine additively. While the amount of research on this topic is quite small, the field is not entirely without knowledge about whether the above mentioned risk factors combine additively or interactively to influence depression. Indeed, the weight of existing evidence from research involving the tripartite model, as well as from research involving other related models (e.g., Eysenck’s personality theory), suggests that these personality or temperament dimensions interact to influence depression and anxiety. In the following paper, I, first, briefly review Clark and Watson’s tripartite model to give a fuller picture of how current theory conceptualizes NA and PA in relation to depression. Second, I address the important distinctions between an interactive and an additive model and review the implications of an explicitly interactive model. Third, I present an extensive
review of the relevant literature to demonstrate that the weight of existing evidence suggests an interaction between NA and PA in the prediction of depression. Finally, I describe an empirical study and discuss the results in the context of the implications of an NA x PA interaction in depression and anxiety.

The Tripartite Model

The literature often characterizes depression as involving a combination of high distress (i.e., high NA) and anhedonia (i.e., low PA) as was proposed by Clark and Watson in their tripartite model of anxiety and depression (Clark, Watson, & Carey, 1988; Clark & Watson, 1991). The tripartite model was designed as a way to account for the high comorbidity and high degree of symptom overlap between anxiety and depression. Clark and Watson (1991) provided evidence that self-report measures, symptom measures, and multiple factor analyses reveal a distress factor common to both disorders, which accounts for the comorbidity and overlap. Additionally, these analyses revealed a factor unique to each disorder, which allowed for differentiation. The common distress factor is NA, which Clark et al. (1988) describe as a general index of subjective distress that subsumes a broad range of negative mood states including fear, anxiety, hostility, sadness, loneliness, and disgust. Individuals with a temperamental predisposition to experience negative emotions are said to be high in trait NA while a tendency to experience peacefulness and calm reflects low trait NA (Clark & Watson, 1991). The depression specific factor, positive affectivity (PA), encompasses mood states such as energetic, joyful, proud, interested, friendly, and pleasurably engaged. Individuals with a temperamental predisposition to experience such positive emotions are said to have high trait PA while low trait PA is characterized
by a tendency toward fatigue, lack of interest or enjoyment, and lack of social or environmental engagement (Clark et al., 1988; Clark & Watson, 1991). The anxiety specific factor in the tripartite model, physiological hyperarousal (PH), is best described as a state factor and is characterized by somatic symptoms such as muscle tension, shortness of breath, racing heartbeat, and dizziness.

The tripartite model has undergone some refinement since its inception. Namely, Mineka, et al. (1998) proposed a revised tripartite model which they call the integrative model. One of the catalysts for this revision was the finding that the original model did not adequately account for the heterogeneity among the anxiety disorders. Specifically, PH was found to be more related to panic than to anxiety disorders in general (Brown, et al., 1998). In light of this evidence, Mineka, et al. (1998) drew from the tripartite model and Barlow’s hierarchical organization of anxiety disorders (Brown & Barlow, 1992; Zinbarg & Barlow, 1996) to propose their integrative model, which diverges from the classic tripartite model in several ways, two of which are important to note within the current discussion. First, each individual distress syndrome (e.g., depression, panic, specific phobia) is hypothesized to contain both a common and a unique element. Accordingly, PH is no longer viewed as characteristic of all anxiety disorders, but rather, as the unique element of panic disorder. Second, the integrative model asserts that rather than viewing any one set of symptoms as entirely unique to any one disorder (e.g., symptoms of low PA are entirely unique to depression), it may be more accurate to describe each disorder as a unique combination of symptoms. For example, low PA symptoms are not confined to depression but play a role in social phobia as well (e.g., Clark, et al., 1988; Watson & Clark, 1995). As recently as 2005, both Clark and Watson
have upheld these revisions (Clark, 2005; Watson, 2005). While these revisions do improve the model’s descriptive and predictive abilities, what is noteworthy for the present discussion is that, first, like the original tripartite model, the integrative model does not explicitly address the question of interactive relations between NA and PA (neither as states or as traits), and, second, the integrated model highlights the finding that high NA and low PA may be involved in some anxiety disorders as well as depression. Indeed, though far less research has been done on the potential interaction between NA and PA in anxiety, research has established that both high neuroticism (which is related to trait NA, see below) and low extraversion (which is related to trait PA, see below) are associated with social phobia (Stemberger, Turner, Beidel, & Calhoun, 1995; Brown, et al., 1998; Bienvenu, 2007; Bienvenu, Hettema, Neale, Prescott, & Kendler, 2007; Kashdan, 2007; Kotov, Watson, Robles, & Schmidt, 2007; Alden, Taylor, Mellings, & Laposa, 2008) and there is some evidence that this combination of temperament traits may be associated with agoraphobia as well (Bienvenu, et al., 2001; Bienvenu, et al., 2007; Rosellini & Brown, 2008). Thus, to the extent possible, this review will include research relevant to the NA by PA interaction in the prediction of anxiety as well.

As mentioned earlier, there is research involving models other than the tripartite model that offer evidence in support of an interactive relation between NA and PA as vulnerability factors for depression. These other models similarly propose high distress or withdrawal (related to high NA) and low positive affect or approach (related to low PA) as vulnerabilities to depression and anxiety and, similarly, do not explicitly address how their respective NA-type and PA-type factors combine to influence depression. In
different models these factors are called by different names and have slightly different definitions. However, the factors from these models have been shown to be closely related to NA and PA. For example, trait NA from Clark and Watson’s tripartite model has been conceptualized as a temperamental affective facet of Eysenck’s broader personality dimension neuroticism (e.g., Clark, et al., 1994; Clark, 2005) and has demonstrated a strong association with neuroticism (e.g., r = .74, Evans & Rothbart, 2007; Watson, Gamez, & Simms, 2005). In parallel fashion, trait PA has been conceptualized as a temperamental affective facet of Eysenck’s broader personality dimension extraversion (e.g., Clark, et al., 1994) and has demonstrated a strong association with extraversion (e.g., r = .67, Evans & Rothbart, 2007; Watson, et al., 2005). Additionally, the issues surrounding the relations between different NA-type and PA-type factors and the implications of those relations are the same across models. Specifically, this proposal will review literature relevant to trait NA and trait PA from Clark & Watson’s (1991) tripartite model, neuroticism (N) and extraversion (E) from Eysenck’s personality model, and behavioral inhibition sensitivity (BIS; related to NA) and behavioral activation sensitivity (BAS; related to PA) from Gray’s reinforcement sensitivity theory (RST; Gray, 1970). While the individual factors from Eysenck’s and Gray’s models will be fully defined later, what is important to the present point is that even though factors from these other models are not isomorphic with Clark and Watson’s NA and PA, they are similar enough that the issues surrounding their relations and the implications of their potential interaction are the same as for NA and PA. Therefore, when not discussing model-specific research, I will refer to the trans-model
NA-type component as general negative emotionality, abbreviated NE, and the trans-model PA-type component as general positive emotionality, abbreviated PE.

Interactive Versus Additive Model

The distinction between interactive and additive relations is often challenging in the context of vulnerability models because the question is not whether a combination of two factors increases vulnerability but rather whether the increase due to the combination can be fully accounted for by the independent effects of each factor. For example, an interactive tripartite model would predict that the combination of low PE and high NE results in greater risk for depression/anxiety than could be accounted for by the sum of the individual effects of each factor (i.e., high NE is much more strongly associated with depression when PE is low than when PE is moderate or high). Alternately, an additive tripartite model would predict that the risk for depression and anxiety could be entirely accounted for by the sum of the independent effects of NA and PA.

The same distinction can be made with regard to decreased vulnerability. In an interactive model, high NE would be much less strongly associated with depression/anxiety in the presence of moderate to high PE than low PE. For example, work by Fredrickson and colleagues has demonstrated that positive emotions can greatly reduce the physiological effects of negative emotions (e.g., increased heart rate) and can even buffer the depressogenic effects of psychological trauma (Fredrickson & Levenson, 1998; Fredrickson, Mancuso, Branigan, & Tugade, 2000). While Fredrickson’s model has been tested only additively, it potentially implies an NE by PE interaction.
Implications of an Explicitly Interactive Model

There are several important implications of a model in which NE and PE interact to predict depression and anxiety. Indeed, refining models that conceptualize high NE and low PE as risk factors for depression and anxiety so that they explicitly reflect the interaction between NE and PE could improve the utility of these models for research on the etiology, treatment, and prevention of depression. Specifically, as discussed at length below, explicitly considering the potential for an interaction between NE and PE has the potential to increase the utility of models by allowing statistical models to account for a greater proportion of variance, providing a clearer picture of how other risk and protective factors are related to depression and anxiety, improving treatment programs and their implementation, improving prevention programs and their implementation, and allowing for more effective tests of those prevention programs. In the area of research, including the NE x PE interaction term in statistical analyses has the potential to pull systematic variance out of the error term and reduces model specification error, thus allowing for better predictions (i.e., accounting for more variance) of both depressive and anxious symptoms. For example, in a clinical sample of children and adolescents, Joiner and Lonigan (2000) found that the addition of the NA x PA interaction allowed their model to account for an additional 12% of the total variance in change in depression symptoms across time compared to a model that included only the main effects. Admittedly, the interaction is not always of such impressive size. For example, in a study by Loney, Lima, and Butler (2006) the NA x PA interaction only account for an additional 2% of the variance in depression above and beyond the rest of their model. However, the small amount of variance accounted
for by the interaction in some studies should not be cause for their dismissal. Monte Carlo simulations have shown that interactions accounting for as little as 1% of the variance may be of critical importance (McClelland & Judd, 1993).

Second, establishing how high NE and low PE combine to increase risk for depression and anxiety opens lines of inquiry into the context in which other risk and protective factors operate. For example, evidence suggests that the construct of effortful control (EC; the ability to suppress a dominant response in order to perform a subdominant response, Rothbart, Ellis, Rueda, & Posner, 2003) is protective in that it moderates the associations between high levels of NE and symptoms of anxiety and depression and between low levels of PE and symptoms of depression. Specifically, several studies have found that high NE is more weakly related to depression and anxiety at high EC (e.g. Dinovo, 2005; Dinovo, Vasey, & Bills, 2007; Baumann, Kaschel, & Kuhl, 2007; Martel & Nigg, 2006; Verstraeten, Vasey, Raes, & Bijttebier, 2009). However, there is some evidence to suggest that fully understanding the effect of EC requires looking at EC in the context of an NE by PE interaction. Dinovo and Vasey (in preparation) found that this NE by EC interaction is further moderated by PE such that it is non-significant at low levels of PE. That is, it may be that high EC can buffer the depressogenic and anxiogenic effects of high NE, but only at moderate to high levels of PE, suggesting that high EC may be a potential protective factor for individuals with moderate to high levels of PE, but not for individuals with low PE. Thus, one can see how considering the possibility of an interaction between NE and PE can provide a clearer picture of how other factors are associated with depression and anxiety.
Another benefit of an interactive model is that it contains an additional layer of complexity that has implications for making treatment programs and their implementation more effective. For example, an interactive model may help explain treatment failures, thereby, suggesting guidelines along which treatment matching can occur. When a treatment that is expected to be effective is ineffective, or when it is effective in one group of individuals, but not in another, this suggests that there may be some factor moderating treatment response. One possibility that is suggested in work by Quilty et al. (2008) is that level of E may moderate the efficacy of treatment in reducing depressive symptoms. Quilty et al. (2008) found that an N x E interaction qualified treatment response such that at lower levels of E, treatment response was poor regardless of level of N whereas at higher levels of E, N was strongly associated with treatment response such that low N predicted better response than high N. Therefore, it seems that different combinations of these personality traits may differentially predict treatment response. This has the potential to not only aid in treatment matching, but also to pinpoint the groups of individuals for whom new and different treatments need to be developed.

Additionally, considering how PE and NE combine highlights the possibility that they may combine differently under different conditions which has implications for treatment implementation. For instance, as was found in a study by Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapil (2007), PE and NE may work together differently depending on level of stress. In this study, high NE was more weakly related to anhedonic depressive symptoms at moderate/high PE, but only when life stress was low. This suggests that clinicians may find an intervention aimed at increasing PE to be
more effective if it is implemented after a stress reduction intervention. That is, determining the conditions that moderate particular ways in which NE and PE work together may help therapists determine the optimal order and timing of interventions.

An interactive model may aid prevention efforts by facilitating the identification of different causal mechanisms that operate in individuals with different combinations of temperamental risk factors. Based on the concept of equifinality, the same outcome may result via different causal processes and identification of these processes may improve the efficacy of prevention programs by indicating which therapeutic targets to address in which individuals. In other words, matching individuals with certain temperamental risk factors to prevention programs tailored to address the specific causal processes associated with those risk factors may make prevention more effective. For example, in a recent longitudinal study of adolescents, Wetter and Hankin (2009) found that the relation between baseline NE and anhedonic depressive symptoms was mediated by dependent stressors and the relation between baseline PE and anhedonic depressive symptoms was mediated by supportive relationships. However, neither of these factors mediated the relation between the baseline NE x PE interaction and anhedonic depressive symptoms. This suggests that anhedonic depression may develop via different mechanisms in individuals who are both high in NE and low in PE than in individuals with only one of these vulnerabilities. Thus, prevention efforts may be enhanced by identifying the mediators of the NE x PE interaction and matching individuals to programs that target the mediator(s) specific to their category of temperamental risk (e.g., high NE, low PE, and the combination of high NE and low PE).
An interactive model could also have implications for research aimed at testing prevention programs. As discussed by Cuijpers (2003), most research on prevention programs looks at whether a particular program can increase factors known to protect against mental disorders (e.g., social skills, stress management) rather than looking at whether the program actually reduces the incidence rate of a particular mental health disorder. He posits that this is, at least in part, because extremely large sample sizes are needed to achieve sufficient statistical power to detect changes in incidence rates. One of the ways that Cuijpers (2003) suggests to increase power to detect such changes is to target high-risk groups by focusing on multiple risk factors. That is, a prevention program implemented in a group with a higher expected incidence rate (as determined by a combination of risk factors) will achieve a larger effect than the same program implemented in a group with a lower expected incidence rate (as determined by only a single risk factor). It follows that while any multi-risk factor model could help prevention efforts by identifying individuals with a higher level of risk, a model that includes multiple risk factors that combine interactively (i.e., the joint effect increases risk above and beyond the effect of each factor independently) could be particularly helpful in this way.
Chapter 2: Literature Review

The following literature review examines what current theory and data have to say about the relation between NE and PE and how that relation influences depression and anxiety. The research is organized into four categories a) research involving NA and PA b) research involving N and E, c) research involving Gray’s response sensitivity theory (RST; Gray, 1970) and d) research from the resilience and coping literature. A final section summarizes the main findings and briefly reviews their implications for research, prevention, and treatment.

