Links Between Depressive Symptoms and Reactive and Effortful Temperament
Mediated by Behavioral Activation

Dissertation

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Abstract

The present study explored the association of depressive symptoms with reactive and effortful temperament as well as behavioral activation (BA) in undergraduate students. Exploratory and confirmatory factor analyses found that items of the Adult Temperament Questionnaire Activation Control subscale (Evans & Rothbart, 2007; Rothbart, Ahadi, & Evans, 2000) and Behavioral Activation for Depression Scale (BADS; Kanter, Mulick, Busch, Berlin, & Martell, 2007; Kanter, Rusch, Busch, & Sedivy, 2009) loaded on different factors, suggesting theses measures of activation control and BA adequately assessed the distinction between these constructs. However, the results also showed strong interfactor correlations between the Activation Control factor and BADS subscale factors confirming that, though distinct, activation control and BA are closely related. Tests of simple mediation in both cross-sectional and prospective samples showed that the association between low positive emotionality (PE) and depressive symptoms was mediated by BA as was the association between negative emotionality (NE) and symptoms. BA also mediated the link between activation control and depressive symptoms in both samples. A proposed model of moderated mediation in which activation control moderated the indirect path from PE to BA to depressive symptoms was not supported. Specifically, activation control was expected to moderate the association between PE and BA. Instead, the association between PE and BA was
consistent across all levels of activation control. An inspection of items of different measures of PE revealed important differences in coverage of goal-oriented behavior. Finally, the results provided evidence of an interaction between NE and PE predicting BA such that high levels of PE were protective against low BA associated with high NE.
Dedication

For Paige and my family, thank you for your continuing love and support.
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as participants.
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Chapter 1: Introduction

Behaviors associated with depression are considered in both temperament/personality and behavioral activation (BA) models of depression. Temperament/personality theories conceptualize vulnerability to depression as a combination of reactive tendencies including low levels of positive emotionality (PE), which is characterized by loss of interest, diminished activity, and sluggishness, and high levels of negative emotionality (NE), which is associated with distress and avoidance (e.g., Clark & Watson, 1991; Mineka, Watson, & Clark, 1998). Some temperament/personality models also include consideration of the capacity for effortful control (EC), which may enable some individuals to compensate for reactive tendencies that confer vulnerability to depression (Dinovo & Vasey, 2011; Rothbart & Bates, 2006). Activation control is a facet of EC representing a capacity to carry out an action despite reactive motivation not to (Evans & Rothbart, 2007; Putnam, Ellis, & Rothbart, 2001). Preliminary research suggests that activation control may have a protective effect against depressive symptoms associated with low PE (Buffington, 2009; Vasey, Buffington, Harbaugh, Dinovo, & Hyland, 2009). Individuals with a high capacity for activation control may continue to actively engage with their environment even at low levels of PE.

BA theories of depression focus on changes in behavior to improve mood. BA consists of productive engagement with the environment and reduction of avoidance behaviors (e.g., Lejuez, Hopko, & Hopko, 2001; Martell, Addis, & Jacobson, 2001).
Consistent with expectations of BA conceptualizations, interventions targeting behavioral change have been successful in treating depression (Dimidjian et al., 2006; Mazzucchelli, Kane, & Rees, 2009).

The link between low PE and depression and the success of interventions targeting BA both suggest the importance of active, productive engagement with the environment to prevent or reduce depressive symptoms. Considering these two approaches together suggests individuals with low PE experiencing loss of interest, a feeling that nothing is enjoyable, and low approach motivation are not apt to actively engage in their environment. As such, low BA likely mediates the link between low PE and symptoms of depression. Avoidance motivation associated with high NE may also reduce activation, suggesting BA also likely mediates the link between NE and depressive symptoms. Activation control is characterized by a capacity for engagement even when an individual feels low reactive motivation or is motivated toward avoidance. This may represent a naturally occurring tendency to engage in the types of behaviors that have been successful for treating depression in BA interventions. This connection suggests the association between activation control and reduced depression is also likely mediated by BA. Finally, if the link between low PE and depression is mediated by BA, this effect may be moderated by activation control such that the indirect effect is weaker at high levels of activation control. That is, at high levels of activation control, low PE would not predict low BA or, in turn, increased depressive symptoms.

Temperament/Personality Theories of Depression

There is an extensive body of research exploring the association between temperament and a wide range of psychological symptoms and disorders, including
depression. Evidence suggests that temperament/personality affects the etiology and course of depression as well as treatment response (e.g., Clark, Watson, & Mineka, 1994; Klein, Durbin, & Shankman, 2009; Mineka et al., 1998; Muris, 2006). Temperament describes individual differences in reactivity and capacity for self-regulation that reflect the sum total of genetic, developmental, and experiential influences (Rothbart & Bates, 1998). Although personality theories historically focused on a structural approach to explaining behavior without a focus on etiology, the contemporary literature generally recognizes that core personality traits map well onto the major dimensions of temperament and that there is little difference between these two approaches (Clark & Watson, 1999). Temperament/personality models of psychopathology generally include reactive components of positive and negative emotionality. NE describes a broad tendency toward unpleasurable engagement with the self and the world characterized by the experience of emotions such as sadness, fear, and irritability. Negative affectivity, neuroticism, and Gray’s behavioral inhibition system sensitivity are all similar constructs representing NE (Clark, 2005; Lonigan, Vasey, Phillips, & Hazen, 2004; Nigg, 2006).

Conversely, PE describes a broad tendency toward pleasurable engagement with the self and the world characterized by interest, curiosity, and, at high levels, enthusiasm. Conceptualizations of PE include positive affectivity, extraversion, and Gray’s behavioral activation system sensitivity (Clark, 2005; Lonigan et al., 2004; Nigg, 2006).

High levels of NE are associated with behavioral inhibition, nonspecific arousal, attentional bias toward threatening stimuli (Gray, 1994), and cognitive processes such as worry and rumination (Muris, Roelofs, Rassin, Franken, & Mayer, 2005). Accordingly, NE has been shown to be a broad predictor of psychopathology including disorders of
mood, anxiety, eating, conduct, and personality as well as substance abuse disorders, somatoform disorders, schizophrenia, and dissociative identity disorder (Malouff, Thorsteinsson, & Schutte, 2005; Mineka et al., 1998). High NE appears to represent a broad vulnerability to general distress and is most strongly associated with disorders characterized by chronic, pervasive distress such as depression and generalized anxiety disorder (Watson, Gamez, & Simms, 2005). Low PE is characterized by low approach motivation (Gray, 1994), fatigue, and general sluggishness, and at very low levels, anhedonia (Mineka et al., 1998). Correspondingly, low levels of PE are associated with depression as well as other disorders including a component of diminished approach motivation such as social anxiety, eating disorders, and schizophrenia (Malouff et al., 2005; Mineka et al., 1998).

Models emphasizing NE and PE, including Clark and Watson’s influential tripartite model of anxiety and depression (Clark & Watson, 1991; Mineka et al., 1998), and Gray’s reinforcement sensitivity theory (Gray, 1970, 1982), propose that vulnerability to depression is characterized by a combination of high levels of NE, reflecting a general distress component, and low levels of PE, reflecting a component of sensitivity to reward and low approach motivation. Research also suggests that this combination of temperaments characterizes some anxiety disorders, such as social phobia and agoraphobia (Bienvenu, Hettema, Neale, Prescott, & Kendler, 2007; Brown, Chorpita, & Barlow, 1998; Mineka et al., 1998).

**Effortful Control**

Links between reactive temperament (i.e., NE and PE) and depression are well established and multiple reviews of the relations of NE and PE with depression are
available (e.g., Anderson & Hope, 2008; Clark, 2005; Watson, 2009). However, the vast majority of research on associations between temperament and psychopathology has not considered temperamental capacity for voluntary control of behavior. Rothbart and colleagues have proposed effortful control (EC) as a separate temperament factor to describe the capacity for regulation of reactive responses in circumstances where the prepotent response would be detrimental to the individual. EC includes the capacity to regulate both cognitive processes and behaviors as necessary to manage or override reactive inclinations. Individuals high in EC are better able to adapt to situational demands and avoid automatic reactions that might lead to undesirable longer-term consequences even when it requires sacrificing immediate rewards. The ability to manage reactive responses enables individuals to focus their attention, cognition, and behavior in ways that best serve their longer-term goals (Rothbart, 1989; Rothbart & Bates, 2006).

EC is an important consideration for any model of temperament and psychopathology because control of cognition and behavior may affect symptomatology. Indeed, research in the developmental literature suggests that low levels of EC are associated with an increase in anxious and depressive symptoms. For example, several studies comparing groups of children with internalizing problems to control groups have found lower average levels of EC in problem groups. Group differences were identified in a large sample of 2,000 Dutch preadolescent schoolchildren (Oldehinkel, Hartman, De Winter, Veenstra, & Ormel, 2004) as well as in other child samples (Eisenberg et al., 2001; John, Caspi, Robins, Moffitt, & Stouthamer-Loeber, 1994), although not without exception (Eisenberg et al., 2005). Further, several regression analyses indicating an association between EC and lower internalizing problems have been reported (Loukas &
Robinson, 2004; Morris & Age, 2009; Verstraeten, Vasey, Claes, & Bijttebier, 2010). Additionally, low EC was shown to predict increases in internalizing problems across four years (Eisenberg, Valiente, et al., 2009). Although these findings offer support for an association between EC and emotional problems, it is important to note that these results do not address this link in relation to reactive temperament.

Several other studies have been reported confirming the association between EC and psychological symptoms and showing that this link is distinct from the relation between reactive temperament and symptoms (e.g., Muris, 2006; Verstraeten, Vasey, Raes, & Bijttebier, 2009). In study of 600 students from 4th to 11th grade, Lonigan, Phillips, and Hooe (1999 in Lonigan et al., 2004) found that NE and EC were each uniquely associated with depressive and anxious symptoms. The results also showed that PE was associated with depressive problems but not anxiety problems, consistent with expectations based on Clark and Watson’s (1991) tripartite model. Additionally, two studies reported an indirect path using structural equation modeling from EC to internalizing problems through resiliency, a personality trait similar to EC that describes how an individual responds and adapts to stress (Eisenberg, Chang, Ma, & Huang, 2009; Eisenberg et al., 2004).

Positive results for unique effects of EC have also been reported in other longitudinal samples. Caspi, Henry, McGee, Moffitt, and Silva (1995) found several prospective associations over a 12-year period between reactivity and self-regulation measured in early childhood and psychological problems assessed in late childhood and adolescence. Children high in lack of control (i.e., low in EC) in early childhood were more likely to experience internalizing problems in later childhood and adolescence.
Similarly, Oldehinkel, Hartman, Ferdinand, Verhulst, and Ormel (2007) found that EC predicted internalizing symptoms two to three years later in a large study of almost 2,000 Dutch adolescents when controlling for symptoms at the initial assessment. In contrast to these positive results, Verstraeten et al. (2009) reported only a trend longitudinal effect for EC predicting depressive symptoms across a one-year period when controlling for baseline symptoms. Additionally, Verstraeten et al. (2010) reported null results for a predictive effect of EC on changes in internalizing problems over one year, although EC was strongly related to baseline symptoms in both studies. Loukas & Roalson (2006) also did not find a predictive effect for EC on internalizing problems over a one-year interval when controlling for baseline symptoms.

In addition to unique associations between psychological problems and both EC and reactive temperament, several authors have introduced models proposing that high levels of EC may have a protective effect against risks associated with high NE and low PE. These models suggest that individuals high in NE or low in PE may not experience psychological symptoms if they are also high in EC. Even when reactive temperaments are associated with emotional problems, these models suggest individuals high in EC are able to compensate for their maladaptive reactive tendencies (Lonigan & Phillips, 2001; Lonigan et al., 2004; Nigg, 2006; Rothbart & Bates, 2006). That is, an individual who is predisposed to depression by virtue of high NE and low PE may experience fewer depressive symptoms if EC is also high.

Multiple studies have demonstrated cross-sectional results consistent with a protective model of EC by showing that EC moderates the association between NE and symptoms of depression such that the link between high NE and depressive symptoms is
weaker when EC is high. Verstraeten et al. (2009) found that NE, PE, and EC were each independently related to depressive symptoms and that EC moderated the association between NE and depressive symptoms. Additionally, Vasey et al. (2002 in Lonigan et al., 2004) also found unique relationships for EC and reactive temperament with symptoms of depression and anxiety as well as a moderation effect of EC on the relationship between depressive symptoms and NE. Two studies of Dutch children and adolescents also found unique associations between both NE and EC and internalizing problems in addition to a moderation effect of EC on the link between NE and internalizing problems (Muris, 2006; Muris, Meesters, & Blijlevens, 2007). Similarly, in a recent study of Australian early adolescents, Yap et al. (2011) also found independent associations between depressive symptoms and both NE and EC and that EC moderated the association between NE and symptoms. Although research exploring the effects of EC on psychopathology has been nearly exclusively focused on children and adolescents, reflecting the origins of the addition of EC to temperament models in the developmental psychology literature, Dinovo and Vasey (2011) found results similar to those reported in the developmental literature in a sample of undergraduate students. EC was associated with measures of both symptoms of general distress and anhedonia in a cross-sectional analysis independent of the effects of NE and PE. The results also showed that EC moderated the association between NE and general distress and well as the association between NE and anhedonic symptoms of depression.

Although the results of several studies have shown evidence of a moderation effect of EC on the association between NE and depressive symptoms consistent with a protective effect of EC, these outcomes have not been found without exception. de Boo
and Kolk (2007) reported null results for an interaction between NE and EC, although depressed mood was used as the outcome measure, instead of depressive symptoms as with the other studies previously reviewed. Miller et al. (2009) also reported null results for the same moderation effect, although the sample of 75 child cancer patients was relatively small compared to the reports previously discussed.

Two studies have tested the moderating effect of EC on the link between NE and depressive symptoms in longitudinal assessments. Oldehinkel et al. (2007) found that both NE and EC predicted internalizing problems two to three years later and that EC moderated the longitudinal association between NE and internalizing problems. However, the interaction between NE and EC was nonsignificant when controlling for initial levels of internalizing problems. Verstraeten et al. (2009) also reported null results for an interaction between EC and NE predicting depressive symptoms when controlling for baseline symptoms in a one-year longitudinal assessment. The authors also found support for moderated mediation in which EC moderated whether rumination mediated the link between NE and depressive symptoms. This finding as well as models of moderated mediation will be more fully described in a later section.

Although several studies have found support for an interaction between NE and EC for depressive symptoms, much less attention has been paid to the interaction between PE and EC. In fact, many studies of NE, EC, and depressive symptoms have excluded consideration of PE altogether (e.g., Muris, 2006; Muris et al., 2007; Yap et al., 2011). However, PE is a key aspect of a full picture of how reactive and effortful temperament relate to depressive symptoms and consideration of an interaction between PE and EC is important. A protective model of EC suggests low PE should be less
strongly related to depression in the presence of high EC. Indeed, Vasey et al. (2002 in Lonigan et al., 2004) found an interaction between PE and EC for symptoms of depression in a cross-sectional analysis. Verstraeten et al. (2009) also reported a cross-sectional moderating effect of EC on the link between low PE and depressive symptoms, although only for girls. Similarly, Dinovo and Vasey (2011) found that EC moderated the association between PE and anhedonia for women, but not for men. The same studies that reported null results for an NE by EC interaction also reported null results for a PE by EC interaction, although one used depressed mood as the dependent variable (de Boo & Kolk, 2007) and the other had a small sample size with limited statistical power to detect interactions (Miller et al., 2009). Finally, in a longitudinal test, Verstraeten et al. (2009) did not find an interaction between PE and EC predicting depressive symptoms one-year later when controlling for baseline symptoms.

In addition to tests of the moderation effect of EC on the association between reactive temperament and depressive symptoms, evidence has also been reported that some observational and self-report measures of EC moderate the link between other risk factors and internalizing and externalizing problems. Risk factors such as low SES, having an adolescent parent, maternal depression, and household density have been shown to be more strongly associated with internalizing and externalizing problems at low levels of EC in both cross-sectional (Lengua, 2002) and prospective analyses (Lengua, Bush, Long, Kovacs, & Trancik, 2008).

Taken together, available evidence supports an association between EC and psychological problems independent of reactive temperament. The evidence also generally supports a protective model of EC. Although some studies have reported null
results, several studies have found a moderating effect of EC on the association between NE and emotional problems such as depression and anxiety. Available evidence also supports a moderating effect of EC on the association between PE and depressive symptoms, although only a few studies have provided strong tests of this effect. These findings will be strengthened by future replication.

Facets of Effortful Control

Consideration of EC as a single, broad construct has been successful, but it is potentially limiting. Dividing EC into different facets may provide a better test of a protective model of EC. Different facets likely have distinct roles in their associations with depression and evidence suggests the moderating effect of EC on the association between NE and PE and depression are dominated by different facets (Buffington, 2009; Vasey et al., 2009). Testing this model with more finely refined components of EC may improve the signal to noise ratio for each interaction relative to a broader conceptualization of EC and thereby improve statistical power, which is an important concern for detecting interaction effects (McClelland & Judd, 1993).

According to Rothbart and colleagues, the higher-order factor EC can be further divided into facets of attentional control, inhibitory control, and activation control in adults and adolescents (Evans & Rothbart, 2007; Putnam et al., 2001). The facets parse EC into components of flexible control of internal cognitive processes as well as behavior. In addition to their theoretical validity, factor analyses by Rothbart and colleagues have confirmed that these facets load on a higher order EC factor with general consistency for a range of ages from childhood to adulthood (Ahadi, Rothbart, & Ye, 1993; Evans & Rothbart, 2007; Putnam et al., 2001; Rothbart, Ahadi, & Evans, 2000;
Rothbart, Ahadi, Hershey, & Fisher, 2001). Distinguishing between individual facets of EC may improve our understanding of the associations between NE, PE, EC, and psychological symptoms.

Attentional control. Attentional control is the capacity to flexibly focus and shift attention between stimuli as well as to focus thought and resist distraction. It is thought to play a key role in regulating emotion by enhancing or minimizing focus on positive and negative thoughts or stimuli (Derryberry & Reed, 1994, 1996, 2002; Eisenberg, Fabes, Guthrie, & Reiser, 2000; Posner & Rothbart, 2000). Attentional control is a primary component of EC as voluntary deployment of attention enables individuals to control reactive tendencies and optimally manage behavior (Rothbart & Rueda, 2005). Indeed, several reports on EC have used measures of attentional control to assess EC (Meesters, Muris, & van Rooijen, 2007; Muris, de Jong, & Engelen, 2004; Muris, Mayer, van Lint, & Hofman, 2008; Muris, Meesters, & Rompelberg, 2006; Vasey et al., 2002 in Lonigan et al., 2004). Attentional control consistently loads on an EC factor in factor analyses for children (Rothbart et al., 2001), adolescents (Putnam et al., 2001), and adults (Evans & Rothbart, 2007; Rothbart et al., 2000), although it did not load on an EC factor in one reported factor analysis with a child sample (Ahadi et al., 1993).

Like EC, self-report measures of attentional control have also been shown to be negatively correlated with reports of symptoms of anxiety and depression (Healy & Kulig, 2006; Lengua, West, & Sandler, 1998; Moriya & Tanno, 2008; Muris et al., 2008; Muris et al., 2006). Several studies have also found that attentional control is uniquely related to emotional problems independent from reactive temperament (Meesters et al., 2007; Muris et al., 2004; Muris et al., 2007; Sportel, Nauta, de Hullu, de Jong, &
Hartman, 2011; Vasey et al., 2002 in Lonigan et al., 2004). Further, attentional control was shown to moderate the association between NE and depressive symptoms in several studies. Consistent with expectations of a protective model of EC, Vasey et al. (2002 in Lonigan et al., 2004) found an interaction between NE and attentional control for symptoms of depression. Similarly, a recent study of Dutch adolescents also found that attentional control moderated the link between NE and depressive symptoms (Sportel et al., 2011). No studies to date have reported null results for a moderation effect of attentional control on the association between NE and depressive symptoms.

Additionally, although only one test of the PE by attentional control interaction has been reported to date, Vasey et al. (2002 in Lonigan et al., 2004) found that attentional control moderated the association between PE and depressive symptoms.

Although a number of studies reported results for attentional control, none of the reports previously reviewed considered multiple facets of EC in the same analysis. Thus, these studies did not provide a test of whether the association between attentional control and emotional symptoms is unique from the other facets of EC. In a sample of undergraduate students, Buffington (2009) analyzed all three facets of EC (i.e., attentional control, inhibitory control, and activation control) simultaneously along with reactive temperament, allowing a direct comparison of each facet relative to the other facets. The results showed that attentional control was associated with general distress and depressive symptoms independent of reactive temperament, inhibitory control, and activation control. Additionally, attentional control moderated the association between NE and both symptom groups. Similarly, Vasey et al. (2009) found that attentional control was uniquely related to depressive symptoms in an analysis.
including reactive temperament, attentional control and persistence, a construct similar to activation control. Further, the results showed that only attentional control moderated the association between NE and depressive symptoms.

**Inhibitory control.** Inhibitory control is the capacity to intentionally suppress motor behavior when desirable. Although the term inhibitory control is sometimes used to describe cognitive inhibition of thought or emotion, those capabilities are more theoretically similar to attentional control than to inhibitory control in the framework of the three facets of EC. It is also important to distinguish inhibitory control from inhibition due to NE, such as freezing in fear situations. Although reactive temperament can suppress behavior, it is different from the inhibitory control discussed here because it not flexible. In fact, behavioral inhibition due to reactive temperament may cause suppression of desired behavior as well as undesired behavior (Derryberry & Rothbart, 1997). Measures of inhibitory control have not consistently performed as expected. In factor analyses, inhibitory control consistently loads primarily on an EC factor, but it also loads on reactive factors in samples of children (Ahadi et al., 1993; Rothbart et al., 2001), adolescents (Putnam et al., 2001), and adults (Evans & Rothbart, 2007). The inhibitory control scale has demonstrated a low internal reliability for both adults (Evans & Rothbart, 2007) and adolescents (Verstraeten et al., 2010).

Inhibitory control of behavior is not likely to be an important factor for depression as high reactive control is common for internalizing disorders (Eisenberg et al., 2001; Eisenberg et al., 2005). In fact, depressed individuals are likely to demonstrate low activity or anhedonia (Mineka et al., 1998). Children with internalizing problems do not have a tendency to engage in inappropriate behavior, so there is little need to exercise
inhibitory control (Derryberry & Rothbart, 1997; Eisenberg & Morris, 2002; Muris & Ollendick, 2005). Consistent with predictions, empirical data show children with internalizing problems do not differ from non-disordered children in inhibitory control. Despite the behavioral focus of inhibitory control in this context and the theoretically meaningful distinction between internal and external inhibition (see also Nigg, 2000), there is some evidence that cognitive and behavioral inhibition cannot be meaningfully separated because areas of the brain responsible for motor control are also involved in cognitive tasks traditionally used to test cognitive inhibition (Aron, 2007). Even if behavioral inhibition is not theoretically an important factor for depression, if inhibitory control of behavior and cognition are not separable, measures of inhibitory control of behavior may account for aspects of cognitive control and yield positive results. Indeed, Buffington (2009) found that inhibitory control was associated with distress symptoms common to anxiety and depression. Inhibitory control has also been shown to be negatively correlated with depressive symptoms (Moriya & Tanno, 2008). Additionally, in a recent study of adolescents, Raes, Verstraeten, Bijttebier, Vasey, and Dagleish (2010) found that inhibitory control partially mediated the association between overgeneral memory recall of autobiographical memories and depressive symptoms.

**Activation control.** Activation control is the capacity to perform an action despite motivation not to act (Evans & Rothbart, 2007; Putnam et al., 2001). However, measurement items for activation control such as “I often make plans that I do not follow through with” or “I am often late for appointments” are also consistent with a broader definition including taking action in the absence of immediate rewards even if there is no specific motivation not to act. As with inhibitory control, it is important not to confuse
activation control with reactive behavioral influences, such as action due to impulsivity in
a case of high PE. Activation control consistently loads significantly and exclusively on
an EC factor in factor analyses for adolescents (Putnam et al., 2001) and adults (Evans &
Rothbart, 2007). Activation control has received very little attention in the literature,
although reliable measures for activation control have not been available for as long as
for the other facets. More recently though, the scale has been reworked and now
demonstrates improved internal reliability as well as expected loadings in factor analyses
(Evans & Rothbart, 2007; Putnam et al., 2001).

Activation control is likely important to regulation of diminished activity
characteristic of depression. Depressed individuals also often engage in avoidant
behaviors to escape from potential stressors. Avoidance can include reduced
socialization, low attendance at work, or remaining in bed throughout the day (Jacobson,
Martell, & Dimidjian, 2001). Low approach and anhedonia associated with depression
are also related to low levels of PE (Clark & Watson, 1991; Mineka et al., 1998). The
association between low PE, low activity or anhedonia, and depressive symptoms
suggests that a potential protective effect of activation control may operate by moderating
the association between PE and depression. That is, individuals with low PE and a
tendency toward low activity may not experience depressive symptoms if they are also
high in activation control and are thus able to continue to engage with the environment
despite their reactive tendencies. An extension of the definition of activation control to
include action in the absence of reactive approach motivation offers further theoretical
support for a protective effect against general malaise associated with depression.
Consistent with expectations, activation control has been shown to negatively correlate with depressive symptoms (Moriya & Tanno, 2008). Buffington (2009) found an association between activation control and depressive problems unique from reactive temperament or the other facets of EC. Further, activation control moderated the link between PE and depressive symptoms such that high levels of activation control weakened the association between low PE and increased symptom reports. Vasey et al. (2009) examined links between depressive symptoms and persistence, a construct similar to activation control. Persistence was measured using the Persistence-Low Distractibility subscale of the Effortful Control Scale (Lonigan & Phillips, 2001), which includes items such as “When an activity or task is difficult, I give up (reverse scored)” that are clearly relevant to activation control. The authors found that persistence moderated the association between PE and depressive symptoms, although surprisingly there was no main effect for persistence. Only a few empirical reports of activation control have been published to date, but promising early results from these tests supporting a protective effect against depression indicate that this is an important area for further research as in the present study. Additionally, testing a PE by activation control interaction may provide a more specific test of the PE by EC interaction and improve power to find the expected pattern of results.