Temperamental Negative and Positive Affectivity Research

Only four studies that tested an NA x PA interaction in the prediction of depression could be located. The first of these was conducted by Joiner and Lonigan in 2000. In this study, the authors tested the NA x PA interaction cross-sectionally and longitudinally and they appear to take for granted that such an interaction is expected under the tripartite model. Specifically, the authors predicted that an NA x PA interaction would predict depression status cross-sectionally as well as changes in depression over time. These predictions were supported in their samples of 33 (for the cross-sectional analysis) and 35 (for the longitudinal analysis) psychiatric inpatient children and adolescents with primary diagnoses of either depressive disorder, ADHD, or conduct disorder after controlling for baseline symptomatology. The authors conclude that their results supported the tripartite model. Curiously, although several studies cite Joiner and Lonigan (2000) only two have tested this interaction. Loney, et
al. (2006) examined the relations between temperament and conduct problems and depression in a sample of adolescents. Like Joiner & Lonigan (2000), the authors appear to take for granted that the tripartite model predicts such an interaction. Specifically, they predicted that the NA x PA interaction would predict conduct problems and depression, such that high NA, high PA adolescents would endorse the most conduct problems and that high NA, low PA adolescents would endorse the most depressive symptoms. Results supported these predictions. That is, controlling for depression, the NA x PA interaction significantly predicted conduct problems and, controlling for conduct problems, the NA x PA interaction significantly predicted depression.

In a very recent study, Wetter and Hankin (2009) also cited Joiner and Lonigan (2000)’s results and tested the interaction between temperamental vulnerabilities in the prediction of anhedonic depressive symptoms in a community sample of 345 adolescents aged 11-17 years. They used an adolescent measure of temperamental negative and positive emotionality (NE and PE, respectively) at baseline to predict anhedonic depressive symptoms measured 5 months later. The authors predicted that there would be main effects of both NE and PE and that the interaction between NE and PE at baseline would predict prospective anhedonic depressive symptoms. Results indicated that indeed high NE and low PE were each independently related to increased anhedonic depressive symptoms and the NE x PE interaction predicted symptoms such that individuals with both high NE and low PE reported a higher level of symptoms than could be accounted for by the independent effects of NE and PE.

DeBoo and Kolk (2007) examined the relation between temperament and
depressed and aggressive moods in a large (N = 423) sample of 9 – 13 year old Dutch children from four different ethnic/national background including Caucasian, Moroccan, Turkish, and mixed ethnicity. The authors hypothesized that high NA and low PA would additively predict depressed mood. Additionally, they tested the NA x PA interaction citing Shahar et al.’s (2004) assertion that the effects of one temperament dimension may be attenuated or amplified by the effect of another. While some of the results were rather odd and difficult to explain (e.g., PA was positively related to depression), the NA x PA interaction predicted depressed mood such that at low NA PA was not related to depressed mood but at high NA, PA accounted for a significant proportion of variance in depressed mood.

In summary, a strikingly small number of studies have addressed the NA x PA interaction in the prediction of depression and no studies could be located that tested this interaction in the prediction of anxiety. The tripartite model and NA and PA in general are most often examined and discussed in terms of the factor structure of the model and the correlations between those factors and anxiety and depression. Research dealing with the model does mention and demonstrate that depression and some anxiety disorders (e.g., social phobia and agoraphobia) are related to both high NA and low PA, but they are silent with respect to the interactive versus additive nature of NA and PA (e.g., Brown, et al., 1998; Kiernan, Laurent, Joiner, Catanzaro, & MacLachlan, 2001; Jacques & Mash, 2004; Rosellini & Brown, 2008). For example, researchers often test the effects of NA while statistically controlling for PA, or vice versa (e.g., Rosellini & Brown, 2008). Although, it is possible that some researchers have tested this interaction, but not reported it (presumably due to non-significant results), it seems, based on the
lack of discussion of a potential interaction, more likely that most authors simply have not considered the possibility.

Neuroticism and Extraversion Research

Given the paucity of research on the interaction between NA and PA and the close relation between NA/PA and N/E, the literature on N and E as risk factors for depression and anxiety provides another source of evidence about the interactive versus additive nature of these vulnerabilities to depression and anxiety. While the main effects approach still dominates in this area of personality research, a handful of investigators have explored whether N and E interact to influence depression and anxiety. Hotard, et al. (1989; Study 2) examined the relation between Eysenck’s personality traits N and E, quality of social interactions, and subjective well-being (SWB; an operationalization of happiness) in a sample of 131 college students. Hotard et al. (1989) interpreted Eysenck’s model to predict interactive rather than additive relations between traits and as such tested the interactions between their predictor variables. Based on Eysenck’s model, they predicted that neurotic introverts (i.e., individuals with high N and low E) would report lower SWB than would be expected based on the additive effects of these two personality traits. Results indicated that indeed N and E interacted to predict SWB such that particularly low SWB tended to be characteristic of neurotic introverts whereas relatively high SWB tended to characterize all other personality combinations. Because this interaction had not been described in the well-being literature before, the authors collected data from a replication sample (N = 109) and reanalyzed data from two other studies (N = 319 and N = 160). All three samples were comprised of college students. The pattern of results in the replication sample was identical to that in the
original study. The two data sets reanalyzed did not include measures of SWB, but they
did include a measure of depression (Beck Depression Inventory; BDI), which the
authors considered to be a negative reflection of SWB. Additionally, one of the samples
used alternate measures of N and E, the Sociability Scale of the Guilford-Zimmerman
Temperament Survey (Guilford & Zimmerman, 1949, as cited in Hotard et al., 1989) as
a measure of E and the Psychoasthenia scale from the Minnesota Multiphasic
Personality Inventory as a measure of N. In both re-analyses the N x E interaction
predicted BDI score such that introversion (low E) was only associated with depression
at high levels of N. In sum, Hotard et al. (1989) interpreted Eysenk’s model to be
interactive and presented results from four data sets to support their interpretation.

Using similar measures, McFatter (1994) replicated Hotard et al.’s (1989) main
findings in another sample of 384 college students. Specifically, he found an NxE
interaction such that E was significantly related to BDI score and a measure of well-
being only at high levels of N. Importantly, McFatter (1994) argued strongly for
researchers to begin considering the possible interactive relations among personality
dimensions. Some researchers took this argument to heart and began testing the N x E
interaction in their data citing Hotard et al. (1989) and McFatter’s (1994) findings as
their rationale. One such set of researchers, Gurshey and Sher (1998), conducted a
longitudinal study of the relation between personality and depression and global
anxiety. The 4 year longitudinal study utilized data collected annually from a sample of
undergraduates who were first time college freshmen at Year 1. All analyses were
conducted on data from the 466 students who participated at all 4 time points. Cross
sectional analysis of the data from Year 1 revealed no significant interaction between N
and E in the prediction of depression or anxiety. However, in the prospective analysis, which predicted Year 4 depression and anxiety from the Year 1 interaction between N and E while controlling for symptoms at Year 1, this interaction was significant for both anxiety and depression. In explanation, the authors suggest that since the first two years of college are typically marked by higher distress than the last two some of the symptoms reported at Year 1 may be situational and are naturally reduced for some participants as they adjust to college life and build social support networks. However, for those who are more introverted (lower in E) the same type of social support may not be developed leaving these individuals at increased risk for depressive or anxious symptoms as time goes on. In other words, an interaction between N and E may become more salient and apparent in the prediction of depression and anxiety over the course of a few years because individuals high in N but low in E may have fewer coping resources (e.g., social support) available to them than their higher E counterparts and, thus, their symptoms are not reduced over time. In sum, it may be that the N x E interaction did not predict depression or anxiety at Year 1 because situational distress, at least in part, masked this personality effect. It is noteworthy that these authors found a significant N x E interaction in the prediction of anxiety given that they used a measure of global anxiety, which was composed of items asking about general anxiety, phobic anxiety, and obsessive compulsive anxiety, rather than one specifically tapping social anxiety or agoraphobia.

The N x E interaction has also been shown to predict response to treatment for depression. Quilty, et al. (2008) reported results from a double-blind, randomized trial in which depressed participants underwent combination treatment with antidepressant
medication and psychotherapy for 6 months. High E and low N were each independently related to better response. A significant N x E interaction qualified these main effects such that at low E, level of N was more weakly associated with response than it was at higher levels of E. That is, low E was predictive of poor response regardless of level of N whereas at higher levels of E, lower N predicted better treatment response.

In a review paper on the relation between personality and the expression of PTSD, Miller (2003) presented research that implies a model of vulnerability to PTSD in which an individual’s constellation of pre-trauma negative emotionality (NEM), positive emotionality (PEM), and constraint/inhibition (CON) predispose him/her to a specific manifestation of post-trauma reaction. Specifically, Miller hypothesizes that internalizing presentations of PTSD (i.e., presentations marked by high occurrence of depressive and anxious symptoms) can be accounted for by an interaction between high NEM and low PEM and externalizing presentations of PTSD (i.e., presentations marked by high occurrence of aggressive and antisocial symptoms) can be accounted for by an interaction between high NEM and low CON. In other words, high NEM is conceptualized as a generalized vulnerability to PTSD (and psychopathology in general) but it is pre-trauma level of PEM and/or CON that determines the exact presentation of PTSD such that low PEM is more strongly associated with PTSD with prominent internalizing symptoms – specifically, anhedonia, withdrawal, and avoidance – than is moderate or high PEM. While Miller offers no direct empirical test of his hypothesis, the research and arguments he presents suggest that such a test is warranted.
Not everyone who has looked for an N x E interaction in the prediction of depression and anxiety has found it, however. A set of studies by Jorm, et al. (2000) failed to replicate the N x E interaction as a predictor of depressive or anxious symptoms in a cross sectional study using a very large (N = 2677) community sample ages 18 – 79 and in cross sectional and longitudinal examinations of an elderly sample of individuals over the age of 70 (N = 668 for cross sectional analyses; N = 441 for longitudinal analyses. After failing to find an N x E interaction in the community samples, Jorm et al. (2000) ran a regression analysis on subsamples of young adults (ages 18 – 24; N = 292) and of individuals with a university degree (N = 826) in an attempt to approximate the college samples used in Hotard et al. (1989) and Gershuny & Sher (1998). Still, they found that the interaction accounted for no additional variance whatsoever above and beyond the main effects. The authors offer two possible explanations for why their findings may differ from those of Hotard et al. (1989) and Gershuny & Sher (1998). First, they cite the sample differences suggesting that perhaps the interaction does not generalize outside of college populations. Second, Jorm et al. (2000) cited differences in depression symptom measures. Hotard et al. (1989) used the BDI and Gershuny & Sher (1998) used the Brief Symptom Inventory while Jorm et al. (2000) used the Anxiety and Depression Scales of Goldberg (Goldberg, Bridges, Duncan Jones, & Grayson, 1988). Notably, while the Scales of Goldberg (Goldberg, et al., 1988) are intended for oral administration, they were given in a self-completion format in the elderly sample. The correlation between the oral and self-completion formats is not known. Additionally, it could be that Jorm et al.’s (2000) failure to find an interaction in the prediction of anxiety was because the Anxiety Scales of Goldberg
(Goldberg, et al., 1988) were validated against diagnoses of generalized anxiety disorder, which implies that the Scales primarily tapped symptoms of worry, rather than symptoms more related to anxiety in general or anxiety disorders that are specifically associated with high N and low E such as social anxiety and agoraphobia.

In a longitudinal study of personality determinants of post-partum depression (N = 277), Verkerk, Denollet, Van Heck, Van Son, & Pop (2005) found that while high N and high I (i.e., low E) each independently predicted self-reported depressive symptoms at every time point measured (i.e., 34 weeks into pregnancy and 3, 6, and 12 months post-partum) there was no statistically significant N x E interaction. The results of this study appear to support an additive tripartite model of depression to the extent that the model extends to post-partum depression.

Some studies examining the relation of personality to SWB, which has been considered a negative index of depression (Hotard et al., 1989), have produced confusing and null results. In an effort to extend the extraversion – well-being relation to non-self report methods, Pavot, Diener, & Fujita (1990) collected self report personality and state affect data from 136 college student participants and had the participants’ family and friends complete three non-self-report measures of well-being. Analyses revealed that for two of the three non-self-report well-being measures the N x E interaction appeared to be present but failed to reach statistical significance. The third well-being measure was predicted by a statistically significant N x E interaction. The authors conclude that the additive versus interactive nature of N and E in relation to well-being needed to be examined further in larger samples and with more diverse self report and non-self report measures.
It is clear from the studies reviewed above that results have not been entirely consistent. There may be a number of reasons for this. First, as Jorm et al. (2000) point out, inconsistent findings may be due to the use of different samples. For example, in the literature reviewed above, the N x E interaction effect for depression is found more often in college and adolescent samples (Hotard, et al, 1989; McFatter, 1994; Gurshey & Sher, 1998; Joiner & Lonigan, 2000; Loney, et al., 2006; DeBoo & Kolk, 2007; Wetter & Hankin, 2009) than in community adult samples. Indeed, two of the three studies that used non-college adult samples failed to replicate the N x E interaction (Verkerk et al., 2005; Jorm et al., 2000). The third study conducted with community adults found an interaction predicting treatment response, but this study did not test the interaction in the prediction of presence or severity of depressive symptoms (Quilty et al., 2008). While it is possible that sample differences may account for discrepant results, there are too few studies testing the interaction in any type of sample to draw firm conclusions at this time. Differences in sample size do not appear to be responsible for discrepancies as some of the larger studies reviewed (e.g., Jorm et al., 2000) did not find an interaction.¹ However, power to find an N x E interaction may be decreased by the additional error variance introduced when using measures that inadequately capture the symptom dimensions relevant to an N x E interaction. For example, the unvalidated self-report version of the Goldberg scales used by Jorm et al. (2000) may have inadequately tapped anxious and depressive symptoms making an N x E interaction undetectable. Likewise, it is possible that the inconsistency between Gershuny & Sher

¹ However, it is important to keep in mind when reviewing this literature that, in general, power to find this interaction effect is low (McClelland & Judd, 1993).
(1998) and Jorm et al. (2000) in finding an N x E interaction in the prediction of anxiety was due to differences in the symptoms tapped by different anxiety measures. That is, perhaps the anxiety measure used by Gershuny & Sher (1998) tapped symptoms more related to low E (e.g., symptoms related to social anxiety or agoraphobia) than did the worry-focused anxiety measure used by Jorm et al. (2000). In sum, the discrepant results of extant studies on the topic serve to illustrate the need for more research on the N x E interaction in depression and anxiety in a variety of samples using a variety of well-validated measures.

Behavioral Activation and Inhibition Sensitivity Research

An examination of research on the Behavioral Inhibition System (BIS) and Behavioral Activation System (BAS), which are analogous to N and E, also provides some clues as to the interactive versus additive nature of personality vulnerabilities to depression and anxiety. The BIS/BAS model of personality is often called Reinforcement Sensitivity Theory (RST) and is credited to Gray (1981, 1987). There have been several clarifications, interpretations, and revisions of this model (e.g., Wallace, Newman, & Bachorowski, 1991; Gray & McNaughton, 2000, Corr, 2008), but the fundamental tenants of the theory have all but remained the same. Additionally, most of the research on the BIS/BAS model has been informed by the original iteration of RST. Therefore, in order to remain within the scope of this proposal and to maintain contact with the relevant empirical literature, the following review will be limited as much as possible to the issue of how the relatively unchanged fundamentals of Gray’s original RST predict that BIS and BAS combine to influence anxiety and depression.

Before reviewing the empirical literature, a brief description of RST and its
relation to Eysenck’s personality theory of N and E is instructive. Gray’s model holds
that there are two motivational systems, one aversive motivational system, the BIS, and
one appetitive motivational system, the BAS. Individual differences in the sensitivity of
these systems to relevant environmental stimuli represent individual differences in two
personality dimensions, which Gray calls anxiety proneness and impulsivity,
respectively. The BIS is sensitive to signals of punishment, non-reward (or reward
removal), and novelty while the BAS is sensitive to signals of reward, non-punishment,
and escape from punishment. In broad terms, RST predicts that strong BAS individuals
(i.e., highly impulsive individuals) should be most sensitive to signals of reward,
compared to weak BAS individuals and strong BIS individuals (i.e., highly anxious
individuals) should be most sensitive to signals of punishment, compared to weak BIS
individuals (Corr, 2001). Gray argued that these systems are part of the neurological
underpinnings of behavior and affect. The BIS is said to be responsible for the
experience of negative emotion and the BAS is said to be responsible for the experience
of positive emotion (c.f., Corr, 2002). Gray’s original model holds that the BIS and
BAS axes are orthogonal and intersect to make a 4 quadrant personality space that is a
30-degree rotation of Eysenck’s N and E personality space. The BIS axis runs from
neurotic introvert to stable extravert (i.e., high anxiety to low anxiety), though it is
conceptually closer to N than to E. The BAS axis runs from neurotic extravert to stable
introvert (i.e., high impulsivity to low impulsivity), though it is conceptually closer to E
than to N. While the two sets of factors are clearly related, BIS/BAS and N/E are not
isomorphic. As mentioned in the beginning of this proposal, what is important for the
present discussion is, first, that the relation between BIS/BAS and depression is
analogous to the relation between NE and PE and depression. Specifically, depression is characterized by high BIS and low BAS (e.g., Kasch, Rottenberg, Arnow, & Gotlib, 2002; Pinto-Meza, et al., 2006). Second, the implications of an interaction between any of these sets of factors transcend the particular model under consideration. That is, an interaction between BIS and BAS has the same implications for research, treatment, and prevention as does an interaction between N and E or NA and PA.

In contrast to personality and temperament researchers, BIS/BAS researchers, including Gray himself, have been vocal about the relation between BIS and BAS effects as predicted by RST. Specifically, the proposed functional independence of the BIS and BAS has been the subject of recent theorizing and research. This issue has been most thoroughly explicated by Corr (2001, 2002, 2004) whose basic notion is that many of the predictive failures of RST can be traced to the hypothesized functional independence of BIS/BAS effects. He goes on to propose two hypotheses intended to address this complex issue. They are as follows: If the BIS and BAS are indeed orthogonal and operate independently of one another, then response to reward should be the same at all levels of BIS and response to punishment should be the same at all levels of BAS. This is the separable subsystems hypothesis (SSH; Corr, 2001). Alternately, if these systems are functionally dependent (i.e., the BIS and BAS inhibit or facilitate each other depending on the environmental context) as is proposed by the joint subsystems hypothesis (JSH; Corr, 2001), then one would expect response to reward and punishment to depend on one’s levels of both BIS and BAS. Specifically, the JSH predicts that under the conditions most likely to be present in the human psychological lab (i.e., participants with non-extreme levels of BIS and BAS, stimuli with mixed
and/or non-extreme valances, and conditions that require rapid attentional and behavioral shifts between aversive and appetitive stimuli) the BAS facilitates responses to appetitive stimuli and antagonizes responses to aversive stimuli while the BIS facilitates responses to aversive stimuli and antagonizes responses to appetitive stimuli. Thus, when the two systems act in combination, the result is a function of their combined effects (Corr, 2004). Corr (2004) stresses that the JSH and SSH are complementary rather than competing hypotheses meaning that under certain conditions (i.e., when BIS/BAS levels are extreme, when stimuli are very strong and pure, and when there is no need for rapid attentional or behavioral shifts between aversive and appetitive stimuli) separable effects of the BIS and BAS are to be expected. Indeed, while work by researchers who found unexpected joint effects of the BIS and BAS (e.g., Pickering, Diaz, & Gray, 1995; Gray, Corr, Fotiadou, & Wilson, 1994 [as cited in Corr, 2004]) as well as more recent research intended to directly test the JSH have provided evidence in support of the interdependence of the BIS and BAS (see Corr, 2004 for a review), not all studies have produced definitive support for JSH (e.g., Hundt, Kimbrel, Mitchell, Nelson-Gray, 2008) and some even better support SSH (e.g., Gomez, Cooper, McOrmond, & Tatlow, 2004).

That said, several studies have found clear statistical BIS x BAS interactions in the prediction of several outcomes (e.g., reactions to emotional stimuli, Corr, 2002; information processing of emotional stimuli, Gomez, et al., 2004; substance abuse, Genovese & Wallace, 2007; religious attitudes, Jackson & Francis, 2004), thus,
providing solid support for JSH.\textsuperscript{2} However, similar to the literature on NA/PA and N/E, the number of empirical studies that test the statistical interaction between BIS and BAS in the prediction of depression and anxiety is quite small. Only two such studies could be located for this review. In a study of 768 mainstream Russian adolescents, Knyazev & Wilson (2004) measured how BIS, BAS, N, E, and psychoticism (P) relate to conduct disorder symptoms and emotional symptoms. Among several other complicated results, the authors found that the BAS x N interaction predicted self-rated emotional symptoms such that the association of N with emotional symptoms was more pronounced at low levels of BAS. Hundt, et al. (2007) examined how BIS and BAS effects interact with each other and life events to influence internalizing symptoms in a sample of 285 undergraduates. Results showed that across levels of stressful life events, level of BAS only predicted symptoms when in combination with high BIS, suggesting that low BAS alone may not be sufficient for substantial internalizing symptoms. Additionally, there was a BIS x BAS interaction in the absence of stressful life events such that high BIS predicted anhedonic depressive symptoms only when combined with low BAS. Notably, however, the combination of high BIS and \textit{high} BAS predicted mixed anxiety – depression symptoms across levels of stressful life events. The authors explain this finding by hypothesizing that individuals with high BIS and high BAS may frequently find themselves in approach – avoidance conflicts which cause significant distress resulting in mixed symptoms. It may be that while having a moderate (i.e., not low) BAS level prevents the toxic effects of high BIS, high BAS actually adds to the

\textsuperscript{2} See Corr (2004) for a discussion of how dynamic interplay between BIS and BAS that is not reflected in statistical interactions may also be supportive of JSH.
negative emotions engendered by high BIS. This notion is consistent with the fact that while BAS is conceptualized as being responsible for and is related to PA (e.g., Holzwarth & Meyer, 2006; Quilty & Oakman, 2004; Depue & Iacono, 1989), high BAS is also characterized by impulsivity which has been associated with low frustration tolerance (e.g., Corr, 2002). Additionally, some research demonstrates that high BAS is related to negative emotions and externalizing symptoms (Hundt, et al., 2008; Knyazev & Wilson, 2004; Quilty & Oakman, 2004; Quay, 1993). No studies on the BIS x BAS interaction in anxiety could be located.

In sum, it appears that while theorists have largely begun to consider BIS/BAS interaction and interdependence, not much attention has yet been given to the empirical examination of this interaction in relation to depression and no attention, it seems, has been given to the interaction in relation to anxiety. That said, what empirical research is available appears to be generally supportive of a BIS x BAS interaction in depression and theoretical work certainly points towards some kind of important interaction between the two systems with relation to depression and anxiety. Future research will, of course be required to replicate and extend such findings. In this future work, care should be taken to address the difference between moderate and high levels of BAS and the difference between the negative (e.g., impulsivity) and positive (e.g., positive emotionality) characteristics associated with high BAS as Hundt et al.’s (2007) study provides some evidence that, at least in the case of anxiety, high levels of BAS may exacerbate the negative consequences of high BIS rather than protect against them.

Despite a relative paucity of empirical work and some inconsistent findings within the literatures on NA/PA, N/E, and BIS/BAS as risk factors for depression and
anxiety, one thing is clear: there is enough theory and evidence to warrant serious efforts to advance the field’s understanding of how different combinations of NE and PE influence depression and anxiety. What is also clear is that the effect of an NE x PE interaction on depression and anxiety has not been given enough theoretical or empirical attention to date. A few researchers have considered it but, given the effect’s numerous and potentially important implications as discussed above, it has not made the impact on the field that one would expect. Whether the reason for this is that depression and anxiety researchers have skipped over the NE x PE interaction in favor of looking for simple, clean relations between factors (Claridge & Davis, 2001) or that researchers have not been exposed to the existing work on this interaction, continuing to ignore the potential for interaction between these factors could limit the progression of depression and anxiety research as a whole.

Coping and Resilience Research

One other place to look for clues into the nature of the relation between NE and PE in depression and anxiety is the coping and resilience literature. Indeed, research within this area provides a fairly consistent pattern of results. It is not often, if at all, that coping and resilience researchers discuss findings in terms of a statistical interaction between NE and PE, but, at least in some researchers’ work, the pattern of evidence may imply an interactive model. For example, Barbara Fredrickson and her colleagues have done research on the regulating effect of positive emotions on negative emotions. Fredrickson’s relevant empirical work is guided by the “undoing” hypothesis and the broaden-and-build hypothesis. The undoing hypothesis simply says that positive emotions can undo the effects of negative emotions (Fredrickson, et al., 2000). This
hypothesis was formulated as an explanation for the adaptive role of positive emotions. Negative emotions are thought to be adaptive because they trigger specific action tendencies (Frijda, 1986), which are often associated with significant physical arousal, especially increased cardiovascular activity. Fredrickson and colleagues posit that positive emotions are adaptive, at least in part, because they can down-regulate the physiological arousal associated with negative emotions (Fredrickson, et al., 2000). The slightly broader notion of positive emotion as an attenuator or inhibitor of negative emotion is supported in some neuropsychological work as well (e.g., Depue & Collins, 1999). Fredrickson, et al. (2000) point out that down regulation of physiological arousal has important implications for health because recurrent or prolonged cardiovascular reactivity is thought to increase risk for the development or exacerbation of heart disease. This notion has been supported in the health literature (e.g., Steptoe, Wardle, & Marmot, 2005). In a 1998 study, Fredrickson and Levenson demonstrated that positive emotions can speed recovery from the cardiovascular sequelae of negative emotions.

Sixty female undergraduate subjects viewed a fear-provoking film and then were randomly assigned to view a second film designed to elicit one of four other emotions: contentment, amusement, neutrality, or sadness. Compared to subjects who viewed the sad and neutral secondary films, subjects who viewed the films intended to induce contentment or amusement made a faster return to pre-film cardiovascular activation. This effect was replicated using a similar paradigm with a speech preparation task to induce anxiety (Fredrickson, et al., 2000).

It is worth noting that the undoing hypothesis is not necessarily interactive, but rather could refer to an additive “off-setting” of negative emotions by positive
emotions. An explicitly interactive test of the undoing hypothesis could be conducted by reversing the order of film presentation in the Fredrickson and Levenson (1998) study. If the cardiovascular response to viewing a negative film after viewing a positive film was reduced compared to the cardiovascular response to viewing a negative film after viewing a neutral film, then this might suggest an interactive relation between positive and negative emotions in which positive emotions buffer against the cardiovascular effect of negative emotions. Indeed, this type of buffering effect has received support in the health literature.

Some researchers have begun to study how PE can act as a buffer against the negative emotions that result from physical pain. For example, in a study of patients with rheumatoid arthritis, Strand, et al. (2006) found that PA was influential in reducing NA during weeks when patients experienced more pain. That is, higher levels of PA helped patients experience higher levels of pain as less distressing. Similarly, spikes in pain increased NA less during weeks of higher PA in a sample of women with fibromyalgia and/or osteoarthritis (Zautra, Johnson, & Davis, 2005). This has potentially important implications for physicians and mental health professionals who work with chronic pain patients as using PA to reduce pain induced NA may positively influence disease course, prevent increases in frequency of pain flares, and preserve coping resources (Strand et al., 2006). Further, a study by Lightsey (1994) demonstrated that experiencing certain types of automatic positive thoughts may weaken the relation between stressful life events in general and depression (Lightsey, 1994) suggesting that perhaps positive thoughts and the positive emotions with which they are associated can
help protect chronic pain patients from potential depressogenic effects of their stressful health issues.

While Fredrickson’s undoing hypothesis is concerned with how positive emotions influence the physiological effects of negative emotions, Fredrickson and colleagues’ second guiding hypothesis, the broaden-and-build hypothesis, is focused on the more cognitive and psychosocial effects of positive emotions. This hypothesis holds that whereas many negative emotions narrow individuals’ momentary thought-action repertoires by triggering specific action tendencies (e.g., attack, run away), many positive emotions broaden individuals’ momentary thought-action repertoires. Support for the notion that this type of cognitive broadening is a unique consequence of positive emotions comes from research that has shown that these effects do not occur when negative features of experimental stimuli or outcomes are made salient (Ashby, Isen, & Turken, 1999). Work by Isen and colleagues suggests that this cognitive effect of positive emotions may be mediated by increased dopamine levels in the brain (Ashby, et al., 1999; Isen, 2002), although more recent work has failed to replicate this effect (Burns, et al., 2008). Cognitive broadening is posited to promote a wider range of thoughts and actions than is typical (e.g., explore, play, savor), thereby, over the course of time, building enduring personal resources. Fredrickson and colleagues hypothesize that these resources can be utilized in later times of need and that they range from physical (e.g., health, longevity) and intellectual (e.g., expert knowledge, intellectual complexity) resources to social (e.g., social support networks) and psychological (e.g., resilience, optimism) resources (Fredrickson, 1998, 2001). Recent research has shown that these types of resources may indeed have an impact on psychological and health’
outcomes via physiological response to stress. Specifically, a study looking at how psychosocial resources moderate the cortisol stress response (which has been positively associated with outcomes such as hypertension, insulin resistance, and depression) found that individuals who reported higher psychosocial resources (e.g., optimism, extraversion, self-esteem) exhibited significantly less cortisol reactivity following a laboratory stress task (Taylor, et al., 2008). This suggests that one pathway by which positive emotions may buffer against negative outcomes is by promoting broader thought-action repertoires, which can enhance the type of psychosocial resources that are associated with a more positive response to stress (i.e., decreased cortisol reactivity).