Other Interactions

In addition to tests of a protective model of EC, considering an interaction between NE and PE may also help clarify links between temperament and psychopathology. Despite extensive research on how NE and PE relate to emotional problems, current models have not specifically addressed whether NE or PE moderates
the others’ association with psychological symptoms. Risk for emotional problems should be especially high when NE is high and PE is low. Additionally, high PE may have a buffering effect against depressive symptoms associated with high NE, such that the relation between high NE and depression is weakened when PE is high. Several studies of child and adolescent samples have demonstrated an interaction between NE and PE for depressive symptoms consistent with this pattern (e.g., Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapis, 2007; Joiner & Lonigan, 2000; Wetter & Hankin, 2009).

Consistent with a protective model of PE, research by Frederickson and colleagues suggests a buffering effect of positive emotions against depressive symptoms. Employees of a software company who were randomly assigned to a meditation workshop experienced more positive emotions than a waitlist comparison group. Additionally, increases in positive emotions predicted lower depressive symptoms (Fredrickson, Cohn, Coffey, Pek, & Finkel, 2008). Further, experiences of positive emotion mediated a link between high resiliency before the September 11th terrorist attacks and lower levels of depressive symptoms after the attacks in a sample of undergraduate students (Fredrickson, Tugade, Waugh, & Larkin, 2003). These results suggest positive emotions can buffer against depressive symptoms after psychological distress.

An NE by PE interaction suggests the moderating effect of EC may be most apparent when the risk for depressive symptoms is the highest, i.e., when NE is high and PE is low (Harbaugh et al., 2011). Indeed, Dinovo and Vasey (2011) found evidence for a three-way interaction such that EC was most strongly related to general distress and anhedonia when NE was high and PE was low. That is, support for a protective effect of
EC was more apparent at higher-risk levels of reactive temperament indicating high avoidance and low approach motivation. These results are consistent with expectations based on a protective model of EC because a capacity for regulating reactive response should be less important when levels of NE and PE are not associated with risk for depression.

Behavioral Activation Treatments for Depression

The association between low PE and depression is likely due in part to diminished activity associated with lack of interest and low approach motivation that are characteristic of low PE. Additionally, a protective effect of activation control against depression that is especially pertinent at low levels of PE suggests individuals high in activation control are less likely to decrease activity despite reduced interest. High NE is also associated with depression as well as avoidance, which may also lead to decreased activity. Consistent with the importance of behavior to links between PE, NE, activation control, and depression, contemporary BA treatments for depression (Lejuez, Hopko, & Hopko, 2001; Martell et al., 2001) emphasize BA to reduce depressive symptoms. BA consists of engagement in goal-oriented activities to naturally elicit positive reinforcement from the environment instead of resorting to avoidance behaviors typical to depression.

The behavioral focus of BA treatments is especially relevant to temperament/personality conceptualizations of depression because it focuses on stimulating the type of diminished activity associated with low PE and high NE. BA treatments provide a potential mediator of links between depression and low PE, NE, and activation control. That is, low PE and high NE are likely associated with low BA, which
has been identified by BA treatments as a contributing factor to maintaining depression. Further, high levels of activation control may naturally facilitate the same behaviors identified by BA as important for symptom relief even when PE is low. If so, BA should mediate the links between low PE and high NE with depression as well as the association between activation control and depression.

BA treatment for depression (Dimidjian, Martell, Addis, & Herman-Dunn, 2008; Jacobson et al., 2001; Martell et al., 2001; Martell, Dimidjian, & Herman-Dunn, 2010) encourages engagement in activities that provide natural reinforcement from the environment to improve mood and encourage future activation in a continuous cycle. In this model, conceptualization of depression is based on the relationship between activity and mood. In addition to the benefits of rewarding activities, BA focuses on the detrimental effects of avoidance and escape behavioral patterns common to depression. Treatment is designed to interrupt avoidance patterns and replace them with more adaptive behaviors that are rewarding or serve long-term goals. Functional analysis of behavior is a core component of BA used to identify both reward and avoidance behaviors in a particular client’s life. The combination of activating reward behaviors and decreasing avoidance is thought to have an antidepressant effect.

BA treatments focus on modifying avoidance and escape behaviors. Behavior of depressed individuals is often motivated by short-term desire to escape aversive consequences (i.e., negative reinforcement) rather than achieving long-term goals (i.e., positive reinforcement). Focus on immediate relief interferes with long-term rewards and goals and can narrow behavioral options (Martell et al., 2001; Martell et al., 2010). For example, someone with depression may elect to stay in bed to avoid the difficulty of
having to go to work, which would provide short-term relief at the expense of longer-term job security. Long-term difficulties caused by short sighted avoidance behaviors subsequently increase depressive mood, which further sharpens the maladaptive focus on immediate relief and reduces opportunities to engage in rewarding behaviors. This sequence further lowers mood and can repeat indefinitely in a continuous downward cycle.

BA treatment teaches behaviors aimed at disrupting and reversing this downward cycle. Activation is thought to lead to positive emotion and increased future activation in a continuous upward cycle (Martell et al., 2001; Martell et al., 2010). BA encourages goal-directed behavior instead of mood-directed behavior and emphasizes to clients that changes in mood do not have to precede changes in behavior. Activation begins with small behavioral changes and strict routine before progressing to more difficult behaviors.

The renewed interest in a behavioral focus for treating depression began following encouraging results from a large-scale treatment study of adults suffering from major depressive disorder (Jacobson et al., 2001). In the study, Jacobson et al. (1996) found that treatment based on the BA component of cognitive therapy for depression by Beck et al. (1979) was successful in treating depression at both post-treatment and six-month follow-up. The same results were confirmed at 12, 18, and 24-month follow-up evaluations (Gortner, Gollan, Dobson, & Jacobson, 1998). Another large-scale study testing BA in a clinically depressed sample also reported successful outcomes for participants at post-treatment (Dimidjian et al., 2006) as well as at one and two-year follow-up assessments (Dobson et al., 2008).
Other treatments for depression focusing on increasing behavior have also been successful. Similar to BA, behavioral activation treatment for depression (BATD; Lejuez, Hopko, & Hopko, 2001, 2002; Lejuez, Hopko, LePage, Hopko, & McNeil, 2001) also focuses on increasing engagement with the environment and reducing avoidance. BATD has shown promise in a number of different populations including clinically depressed inpatients (Hopko, Lejuez, LePage, Hopko, & McNeil, 2003), moderately depressed undergraduate students (Gawrysiak, Nicholas, & Hopko, 2009), and depressed cancer patients (Hopko et al., 2008; Hopko, Bell, Armento, Hunt, & Lejuez, 2005). Additionally, several meta-analyses of randomized trials of a wider range of behavioral treatments of depression have reported efficacy for behaviorally focused treatments (Cuijpers, van Straten, Andersson, & van Oppen, 2008; Cuijpers, van Straten, & Warmerdam, 2007; Ekers, Richards, & Gilbody, 2008; Mazzucchelli et al., 2009).

Taken together, this large body of research including a detailed theoretical foundation, large-scale trials of BA, and multiple meta-analyses all point to the efficacy of behavioral treatments for depression. However, although the success of behaviorally-focused treatments suggests a direct impact of behavioral changes on depression, it is important to note that this promising hypothesis requires further research (Martell, Addis, & Dimidjian, 2004). The Behavioral Activation for Depression Scale (BADS; Kanter, Mulick, Busch, Berlin, & Martell, 2007; Kanter, Rusch, Busch, & Sedivy, 2009) was specifically designed to assess progress toward goals targeted by BA treatment including activation, avoidance, rumination, and occupational and social functioning and may aid future research into mediators of treatment effects. Regardless of whether behavioral change mediates the efficacy of BA treatments, BA is relevant to
temperament/personality theories of depression as a potential mediator of links between low PE, high NE, activation control, and depression. Activation, avoidance, and improvements in social and work functions as measured by the BADS reflect the focus of BA on goal-oriented behaviors that lead to accomplishment and are all important considerations for how reactive and effortful temperament relate to depression. Measuring BA as defined by BA treatments using the BADS allows tests of the proposed meditational effects in the present study and thereby offers a bridge between temperament/personality and BA theories of depression.

*Moderated Mediation*

Analyses of moderator and mediator variables are common in psychological research, as illustrated by many of the studies previously discussed. A moderator variable alters the association between two variables such that the strength of the relationship depends on the level of the moderator variable. A mediator variable carries the association between an independent variable (X) and an outcome variable (Y) such that the independent variable (X) affects the outcome variable (Y) indirectly through its effect on a mediator variable (M), which in turn affects the outcome variable (Y) as in Figure 1 (Baron & Kenny, 1986). Links between variables are often more complicated than simple moderation or mediation and the two functions are not mutually exclusive. Moderated mediation occurs when a mediation effect depends on the level of a moderator variable. That is, the path \(a\) from the independent variable (X) to the mediator variable (M), the path \(b\) from the mediator variable (M) to the outcome variable (Y), or both may be moderated by one or more variables (James & Brett, 1984; Muller, Judd, & Yzerbyt, 2005). The present study focuses on a model of moderated mediation in which the path
(a) from the independent variable (X) to the mediator variable (M) is moderated by a fourth variable (W) as in Figure 2. In this case, the moderator (W) interacts with the independent variable (X) to predict the mediator (M) so that the indirect effect ($ab$) changes at different levels of the moderator (W).

Models of moderated mediation such as these may be relevant to improving our understanding of the protective effects of EC by enabling an exploration into potential mechanisms of this effect. Vasey, Harbaugh, Buffington, Bills, and Dinovo (2008) performed a test of moderated mediation in a sample of undergraduate students. Data was collected in a prospective design at three assessment points over an eight-week period. The results showed that rumination at time two mediated the association between NE at time one and depressive symptoms at time three when controlling for baseline symptoms. Rumination is the tendency to repetitively dwell on negative emotion and the causes, meaning, and consequences of negative aspects of life. It is characteristic of depression and has been linked to increased severity and duration of depressive episodes (Nolen-Hoeksema, 1991, 2000). In addition to the mediation effect of rumination, EC at time one moderated the path between NE and rumination such that mediation effect of rumination only occurred at low levels of EC. That is, the mediation effect of rumination on the association between NE and depressive symptoms depended on the level of EC.

Verstraeten et al. (2009) also tested whether EC moderated the mediation effect of rumination on the association between NE and depressive symptoms in a sample of Belgian children and adolescents. In cross-sectional data, rumination mediated the association between NE and depressive symptoms and EC moderated the path between NE and rumination. In prospective data including a follow-up assessment one year later,
a different pattern of moderated mediation was found. Rumination mediated the association between NE and depressive symptoms when controlling for baseline symptoms, but EC moderated the path between rumination and depressive symptoms, not the path between NE and rumination as in the cross-sectional analysis.

The Present Study

Both temperament/personality theories and BA conceptualizations of depression identify reduced interest in activity and increased avoidance as common characteristics of depression (Clark & Watson, 1991; Martell et al., 2001; Mineka et al., 1998). Temperament/personality theories associate low approach motivation and avoidance with a vulnerability for depressive symptoms and indeed, BA treatments, which target change in behavior and emphasize increased activation and reduced avoidance, have been successful (e.g., Dimidjian et al., 2006; Jacobson et al., 1996). These results suggest increased BA as prescribed by BA treatments is associated with a decrease in depressive symptoms and are consistent with a conceptual link between diminished activity and depression as proposed by temperament/personality theories.

Individuals high in activation control may be naturally more likely to engage in the types of approach behaviors prescribed by behavioral treatments for depression even at lower levels of PE when reactive motivation is diminished. A capacity for BA may have a protective effect against depressive symptoms by compensating for low levels of PE normally associated with depressive symptoms. Only a few tests of this protective model have been reported, but initial evidence is promising. Activation control, and not attentional control, has been shown to moderate the association between PE and depressive symptoms in two samples of undergraduate students (Buffington, 2009; Vasey
et al., 2009). Also, as previously discussed, several studies have reported a moderating effect of EC on the association between PE and depressive symptoms (Dinovo & Vasey, 2011; Verstraeten et al., 2009; Vasey et al., 2002 in Lonigan et al., 2004). In contrast, two other studies found null results for this interaction, although one study measured depressed mood instead of symptoms (de Boo & Kolk, 2007) and the other utilized a small sample that may have had insufficient power to detect the interaction (Miller et al., 2009). These studies may have also benefited from testing an interaction between PE and activation control instead of between PE and broad EC to provide a more targeted test.

Multiple studies have also identified a link between NE and depression (e.g., Muris, 2006; Muris et al., 2007; Verstraeten et al., 2009). In addition to sadness, fear, and irritability, high NE is also associated with avoidant behaviors, which may be another path to reducing BA and may in turn lead to an increase in depressive symptoms.