Fredrickson (2001) cites work by Isen and colleagues as foundational evidence for their broaden-and-build hypothesis (see Fredrickson, 2001 for details or Isen, 2000 for a review). Several examples of novel empirical support for the hypothesis follow. In a 2001 study by Fredrickson, subjects were randomly assigned to watch one of five film clips designed to induce either contentment, joy, fear, anger, or neutrality and then were asked to list the things they would like to do right then in light of their emotion. Subjects listed their responses on up to 20 blank lines that began with the phrase “I would like to.” Subjects who viewed one of the two positive emotion clips listed more things they would like to do than subjects who viewed the negative emotion or neutral clips. Additionally, the subjects who viewed the negative emotion clips listed fewer things than subjects who viewed the neutral clips. This result is interpreted as supportive of the hypothesis that positive emotions produce a broader thought-action repertoire than neutral states or negative emotions. In a 2002 study, Fredrickson and Joiner demonstrated in a sample of college students that baseline positive emotions
predicted increases in broad-minded coping (i.e., having a broad perspective on problems and being able to generate multiple potential solutions) 5 weeks later. This result was replicated and extended in a 2008 study by Burns et al., in which they found that, over the course of 8 weeks, positive emotions predicted not only broad-minded and positive coping, but also interpersonal trust.

Further, Fredrickson, Cohn, Coffey, Pek, and Finkel (2008) attempted to experimentally test the broaden-and-building hypothesis. In a 10 week study, the authors experimentally manipulated positive emotions using a type of meditation called Loving-Kindness Meditation (LKM), which involves discrete periods of time spent in quiet while focusing on feelings of love and kindness one has for close friends or family and then mentally extending those feelings to oneself and to others. Results indicated that LKM significantly increased daily experience of positive emotions and increments in positive emotions were related to increases in a number of resources including mindfulness, purpose in life, social support received, self-acceptance, savoring the future, environmental mastery, positive relations with others, pathways thinking, and decreased illness symptoms. Fredrickson et al. additionally tested the direct path from experimental condition (LKM or control) and time spent meditating to increases in resources and found that this path accounted for a very small portion of the total variance, suggesting that LKM had its effect on resources via its impact on positive emotions. The authors also demonstrated that increases in positive emotions were positively associated with life satisfaction by way of the resources that increased with positive emotions and that increases in positive emotions were negatively associated with depressive symptoms by way of increases in those same resources. Further, the
authors tested a model in which increases in resources mediate the relation between positive emotions and life satisfaction and depressive symptoms. Specifically, they demonstrated that increases in positive emotions were positively associated with life satisfaction by way of the resources that increased with positive emotions and that increases in positive emotions were negatively associated with depressive symptoms by way of increases in those same resources.

To reiterate the main thrust of Fredrickson and colleagues’ work within the framework of the NE x PE interaction: Positive emotions moderate the effect of negative emotions (e.g., prolonged cardiovascular activity, narrowed thought-action repertoire) such that in the presence of positive emotions, negative emotions have a reduced effect (e.g., cardiovascular activity is attenuated more quickly, thought-action repertoires are broadened resulting in increased personal resources). While the evidence reviewed above does support this assertion, it does not directly speak to the specific hypothesis asserted at the beginning of this proposal, which states that NE and PE interact to confer vulnerability to depression and anxiety such that high PE can reduce the depressive and anxiety inducing effects of high NE (or vice versa). Among work by Fredrickson and colleagues, a study conducted in the aftermath of the 9/11 terrorist attacks on the United States provides perhaps the most relevant evidence in support of this specific hypothesis. Fredrickson, Tugade, Waugh, & Larkin (2003) collected self report data from a sample of college undergraduates in early 2001 and again in the weeks following the 9/11 terrorists attacks which occurred in 2003. Forty-seven participants reported on their personality, depressive symptoms, resilience, and reactions to the events of 9/11. One of the hypotheses tested was that resilient
individuals, defined as individuals who tend to be optimistic, zestful, energetic, curious and open to new experiences, and tend to have high positive emotionality, would be buffered from depressive symptoms by positive emotions. Results indicated that, in general, regardless of their level of resiliency, individuals did not differ on the number or type of negative emotions experienced in reaction to 9/11. The only exception to this was that non-resilient individuals tended to endorse slightly more sadness than resilient individuals. While non-resilient individuals did experience some positive emotions (gratitude and love) to a similar degree as resilient individuals, resilient individuals experienced a wider variety of positive emotions (e.g., interest, joy, hope, contentment) and experienced them more frequently than did individuals who scored lower on resilience. Further, results supported the main hypothesis indicating that resilience was negatively related to depressive symptoms and this relation was fully mediated by positive emotions. The path from resilience to depressive symptoms was reduced to non-significance when positive emotions was entered as a mediator. In short, Fredrickson et al. (2003) found that the depressive effect of the 9/11 terrorist attacks was reduced in resilient individuals because they experienced more positive emotions than non-resilient individuals (i.e., positive emotions buffered individuals from depressive symptoms in response to a life stressor).

Wichers, et al. (2007) cite Fredrickson et al. (2003) as the only study to date to examine the role of PA as a buffer against depression. Wichers et al. (2007) addressed the paucity of research by conducting a twin study to investigate whether PA buffers NA reactivity in response to daily life stress. Previous research suggests that NA reactivity to daily life stress is an endophenotype of depression meaning that the extent
to which small daily life stressors produce NA reflects the genetic liability to develop a depressive disorder. The authors reasoned that an interaction between PA and NA reactivity in response to stress would provide evidence that PA moderates the genetic predisposition to depression (i.e., the experience of PA reduces the effect or expression of the genetic vulnerability). Undergoing an experience sampling paradigm, the participants, 279 Belgium female twin pairs, were prompted to record stress, mood, thoughts, current context (e.g., activity, persons present, location), and appraisals of current situation 10 times throughout the day for 5 days. After controlling for average level of PA and level of depressive symptoms, results indicated that there was a strong and statistically significant interaction between stress and PA in the association with NA such that participants with low PA experienced more NA in response to stressful (i.e., unpleasant) events than participants with mid-level or high PA. Further results showed that the experience of mid-level and high PA, compared to low PA, significantly reduced the effect size of the proband NA reactivity to stress by co-twin lifetime depression interaction. In other words, compared to low PA, mid-level and high PA reduced the amount of NA in response to stress (i.e., reduced the expression of an endophenotype of depression) for participants with a shared genetic vulnerability to depression. Sharing a genetic vulnerability to depression matters less when individuals experience more PA during moments of stress in the flow of daily life.

Wichers and colleagues extended these findings in a study of how PA moderates the expression of brain derived neurotrophic factor (BDNF) polymorphisms via stress sensitivity (i.e., NA) in a sample of Belgian twins (Wichers, et al., 2008). BDNF is important for neuronal survival and synaptic plasticity. Levels of BDNF can be
influenced by stress and, thus, it is thought that BDNF may play a role in stress related disorders like depression. The Valine (Val) variant of the BDNF gene is associated with higher neuronal BDNF secretory activity than the Methionine (Met) variant, suggesting that BDNF “Met” carriers are more sensitive to stress-induced BDNF depletion than their Val/Val counterparts. Thus, Wichers, et al. (2008) hypothesized that the BDNF Val/Met polymorphism would moderate stress-related NA (i.e., individuals with the Val/Met polymorphism are predicted to experience greater NA in response to stress) and that PA would reduce the moderating effect of BDNF genotype on NA response to stress (i.e., the association between the Val/Met polymorphism and stress-induced NA is predicted to be weaker at higher levels of PA). Results supported both hypotheses except only in the case of social stress, which is in agreement with recent work that has shown that BDNF plays a primary role in mediating behavioral plasticity in response to aversive social experiences (Berton, et al., 2006, as cited in Wichers, et al., 2008). The authors conclude that the evidence supporting the first hypothesis suggests a possible mechanism for the association between the Val/Met BDNF polymorphism and depression. That is, it may be that “Met” carriers are more vulnerable to depression because of an increased NA response to social stress associated with the polymorphism. Further, the evidence supporting the second hypothesis suggests that because PA is negatively associated with the expression of the Val/Met polymorphism, interventions aimed at increasing the experience of PA in the face of social stress may reduce the development of depression in vulnerable individuals (i.e., individuals with the Val/Met polymorphism who are, thus, more prone to experiencing NA in response to social stress). In sum, this study supports the notion that high PA can reduce the expression of
a brain-based, NA-related marker of risk for depression.

A recent study by Hughes and Kendall (2008) provides support for the hypothesis that PA can protect against the anxiety specific effects of high NA. Children with a principle diagnosis of an anxiety disorder (AD) and non-disordered control (ND) children were randomly assigned to undergo a positive or neutral emotion induction followed by a measure of tendency to interpret ambiguous information as threatening. This type of threat bias is a classic characteristic of individuals with anxiety disorders (e.g., Mathews, Mogg, Kentish, Eysenck, 1995). The threat bias measure included a series of ambiguous scenarios, which the children were instructed to complete (i.e., write an ending to the situation). Completions were coded as either threatening (e.g., mom is late because she got into an accident) or non-threatening (e.g., mom is late because she is picking up dinner). Results indicated that for AD children, those in the positive emotion induction condition rated significantly fewer scenarios as threatening than those in the neutral condition. This difference was not observed for ND control children who were not prone to threat interpretations in either condition. Further, for both groups of children subjective ratings of distress pre to post emotion induction were significantly decreased in the positive emotion induction condition but not in the neutral condition. The authors concluded that results suggest a positive emotional state may mitigate or reduce the tendency to interpret ambiguous information in a threatening manner for AD children and that positive emotional states may have the ability to meaningfully reduce state anxiety in children.

The presence of an interaction between BIS and BAS or N and E in the coping, resilience, and health literatures would take the form of attenuated negative effects of
high BIS/N for individuals high in BAS/E. This hypothesis is difficult to address at this point in time, however, as research concerning BIS/BAS and N/E and resilience, coping, and health outcomes is very limited and most of the extant research is inconclusive (e.g., Muris, et al., 2007; Heponiemi, Keltikangas-Jarvinen, Kettunen, Puttonen, & Ravaja, 2004; van der Linden, Beckers, & Taris, 2007; van der Linden, Taris, Beckers, & Kindt, 2007).

Summary of Literature Review

While it appears that only a relatively small number of researchers have investigated the NE x PE interactions in the prediction of anxiety and depression, those that have done so have found generally promising resulting. Specifically, four studies have demonstrated a clear NA x PA interaction (Joiner & Lonigan, 2000; Loney, et al., 2006; DeBoo & Kolk, 2007; Wetter & Hankin, 2009). It is worth noting, however, that these four studies were conducted in child and adolescent samples and no published studies of the NA x PA interaction in college, adult, or elderly samples could be located. More research has been done concerning the interaction between N and E in the prediction of depression and anxiety, though there are some inconsistencies in results. Evidence in support of E as a buffer against the depressive and anxious effects of high N comes mostly from studies of undergraduates (Hotard, et al., 1989; McFatter, 1994; Gurshey & Sher, 1998). One study demonstrates that the N x E interaction may predict response to treatment for depression in a community sample (Quilty et al., 2008).

However, some other studies failed to replicate the N x E interaction (Jorm et al., 2000; Verkerk et al., 2005; Pavot et al., 1990). No studies of the N x E interaction in child or adolescent samples could be located. How the effects of the BIS and BAS combine in
general has received recent theoretical and empirical attention (e.g., Corr, 2001, 2002, 2004) and how they combine in relation to depression has been the focus of some recent work (e.g., Tomarken & Keener, 1998; Shankman & Klein, 2003). Specific to the statistical BIS x BAS interaction, two relevant studies that could be located generally support the hypothesis that BIS and BAS interact to predict depressive symptoms, though there is some evidence that moderate levels of BAS may be more protective than high levels (Knyavez, Slobodskaya, & Wilson, 2004; Hundt et al., 2007). Future research will need to address this and other hypotheses to continue to clarify the exact nature of the interaction between BIS and BAS in depression.

Research within the areas of coping and resilience also support the role of PE as a buffer against the deleterious effects of NE. Work by Fredrickson and colleagues demonstrates that positive emotions can reduce the cardiovascular and emotional consequences of negative emotions (Fredrickson, 1998, 2001) and that the experience of positive emotions can account for reduced depressive symptoms following a traumatic event (Fredrickson, et al., 2003). Other researchers have shown that PA can help patients experience less NA in response to chronic pain (Zautra et al., 2005; Strand et al., 2006), that moderate to high PA is associated with reduced expression of a marker of genetic risk for depression (Wichers et al., 2007; Wichers et al., 2008), and that a positive emotion induction can reduce threat bias and subjective anxiety among children with anxiety disorders (Hughes & Kendall, 2008). Research on BIS/BAS and N/E within coping and resilience is limited and yields inconclusive results.

It is clear from the foregoing review and summary that the NE x PE interaction has been demonstrated by several different researchers, in different samples, using
different measures and different constructs (i.e., NA/PA versus N/E). That said, it is also clear that studies of this important idea are still very few and some of the results are contradictory or counterintuitive. Indeed, further research is necessary to determine the complete and true picture of the influence of these characterological dimensions on the development, presentation, and maintenance of depressive and anxious disorders.

Results from existing research suggest some potential implications for research, prevention, and treatment. For research, adding the interaction into statistical tests could increase efficiency by removing systematic variance from the error term. Additionally, establishing how high NE and low PE combine to increase risk for depression throws light onto the context in which other risk and protective factors operate. Implications for prevention and treatment, while no more important, are perhaps more exciting. For example, the finding that high PA or positive emotions can attenuate the depressive or anxious effects of a traumatic event (Fredrickson, et al., 2003) suggests that interventions to increase PA or positive emotional experiences in high NA low PA individuals at risk for trauma (e.g., soldiers, females) may help to prevent depressive disorders in the wake of such events. Work with chronic pain patients (Zautra et al., 2005; Strand et al., 2006) suggests that interventions aimed at teaching these patients how to increase positive emotions during times of heightened pain may help to reduce their level of NA which, in turn, may reduce their risk for depressive symptoms. Further, as discussed in the work of Cuijpers (2003), using the NE x PE interaction to identify groups with high expected incidence rates of depressive and anxious symptoms (i.e., high NE low PE individuals) would allow for more effective tests of such prevention programs and allow administrators to focus limited resources on the groups
at highest risk for disorder. Additionally, as suggested by Wetter and Hankin’s (2009) work, different causal mechanisms may be operating in individuals with the combination of high NE and low PE and matching these individuals to prevention programs tailored to address the specific causal processes associated with this combination of interactive risk factors may make prevention more effective.

Additionally, the NE x PE interaction could be used to inform and improve treatment. For example, some researchers have posited a causal relation between threat biases and anxiety disorders (Matthews & MacLeod, 2002) and some studies have shown that reducing threat bias in individuals with anxiety disorders significantly reduces subjective experience of anxiety (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002; Li, Tan, Qian, & Liu, 2008). Additionally, reductions in threat bias are associated with successful treatment of anxiety disorders (e.g., Mathews, et al., 1995; Waters, Wharton, Zimmer-Gremeck, & Craske, 2008; Li, et al., 2008). Hughes and Kendall’s (2008) work suggests an easily distributable and cost effective method for reducing threat bias associated with anxiety disorders: teaching individuals how to increase their experience of positive emotions, especially in times of high anxiety. Finally, Quilty et al.’s (2008) work on how the N x E interaction influences response to treatment for depression may have implications for future work on personality based treatment matching.
Chapter 3: The Present Study

While definitive conclusions await substantial longitudinal and, to the extent possible, experimental, research in a variety of samples, especially clinical ones, the purpose of the proposed study was to provide a step towards gaining a better understanding of the nature and reliability of the proposed NE x PE interaction in the prediction of depressive and anxious symptoms. To that end, the present study provided a cross sectional test of the hypothesis that the NA x PA, N x E, and BIS x BAS interactions predict depressive symptoms, anhedonic symptoms, and symptoms common to anxiety and depression (i.e., mixed anxious-depressive symptoms).³ Specifically, it was hypothesized that at low levels of PE, high NE would be more strongly associated with depressive and mixed anxious-depressive symptoms than at high levels of PE and that at high levels of NE, low levels of PE would be more strongly associated with anhedonic symptoms than at low levels of NE.

Methods

Participants.

Participants were 1277 non-selected, undergraduates recruited from the Ohio State University (OSU) psychology participant pool. Data from 7 participants were excluded from the analyses due to suspicious patterns of responding. Additionally, data

³ A measure of symptoms common to anxiety and depression was chosen over a measure of purely anxious symptoms because, at this point, the literature does not support hypotheses about an NE x PE interaction in anxiety specific symptoms, such as those associated with panic.
from 28 participants were excluded due to excessive non-response (greater than 25% of data missing). Analyses were conducted on the remaining 1242 (687 females) participants (M age = 19.11, SD = 2.05, range = 18-52). The sample was 6.4% African American, 0.2% American Indian or Alaskan Native, 6.1% Asian, 3.1% Hispanic, 0.3% Native Hawaiian or Pacific Islander, 81.7% non-Hispanic White, and 1.9% who described their ethnicity as “other”. Participants received course credit for their participation.

Measures.

*Positive and Negative Affect Schedule (PANAS – Trait Form).* The PANAS – Trait Form (Watson, Clark, & Tellegen, 1988) is a widely used measure that includes two 10 items scales, one for positive affectivity (PA) and one for negative affectivity (NA). Participants indicate on a 1 (“Very slightly or not at all”) to 5 (“Extremely”) Likert scale the degree to which they generally experience each of 20 affective descriptors (e.g., guilty, excited, distressed, proud). Scores for each scale range from 10 to 50 with higher scores indicating more NA or PA. Research has demonstrated that the PANAS is a reliable and valid measure (e.g., Watson, Clark, Tellegen, 1988; Crawford & Henry, 2004).

*Behavioral Inhibition Sensitivity/Behavioral Activation Sensitivity Scales (BIS/BAS Scales).* The BIS/BAS Scales (Carver & White, 1994) comprise a 20 item, four-point Likert type measure that assesses emotional responding to potentially negative or rewarding events. There is a single BIS scale, which captures the tendency to experience negative affect or behavioral inhibition in the presence of aversive or threat cues. The total BAS Scale assesses the tendency to experience positive affect or
approach motivation in the presence of incentive cues. There are three related BAS subscales: BAS-Drive, BAS-Reward Responsivity, and BAS-Fun Seeking. BAS-Drive emphasizes motivation to pursue goals regardless of whether those goals are inherently pleasurable. BAS-Reward Responsivity assesses the tendency to respond with energy and positive affect in the context of desired events or cues of future reward. BAS-Fun Seeking emphasizes novelty seeking and impulsive pursuits of pleasure. The BIS/BAS Scales have moderate internal consistency and good convergent and discriminant validity (Campbell-Sills, Liverant, & Brown, 2004).

*Adult Temperament Questionnaire – Short (ATQ – Short).* The ATQ – Short (Rothbart, Ahadi, & Evans, 2000) is a 77-item self-report measure of adult temperament. Participants rate the items based on how well they think each item describes them. A 1 (“extremely untrue”) to 7 (“extremely true”) Likert scale is used. Some items are reverse scored. Four general factors are assessed: Effortful Control, Negative Affect, Extraversion/Surgency, and Orienting Sensitivity. In the present study the Negative Affect and Extraversion/Surgency scales were used as measures of neuroticism and extraversion, respectively. The ATQ - Short Negative Affect correlates highly with Big Five Neuroticism ($r = .74$) and ATQ – Short Extraversion/Surgency correlates highly with Big Five Extraversion ($r = .67$; Evans & Rothbart, 2007). Both the Extraversion and Negative Affect scales contain subscales. The subscales of Negative Affect are Sadness, which measures negative affect and lower mood and energy related to suffering, disappointment, and loss; Discomfort, which measures negative affect related to sensory experiences; Fear, which measures negative affect related to the anticipation of distress; and Frustration, which measures negative affect in
response to interruption of ongoing tasks or goal blocking. The subscales of Extraversion are Positive Affect, which measures frequency, duration, and intensity of positive emotional experiences; Sociability, which measures degree of pleasure derived from social interactions; and High Intensity Pleasure, which measures enjoyment related to high intensity activities. It should be noted that several items were omitted from the ATQ due to an error in copying the measure. These included one item from the Fear subscale of the Negative Affect scale and two items from the High Intensity Pleasure subscale of the Extraversion scale. Given this omission, the Negative Affect scale was made up of 25 items and the Extraversion scale was made up of 15 items.

The Depression, Anxiety, and Stress Scales (DASS). The DASS (Lovibond & Lovibond, 1995) is a 42-item self-report measure of symptoms of anxiety and depression. These symptoms are represented on three 14-item subscales. The DASS-Depression subscale represents symptoms of depression, particularly dysphoric mood and anhedonic symptoms; the DASS-Anxiety subscale represents symptoms of physiological hyperarousal; and the DASS-Stress scale represents symptoms of a generalized type of anxiety that is characterized by worry, nervous overarousal, and general tension. The DASS-Depression scale was used in the present study to measure depressive symptoms. A composite variable created by averaging the DASS-Stress and DASS-Depression scales was used in the present study to measure mixed anxious and depressive symptoms. The DASS-Anxiety scale was not considered in this study.

Participants rate the items based on how much or how often they applied during the past week. The rating scale is a 0 (“Did not apply to me at all”) to 3 (“Applied to me very much, or most of the time”) Likert scale. Example items include “I find myself getting
upset by quite trivial things;” “I felt sad and depressed;” and “I felt scared without any good reason.” This measure has been shown to possess adequate psychometric properties (Lovibond & Lovibond, 1995).

Missing Data.

Missing data analysis revealed that 309 participants (24.9%) had one or more missing items on one or more measures. Items were considered missing if the participant failed to give a response or gave more than one response to a single item. To avoid the loss of power associated with the traditional listwise deletion method of handling missing data, missing values were imputed using a multiple imputation (MI) procedure as described by Allison (2002). In short, MI generates multiple complete datasets in which values for missing data are estimated based on the relations between the variables included in the imputation model and random error.

MI was chosen for the present study because of its advantages over single imputation methods. Single imputation methods underestimate variability and yield downwardly biased standard error estimates for two reasons. First, most single imputation methods estimate missing values without taking into account the random error associated with a random sample. MI achieves a more accurate estimate of the standard error by adding random error to each imputed value. Additionally, the amount of random error added to an estimate of a particular value is varied across datasets. Second, single imputation methods underestimate variability because they use just one estimate of each missing value, which is based on the parameters of the present sample

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4 It should be noted that this can be achieved by some single imputation methods as well.
rather than the population. MI generates multiple estimates of each missing value by adding random error to the parameters of the estimation model, thus correcting for this bias by creating variability across datasets. Because of the addition of random error to the parameters of the estimation model and to imputed values themselves, each of the multiple datasets contains a slightly different estimate of any particular value. However, each data set is an equally probable estimation of the complete data.

MI was conducted at the total scale score level because there were too many items to fit in a single MI model. Rather than computing total scale scores only for cases with complete data, the number of cases for which imputation was necessary was minimized by using weighted mean scale scores (Graham, 2009; Schafer & Graham, 2002). A weighted mean of items for each scale was computed for cases that had a minimum number of items completed. This minimum number was determined based on the reliability of the scale such that higher percentages of non-missing items were required for scales with lower alpha levels. For scales with $\alpha \geq 0.90$, 50% of items were required, for scale with $\alpha \geq 0.80$, 67% were required, and for scales with $\alpha \geq 0.70$, 80% of items were required. Based on these scale scores all interaction terms necessary for the initial hypotheses were computed. This process yielded a complete set of mean scale scores for all but 102 participants (8.2%) and a complete set of interaction terms for all but 100 (8.1%) participants.

To facilitate understanding and allow for comparison with other studies using the DASS, all results were transformed back onto the original DASS scoring scale. Each DASS scale consists of 14 items, such that a mean DASS scale score is the sum of the items divided by 14. For the DASS-Stress/DASS-Depression composite, a mean
scale score is the sum of the items on these two scales divided by 28. Thus, results from analyses predicting DASS-Depression were multiplied by 14 and results from analyses predicting the DASS-Stress/DASS-Depression composite were multiplied by 28 to return them to the original DASS scoring scale.

The distribution of several scale scores and all of the interaction terms were positively skewed, as expected, which violates the normality assumption of MI. However, data were not transformed because MI has been shown to be robust to violations of this assumption, particularly when the amount of missing information is low as is the case in the present dataset (Allison, 2002; Graham & Schafer, 1999; Schafer & Graham, 2002).

Based on results from Graham, Olchowski, and Gilreath’s (2007) study of the relation between amount of missing information, power, and number of imputations, it was determined that 20 imputations would yield optimal power retention in this data set. Specifically, the results of this study suggest that 20 imputations are required to achieve a less than 1% decrease in power (compared to the power achieved by using 100 imputations). Additionally, using too few imputations has a minimal impact on power when effect sizes are large, but can impact power substantially when effect sizes are small. Thus, a large number of imputations was particularly desirable in the present study because the expected sizes of the effects of interest are quite small (McClellend & Judd, 1993).

MI assumes that the missing data are ignorable meaning that the pattern of missing data is either not predictable (i.e., random) or can be predicted only by variables included in the model. Missing data that cannot be predicted are said to be
Missing Completely At Random (MCAR), while missing data that can be predicted only by variables included in the model is said to be Missing At Random (MAR; Schafer & Graham, 2002). Missing data in the present sample may be have been influenced by individual differences in motivation or perseverance, as reflected in measures such as PA, E, and the BAS-Drive subscale, that affected participants' decisions to skip items or quit filling out the packets before completing all of the measures. This is an example of MAR because measurements of PA, E, and BAS-Drive, which are the potential sources of influence, are included in the MI model. No direct test of the MAR assumption is possible because it would require unavailable data. Further, most data sets likely included some departure from MAR (Schafer & Olsen, 1998). However, failing to consider unforeseen sources of influence on missingness will likely have only a minor impact on results (Collins, Schafer, & Kam, 2001). In the present sample, there were no obvious potential sources of bias in the pattern of missingness that were not included in the study, and the MAR assumption was assumed to be valid.

All of the interaction terms relevant to the present study were created in the original dataset by standardizing the main effect variables and then computing product terms. These product terms were included in the MI model because the non-linearity represented by interactions cannot be properly estimated by the linear model that underlies MI. Including the interaction terms in the imputation model allows the model to account for this non-linearity and reliably estimate missing interaction values (Graham, 2009). Additionally, the imputation process must include all combinations of variables that will be tested because the model assumes the correlation between
variables in the model and omitted variables is zero. Excluding variables will bias their correlation with other variables, including the outcome variable, toward zero. Thus, all total scale scores and all interaction terms to be tested in the initial analyses were collectively subjected to MI, producing a total of 21 complete datasets (1 original dataset and 20 imputation datasets). Analyses were conducted on the set of 20 imputation datasets. Pooled results of analyses are reported below. Descriptive statistics were, however, computed in the original dataset using listwise deletion. The N that was used to compute these statistics for each measure is reported below.

Data Analysis.

Significant interactions were interpreted following the recommendations of Aiken and West (1991). Specifically, we tested the significance of slopes at selected values (i.e., simple slopes) and examined regions of significance for such effects. To maintain contact with the relevant literature, separate regression analyses were conducted for each set of NE/PE variables. To test the main hypotheses, the interactions between each set of these variables was tested in the prediction of two dependent variables: depressive symptoms as measured by the DASS-Depression scale and mixed anxious-depressive symptoms as measured by a composite of the DASS-Depression and DASS-Stress scales. Additionally, each interaction was tested in the prediction of anhedonic depressive symptoms as measured by the DASS-Depression scale while controlling for the DASS-Stress scale. For each analysis, the NE/PE variables and Sex were entered on Step 1 and the NE x PE interaction was entered on Step 2. Additional regression analyses were conducted as post-hoc follow up analyses. Because there were
no meaningful changes in the main effects from Step 1 to Step 2 for any of the analyses, only results from Step 2 of each analysis are reported.

While a total of 13 regression analyses were conducted, no steps were taken to constrain Type 1 error rate. Because so little research has been done in the area of NE x PE interactions in anxiety and depression, all of the present analyses are considered at least somewhat exploratory meaning that Type 2 error is of particular concern. Additionally, as discussed above, power to find the hypothesized interaction effects is already quite low and constraining Type 1 error could reduce power to an unacceptable level. Thus, the traditional alpha = .05 significance level was be retained for all analyses.
Chapter 4: Results

Descriptive Statistics.

Mean scores, standard deviations, and reliabilities of all measures are presented in Table 1. Scale intercorrelations and associations with Sex are presented in Table 2.

NA and PA.

Depressive Symptoms. To address the hypothesis that an NA x PA interaction predicts depressive symptoms a regression analysis predicting DASS-Depression from the NA x PA interaction was conducted. Results of this analysis are presented in Table 3. Results revealed significant effects for Sex, NA, PA, and the NA x PA interaction. Consistent with expectation, the form of the NA x PA interaction was such that the positive correlation between NA and depressive symptoms was significantly stronger at low PA than at high PA, as illustrated by differences in simple slopes (Figure 1). The simple slope for the predicted line of NA at low PA (-1 SD) was 4.23, t(1237) = 34.35, p = .000, whereas at high PA (+1 SD) it was 2.79, t(1237) = 10.02, p = .000. Examination of the region of significance revealed that the simple slope remains significant at all possible values of PA.