These results suggest several interesting models of mediation and moderated mediation between PE, NE, activation control, BA, and depression. However, before addressing those possibilities, it is important to establish a distinction between activation control and BA. Although closely related, activation control and BA are separate constructs. Activation control is a temperamental capacity to perform an action despite motivation not to act, while BA represents actual activity. To ensure the theoretical differences between these two constructs are represented by their measures, an exploratory factor analysis (EFA) and follow-on confirmatory factor analysis (CFA) will be carried out to determine whether items from the scales measuring activation control and BA load on different common factors. This expected pattern would indicate that the theoretical separation of these constructs is reflected in their measures.
The implication of temperament/personality theories is that the association between low PE and depression is mediated by diminished activity as a result of reduced interested, sluggishness, and low approach motivation. Additionally, the link between high NE and depression may be due in part to diminished activity as a result behavioral avoidance. However, these models have not been explicitly tested. BA models focus more specifically on increased activation and reduced avoidance leading to accomplishments. Combining these approaches suggests that BA may mediate the links between low PE, high NE, and depressive symptoms. Thus, the present study tests the indirect effect of low PE on depressive symptoms through BA as measured by the BADS, which was designed to assess BA as defined by BA treatments. Additionally, an indirect effect between high NE and depressive symptoms mediated by BA will also be tested. A protective effect of activation control on depressive symptoms would also be expected to be mediated by an increase in BA. As such, an indirect effect of low activation control on depressive symptoms via BA as measured by the BADS will also be tested.

Consideration of the indirect effect from low PE to low BA to depressive symptoms in combination with the protective model of activation control suggests the protective effect of activation control would likely function by increasing BA despite low PE. That is, activation control may moderate an indirect path between low PE and depressive symptoms mediated by low BA by altering the link between PE and BA. This pattern will be tested using a model of moderated mediation in which the path from low PE to low BA to depressive symptoms is moderated by activation control (see Figure 3). The indirect path between low PE and depressive symptoms is expected to be mediated by low BA, but only when activation control is low. That is, the indirect effect is
expected to be weaker when activation control is high because low PE may not be associated with low BA when activation control is high.

The potential effects of low PE and high NE on reducing BA suggest low PE may be associated with especially low BA when NE is also high. Other studies have reported an interaction between NE and PE for depression with a similar pattern (e.g., Joiner & Lonigan, 2000; Wetter & Hankin, 2009). Thus, an NE x PE interaction term for predicting BA will be tested. Further, a recent review of the potential importance of a three-way NE x PE x EC interaction (Harbaugh et al., 2011) suggests consideration of whether the effect of activation control is more apparent at low PE and high NE. Indeed, a recent report of a test of an NE x PE x EC interaction for depressive symptoms suggested the association between EC and depressive symptoms was strongest when PE was low and NE was high (Dinovo & Vasey, 2011). An exploratory test will be done to examine the importance of a three-way interaction between NE, PE, and activation control. NE may further moderate the association between the PE x activation control interaction and BA such that the effect of activation control is maximized when NE is high and PE is low. Conversely, the effect of activation control may be much smaller when little reactive vulnerability exists as in cases where PE is high and NE is low.

In Study 1, a large cross-sectional sample will be used to maximize statistical power to test the proposed interactions as well as provide an adequate number of cases for an EFA and follow-on CFA to verify the distinction between the activation control and BA measures. However, the cross-sectional design does not allow a test of mediation over time in which the predictive power of the independent and mediation variables on change in the dependent variable can be assessed. As such, Study 2 will test the proposed
models of mediation and moderated mediation in a separate prospective sample to confirm the results of Study 1.

Summary

The aims of the present study are to first, determine whether the measures of activation control and BA reflect the theoretical distinction between the two constructs by submitting the measure items to an EFA and a CFA. Items from the scales measuring activation control and BA are expected to load on different common factors. Second, models of simple mediation will be tested in which BA is expected to mediate the links between depressive symptoms and PE, NE, and activation control. Finally, a model of moderated mediation will be tested in which activation control is expected to moderate the indirect effect from PE to BA to depressive symptoms by moderating the path between PE and BA (see Figure 3).
Chapter 2: Methods

Participants

Study 1. 1277 undergraduate students enrolled in an introductory psychology class participated in Study 1. Participants in this unselected sample were not prescreened or selected based on any individual characteristics. The sample was 55.3% female with a mean age of 19.1 years.

Study 2. A separate sample of 288 undergraduate students enrolled in an introductory psychology class participated in Study 2. The sample was collected during a different academic year than Study 1. The sample was 67.8% female with a mean age of 19.4 years.

To improve statistical power, participants were prescreened for NE, PE, and EC. Invitations were disproportionally offered to improve the chance of detecting the predicted effects by favoring students in the upper or lower tertiles for NE, PE, or EC. The focus of the interaction effects between these variables are at the confluence of high and low levels of NE, PE, and EC. Oversampling the population of interest was expected to increase the variance in the interaction terms and indeed the standard deviations of the interactions were 5-15% larger than in Study 1. Descriptive statistics for both samples are listed in Tables 1 and 2 and correlations are listed in Tables 3 and 4. Although oversampling can be a useful design technique, the results must be interpreted with the understanding that the interaction terms may account for a larger proportion of variance
in the oversampled group than in the general population (McClelland & Judd, 1993; Whisman & McClelland, 2005).

Measures

Positive and Negative Affect Schedule (PANAS) – Trait Form. The PANAS (Watson, Clark, & Tellegen, 1988) is a self-report measure of affect comprised of two 10-item subscales measuring negative affect (NA) and positive affect (PA). The scales are made up of emotion words (e.g., “distressed” or “excited”), which respondents are asked to answer based on the degree to which they generally feel that way. The scales have shown high internal reliability and test-retest reliability with undergraduates. Each scale also correlates highly with similar measures, but they are not highly correlated with each other (Watson et al., 1988).

Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) Scales. The BIS/BAS scales (Carver & White, 1994) are designed to measure an individual’s sensitivity to punishment and reward. Individuals reporting high BIS sensitivity should be especially responsive to punishment cues and those with high BAS are expected to be particularly sensitive to reward cues. The scale includes seven BIS items and 13 BAS items with subscales for reward responsiveness, drive, and fun seeking. Although there are subscales, a total BAS score can be derived from the simple sum of the BAS items. The BAS scale was treated as a single measure in the present study. The BIS/BAS scales have shown adequate validity and reliability (Carver & White, 1994).

Affect Intensity Measure (AIM). The AIM (Larsen & Diener, 1987) is designed to measure the subjective strength of emotional responses. The AIM was originally designed as a unidimensional construct, but later analyses identified multiple factors
distinguishing between positive and negative valence as well as reactivity and intensity of emotion that are best represented by a 27-item subset of the original items (Bryant, Yarnold, & Grimm, 1996; Weinfurt, Bryant, & Yarnold, 1994). Emotional reactivity refers to the strength of emotional response and intensity is the magnitude of emotion an individual feels. These are conceptually distinct as individuals with a high capacity for self-regulation may experience low negative intensity despite high negative affectivity. Although conceptually relevant, the AIM does not adequately distinguish between positive reactivity and intensity as evidenced by nearly perfect correlations between the subscales in several samples (Bryant et al., 1996; Weinfurt et al., 1994). Both the total score and individual factors have shown moderate to good internal reliability (Bryant et al., 1996; Larsen & Diener, 1987; Weinfurt et al., 1994).

*Adult Temperament Questionnaire (ATQ) – Short Form.* The ATQ (Evans & Rothbart, 2007; Rothbart et al., 2000) was developed by Rothbart and colleagues as a self-report temperament measure for adults. The short form consists of 77 items and includes three EC subscales to measure attentional control, inhibitory control, and activation control. The activation control subscale has shown good internal reliability and loads on the expected EC factor in factor analyses of the items (Evans & Rothbart, 2007).

*Behavioral Activation for Depression Scale (BADS).* The BADS (Kanter et al., 2007; Kanter et al., 2009) was designed to measure activation and avoidance in BA treatment clients during the past week. The 25-item scale consists of four subscales: Activation, Avoidance/Rumination, Work/School Impairment, and Social Impairment. Items on the last three subscales are reverse-scored so that higher scores reflect increased activation. The BADS has demonstrated high internal consistency in both undergraduate
and community samples as well as significant correlations in the expected direction with other measures of both cognitive and behavioral avoidance, rumination, social support, and depression (Kanter et al., 2007; Kanter et al., 2009).

**Depression Anxiety Stress Schedule (DASS).** The DASS (Lovibond & Lovibond, 1995) is a 42-item self-report questionnaire designed to measure levels of depression, anxiety, and stress on three separate subscales. Each subscale consists of 14 items. Respondents are instructed to rate a series of statements describing negative emotions based on how much each item applies to them over the past week. Each of three subscales has shown high internal reliability in samples of college students (Lovibond & Lovibond, 1995) and nonclinical adults (Crawford & Henry, 2003), as well as in clinical samples from an array of mood and anxiety disorders (Antony, Bieling, Cox, Enns, & Swinson, 1998; Brown, Chorpita, Korotitsch, & Barlow, 1997; Clara, Cox, & Enns, 2001).

The DASS depression subscale was developed to maximize consensus with clinical diagnoses as a whole instead of addressing each criterion for a diagnosis like other popular measures such as the Beck Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996). The measure was developed in this way to maximize its ability to distinguish between anxiety and depression by minimizing items describing overlapping symptoms. Additionally, the DASS depression subscale and an earlier version of the BDI have been shown to be highly correlated (r > .74; Brown et al., 1997; Lovibond & Lovibond, 1995).
Procedure

Study 1. Participants were recruited through announcements posted to an online register of research participation options to fulfill a course requirement. All participants signed an informed consent agreement prior to beginning the experiment. A battery of self-report measures was administered to participants in groups of approximately 30. The questionnaire battery was distributed as a packet with one of ten random orders of the measures. A subset of the battery questionnaires was included in the present study. Due to an error in compiling the packets, one item was left off of both the inhibitory control and activation control subscales. The correlations between the shortened and full scales among the small portion of the sample who completed the full scale (n = 57) were very high for both inhibitory control (r = .97) and activation control (r = .98). Therefore, the unintentionally shortened scales were used for the analyses. Response packets with 25% or more missing items were assumed to represent unreliable responses and were dropped from further analysis. The remaining 1242 participants were included in the analyses. Analyses of the direct effects of reactive and effortful temperament, and relevant interaction terms on depressive symptoms was conducted using the same sample used by Buffington (2009). The present study seeks to extend these findings by testing various models of mediation and moderated mediation of the observed associations between PE, NE, activation control, and depressive symptoms.

Study 2. Participants were recruited through announcements posted to an online register of research participation options to fulfill a course requirement. Informed consent was obtained from all participants. Self-report questionnaires were administered online to participants over the course of a ten-week academic term. A subset of the administered
questionnaires was included in the present study. The PANAS, BIS/BAS, AIM, and ATQ temperament measures were administered during first and second week of the term. The DASS was administered within the week following the initial session to establish baseline depressive symptoms. Due to the close spacing of the measure administration, the PANAS, BIS/BAS, AIM, ATQ, and DASS were grouped as time one measures for the analyses. BADS data was collected at time two, which occurred during the fifth and sixth weeks of the term. Finally, the DASS was administered again at time three during the ninth week of the term. 242 participants completed measures at each of three assessment points and were included in the analyses.
Chapter 3: Study 1 Results

*Exploratory Factor Analysis*

Prior to performing tests of simple mediation and moderated mediation, an exploratory factor analysis (EFA) of the items on the Activation Control subscale and the BADS was carried out to identify potential construct overlap between the scales. The sample was divided to perform an EFA and a subsequent confirmatory factor analysis (CFA) to validate the model specified by the EFA. Several rules of thumb for determining the appropriate sample size of factor analyses have been published based on the ratio of parameters to factors (e.g., Bentler & Chou, 1987; Marsh, Hau, Balla, & Grayson, 1998). However, these rules of thumb are inconsistent, making it difficult to know which rule to apply to a particular study. Additionally, these rules have limited value because they are based on an assumption that the optimal sample size for a given parameter to factor ratio is equivalent in all studies (MacCallum, Widaman, Zhang, & Hong, 1999). Thus, it is important to also consider the communalities of the variables to determine the sample size for a particular study (Fabrigar, Wegener, MacCallum, & Strahan, 1999; MacCallum, Widaman, Preacher, & Hong, 2001; MacCallum et al., 1999). The communality of a parameter is the amount of variance accounted for by the common factor. For samples with high communalities in which the common factors account for a large amount the variance, large samples are unnecessary. Larger sample sizes are
required for a satisfactory solution when communalities are low and much of the parameter variance is accounted for by unique factors (MacCallum et al., 1999).

Based on the inclusion of several parameters in the present study with low communalities (see Table 5) and the large number of factors expected, 600 cases from the original sample were assigned to the EFA in accordance with the recommendations of MacCallum et al. (1999). After listwise deletion of missing data, the EFA sample size was 575. The remaining 642 cases were allocated to the CFA. Cases from the original sample were assigned randomly between the EFA and CFA samples.

The data were analyzed using the Comprehensive Exploratory Factor Analysis (CEFA) program (Browne, Cudeck, Tateneni, & Mels, 2010) using the maximum likelihood method (Fabrigar et al., 1999). An oblique target rotation was employed to allow common factors to correlate (Fabrigar et al., 1999; Preacher & MacCallum, 2003) and to partially specify the factor matrix based on prior knowledge of the parameters (Browne, 2001). The target matrix specified nonzero loadings on separate factors for items on the Activation Control subscale and each of the four BADS subscales. Although a target rotation is based on a specified loading pattern, parameters are free to load on any factor, and misspecifications can be easily detected (Browne, 2001). No variables violated the assumption of normality and all variables were below recommended thresholds for skewness (< 2) and kurtosis (< 7) for structural equation models (West, Finch, & Curran, 1995). Descriptive statistics for each measure are listed in Table 1 and correlations are listed in Table 3.