Anhedonic Depressive Symptoms. Next, a regression analysis predicting DASS-Depression from the NA x PA interaction while controlling for DASS-Stress was conducted to address the hypothesis that an NA x PA interaction predicts variance unique to depressive symptoms (i.e., anhedonic depressive symptoms). Results of this
analysis are presented in Table 4. Results revealed significant effects for Sex, NA, PA, and the NA x PA interaction. The form of this interaction was such that the relation between PA and anhedonic depressive symptoms was stronger at high NA than at low NA (Figure 2), as illustrated by differences in simple slopes. The simple slope of the predicted line of PA at low NA (-1 SD) was -1.17 (t(1237) = -5.77, p = .000), whereas at high NA (+1 SD) it was -2.44 (t(1237) = -12.18, p = .000). Examination of the region of significance revealed that the simple slope became non-significant at values of NA < 1.92 standard deviations.

**Mixed Anxious-Depressive Symptoms.** To test whether the NA x PA interaction predicts variance associated with mixed anxious-depressive symptoms, a regression analysis predicting the DASS-Stress/DASS-Depression composite from the NA x PA interaction was conducted. Results are presented in Table 5. Results indicate that the NA x PA interaction indeed predicts mixed anxious-depressive symptoms such that the relation between NA and such symptoms was weaker at high PA than at low PA (Figure 3), as illustrated by the differences in simple slopes. The simple slope of the predicted line of NA at low PA (-1 SD) was 10.08 (t(1237) = 26.59, p = .000), whereas at high PA (+1 SD) it was 8.15 (t(1237) = 15.91, p = .000). Examination of the region of significance revealed that the simple slope remains significant at all possible values of PA.

**N and E.**

**Depressive Symptoms.** To address the hypothesis that an N x E interaction predicts depressive symptoms, a regression analysis predicting DASS-Depression from the N x E interaction was conducted. Results are presented in Table 6. Results revealed
significant effects for Sex, N, E, and the N x E interaction. Consistent with expectation, the form of the N x E interaction was such that the positive correlation between N and depressive symptoms was significantly stronger at low E than at high E, as illustrated by differences in simple slopes (Figure 4). The simple slope for the predicted line of N at low E (-1 SD) was 3.47, t(1237) = 11.80, p = .000, whereas at high E (+1 SD) it was 2.03, t(1237) = 7.16, p = .000. Examination of the region of significance revealed that the simple slope became non-significant at value of E > 2.47 standard deviations.

**Anhedonic Depressive Symptoms.** To test whether the N x E interaction predicts anhedonic depressive symptoms, a regression analysis predicting DASS-Depression while controlling for DASS-Stress was conducted. Results are present in Table 7. Contrary to expectation, result indicated that the N x E interaction was not significant when predicting anhedonic depressive symptoms.

To explore why the N x E interaction was not significant when predicting anhedonic depressive symptoms, three regression analyses using the facets of N and E were conducted. These analyses were conducted in a new set of MI datasets because they involved interaction terms not included in the initial MI model. All of the previously reported results were obtained in this new set of datasets. Results revealed that the N x ATQ-PA (Table 8), ATQ-Sadness x E (Table 9) and ATQ-Sadness x ATQ-PA (Table 10) interactions significantly predicted anhedonic depressive symptoms. These interactions were all of the same form as the NA x PA interaction. Of these three interactions, the ATQ-Sadness x ATQ-PA interaction (Figure 5) accounted for the most variance (ΔR² = .009). The simple slope of the predicted line for ATQ-PA at low ATQ-Sadness (-1 SD) was -1.15, t(1237) = -5.57, p = .000, whereas at high ATQ-Sadness (+1
SD) it was \(-2.45, t(1237) = -12.65, p = .000\). Examination of the region of significance revealed that the simple slope became non-significant at values of ATQ-Sadness < 1.88 standard deviations.

**Mixed Anxious-Depressive Symptoms.** To test whether the N x E interaction predicts mixed anxious-depressive symptoms, a regression analysis predicting the DASS-Stress/DASS-Depression composite from the N x E interaction was conducted. Results are presented in Table 11. Results indicate that the N x E interaction indeed predicts mixed anxious-depressive symptoms such that the relation between N and such symptoms was weaker at high E than at low E (Figure 6), as illustrated by the differences in simple slopes. The simple slope of the predicted line of N at low E (-1 SD) was 8.96 \((t(1237) = 16.186, p = .000)\), whereas at high E (+1 SD) it was 5.84 \((t(1237) = 10.96, p = .000)\). Examination of the region of significance revealed that the simple slope remains significant at all values of E represented in the present sample.

**BIS and BAS.**

**Depressive Symptoms.** A regression analysis predicting DASS-Depression from the BIS x BAS interaction was conducted to address the hypothesis that a BIS x BAS interaction predicts depressive symptoms. Results are presented in Table 12. Results revealed significant effects for Sex, BIS, BAS, and the BIS x BAS interaction. Contrary to expectation, the form of the BIS x BAS interaction was such that the positive correlation between BIS and depressive symptoms was significantly stronger at high BAS compared to low BAS (Figure 7). The simple slope for the predicted line of BIS at low BAS (-1 SD) was 1.65, \(t(1237) = 5.98, p = .000\), whereas at high BAS (+1 SD) it
was 2.66, $t(1237) = 8.80$, $p = .000$. Examination of the region of significance revealed that the simple slope remains significant at all possible values of BAS.

**Mixed Anxious-Depressive Symptoms.** The form of the BIS x BAS interaction that predicted depressive symptoms was contrary to expectation and different than the NA x PA and N x E interactions. That is, high BAS was associated with a *stronger* positive relation between BIS and depressive symptoms whereas high PA and high E were associated with *weaker* positive relations between NA and N and depressive symptoms, respectively. Though not predicted in the present study, the form of the BIS x BAS interaction is similar to what was found by Hundt et al. (2007). These authors found that a BIS x BAS interaction predicted mixed anxious-depression symptoms such that high BAS strengthened the positive relation between BIS and these symptoms. In an attempt to replicate this finding in the present study, a regression analysis predicting the DASS-Stress/DASS-Depression composite from the BIS x BAS interaction was conducted. Results are presented in Table 15. Results indicate that a significant BIS x BAS interaction predicted mixed anxious-depressive symptoms and that the form of this interaction was the same as that found by Hundt et al. (2007), as illustrated by differences in simple slopes (Figures 9 and 10). The simple slope for the predicted line of BIS at low BAS (-1 SD) was 3.99, $t(1237) = 7.46$, $p = .000$, whereas at high BAS (+1 SD) it was 7.15, $t(1237) = 12.35$, $p = .000$. Examination of the region of significance revealed that the simple slope remains significant at all possible values of BAS.

**Anhedonic Depressive Symptoms.** It was expected that the BIS x BAS interaction would significantly predict anhedonic depressive symptoms and that the form of this interaction would be such that the relation between BIS and this variance
was stronger at low BAS than high BAS. This hypothesis is based on results from Hundt et al. (2007). Specifically, they found a significant BIS x BAS interaction of this form when predicting anhedonic depressive symptoms in their study. To test this hypothesis, an analysis predicting DASS-Depression controlling for DASS-Stress was conducted. Results of the analysis in the present study are presented in Table 13. These results indicate that, contrary to expectation, the BIS x BAS interaction was not significant when predicting anhedonic depressive symptoms although the sign on the BIS x BAS regression coefficient was in the predicted (i.e. negative) direction.

One hypothesis to describe why the BIS x BAS interaction significantly predicted anhedonic depressive symptoms in Hundt et al.’s (2007) study, but did not predict anhedonic depressive symptoms in the present study is that Hundt et al. (2007) used composite measures of BIS and BAS. Specifically, participants’ BIS scores were combined with their scores on a measure of punishment sensitivity and participants’ BAS scores were combined with their scores on a measure of reward sensitivity. Thus, the constructs used to reflect BIS and BAS in Hundt et al.’s (2007) study heavily emphasized punishment and reward sensitivity, respectively. It was not possible in the present study to approximate Hundt et al.’s (2007) BIS construct, but the Reward Responsiveness subscale of the BAS scale is a reasonable approximation of Hundt et al.’s (2007) composite BAS construct because, like this construct, the Reward Responsiveness subscale emphasizes sensitivity or responsivity to reward. It was hypothesized that, of the interactions between BIS and the three subscales of BAS, the BIS x BAS-Reward Responsiveness interaction would predict anhedonic depressive symptoms and that the sign on the regression coefficient of this interaction would
negative. This hypothesis was confirmed in a new set of MI datasets\(^5\) by a regression analysis predicting DASS-Depression from BIS x BAS-Reward Responsiveness, BIS x BAS-Fun Seeking, and BIS x BAS-Drive while controlling for DASS-Stress. Results are presented in Table 14. Specifically, the relation between BIS and anhedonic depressive symptoms was stronger at low BAS-Reward Responsiveness than at high BAS-Reward Responsiveness, as illustrated by differences in simple slopes (Figure 8). The simple slope for the predicted line of BAS-Reward Reward Responsiveness at low BIS (-1 SD) was -0.69, \(t(1237) = -3.10, p = .002\), whereas at high BIS (+1 SD) it was -1.49, \(t(1237) = -4.83, p = .000\). Examination of the region of significance revealed that the simple slope was non-significant at values of BIS < 1.46 standard deviations.

\(^5\) A new set of MI datasets was used because this analysis involved interaction terms not included in the initial MI model. All of the previously reported results were obtained in this new set of datasets.
Chapter 5: Discussion

The present study assessed the relations between the NA x PA, N x E, and BIS x BAS interactions and three types of psychological symptoms: symptoms of depression, broadly construed; symptoms of anhedonic depression; and mixed anxious-depressive symptoms. Results indicated that these interactions are indeed associated with such symptoms, but that the exact nature of the association depends on the set of temperament constructs and the type of symptoms considered. Specifically, results indicated that the NA x PA interaction predicted all three types of symptoms such that high NA was more strongly associated with depressive and mixed anxious-depressive symptoms when PA was low and low PA was more strongly associated with anhedonic depressive symptoms when NA was high; that the N x E interaction predicted depressive and mixed anxious-depressive symptoms such that high N was more strongly associated with symptoms when E was low; and that while the BIS x BAS interaction also predicted depressive and mixed anxious-depressive symptoms, the form of this interaction was unexpected. Specifically, high BIS was more strongly associated with symptoms than low BIS when BAS was high. However, the expected pattern was found for anhedonic symptoms, when the interaction between BIS and the Reward Responsiveness facet of BAS was considered. That is, anhedonic symptoms were more strongly associated with low levels of Reward Responsiveness at high versus low levels of BIS. In sum, the present study contributes to the literature by replicating the presence
and form of the NA x PA, N x E, and BIS x BAS interactions in depressive, anhedonic, and mixed anxious-depressive symptoms and by providing some preliminary evidence about how these interactions function differently across different types of symptoms.

NA and PA.

Results of the present study cross-sectionally replicated the longitudinal youth-sample-based results of Joiner and Lonigan (2000), Loney et al. (2006), and Wetter and Hankin (2009) in a non-clinical sample of adults. Specifically, the present study replicated the NA x PA interaction in the prediction of depressive and anhedonic depressive symptoms. To our knowledge, this is the first study to investigate the NA x PA interaction in depressive or anhedonic symptoms in adults. These findings have potential implications for the tripartite model, for understanding the etiology of depression, and for prevention.

First, the main effects approach used to develop and test the tripartite model addresses the association between NA and depressive symptoms at an average level of PA and the association between PA and depressive symptoms at an average level of NA. The interactive approach used in the present study, however, is more fine grained and represents the next step in explicating the tripartite model. Specifically, the present study addresses the question, “Given that NA and PA are associated with depressive symptoms, are those associations consistent across levels of PA and NA, respectively?” Results indicated that the association of each factor with depressive symptoms was indeed different across levels of the other factor. The association between NA and depressive symptoms was stronger at low PA than at high PA, implying that the risk for depression associated with high NA may be greater for individuals low in PA than for
individuals high in PA. Similarly, the NA x PA interaction that predicted anhedonic depressive symptoms in the present study suggests an additional layer of complexity that could make the tripartite model more nuanced and improve its predictive utility. Several studies of the tripartite model have demonstrated that PA specifically accounts for variance associated with anhedonic symptoms (e.g., see Watson & Clark, 1991; Watson, 2005; Mineka et al., 1998). Indeed, in the present study, there was a significant main effect of PA predicting such symptoms. However, examination of the simple slopes of the NA x PA interaction revealed that at high NA, the association between PA and anhedonic symptoms was about twice as strong as it was at low NA. These results are thus consistent with the tripartite model in that they demonstrate that PA is associated with anhedonic symptoms, but they add to the tripartite model by demonstrating that the strength of that association depends on level of NA.

A tripartite model which recognizes that low PA and high NA related risk for depression varies across levels of NA and PA, respectively, has potential implications for understanding the etiology of depression and, as a consequence, for prevention efforts. First, as suggested by Wetter and Hankin’s (2009) study of mediators of the NA x PA interaction, mediating factors may be different for individuals with both high NA and low PA than for individuals who are extreme on only one of these dimensions. Thus, it will be important to consider the NA x PA interaction in future research aimed at identifying mediating and causal mechanisms. Results of the present study, of course, are but one very preliminary step toward drawing causal inferences related to this interaction. However, if future research does establish that there are unique causal mechanisms associated with particular combinations of temperament traits, then that
could have exciting implications for understanding the etiology of depression. That is, in the same way that examining how etiology or presentation of depression is different across cultures or sexes has brought increased understanding of depression (e.g., Nolen-Hoeksema, 2006) examining depression within the context of temperament or personality differences may further increase our understanding of this disorder.

The NA x PA interaction also has implications for prevention. As noted by Cuijpers (2003), samples with high incidence rates for a particular disorder are necessary for optimal tests of prevention programs. Because the NA x PA interaction identifies high NA low PA individuals as being at highest risk, testing prevention programs in samples comprised of such individuals may be highly effective. However, if the causal/mediating factors relevant to high NA low PA individuals are indeed different than those relevant to individuals with other combinations of NA and PA, programs developed and tested only in high NA low PA samples may not be effective in these other individuals. Thus, it may be necessary to develop multiple prevention programs, or to create multiple modules within a single prevention program, based on the specific mechanisms that future research identifies as most relevant for each temperament group.

The NA x PA interaction that predicted mixed anxious-depressive symptoms also has potential implications for the tripartite model. This model holds that low PA is unique to depression, which implies that low PA is minimally related to symptoms that are common to anxiety and depression. Results of the current study are consistent with this in that the main effect of NA on mixed symptoms is 3 times as large as the main effect of PA on such symptoms (i.e., at an average level of NA, PA is weakly associated...
with mixed symptoms). However, the interaction reveals that PA moderates the relation between NA and mixed symptoms such that NA is more strongly associated with symptoms when PA is low than when PA is high. Thus, it appears that for individuals with an average or low level of NA, PA is only minimally predictive of symptoms, but for individuals high in NA, level of PA is an important predictor of mixed symptoms. Together with the results discussed above, this could imply that for individuals high in NA, low PA is a more general predictor of symptoms than is suggested by the tripartite model. The integrative model hints at this by recognizing that low PA may play a role in some anxiety disorders (Mineka, et al., 1998). The two anxiety disorders that research has identified as associated with low PA, social phobia and agoraphobia, are both characterized by a high degree of distress (i.e., high NA) and a high degree of generalized withdrawal/avoidance (i.e., low PA) (American Psychological Association, 2000), a characterization that fits depression as well. Thus, perhaps it is not surprising that the present study found that low PA predicted mixed symptoms. That is, to the extent that the measure of mixed symptoms used in the present study reflected the low PA related symptoms that are common to depression and social phobia/agoraphobia, one would expect that PA would be a significant predictor of such symptoms. However, results of the present study go one step further by demonstrating that PA is more strongly related to mixed symptoms when NA is high than when it is low. Future research could examine the generality versus specificity of low PA as a vulnerability for depression and different anxiety disorders at different levels of NA. If what is suggested by the results of the present study holds true, then it would be expected that for individuals high in NA, low PA would emerge as a general vulnerability to depression.
and to anxiety disorders that have a significant PA related component, whereas for individuals with lower levels of NA, low PA would be more specific to depression. A caveat worth mentioning here is that although the NA x PA interaction was significant in predicting mixed symptoms, the proportion of variance that it accounted for in depressive and anhedonic symptoms was substantially higher than in mixed symptoms. This could suggest that the NA x PA interaction may be somewhat more relevant for symptoms that are most strongly related to depression. Alternatively, it could be a function of overlap between the measure of NA and the measure of mixed symptoms used in the present study. Examination of the instructions and items on the PANAS suggests that the NA scale could capture level of symptoms as much as it captures temperament. Specifically, the instructions direct participants to indicate the degree to which several adjectives describe how they generally feel. The degree to which an individual feels, for example, “nervous,” “distressed,” or “sad” might reflect the degree to which he or she is presently experiencing anxious or depressive symptoms as much as it reflect his or her propensity to experience negative affect (i.e., temperament). Therefore, the small amount of variance accounted for by the NA x PA interaction in predicting mixed symptoms could be attributable to the fact that NA accounted for a very large portion of the variance in symptoms, leaving less room for PA to matter.

For the N x E interaction, results of the present study replicated findings by Hotard et al. (1989) and McFatter (1994) who found N x E interactions in the prediction of depressive symptoms in college samples. However, the present results are somewhat in contrast with Gershuny and Sher’s (1998) study in which they failed to find an N x E
interaction predicting depressive or anxious symptoms cross-sectionally in a sample of college freshmen. However, these authors did find that the N x E interaction predicted both types of symptoms 4 years later. Additionally, they tested the interaction in predicting symptoms at 2 and 3 years follow up and found that the strength of the interaction increased as their participants progressed through college. Gershuny and Sher (1998) hypothesized that the situational distress characteristic of the first year of college may have masked the cross-sectional interaction effect and that the increase in strength of this interaction over time may have been due to a correlated decrease of situational distress. Based on this reasoning, it could be that Gershuny and Sher (1998) failed to find a cross-sectional N x E interaction predicting anxiety and depression symptoms because this effect was too small to be detected given their relatively small sample size (N = 466). In the present study, however, the very large sample size allowed these effects to be detected.

Results of the analyses involving the N x E interaction are consistent with results of the analyses involving the NA x PA interaction, except in the case of anhedonic symptoms. However, while the N x E interaction did not predict such symptoms, the interaction between the ATQ-Sadness and ATQ-PA facets of N and E did. Examination of the items on the ATQ-Sadness and ATQ-PA scales revealed that they primarily tap propensity to experience sadness and happiness, respectively. Thus, ATQ-Sadness and ATQ-PA seem similar to PANAS NA and PA in that they heavily reflect tendencies to experience positive and negative affect. This could suggest that the specific tendencies to experience positive and negative affect are what interact to predict anhedonic symptoms whereas the broader constructs of N and E and BIS and
BAS are more relevant for symptoms of depression in general and mixed anxious-depressive symptoms. It could also be that the NA x PA and ATQ-Sadness x ATQ-PA predict anhedonic symptoms because anhedonia involves states of high negative affect and low positive affect. Research has shown that, in general, measures of personality/temperament tap both trait and state variance (Clark, Vittengl, Kraft, & Jarrett, 2003) and it seems likely that measures, such as the PANAS and the ATQ-Sadness and ATQ-PA scales, that are heavily focused on the experience of emotions may be most susceptible to influence by state affect. If this is the case, then a hypothesis to explain why the ATQ-Sadness x ATQ-PA and NA x PA interactions predicted anhedonic depressive symptoms while N x E and BIS x BAS did not could be that the interaction between trait levels of positive and negative emotionality is not as important for anhedonic symptoms as is the interaction between current positive and negative emotional states. This hypothesis could be tested in future research by using statistical techniques similar to those used by Clark et al. (2003) to separate the trait versus state variance captured by personality measures.

**BIS and BAS.**

Contrary to prediction, results of the BIS/BAS analyses indicated that while BIS was associated with mixed symptoms across levels of BAS, this association was stronger when BAS was high than when it was low. However, this interaction replicated the BIS x BAS interaction that predicted mixed symptoms under conditions of high life stress in Hundt et al’s (2007) study. It is perhaps not surprising that Hundt et al’s (2007) high life stress interaction was replicated rather than the low life stress interaction because the majority of participants in the present study were in their first year of
college (many in their first quarter) a time that is associated with increased stress (Sher, Wood, & Gotham, 1996). The interpretation of results offered by Hundt et al. (2007) was based on revised Reinforcement Sensitivity Theory (rRST; Gray & McNaughton, 2000), which conceptualizes anxiety as the result of goal or motivation conflict (Corr, 2008) such that anxiety occurs when both appetitive/approach and aversive/avoidance motivations are simultaneously high. Thus, in the present study, rRST would predict that mixed anxious-depressive symptoms result from the distress associated with frequently experiencing approach-avoidance conflict (i.e., conflicts between high BIS and high BAS). However, neither Hundt et al.’s high life stress BIS x BAS interaction nor the BIS x BAS interaction in the present study is entirely consistent with this interpretation. That is, under rRST, one would expect there to be no relation between BIS and mixed symptoms when BAS was low. However, as is illustrated in Figure 10, at low BAS, high BIS is associated with a greater level of symptoms than low BIS. One possible interpretation, then, could be that there are two reasons that individuals experience mixed anxious-depressive symptoms. First, it could be that high BAS individuals experience mixed symptoms if they also have high BIS, because they encounter frequent approach-avoidance conflicts, which are quite distressing. Second, it could be that high BIS individuals who are low in BAS experience mixed symptoms because they are unable to get motivated and find the lack of reward/productivity that results from their low motivation to be highly distressing. In other words, individuals may experience a high degree of mixed symptoms because they are frequently distressed by approach-avoidance conflicts or because they are highly distressed by their inability to get motivated and the lack of productivity and reward that results.
The present study is obviously not able to support or refute this interpretation and alternative interpretations could be made. For example, the BIS x BAS interaction could be interpreted to suggest that when BIS is high, level of BAS does not matter (e.g., high BAS is not sufficient to attenuate the mixed anxious-depressive symptoms that are associated with high BIS), such that only when BIS is low does BAS predict symptoms. This could be consistent with other research that suggests that the main effect of BAS may not predict symptoms common to anxiety and depression (see Bijttebier, Beck, Claes, & Vandereycken, 2009 for review). Further, results of Hundt et al. (2007) demonstrate that the form of the BIS x BAS interaction that predicts mixed symptoms is dependent upon level of stress such that at low levels of stress, the interaction is consistent with the rRST interpretation described above (i.e., symptoms results from distress associated with motivation conflict), but that at high levels of stress, a more complex interpretation, such as the “two reasons” interpretation described above (i.e., there may be two reasons that individuals experience symptoms), is required. Thus, a fruitful direction for future research aimed at explicating rRST will be to further examine how this interaction is associated with mixed anxious-depressive symptoms, how that association differs across levels of stress, and what factors mediate the interaction. Regardless of the precise results of future research, however, the implications for prevention and treatment that are associated with an NE x PE interaction will apply. That is, regardless of why or how BIS and BAS interact to predict anxious and depressive symptoms, the interaction suggests that different mediators/causal processes could be operating for individuals with different combinations of BIS and BAS such that developing multiple prevention and treatment
programs aimed specifically at those mediators/causal processes could improve our ability to prevent and treat anxious and depressive disorders.

Whereas Hundt et al. (2007) found that at high levels of BIS, low BAS was more strongly associated with anhedonic depressive symptoms than high BAS, in the present study, the BIS x BAS interaction was not significant, although it was in the predicted direction. Follow up analyses involving the facets of BAS revealed that only BAS-Reward Responsiveness interacted significantly with BIS to predict anhedonic symptoms. Specifically, at high levels of BIS, low BAS-Reward Responsiveness was more strongly associated with symptoms than high BAS-Reward Responsiveness. One possible explanation for the discrepant findings was that Hundt et al. (2007) used composite BIS and BAS measures which were a combination of participants’ BIS and BAS scores from Carver and White’s measure and their scores on a measure of punishment and reward sensitivity, respectively. Thus, the BAS score used by Hundt et al. (2007) was particularly skewed to reflect reward sensitivity. This may explain why, in the current study, only the BIS x BIS-Reward Responsiveness interaction predicted symptoms unique to depression. Indeed, while a wealth of literature demonstrates that low BAS in general is associated with anhedonic symptoms, at least some of that literature seems to suggest that responsiveness or sensitivity to reward may be a particularly important predictor of anhedonic depressive symptoms (e.g., Forbes, 2009).

For example, Pizzagalli, Iosifescu, Hallett, Ratner, & Fava (2009) used a signal detection task to assess reward responsiveness in a sample of clinically depressed patients. Results indicated that, compared to control subjects, the depressed patients showed reduced responsivity to reward and that impairment was correlated with
anhedonic symptoms even after controlling for symptoms of anxiety and general
distress. Thus, as is suggested by Bijttebier, et al. (2009), it will be important for future
research to take into consideration the possible differential relations of BAS and its
subscales to depressive versus anhedonic symptoms.

The difference in the amount of variance accounted for by the BIS x BAS
interaction between the mixed anxious-depressive, depressive, and anhedonic symptoms
also highlights the importance of differentiating between these types of symptoms for
clarifying exactly what role BIS and BAS play in depression. Importantly, depressive
symptoms as tapped by the DASS-Depression scale share a substantial amount of
variance with anxiety and distress symptoms as measured by the other two DASS scales
(e.g., Henry & Crawford, 2005). Therefore, it is not surprising that the form of the BIS
x BAS interaction that predicted depressive symptoms in the present study was of the
same form as the interaction that predicted mixed anxious-depressive symptoms.
However, the interaction accounted for about half as much variance in depressive
symptoms compared to mixed symptoms and was not significant once DASS-Stress
scores were partialled out suggesting that the interaction may be most important for the
portion of symptoms that DASS-Depression also shares with anxiety and distress.
Future research using alternative measures of depression, anhedonic symptoms, and
anxiety will be necessary to determine the true form of the BIS x BAS interaction as it
relates to each type of symptoms.

The difference between the forms of the BIS x BAS interaction compared to the
NA x PA and N x E interactions in predicting both depressive and mixed anxious-
depressive symptoms highlights that, whereas the present study has grouped these three
sets of constructs under the same category, there appear to be important differences between them. Most obviously, while the BAS main effect was in the same direction (i.e., negative) as the PA and E main effects for all types of symptoms, when in interaction with BIS, BAS’s association with symptoms was different than the association between PA and E and symptoms when they interacted with NA and N, respectively. In other words, it is at high BIS that BAS is differently related to symptoms compared to PA and E. One possible explanation for this could be that BAS is more strongly associated with approach motivation than are PA and E. Indeed, as previously discussed Hundt et al. (2007) and rRST suggest that it is approach motivation in particular that interacts with high BIS to predict elevated anxious-depressive symptoms. One specific type of approach motivation to which BAS has been consistently linked is impulsivity (e.g., Corr, 2002; Quilty & Oakman, 2004). Given that both high BAS and impulsivity have been related to a number of negative outcomes such as drug and alcohol use and other externalizing behavior problems as well as symptoms of ADHD (for review see Bijttebier, et al., 2009; also see Hundt, et al., 2008; Knyazev & Wilson, 2004), one can imagine how the interaction between impulsivity and high BIS might be associated with depressive and anxious symptoms. For example, consider a high BAS (i.e., high impulsivity) individual who uses drugs and alcohol and has symptoms of ADHD. It is likely that he or she would experience negative consequences of these characteristics and behaviors (e.g., problems in school/work, interpersonal problems). Now imagine that this individual has high BIS. What might his or her reaction to those negative consequences be? Given the conceptualization of BIS as punishment sensitivity (e.g., Gray, 1981; 1987; Wallace, Newman, & Bachorowski,
one might predict that he or she would experience significant negative emotion and, perhaps, symptoms of depression and anxiety in response to the negative consequences of his or her high BAS related behaviors. Thus, to the extent that BAS reflects approach motivation, particularly impulsivity, more than PA and E do, this difference could account for the different forms of the BIS x BAS interaction compared to the NA x PA and N x E interactions in the present study.

Proportion of Variance Accounted For. The proportions of variance accounted for by the interactions in the present study were very small. As mentioned above, research by Gershuny & Sher (1998) suggests that the effect of the NE x PE interaction may be masked in young college students by the situational distress that is characteristic of the first and second year of college and that the size of the interaction effect may increase as that distress lessens over time. However, even in their longitudinal analysis, the interaction only accounted for 2% of the variance in depressive symptoms. Further, studies in children and adolescents have found similar sized effects. Specifically, Wetter and Hankin (2009) and Loney, et al. (2006) both found interactions accounting for as little as 1% - 2% of the variance. Thus, the size of the interaction effects in the current study is on par with other studies in non-clinical samples. Effects may be more substantial in clinical samples, however. Joiner and Lonigan’s (2000) longitudinal study in psychiatric inpatient children and adolescents revealed that the NA x PA interaction accounted for 15% of the variance in change in depressive symptoms, even after controlling for baseline depression symptoms and anxiety symptoms. This difference in effect sizes could be explained by restriction of range (McClelland & Judd, 1993).
Specifically, it could be that the NE and PE interaction has a strong association with depressive symptoms only when NE and PE are extreme and because traditional non-clinical samples do not have high representation of these extremes, a strong association cannot be demonstrated. Stated another way, for the majority of individuals the NE and PE main effects adequately predict their level of risk for such symptoms, but for individuals at the extremes on NE and PE, the NE x PE interaction may be a very important predictor of symptoms. Therefore, when the interaction is examined on average (i.e., in a sample with low representation of the extremes) it will appear as though the interaction accounts for only a small amount of variance. This hypothesis could be tested in a non-clinical sample that has been over-sampled on the tails of the NE and PE distributions (i.e., a sample that contains a higher than chance proportion of high NA, low PA individuals) or in a mixed sample including clinical and community members.