Multiple criteria were considered to determine the appropriate number of common factors to extract from the data including a scree plot, parallel analysis, and model fit.
(Brown, 2006; Fabrigar et al., 1999; Preacher & MacCallum, 2003). A scree test uses a plot of the eigenvalues of each factor in descending order to determine the number of factors to extract. The plot will drop quickly after the first few factors before settling into a gradual slope. The first factor after the substantial drop should be the last one extracted (Cattell, 1966; Cattell & Jaspers, 1967). Although the scree test is sometimes criticized for being too subjective, it is a useful tool when used in conjunction with other criteria (Fabrigar et al., 1999). The scree plot for the present study (see Figure 4) suggests seven factors should be extracted.

In a parallel analysis, the scree plot is compared to a plot of average eigenvalues from multiple random datasets with the same number of cases and parameters as the sample data. The number of factors to extract is determined by the number of factors that account for more variance than the random data (Brown, 2006; Reise, Waller, & Comrey, 2000; Russell, 2002). A comparison of the present sample with the average eigenvalues of 100 random samples (see Figure 4) suggests five factors should be extracted.

Model fit is also an important consideration for determining the number of factors to extract (Brown, 2006; Fabrigar et al., 1999; Preacher & MacCallum, 2003). Root Mean Square Error of Approximation (RMSEA) is a measure of model error per degree of freedom (Steiger, 1990). The fit of a model is generally improved as parameters are added, but model complexity also increases. RMSEA balances the importance of reducing error with model parsimony by evaluating error in terms of degrees of freedom. Therefore, a model with a smaller error but more parameters may actually have a poorer fit, as indicated by RMSEA, than a simpler model with slightly more error (Browne & Cudeck, 1993; Steiger, 1990). Generally, RMSEA values larger than 0.08 are considered
a poor or marginal fit, values between 0.05 and 0.08 are considered an adequate fit, and values less than 0.05 constitute a good fit (Browne & Cudeck, 1993). In the present sample, the five-factor solution (RMSEA = 0.065, 90% CI [0.061, 0.069]), six-factor solution (RMSEA = 0.059, 90% CI [0.055, 0.064]), and seven-factor solution (RMSEA = 0.055, 90% CI [0.050, 0.060]), all provided an adequate fit to the data.

The structure of the factor loadings is also important for determining appropriateness of the solution. Ideally, each factor should have multiple parameters with strong primary loadings. That is, parameters loading on a particular factor, but not on other factors (Brown, 2006). Multiple parameters loading on different factors is an indication of underfactoring, wherein multiple common factors collapse on the same factor. Structures with factors that contain few primary loadings may indicate underfactoring, which means that too many factors are retained. Additionally, most parameters should load on one primary factor with near-zero loading on the remaining factors (Brown, 2006; Browne, 2001). There are no firm rules regarding what constitutes a high or a low loading and it is highly dependent on the research context, but generally loadings above 0.30 or 0.40 are considered meaningful (Brown, 2006).

The combination of the Activation Control subscale and four subscales of the BADS suggest a five-factor solution, which is consistent with the parallel analysis and model fit comparison as a reasonable number of factors to extract. The five-factor solution (see Tables 5 and 6) included one factor with no strong or single-factor parameter loadings, indicating a poor fit for the data. This structure can indicate overfactoring. However, the four-factor solution showed a large number of parameters loading on multiple factors, which is clear evidence of underfactoring in that solution.
Additionally, the Activation Control subscale items loaded on two primary factors shared by the BADS Activation and Work/School Impairment subscales. This pattern conflicts with prior expectations and may indicate underfactoring (Fabrigar et al., 1999), suggesting a solution with six or more factors may be more appropriate. Further, it is important to test larger models as overfactoring is preferable to underfactoring (Fabrigar et al., 1999; Gorsuch, 1983; Reise et al., 2000). It is also important to note that the factor loadings in this model may also reflect construct overlap between the measures. If so, solutions with six or more factors should continue to show Activation Control subscale items loading on factors shared by BADS subscale items.

Based on the scree test and model fit comparisons, a six-factor solution is appropriate for the data. Consistent with prior expectations, the six-factor solution (see Tables 7 and 8) showed the items on the Activation Control subscale and individual BADS subscales loaded on separate factors. In this model, the BADS Activation subscale loaded on two separate factors. This pattern is consistent with the possibility that the five-factor model was underfactored and contradictory to the possibility that the factor pattern in the previous model was due to construct overlap. The factor structure of this solution is also an improvement over the five-factor solution because there were no factors without strong, single-factor loadings.

The scree test and model fit comparisons also indicate a seven-factor solution may be suitable. In the seven-factor solution (see Tables 9 and 10), the items on the Activation Control subscale and individual BADS subscales again loaded on separate factors. The BADS Activation subscale loaded on two factors and Work/School Impairment subscale included double loadings on an additional factor. Similar to the six-factor solution, this
factor structure provided additional evidence of construct separation between the Activation Control subscale and BADS subscales. However, the more complex model provided only a slight improvement to the statistical model fit over the six-factor solution and the slight differentiation in the BADS Work/School Impairment subscale items is not relevant to the present inquiry. Therefore, the more parsimonious six-factor model was considered the best fit for the present study.

**Confirmatory Factor Analysis**

A CFA was completed with the remaining 642 cases from the original sample to confirm the six-factor solution identified by the EFA. After listwise deletion of missing data, the sample size for the CFA was 623. This satisfies the recommended sample size based on the communalities of the variables as well as the parameter to factor ratio, as previously discussed (MacCallum et al., 2001; MacCallum et al., 1999). The sample also well exceeds requirements for sufficient power (0.80) to detect a close-fit model (Kim, 2005; MacCallum, Browne, & Sugawara, 1996; Preacher & Coffman, 2006).

The data were analyzed using the RAMONA function in SYSTAT 12.0 (Browne, 2007). A six-factor model was specified to reflect the EFA solution. The model contained no double-loading parameters. The six factors were allowed to correlate because the factors were expected to be related. Goodness-of-fit was evaluated using RMSEA. As previous described, there were no departures from normality among the model parameters.

The six-factor model (see Tables 11 and 12) provided an adequate fit for the data (RMSEA = 0.066, 90% CI [0.062, 0.069]). The residual matrix did not indicate areas of poor fit in the solution. Factor loading estimates showed that parameters had moderate to
strong relations to the common factors. The solution also indicated moderate relationships between the Activation Control and BADS Activation factors (0.668) as well as between the Work/School Impairment factor and both the Activation Control (0.701) and BADS Activation (0.754) factors. The high factor correlations were consistent with expectations and were below the 0.80 to 0.85 range that would suggest they should be collapsed into a single factor (Brown, 2006). Further, models with fewer factors were tested and found to provide a less optimal fit to the data, which also indicates that the factors should not be collapsed. It is important to note that error variance has been partialled out of the latent factors, so traditional thresholds for assessing correlations between zero-order variables may be too low. Taken together, the results of the CFA confirm a six-factor solution as the best fit for the model including the Activation Control subscale and the BADS in the present study.

Model Variables

PE was operationalized as PANAS PA, BAS, and AIM PA in separate tests to provide multi-measure support for the results given the number of available measures for PE. A composite scale of these three measures was also created as all three scales are designed to assess PE. Aggregate measures also reduce measurement error (Rushton, Brainerd, & Pressley, 1983). Additionally, the zero-order correlations between the measures were medium to large (r = 0.30 to 0.44; see Table 3). The composite scale was created by averaging the standardized scores from each of the individual PE measures (α = 0.65). The DASS Depression subscale was used as the outcome variable to measure depressive symptoms.
The results of the factor analyses indicated the Activation Control subscale and BADS subscales represent separate factors. Based on these findings, activation control was measured using the Activation Control subscale. The factor analyses also identified multiple factors from the BADS items, which was consistent with expectations as the BADS contains multiple subscales identified by the original authors. As such, the BADS items were used to create a single variable and total BADS score was used to operationalize BA. Individual factors identified by the factor analyses from the BADS items were not considered separately because the measure was designed to assess a broad conceptualization of BA as defined by BA treatment for depression with multiple subscales (Kanter et al., 2007; Kanter et al., 2009). BA was operationalized as total BADS score because this broad conceptualization of BA was the focus of the present study and the results of the factor analyses showed total BADS score could be used to assess BA without excessive construct overlap between the Activation Control subscale and any of the BADS subscales.

In addition to the variables in the simple mediation and moderated mediation models, covariates were included in each analysis to account for effects of variables commonly addressed in the literature on reactive and effortful temperament and depression. Corresponding measures of PE and NE, i.e., PANAS, BIS/BIS, AIM, and Composite scales, were included in each model. The Composite NE scale was created by averaging the standardized scores of the PANAS NA, BIS, and AIM NR scales ($\alpha = 0.65$). All three facets of EC were included in each model. Interaction terms between reactive temperament and corresponding facets of EC including NE x Attentional Control
and PE x Activation Control were also included (Buffington, 2009; Vasey et al., 2009). Finally, NE x PE was included in each model.

All variables were normally distributed with the exception of DASS Depression (skew = 1.69), although its skew did not represent a large departure from normality. Although a skew value below one is recommended for a linear regression analyses, slight to moderate violations of normality do not bias results for large samples (Cohen, Cohen, West, & Aiken, 2003). The skew for all other variables was less than one. The temperament and BA variables were standardized so that zero would represent the average level of each variable. Transforming the variables so that zero is a meaningful value eases interpretation as regression coefficients can be interpreted as the change in the dependent variable for a one-unit increase in an independent variable when all the other variables in the model are at their averages (Whisman & McClelland, 2005). Standardization also simplifies interpretation of coefficients for variables using different scales. Missing data were removed by listwise deletion. Descriptive statistics for all variables are listed in Table 1. Correlations are listed in Table 3.

Simple Mediation

To test the hypotheses that BA mediates the associations between PE, NE, and activation control with depressive symptoms, a statistical test of the indirect effect was completed using an SPSS macro developed by Preacher and Hayes (2008). A bootstrapping procedure was used to create 5,000 resampled data sets to estimate indirect effects and 95% confidence intervals (Hayes, 2009; MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2008; Shrout & Bolger, 2002). Mediation was determined by a significance test of the indirect effect. In addition to the indirect path (ab), other path
coefficients are provided for context in Appendix B for each mediation model. The paths from the independent variable to the mediator ($a$), from the mediator to the dependent variable ($b$), the total effect of the independent variable on the dependent variable not accounting for the mediator ($c$), and the direct effect of the independent variable on the dependent variable after accounting for the mediator ($c'$) are included. Although the traditional casual steps approach for testing mediation effects (Baron & Kenny, 1986) includes requirements for significant $a$, $b$, and $c$ paths, this approach has low statistical power to detect effects because an indirect effect may be significant even when one of these paths is nonsignificant. More recently available techniques for directly testing the significance of the indirect effect such as bootstrapping are therefore preferable (Hayes, 2009; MacKinnon, Fairchild, & Fritz, 2007). Effect sizes were calculated using kappa-squared ($\kappa^2$), a measure of the ratio of the indirect effect of the sample to the maximum possible indirect effect (Preacher & Kelley, 2011). General guidelines for determining small, medium, and large effect sizes for $\kappa^2$ are 0.01, 0.09, and 0.25, respectively (Preacher & Kelley, 2011).

Consistent with expectations, the indirect path from PE to depressive symptoms through BA was significant for each of the three models that were tested. The indirect effect of PANAS PA on DASS Depression via the BADS was $-0.84$, 95% CI $[-1.09, -0.63]$, $\kappa^2 = 0.28$ (see Figure 5). PANAS NA, Attentional Control, Inhibitory Control, Activation Control, PANAS NA x Attentional Control, PANAS PA x Activation Control, and PANAS NA x PANAS PA were included in the analysis as covariates. The indirect effect for BAS was $-0.60$, 95% CI $[-0.89, -0.30]$, $\kappa^2 = 0.11$ (see Figure 6). BIS, Attentional Control, Inhibitory Control, Activation Control, BIS x Attentional Control,
BAS x Activation Control, and BIS x BAS were included as covariates. For AIM PA, the indirect effect was -0.86, 95% CI [-1.21, -0.55], \( \kappa^2 = 0.10 \) (see Figure 7). AIM NR, Attentional Control, Inhibitory Control, Activation Control, AIM NR x Attentional Control, AIM PA x Activation Control, and AIM NR x AIM PA were included as covariates.

Based on the similar performance of the different measures of PE (i.e., PANAS PA, BAS, and AIM PA) in the mediation tests, a similar test was carried out using the Composite PE scale. The indirect effect of Composite PE on DASS Depression via the BADS was -1.10, 95% CI [-1.41, -0.85], \( \kappa^2 = 0.22 \) (see Figure 8). Composite NE, Attentional Control, Inhibitory Control, Activation Control, Composite NE x Attentional Control, Composite PE x Activation Control, and Composite NE x Composite PE were included as covariates.

The indirect effect from Composite NE to the BADS to DASS Depression (1.26, 95% CI [1.03, 1.52], \( \kappa^2 = 0.24 \); see Figure 9) was also significant. Composite PE, Attentional Control, Inhibitory Control, Activation Control, Composite NE x Attentional Control, Composite PE x Activation Control, and Composite NE x Composite PE were included as covariates.