Future Research and Potential Treatment Implications. By demonstrating that the interaction between positive and negative temperament variables matter statistically, the present study has laid the groundwork for future research to determine its clinical importance. Results suggest several potentially fruitful avenues for future research. Because the present study was purely correlational, treatment implications will be discussed within the context of this future research. Important next steps in NE x PE research involve, among other things, replicating directionality, examining the role of interactions between states versus traits, examining mediators, and determining causality. Results from longitudinal studies by Gershuny & Sher (1998), Lonigan & Joiner (2000), and Wetter & Hankin (2009) provide some support for directionality (i.e.
temperament variables predict symptoms rather than symptoms predict temperament variables), but more research in more varied samples, particularly clinical samples and samples that have high representation of the extremes on NE and PE, is needed for replication. Further, as mentioned above, research in samples that have high representation of the extremes will be necessary for gauging the importance of the NE x PE interaction in determining depressive and anxious symptoms in high risk populations.

Longitudinal studies will provide the necessary context in which to examine the state versus trait components of NE and PE and how interactions between states versus traits are related to symptoms. For example, Clark et al. (2003) found that only state levels of NE and PE predicted change in depression severity from pre- to post-treatment, but that both state and trait levels of NE and PE predicted depression severity measured at any single time point. Thus, it could be (assuming interactions between both state and trait NE and PE are important for depressive and anxious symptoms) that the interactions between state and trait NE and PE predict different aspects of depression and anxiety. Determining the role of state versus trait variance in the NE x PE interaction could have implications for treatment as well. For example, if results of this research support the role of an interaction between state NE and PE, then increasing PE via manipulations like Loving Kindness Meditation (Fredrickson et al., 2008) may be an effective method for treatment and prevention, particularly amongst individuals who are also high in NE (i.e., individuals for whom low PE would have the most impact on symptoms).
Following the lead of Wetter & Hankin (2009), future research should examine the potential mediators of the NE x PE interaction, which will have implications both for understanding depression and anxiety and for prevention and treatment efforts. For example, suppose the BIS x BAS interaction that predicts mixed symptoms is mediated by the stress associated with impulsive behaviors. This might suggest that prevention programs could focus on teaching high BIS, high BAS individuals how to better cope with stress or that treatment could influence symptoms by decreasing impulsivity. Further, as suggested by Wetter & Hankin’s (2009) results, mediating or causal factors may differ based on different combinations of NE and PE, which might suggest unique etiological pathways. If this is the case, then researching the etiology of depression and anxiety within the context of the NE x PE interaction may help clarify our understanding of how these disorders develop. This would also imply that prevention programs could be made more effective by designing and implementing them based on the unique mechanisms operating within each temperament group (i.e., high NE low PE group, moderate NE low PE group). Additionally, as mentioned above, studying such prevention programs in samples with the highest incidence rates as determined by the NE x PE interaction would allow researchers to conduct the most effective tests (Cuijpers, 2003).

The unexpected form of the BIS x BAS interaction suggests that future research examining the differences between BAS, PA, and E is necessary to understand precisely which aspects of positive temperament (i.e., positive emotionality versus approach motivation) interact with which aspects of negative temperament (e.g., negative emotionality versus punishment sensitivity). Further, as mentioned above, additional
research is needed to clarify the BIS x BAS interaction that predicted mixed symptoms. Specifically, determining the mediators of this interaction may have implications for rRST and considering the moderating role of stress may be important for determining the parameters of rRST’s application. For example, Hundt et al.’s (2007) results seem to suggest that rRST may apply differently at low stress compared to high stress. It will also be beneficial to examine this and other NE x PE interactions using different measures of anxiety and depressive symptoms.

Further research into the role of PE and the role of the NE x PE interaction in anxiety is warranted. Specifically, results of the present study might suggest that the specificity of low PA to depression depends on level of NA. That is, for individuals high in NA, low PA may be a more general predictor of depression and anxiety disorders that have large PA related components, but for individuals lower in NA, low PA may be a specific predictor of depression. In the case of BIS/BAS, rRST predicts that conflict between BIS and BAS should specifically produce anxiety, but in the present study, this conflict was associated with mixed anxious-depressive symptoms. If future research replicates this finding and determines causality, then this might suggest that rRST could be refined by stating that approach-avoidance conflicts influences symptoms common to anxiety and depression (i.e., distress related symptoms). Examination of the extent to which motivation conflict also influences anxiety specific symptoms (e.g., panic symptoms) could also further explicate rRST.

The simplest type of future research that is suggested by results of the present study is this: testing the interaction between NE and PE constructs in existing datasets. Given the amount of research on the tripartite model and the relation between positive
and negative temperament/personality and internalizing disorders, there is undoubtedly a large number of existing datasets in which this interaction could be tested. Because these datasets no doubt contain a large variety of measures of NE, PE, and depressive and anxious symptoms, comparing results across datasets could provide a clearer picture of how this interaction differs across NE and PE constructs and across different types and severities of symptoms.

Finally, the results of the present study could potentially inform treatment choice (Zinbarg, Uliaszek, and Alder, 2008) and suggest potential ways to improve current treatments. For example, Jacobson, Dobson, Truax, Addis, Koerner, Gollan et al. (1996) conducted a study in which they compared three treatments for depression: behavioral activation therapy, behavioral activation therapy with modification of dysfunctional thoughts, and cognitive-behavioral therapy, which included all components from the other two therapies plus a focus on modifying core beliefs. Results of the study showed that all three therapies were equally effective. That is, they found that these treatments were equally effective on average. Examining the efficacy of treatments within the context of NE and PE could reveal that temperament traits moderate treatment response. For instance, individuals low in PE may respond best to behavioral activation therapies, individuals high in NE may respond best to therapies that address dysfunctional thinking, and individuals high in NE and low in PE may respond best to the more comprehensive cognitive-behavioral therapy. If this were the case, then it might suggest that choosing treatments based on an individual’s level of NE and PE could increase the likelihood of a positive treatment outcome. Further, if future research determines that PE can attenuate the relation between NE and anxious and depressive symptoms, then
this might suggest that interventions aimed at increasing level of PE could be effective at decreasing symptoms (Fredrickson, et al. 2003), particularly for individuals low in PE and high in NE. Finally, if future research determines that mediating factors are indeed different for individuals with different combinations of NE and PE (Wetter & Hankin, 2009), then designing treatments or treatment modules that specifically target those factors could improve our ability to treat anxiety and depression.

**Strengths and Limitations.** The present study has some notable strengths such as the comparison of results across multiple NE and PE constructs and a very large sample size. Interpretation of these results should, however, be placed in the context of some important caveats and limitations. First, some of the analyses in the present study were purely exploratory and no steps were taken to control for Type 1 error rate. That said, several of the interaction effects that were found would have remained significant even if the \( \alpha \) level had been set much lower than .05. Given the exploratory nature of the analyses and the very large sample size, results that were counterintuitive or difficult to explain should be regarded with particular caution.

This study is also limited by the fact that its results were derived from a group of college students who were mostly freshmen and sophomores (judging by age) and mostly Caucasian. Future research will be necessary to determine the generalizability of the results to populations that are more demographically diverse. Further, the results are limited by the method of assessment. Specifically, these findings were based exclusively on data gathered via self-report measures. While this approach is used quite often in the trait affectivity literature (e.g., Hotard et al., 1989; McFatter, 1994; Loney et al., 2006; Wetter & Hankin, 2009), it has the potential to inflate correlations between
measures due to shared method variance. Future research may benefit from the use of other methods of assessment such as the clinical interview.

Finally, these findings are limited by the modest scope and correlational design of the study. The design precludes causal conclusions and does not come to bear on questions about the mechanisms by which the NE x PE interaction has its effect. However, the purpose of the present study was to replicate the NA x PA, N x E, and BIS x BAS interactions in the prediction of depressive, anhedonic, and mixed anxious-depressive symptoms in an adult sample with the intention of encouraging future research. It is this future research that will hopefully determine causal connections and mediators of these interactions.

**Conclusion.** The present study demonstrated that the interaction between positive and negative temperament/personality is statistically important in the prediction of depressive, anhedonic, and mixed anxious-depressive symptoms. Further, it demonstrated that the association between this interaction and symptoms is different depending on the measure of temperament/personality and on the type of symptoms. Results are discussed within the context of directions for future research and implications for treatment and prevention. The underlying message of the literature review and empirical study presented in this paper is that moving from a main effects approach to an interactive approach in research involving psychological disorders has the potential to reveal important and exciting results that may not otherwise have been discovered.
REFERENCES


Miller, M. W. (2003). Personality and the etiology and expression of PTSD: A three-
factor model perspective. *Clinical Psychology-Science and Practice, 10*, 373-393.


Table 1. Descriptive Statistics

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Note: Alpha = Chronbach’s Alpha; PA = Positive Affectivity; NA = Negative Affectivity; N = Neuroticism; E = Extraversion; BIS = Behavioral Inhibition System; BAS = Behavioral Activation System; Depression = DASS-Depression scale; Str/Dep Comp. = Composite of DASS-Stress and DASS-Depression; ATQ-Fear = ATQ Fear subscale; ATQ-Disc. = ATQ-Discomfort subscale; ATQ-Frust. = ATQ-Frustration subscale; ATQ-Sad = ATQ-Sadness subscale; ATQ-PA = ATQ-Positive Affectivity subscale; ATQ-Soc = ATQ-Sociability subscale; ATQ-HIP = ATQ-High Intensity Pleasure subscale; BAS-RR = BAS-Reward Responsiveness subscale; BAS-DR = BAS-Drive subscale; BAS-FN = BAS-Fun Seeking subscale.
Table 2. Zero Order Correlations

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Note: PA = Positive Affectivity; NA = Negative Affectivity; N = Neuroticism; E = Extraversion; BIS = Behavioral Inhibition System; BAS = Behavioral Activation System; Dep. = DASS-Depression scale; St/Dep. = DASS-Stress/DASS-Depression composite; Fear = ATQ Fear subscale; Disc. = ATQ-Discomfort subscale; Frust. = ATQ-Frustration subscale; Sad = ATQ-Sadness subscale; E-PA = ATQ-PA subscale; E-Soc = ATQ-Soc subscale; E-HIP = ATQ-HIP subscale; RR = BAS-Reward Responsiveness subscale; DR = BAS-Drive subscale; FN = BAS-Fun Seeking subscale.

* = p < .05

** = p < .01
Table 3. NA and PA Regression Analysis Predicting Depressive Symptoms (DASS-Depression as the Dependent Variable)

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Table 4. NA and PA Regression Analysis Predicting Anhedonic Depressive Symptoms (DASS-Depression as the Dependent Variable Controlling for DASS-Stress)

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Table 5. NA and PA Regression Analysis Predicting Mixed Anxious-Depressive Symptoms (DASS-Depression/DASS-Stress Composite)

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Table 6. N and E Regression Analysis Predicting Depressive Symptoms (DASS-Depression as the Dependent Variable)

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Table 7. N and E Regression Analysis Predicting Anhedonic Depressive Symptoms (DASS-Depression as the Dependent Variable Controlling for DASS-Stress)

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Table 8. N and E-Facets Regression Analysis Predicting Anhedonic Depressive Symptoms (DASS-Depression as the Dependent Variable Controlling for DASS-Stress)

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<td>.002</td>
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### Table 9. N-Facets and E Regression Analysis Predicting Anhedonic Depressive Symptoms (DASS-Depression as the Dependent Variable Controlling for DASS-Stress)

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<td>ATQ-Disc</td>
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<td>ATQ-Frust</td>
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### Table 10. ATQ-Sad and ATQ-PA Regression Analysis Predicting Anhedonic Depressive Symptoms (DASS-Depression as the Dependent Variable Controlling for DASS-Stress)

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Table 11. N and E Regression Analysis Predicting Mixed Anxious-Depressive Symptoms (DASS-Depression/DASS-Stress Composite)

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Table 12. BIS and BAS Regression Analysis Predicting Depressive Symptoms (DASS-Depression as the Dependent Variable)

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Table 13. BIS and BAS Regression Analysis Predicting Anhedonic Depressive Symptoms (DASS-Depression as the Dependent Variable Controlling for DASS-Stress)

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- DASS-Str 5.783 (0.168) | .000
- Sex -0.686 (0.168) | .000
- BIS -0.065 (0.168) | .710
- BAS -0.913 (0.168) | .000
- BIS x BAS -0.172 (0.126) | .196

Table 14. BIS and BAS-Facets Regression Analysis Predicting Anhedonic Depressive Symptoms (DASS-Depression as the Dependent Variable Controlling for DASS-Stress)

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<th>ΔR²</th>
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<td>Step 2</td>
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<td>.536</td>
<td>.003</td>
<td>.077</td>
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</table>

- DASS-Str 5.754 (0.168) | .000
- Sex -0.644 (0.168) | .000
- BIS 0.084 (0.182) | .652
- BAS-FN 0.238 (0.182) | .205
- BAS-DR -0.420 (0.182) | .022
- BAS-RR -1.092 (0.224) | .000
- BIS x FN 0.112 (0.182) | .537
- BIS x DR 0.028 (0.168) | .897
- BIS x RR -0.392 (0.154) | .011
Table 15. BIS and BAS Regression Analysis Predicting Mixed Anxious-Depressive Symptoms (DASS-Depression/DASS-Stress Composite)

<table>
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<th>ΔR²</th>
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Figure 1. NA x PA interaction predicting depressive symptoms (DASS-Depression).
Figure 2. NA x PA interaction predicting anhedonic depressive symptoms (DASS-Depression controlling for DASS-Stress).
Figure 3. NA x PA interaction predicting mixed anxious-depressive symptoms (DASS-Depression/DASS-Stress composite).
Figure 4. N x E interaction predicting depressive symptoms (DASS-Depression).
Figure 5. ATQ-Sad x ATQ-PA predicting anhedonic depressive symptoms (DASS-Depression controlling for DASS-Stress.
Figure 6. N x E interaction predicting mixed anxious-depressive symptoms (DASS-Depression/DASS-Stress composite).
Figure 7. BIS x BAS interaction predicting depressive symptoms (DASS-Depression).
Figure 8. BIS x BAS-RR interaction predicting anhedonic depressive symptoms (DASS-Depression controlling for DASS-Stress.)
Figure 9. BIS x BAS interaction predicting mixed anxious-depressive symptoms (DASS-Depression/DASS-Stress composite).
Figure 10. BIS x BAS interaction predicting mixed anxious-depressive symptoms (DASS-Depression/DASS-Stress composite) at 2 standard deviations above and below the mean on BIS and BAS.