A statistical test of the indirect path from activation control to depressive symptoms mediated by BA was also completed. The indirect effect of Activation Control on DASS Depression through the BADS was -1.29, 95% CI [-1.59, -1.04], \( \kappa^2 = 0.32 \) (see Figure 10). Composite NE, Composite PE, Attentional Control, Inhibitory Control, Composite NE x Attentional Control, Composite PE x Activation Control, and Composite NE x Composite PE were included as covariates.
Moderated Mediation

The hypothesized model of moderated mediation was tested in which activation control moderates the indirect effect of low PE on depressive symptoms through BA. Specifically, activation control was expected to moderate the link between PE and BA (see Figure 3). The model was tested using an SPSS macro (Preacher, Rucker, & Hayes, 2007) designed to test models of moderated mediation. Composite PE was used as the independent variable as it yielded significant results in the test of simple mediation. Using the composite scale also reduces type I error by requiring fewer tests and enhances statistical power by reducing measurement error (Rushton et al., 1983).

Contrary to expectations, the hypothesized model of moderated mediation was not supported. In the proposed model, the indirect effect of PE on depressive symptoms through BA was expected to be moderated by activation control such that the path would be nonsignificant when PE is low but activation control is high. That is, low PE would not predict low BADS or, in turn, high depressive symptoms when activation control was high. Low PE was expected to predict low activation and high symptoms only in cases where activation control was also low. To establish the proposed model, the association between PE and BA must be moderated by activation control (Preacher et al., 2007). Consistent with the results of the test of simple mediation, both Composite PE and Activation Control accounted for significant variability in the BADS, but the Composite PE x Activation Control interaction term was nonsignificant (see Table 13). Composite NE, Attentional Control, Inhibitory Control, Composite NE x Attentional Control, and Composite NE x Composite PE were included as covariates.
Exploration of Nonsignificant Results for Moderated Mediation

Low PE was expected to be associated with low BA except when activation control was high. Additionally, at high levels of PE, activation control was expected to have less of an effect on BA because an individual would not need to compensate for low PE. Instead, the results showed that high PE was associated with high BA and low PE was associated with low BA independent of the level of activation control. After the null results of the tests of moderated mediation for the composite PE scale, separate exploratory follow-up tests were conducted using PANAS PA, BAS, and AIM PA for PE to probe for possible differences between the models using different measures of PE. PE and activation control were consistently associated with BA, but none of the models yielded significant results for an interaction between PE and activation control to support the proposed model of moderated mediation.

The results of the test of moderated mediation with PANAS PA (see Table 14) showed that both PANAS PA and Activation Control accounted for significant variability in the BADS, but the product term between the variables was nonsignificant. The test included PANAS NA, Attentional Control, Inhibitory Control, PANAS NA x Attentional Control, and PANAS NA x PANAS PA as covariates. Similar results were found in a test of a model including BAS as the independent variable (see Table 15). BIS, Attentional Control, Inhibitory Control, BIS x Attentional Control, and BIS x BAS were included as covariates. The test of the model using AIM PA also showed that AIM PA and Activation Control accounted for significant variability in the BADS, but the AIM PA x Activation Control interaction was nonsignificant (see Table 16). AIM NR, Attentional Control,
Inhibitory Control, AIM NR x Attentional Control, and AIM NR x AIM PA were included in the model as covariates.

**Differences Between PE Measures**

The link between PANAS PA and Composite PE with the BADS was similar to the link between Activation Control and the BADS. However, the same was not true for BAS or AIM PA. Instead, the association between BAS and AIM PA with the BADS was weaker than the association between Activation Control and the BADS. These differing patterns may suggest important differences between these measures of PE.

The link between PANAS PA and the BADS was similar in strength to the association between Activation Control and the BADS. The regression coefficient for PANAS PA (B = 0.24, SE = 0.02, \( p < .001 \); see Table 14) predicting the BADS was similar to the coefficient for Activation Control (B = 0.25, SE = 0.03, \( p < .001 \)) predicting the BADS. Consistent with these results, the indirect path from PANAS PA to the BADS to DASS Depression (-0.84, 95% CI [-1.09, -0.63], \( \kappa^2 = 0.28 \); see Figure 5) had a similar effect size as the indirect path from Activation Control to the BADS to DASS Depression (-1.29, 95% CI [-1.59, -1.04], \( \kappa^2 = 0.32 \); see Figure 10).

The link between Composite PE and the BADS was also similar in strength to the association between Activation Control and the BADS. The regression coefficient predicting the BADS for Composite PE (B = 0.27, SE = 0.03, \( p < .001 \); see Table 13) was similar to the regression coefficient for Activation Control (B = 0.31, SE = 0.03, \( p < .001 \)). Consistent with these results, the indirect path from Composite PE to the BADS to DASS Depression (-1.10, 95% CI [-1.41, -0.85], \( \kappa^2 = 0.22 \); see Figure 8) had a similar
effect size to the indirect path from Activation Control to the BADS to DASS Depression (-1.29, 95% CI [-1.59, -1.04], $\kappa^2 = 0.32$; see Figure 10).

In contrast to PANAS PA and Composite PE, the link between BAS and the BADS was weaker than the association between Activation Control and the BADS. BAS ($B = 0.12, \ SE = 0.03, p < .001$; see Table 15) was less strongly related to the BADS than was Activation Control ($B = 0.35, \ SE = 0.03, p < .001$) in the regression analysis. Further, the indirect path from BAS to the BADS to DASS Depression (-0.60, 95% CI [-0.89, -0.30], $\kappa^2 = 0.11$; see Figure 6) had a smaller effect size than the indirect path from Activation Control to the BADS to DASS Depression (-1.29, 95% CI [-1.59, -1.04], $\kappa^2 = 0.32$; see Figure 10).

The indirect path from Activation Control to the BADS to DASS Depression was also tested with the BADS Activation subscale removed to provide a more stringent test of the indirect path due to the similarities of the items on the Activation Control and BADS Activation subscale measures. Indeed, this path (-0.98, 95% CI [-1.26, -0.73], $\kappa^2 = 0.27$; see Figure 11) was stronger than the comparable indirect path from BAS to the full BADS to DASS Depression.

AIM PA ($B = 0.18, \ SE = 0.03, p < .001$; see Table 16) was also less strongly related to the BADS than was Activation Control ($B = 0.36, \ SE = 0.03, p < .001$) in the regression analysis. Additionally, the indirect path from AIM PA to the BADS to DASS Depression (-0.86, 95% CI [-1.21, -0.55], $\kappa^2 = 0.10$; see Figure 7) had a smaller effect size than the indirect path from Activation Control to the BADS to DASS Depression (-1.29, 95% CI [-1.59, -1.04], $\kappa^2 = 0.32$; see Figure 10).
Tests of NE x PE and Three-Way Interactions

The NE x PE interaction was included as a covariate in each of the tested models. A trend effect predicting BADS was found for PANAS NA x PANAS PA (B = -0.04, SE = 0.02, *p* = .058; see Table 14) for the BADS. The graph of the interaction shows that the BADS was highest at low PANAS NA and high PANAS PA and that this association was not merely additive (see Figure 12). That is, the association between low PANAS NA and the BADS was stronger at high PANAS PA than would be expected by an additive effect alone. Neither the BIS x BAS nor the AIM NR x AIM PA interactions were significant for BA.

Exploratory tests of an NE x PE x Activation Control interaction for BA were carried out in a subsequent step in each model of moderated mediation. The appropriate NE x Activation Control term was also added in the same step to include all possible two-way interaction combinations between variables in the three-way interaction term (Aiken & West, 1991). The three-way interaction was not supported in any of the tested models, indicating that NE did not moderate the association between the PE x Activation Control interaction and the BADS.

The Composite NE x Composite PE x Activation Control interaction was not significant (B = -0.01, SE = 0.02, *p* = .614; see Table 13) when added to the model of moderated mediation in a subsequent step and therefore did not support a moderating effect of Composite NE on the original model of moderated mediation. Composite NE x Activation Control was also added as a covariate in the same step. In the PANAS PA model, the PANAS NA x PANAS PA x Activation Control interaction was not significant (B = 0.01, SE = 0.02, *p* = .482; see Table 14) when added to the model in a
subsequent step along with PANAS NE x Activation Control. The BIS x BAS x Activation Control interaction was also nonsignificant (B = -0.02, SE = 0.02, p = .292; see Table 15). BIS x Activation Control was added as a covariate in the same step. The AIM NR x AIM PA x Activation Control interaction was also nonsignificant (B = 0.03, SE = 0.02, p = .225; see Table 16). AIM NR x Activation Control was also added as a covariate in the same step.

Summary

An EFA and CFA of the items from the Activation Control and BADS measures supported a six-factor model in which items from the two measures loaded on different factors. The factor analyses suggested the measures of activation control and BA distinguish between the two constructs, although the interfactor correlations showed close associations between Activation Control, the BADS Activation subscale, and the BADS Work/School Impairment subscale. The results also showed support for the hypothesized mediation models in a large cross-sectional sample. BA mediated links between depressive symptoms and PE, NE, and activation control with medium to large effect sizes. However, the proposed model of moderated mediation in which the indirect path from PE to BA to depressive symptoms is moderated by activation control was not supported. Further investigation of the models of moderated mediation showed different patterns for PANAS PA and Composite PE than for the BAS and AIM PA models. Evidence for a trend PANAS NA x PANAS PA interaction predicting BA was found, but similar interactions for the BIS/BAS and AIM models were nonsignificant. Exploratory tests of an NE x PE x Activation Control were null.
To confirm the pattern of results Study 1 over time, the mediation and moderated mediation analyses were replicated in prospective sample in Study 2. The prospective design of Study 2 allowed for longitudinal tests of the predictive power of the variables and mediation effects.
Chapter 4: Study 2 Results

Model Variables

All variables and covariates used in Study 1 were used in Study 2. Additionally, baseline depressive symptoms were included as a covariate to determine the effects of the model variables on change in symptoms over time. Attempts to maximize participants at the high and low levels of NE, PE, and EC did not negatively impact the normality or skew of the distribution. All variables were normally distributed with the exception of T1 DASS Depression and T3 DASS Depression. However, the skew for T1 DASS Depression (skew = 1.25) and T3 DASS Depression (skew = 1.80) did not represent large departures from normality and would not be expected to bias the sample (Cohen et al., 2003). The skew for all other variables was less than one. All temperament and BA variables were standardized as in Study 1. The reliability of the Composite PE scale was $\alpha = 0.74$ and the reliability of the Composite NE scale was $\alpha = 0.64$. Descriptive statistics for all other variables are listed in Table 2. Correlations are listed in Table 4.

Due to the smaller relative size of the sample in Study 2, dropping cases due to listwise deletion had a larger impact on sample size. To mitigate the effects of lost data, all analyses were initially completed using mean imputation. Means of available items were used for missing values for scales with an adequate number of non-missing responses and a reliability of $\alpha \geq 0.70$. The standard for a sufficient number of responses ranged from 50-80% based on the reliability of each measure (Graham, 2009; Schafer &
Graham, 2002). However, the results of the analyses were not significantly altered whether mean imputation or simple listwise deletion was used. Additionally, although mean imputation can reduce the number of cases excluded and increase sample size of the analysis, it can potentially bias results because imputing a fixed scale mean for missing responses artificially reduces the natural variability associated with actual responses (Graham, 2009; Schafer & Graham, 2002). Due to the limitations of mean imputation and similarity of the results, analyses using listwise deletion are reported.

**Simple Mediation**

Although the data from Study 1 supported the hypothesized indirect effects, the prospective data offers an improved way to test mediation by taking into account the change in variables over time, which is consistent with the implication of a meditational relationship. Controlling for baseline levels of the dependent variable also offers a more stringent test as it assesses the indirect effect of the independent variable on change in the dependent variable instead of the level of the dependent variable at a particular point in time. Additionally, baseline levels of the dependent variable are likely to include effects of the independent variable as the basis of the test in an expectation that the independent and dependent variables are associated.

T1 Composite PE was used to test the indirect effect of T1 PE on T3 DASS Depression through T2 BADS based on significant results from Study 1. Additionally, using a composite measure increases statistical power by reducing error measurement (Rushton et al., 1983) and also reduces type I error by requiring fewer tests. Consistent with the significant results from Study 1, the indirect effect of T1 Composite PE on T3 DASS Depression via T2 BADS was significant (-0.52, 95% CI [-1.42, -0.17], κ² = 0.16;
see Figure 13), even when controlling for T1 DASS Depression. T1 Composite NE, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 Composite NE x T1 Attentional Control, T1 Composite PE x T1 Activation Control, and T1 Composite NE x T1 Composite PE were also included as covariates.

The indirect effect from T1 Composite NE to T2 BADS to T3 DASS Depression was nonsignificant at the \( p < .05 \) level (0.33, 95% CI [-0.04, 0.98], \( \kappa^2 = 0.22 \); see Figure 14) when controlling for T1 DASS Depression. When excluding baseline depressive symptoms, the indirect effect was significant as expected (0.90, 95% CI [0.29, 1.83], \( \kappa^2 = 0.22 \); see Figure 15). T1 Composite NE, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 Composite NE x T1 Attentional Control, T1 Composite PE x T1 Activation Control, and T1 Composite NE x T1 Composite PE were included as covariates in both tests.

The indirect effect of T1 Activation Control on T3 DASS Depression via T2 BADS (-0.51, 95% CI [-1.15, -0.04], \( \kappa^2 = 0.22 \); see Figure 16) was also consistent with the results of Study 1. T1 measures of Composite NE, Composite PE, Attentional Control, Inhibitory Control, Composite NE x Attentional Control, Composite PE x Activation Control, Composite NE x Composite PE, and DASS Depression were included as covariates.

**Moderated Mediation**

As expected, based on the nonsignificant results for the hypothesized model of moderated mediation in Study 1, moderated mediation was not supported. The analysis of T1 Composite PE did not yield a significant T1 Composite PE x T1 Activation Control interaction and so was not consistent with moderated mediation (see Table 17). T1
Composite NE, T1 Attentional Control, T1 Inhibitory Control, T1 Composite NE x T1 Attentional Control, T1 Composite NE x T1 Composite PE, and T1 DASS Depression were included as covariates.

Exploration of Nonsignificant Results for Moderated Mediation

After the null results of the tests of moderated mediation for Composite PE, indirect effects of T1 PE on T3 depressive symptoms through T2 BA were tested separately for T1 PANAS PA, T1 BAS, and T1 AIM PA as the basis for follow-up individual tests of moderated mediation. As expected, the indirect effect of the models including T1 PANAS PA and T1 BAS were significant. The indirect effect of T1 PANAS PA to T3 DASS Depression via T2 BADS was -0.28, 95% CI [-0.82, -0.01], $\kappa^2 = 0.21$ (see Figure 17). T1 measures for PANAS NA, Attentional Control, Inhibitory Control, Activation Control, PANAS NA x Attentional Control, PANAS PA x Activation Control, PANAS NA x PANAS PA, and DASS Depression were included as covariates. The indirect effect for T1 BAS was -0.42, 95% CI [-1.08, -0.10], $\kappa^2 = 0.07$ (see Figure 18). T1 BIS, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 BIS x T1 Attentional Control, T1 BAS x T1 Activation Control, T1 BIS x T1 BAS, and T1 DASS Depression were included as covariates. Conversely, the indirect effect of T1 AIM PA on T3 DASS Depression via T2 BADS was nonsignificant (-0.17, 95% CI [-0.79, 0.14], $\kappa^2 = 0.10$; see Figure 19) when T1 DASS Depression was included as a covariate. The indirect effect of T1 AIM PA was tested without including T1 DASS Depression as a covariate in a separate test because baseline depressive symptoms may include effects of T1 AIM PA due to the association between the two variables. Indeed, the indirect effect was significant when T1 DASS Depression was excluded (-0.70, 95% CI [-1.83, -0.08], $\kappa^2 =
0.10; see Figure 20). Both AIM PA analyses included T1 AIM NR, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 AIM NR x T1 Attentional Control, T1 AIM PA x T1 Activation Control, and T1 AIM NR x T1 AIM PA as covariates.

Analyses of the proposed model did not support moderated mediation. Tests of the model including PANAS PA, BAS, and AIM PA as the independent variable in separate analyses were carried out using an identical method to the corresponding tests described in Study 1 with the addition of baseline depressive symptoms as a covariate. The analyses did not yield a significant PE x activation control interaction to support the proposed model of moderated mediation. The regression analyses for each model are summarized in Tables 18, 19, and 20.

Differences Between PE Measures

The results supported an association between T1 PANAS PA and T1 Composite PE with T2 BADS similar in magnitude to the association between T1 Activation Control and T2 BADS consistent with the findings in Study 1. Also, as in Study 1, this pattern did not occur in the BAS or AIM PA models. The link between T1 BAS and T1 AIM PA with T2 BADS was weaker than the link between T1 Activation Control and T2 BADS.

T1 PANAS PA (B = 0.12, SE = 0.07, \( p = .068 \); see Table 18) was less strongly related to T2 BADS than was T1 Activation Control (B = 0.20, SE = 0.07, \( p = .006 \)) when controlling for baseline depressive symptoms. However, the regression coefficients for predicting T2 BADS for T1 PANAS PA (B = 0.24, SE = 0.07, \( p < .001 \)) and T1 Activation Control (B = 0.19, SE = 0.07, \( p = .009 \)) were more similar to the results from Study 1 when T1 DASS Depression was excluded as a covariate to create a more even
comparison to the cross-sectional analysis. The relative differences between the regression coefficients of T1 BAS, T1 AIM PA, and T1 Activation Control did not change when excluding baseline depression in those models. The indirect path from T1 PANAS PA to T2 BADS to T3 DASS Depression (-0.28, 95% CI [-0.82, -0.01], \( \kappa^2 = 0.21 \); see Figure 17) had a similar effect size to the indirect path from T1 Activation Control to T2 BADS to T3 DASS Depression (-0.51, 95% CI [-1.15, -0.04], \( \kappa^2 = 0.22 \); see Figure 16).

The link between T1 Composite PE and T2 BADS was also similar in magnitude to the association between T1 Activation Control and T2 BADS. The regression coefficient for T1 Composite PE (B = 0.20, SE = 0.08, \( p = .008 \); see Table 17) predicting T2 BADS was similar in magnitude to the coefficient for T1 Activation Control (B = 0.18, SE = 0.08, \( p = .023 \)) predicting T2 BADS. The indirect path from T1 Composite PE to T2 BADS to T3 DASS Depression (-0.51, 95% CI [-1.15, -0.04], \( \kappa^2 = 0.22 \); see Figure 13) had the same effect size as the indirect path from T1 Activation Control to T2 BADS to T3 DASS Depression (-0.51, 95% CI [-1.15, -0.04], \( \kappa^2 = 0.22 \); see Figure 16).

Unlike the PANAS PA and Composite PE models, the association between T1 BAS and T2 BADS was weaker than the association between T1 Activation Control and T2 BADS. T1 BAS (B = 0.15, SE = 0.07, \( p = .035 \); see Table 19) was less strongly related to T2 BADS than was T1 Activation Control (B = 0.21, SE = 0.07, \( p = .006 \)) in the regression analysis. Additionally, the indirect path from T1 BAS to T2 BADS to T3 DASS Depression (-0.42, 95% CI [-1.08, -0.10], \( \kappa^2 = 0.07 \); see Figure 18) had a smaller effect size that the indirect path from T1 Activation Control to T2 BADS to T3 DASS Depression (-0.51, 95% CI [-1.15, -0.04], \( \kappa^2 = 0.22 \); see Figure 16).
The indirect path from T1 Activation Control to T2 BADS to T3 DASS Depression with the T2 BADS Activation subscale removed was tested to provide a more stringent test by removing a component of the BADS closely related to Activation Control. The indirect effect (-0.38, 95% CI [-0.97, 0.09], $\kappa^2 = 0.19$; see Figure 21) was also stronger than the comparable indirect path from T1 BAS to the full T2 BADS to T3 DASS Depression. Although the indirect path including the modified BADS was not significant at the $p \leq .05$ level as it was in Study 1, the larger effect size indicates these results are consistent with a stronger association between Activation Control and the BADS.

As in Study 1, a weaker link was observed between T1 AIM PA and T2 BADS than between T1 Activation Control and T2 BADS. T1 AIM PA ($B = 0.11, SE = 0.07, p = .112$; see Table 20) was less strongly related to T2 BADS than was T1 Activation Control ($B = 0.20, SE = 0.08, p = .008$) in the regression analysis. Further, the nonsignificant indirect path from T1 AIM PA to T2 BADS to T3 DASS Depression (-0.17, 95% CI [-0.79, .014], $\kappa^2 = 0.10$; see Figure 19) had a smaller effect size than the indirect path from T1 Activation Control to T2 BADS to T3 DASS Depression (-0.51, 95% CI [-1.15, -0.04], $\kappa^2 = 0.22$; see Figure 16).

Tests of $NE \times PE$

Similar to Study 1, a trend effect predicting T2 BADS was found for T1 PANAS NA x T1 PANAS PA ($B = 0.10, SE = 0.05, p = .061$; see Table 18). High T1 PANAS NA was associated with below average T2 BADS scores as expected, but only when T1 PANAS PA was low. At high T1 PANAS PA, high T1 PANAS NA was associated with above average T2 BADS scores (see Figure 22). This pattern suggests a protective effect
of PANAS PA against low BA associated with high PANAS NA. Neither the T1 BIS x T1 BAS nor the T1 AIM NR x T1 AIM PA interactions was significant for T2 BADS.

Based on the null results for the NE x PE x Activation Control interaction for BADS in Study 1, the interaction was not tested in Study 2 due to its smaller relative sample size.

Summary

Study 2 generally confirmed the results of the mediation analyses from Study 1 in a prospective sample. BA mediated links between depressive symptoms and PE and activation control even when controlling for baseline symptoms. BA also mediated the indirect path from NE to depressive symptoms, although the path was nonsignificant when controlling for baseline symptoms. However, based the medium to large effect size and near exclusion of zero in the 95% confidence interval, this path would likely be significant in a larger sample. As in Study 1, the proposed model of moderated mediation in which the indirect path from PE to BA to depressive symptoms is moderated by activation control was not supported. Similar differences between the measures of PE observed in Study 1 were also supported. A trend T1 PANAS NA x T1 PANAS PA interaction predicting BA was found, but corresponding interaction terms in the BIS/BAS and AIM models were nonsignificant, mirroring the results from Study 1.
Chapter 5: General Discussion

The present study sought to clarify the relationship between reactive temperament (i.e., NE and PE), activation control, BA, and depressive symptoms. First, an exploratory factor analysis (EFA) and a confirmatory factor analyses (CFA) were carried out to determine whether the Activation Control subscale and BADS measures were able to distinguish between activation control and BA, which are closely related constructs. In the optimal-fit solution for the EFA, all of the Activation Control items loaded on a common factor separate from the BADS subscale items. The BADS Activation subscale loaded on two common factors and the remaining subscales loaded on different common factors. The CFA confirmed that this six-factor solution provided an adequate fit to the data using participants not included in the EFA. These results suggest that measures for activation control and BA distinguish between these two constructs as expected. However, the interfactor correlations reflected especially strong associations between the Activation Control, BADS Activation, and BADS Work/School Impairment factors. Taken together, the factor analyses suggest the measures of activation control and BA reflect distinct, but strongly related constructs.

The results strongly supported an indirect path from PE to depressive symptoms mediated by BA. The mediation model was consistently supported cross-sectionally in tests using PANAS PA, BAS, and AIM PA as well as a composite score of these three measures with medium to large effect sizes. The indirect path was also found
prospectively for the composite PE measure, PANAS PA, and BAS even when controlling for baseline depressive symptoms. The indirect path from AIM PA to BA to depressive symptoms was also significant when excluding baseline depression from the model. The prospective results offer strong support of the proposed mediation as it supports theoretical predictions that individual differences in PE can affect BA and ultimately depressive symptoms over time. Additionally, the results offer further evidence of the distinction between activation control and BA because activation control was included as a covariate in each model. That is, the indirect paths were significant even when accounting for common variance accounted for by both constructs.

The indirect path from NE to depressive symptoms was also mediated by BA as expected. The results are consistent with theoretical expectations that avoidance is one pathway by which high NE can lead to depressive symptoms. This effect had a medium to large effect size both cross-sectionally and prospectively, although in the latter case it was not significant when controlling for baseline symptoms of depression. However, based on the effect size and 95% confidence interval, this effect would likely be significant in a larger sample even when controlling for baseline symptoms. It is important to note that the indirect path involving NE was independent of the paths involving PE and activation control.

Consistent with theoretical expectations of a protective effect of activation control against depressive symptoms in which individuals high in activation control are likely to engage in approach behaviors, the results showed that BA also mediated the link between activation control and depressive symptoms. This effect was supported both cross-sectionally and prospectively even when controlling for baseline symptoms. Again, it is
important to note that this indirect path was independent of the paths involving PE and NE.

The meditation paths supported by the results between BA and PE, NE, and activation control add to temperament/personality models of depression and provide important avenues of exploration for potential preventive interventions. Previously observed associations between low PE, high NE, and low activation control with depressive symptoms suggest potential vulnerabilities to depression. The results of the present study expand temperament/personality models of depression by providing one potential pathway for these vulnerabilities. Identifying pathways between temperament/personality vulnerability markers and psychological symptoms suggests possible targets for preventive care. Improving BA among populations with vulnerabilities to depression may prevent or reduce symptoms. Further, BA could be used as an indicator of improvement in at-risk populations even before the actual onset of depressive symptoms.

The results are also consistent with conceptualizations of reduced BA as a maintenance factor for depression. One of the targets of BA treatments for depression is to reduce avoidance because it can lead to negative life consequences that exacerbate depression and further increase avoidance motivation in a continuous downward cycle (Martell et al., 2001; Martell et al., 2010). Similarly, increasing depression may lead to reduced approach motivation and increased avoidance associated with low PE and high NE, which, in turn, could lead to reduced BA and increased depression in a continuous cycle. Conversely, increased BA leading to improvements in depression may increase
approach motivation and reduce avoidance leading to further increases in BA in a continuous cycle.

The proposed model of moderated mediation based on evidence of a protective effect of activation control in which individuals low in PE but high activation control experience fewer or less severe depressive symptoms than are predicted by low PE alone was not supported. In the proposed model, the protective effect of activation control was expected to alter the indirect path from PE to BA to depressive symptoms by moderating the link between PE and BA (see Figure 2). The indirect path was expected to be significant for low levels of activation control, but not at high levels of activation control. That is, low PE was not expected to be associated with low BA or, in turn, depressive symptoms when activation control was high. However, statistical tests of this model did not support the expected relationship. Specifically, the expected interaction between PE and activation control for predicting BA was not found. Instead, the results showed that PE and activation control had an additive effect for predicting BA.

One possible explanation for these results is that the strong similarity between activation control and BA leaves little room to detect an interactive effect. Although the factor analyses demonstrated that the measures used for activation control and BA distinguish between the two constructs and significant results were found for mediation models with BA as the mediator even when controlling for activation control, they remain closely related. Interactions are often small and detecting them can be difficult (McClelland & Judd, 1993). Finding interactions between variables with limited variability can be especially complex. Therefore, despite evidence that activation control
and BA are distinct, their similarity may make an interaction term between them difficult to detect.

Improved measures that better assess the distinctions between activation control and BA may also provide a more accurate test of the expected pattern of moderated mediation by increasing variability between these constructs. Items addressing activation control should focus on an individual’s assessment of their general capacity to perform an action despite a lack of motivation to act, whereas items addressing specific behaviors over a discrete period of time best assess BA. Items from the Activation Control subscale such as “I can keep performing a task even when I would rather not do it” and “I can make myself work at a difficult task even when I don’t feel like trying” gauge a general temperamental capacity to engage in nonspecific behaviors without reactive motivation. However, items such as “I am often late for appointments (reverse scored)” and “I often make plans that I do not follow through with (reverse scored)” refer to more specific behaviors or do not address overcoming a reactive impulse. Indeed, the factor analyses showed that these items had the poorest loadings on the factor made up of items from the Activation Control subscale. New items focused on a general proclivity to engage in productive behaviors without reactive motivation to act over a longer period may better assess the conceptualization of activation control used in the proposed models of the present study. Improved scales could reduce measurement error and sharpen the distinction between measures of activation control as temperamental capacity and BA as specific behavioral outcomes of reactive and effortful temperament.
Measures of PE

Further review of the results for each individual measure of PE showed that the link between PANAS PA and BA was similar in magnitude to the association between activation control and BA. However, the associations between BAS and AIM PA with BA were not as strong as the link between activation control and BA. PANAS PA and activation control had similar regression coefficients when regressed on BA in Study 1 and the same pattern emerged in Study 2 when baseline depression was removed as a covariate. Consistent with these findings, the indirect effect from PANAS PA to BA to depressive symptoms was similar to the strength of the indirect path from activation control to BA to DASS Depression.

The similar strength of the links between PANAS PA and BA and between activation control and BA may reflect a component of goal-directed behavior common to all three constructs. All three scales include items addressing goal-setting, accomplishment, and perseverance in difficult situations. For example, PANAS PA includes other items assessing the frequency an individual feels “interested,” “determined,” and “inspired,” all of which suggest focused behavior. Activation Control items include, “I can make myself work on a difficult task even when I don’t feel like trying” and “I can keep performing a task even when I would rather not do it.” Similarly, representative items from the BADS include, “I did something that was hard to do but it was worth it” and “I was active, but did not accomplish any of my goals for the day (reverse scored).” Indeed, the BADS Activation subscale was explicitly designed to measure behaviors that facilitate goal achievement (Kanter et al., 2007; Kanter et al., 2009).
The BAS and AIM PA models demonstrated a different pattern. Unlike PANAS PA, the association between BAS and AIM PA with BA was weaker than the link between activation control and BA. The regression coefficients for BAS predicting the BA were smaller than the coefficients for activation control predicting the BA. Additionally, the indirect path from BAS to BA to depressive symptoms was weaker than the indirect path from activation control to BA to symptoms. It does not appear that the stronger association between activation control and BA can be accounted for solely by item overlap. Even after omitting the BADS Activation subscale, the subscale with the most similar items to the Activation Control subscale, the indirect path from activation control to the modified BADS to depressive symptoms was still stronger than the indirect path from BAS to depressive symptoms through the full BADS.

This weaker association between BAS and BA may be due to a lack of coverage of goal-directed behavior on the BAS measure. Individuals reporting high BAS are strongly motivated by reward and high in sensation-seeking and fun-seeking, but are not necessarily also motivated by longer-term consequences. That is, their behavior may be particularly impulsive or unfocused. Items such as, “When I get something I want, I feel excited and energized” or “I crave excitement and new sensations” clearly address general activation, but may not lead to the type of constructive activity addressed by the BADS. High BAS alone may not always be a strong predictor of the type of goal-directed, focused behavior measured by the BADS.

In fact, high levels of BAS unfocused toward productive goals may be maladaptive. Hundt et al. (2007) found that at high levels of BIS, high BAS was associated with higher reports of mixed symptoms of anxiety and depression than low
levels of BAS. The authors suggested this unintuitive pattern may indicate distress caused by approach-avoidance conflicts likely experienced by individuals high in both BIS and BAS. Maladaptive impulsivity or low frustration tolerance associated with high BAS might also lead to negative outcomes that are especially distressing in cases of high BIS (Harbaugh et al., 2011). Additionally, the BAS-dysregulation model of bipolar disorders suggests individuals with hypersensitive BAS can be easily over stimulated, which can lead to the onset of hypomanic or manic episodes (Alloy & Abramson, 2010; Urošević, Abramson, Harmon-Jones, & Alloy, 2008).

AIM PA behaved similarly to BAS in the analyses, reflecting a similar difference between unfocused, generalized approach and goal-oriented behavior. Items such as “I get overly enthusiastic” or “When things are going good I feel ‘on top of the world’” reflect this difference. Similar to the BAS, the link between AIM PA and BA was weaker than the link between activation control and BA in the regression analyses. Further, the indirect path from AIM PA to BA to depressive symptoms was weaker than the indirect path from activation control to BA to depressive symptoms.

Depue and colleagues conceptualize sensation-seeking and risk-taking behavior, labeled affective impulsivity, as a result of the combined effects of extroversion and constraint (Depue & Collins, 1999; Depue & Lenzenweger, 2006). The authors proposed that neither extroversion, characterized by general enthusiasm and energy, nor constraint, a trait similar to EC characterized by control of affective systems, can fully account for approach behavior. Although BAS, AIM PA, and PANAS PA are all purported representations of PE, the individual measures may reflect a different balance between extroversion and constraint as described by Depue and colleagues.
Not surprisingly, the composite measure of PE demonstrated a similar pattern of results to PANAS PA. An aggregate score of different measures will emphasize the similarities between the scales, in this case the adaptive aspects of high PE, which is consistent with the observed stronger association with BA. Conversely, differences between the measures, such as the potentially maladaptive aspects of high BAS and AIM PA not assessed by PANAS PA contribute less to the composite score.

*Other Interactions*

The results also showed trend results for an NE x PE interaction for the BADS both cross-sectionally and prospectively. The interactions supported a protective effect of PANAS PA against low BA associated with high PANAS NA. The results did not support an NE x PE interaction in the BIS/BAS or AIM models, which is consistent with the previous discussion regarding the differences between the temperament measures. If BAS and AIM PA do not necessarily represent goal-directed behavior, there is no reason to expect high BAS or high AIM PA to be protective against low BA in cases of high BIS or high AIM NR.

Tests of the three-way interaction between NE, PE, and activation control for predicting BA did not yield significant results. Dinovo and Vasey (2011) reported a three-way NE x PE x EC interaction for general distress and anhedonic symptoms in which EC was most strongly related to symptoms when NE was high and PE was low, but an extension of these results to BA was not supported. However, given the small portion of the population that might be affected by a three-way interactive relationship, the null results here should not be interpreted as definitively ruling out the existence of such an interaction.
Future Directions

The results of the present study offer promising evidence for BA as a mediator of the effects of PE, NE, and activation control on depressive symptoms. Future research should continue to test other possible mediators of the links between reactive and effortful temperament with various psychological problems to lend support for existing treatment targets as well as develop new areas relevant to symptoms improvement. Future prospective studies over a longer time period would also be able to test changes in temperament and symptoms over time to determine potential cyclical associations between them. Further, EC should continue to be tested in adult populations including both undergraduate and community samples. Replication of observed links between temperament and psychological symptoms in clinical populations may help identify treatments best suited to specific temperamental vulnerabilities. For example, behavioral treatments for depression may be most beneficial to individuals with low PE, high NE, and low activation control compared to those with a subset of these vulnerabilities.

Advantages and Limitations

The large sample size in Study 1 provided increased statistical power to identify the effects of interest. Additionally, the larger sample size allowed for a CFA to confirm the results of the EFA using a different group of participants.

The main assets of Study 2 were the longitudinal design and moderate sample size. The design allowed for a truer test of mediation by assessing the variables in a temporal sequence and controlling for baseline symptoms. Although still correlative in nature, the longitudinal design lends further support to theoretical conceptualizations about the relationships between the included variables. The efficiency of the moderate
sample size was also improved by oversampling to increase the chances of including participants most relevant to the present study.

The present study also included some limitations. Despite the large sample size of Study 1 and the oversampling techniques used in Study 2, various combinations of high and low PE, NE, and activation control are not equally represented in the population. Specifically, groups with low PE and high activation control were underrepresented. Approximately four times as many participants reported both high PE and high activation control as those reporting low PE and high activation control in both Study 1 and Study 2. This distribution makes it more difficult to detect moderation as the underrepresented groups are the focus of these effects.

The similarity between the measures of activation control and BA also limited the power of the present study to detect hypothesized effects. Despite the theoretical distinction between these constructs, empirical testing was limited by the precision of available measures. Empirical efforts to examine these measures demonstrated some differentiation, but also confirmed expected overlap. These findings suggest improved measures with better precision would be beneficial to reduce measurement error and improve power to find the expected effects.

The results were also based on participant responses to self-report questionnaires. Although measures with good psychometric properties were used, the results relied on each participant’s subjective assessment of themselves. Finally, the study was correlational in nature in both Study 1 and Study 2 and although many of the results were consistent with theoretical expectations, no causal interpretations can be made.
Conclusions

In summary, an EFA and subsequent CFA of the items of the Activation Control subscale and the BADS found that they loaded on different factors, suggesting the measures of activation control and BA adequately distinguished between the constructs. However, though distinct, activation control and BA are closely related. The results supported the hypothesized mediation model in which the link between low PE and depressive symptoms was mediated by BA. BA also mediated the indirect effect of NE on symptoms of depression. The indirect effect of activation control on depressive symptoms was also mediated by BA. The mediation effects were found both cross-sectionally as well as prospectively. The proposed model of moderated mediation in which activation control moderated the indirect path from PE to BA to depressive symptoms was not supported as activation control did not reliably moderate the association between PE and BA as expected. Instead, PE was associated with BA consistently across all levels of activation control. Consideration of goal-oriented behavior also revealed important differences between PANAS PA, BAS, and AIM PA. A review of the measure items revealed items related to goal-oriented behavior on the PANAS PA scale, but not on the BAS or AIM PA scales. Finally, the results provided preliminary evidence of an interaction between PANAS NA and PANAS PA predicting BA such that high levels of PANAS PA were protective against low BA associated with high PANAS NA.
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### Table 1: Descriptive Statistics of Variables in Study 1

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<th>α</th>
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**Notes:** PANAS = Positive and Negative Affect Schedule, NA = Negative Affect, PA = Positive Affect, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, AIM = Affect Intensity Measure, NR = Negative Reactivity, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Table 2: Descriptive Statistics of Variables in Study 2

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*Notes:* T1 = Time 1, T2 = Time 2, T3 = Time 3, PANAS = Positive and Negative Affect Schedule, NA = Negative Affect, PA = Positive Affect, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, AIM = Affect Intensity Measure, NR = Negative Reactivity, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Table 3: Variables Correlations in Study 1

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* *p < 0.05

Notes: NE = Negative Emotionality, PE = Positive Emotionality, PANAS = Positive and Negative Affect Schedule, NA = Negative Affect, PA = Positive Affect, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, AIM = Affect Intensity Measure, NR = Negative Reactivity, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Table 4: Variable Correlations in Study 2

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*p < 0.05

Notes: T1 = Time 1, T2 = Time 2, T3 = Time 3, NE = Negative Emotionality, PE = Positive Emotionality, PANAS = Positive and Negative Affect Schedule, NA = Negative Affect, PA = Positive Affect, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, AIM = Affect Intensity Measure, NR = Negative Reactivity, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Table 5: Factor Loadings of Five-Factor Exploratory Factor Analysis

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<td>BADS 25R</td>
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Table 6: Factor Intercorrelations of Five-Factor Exploratory Factor Analysis

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Notes: RMSEA = 0.065, 90% CI [0.061, 0.069], n = 575. Comm = Communality, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and R = reversed item.
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<td>0.173</td>
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**Notes:** RMSEA = 0.059, 90% CI [0.055, 0.064], n = 575. Comm = Communality, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and R = reversed item.

Table 8: Factor Intercorrelations of Six-Factor Exploratory Factor Analysis

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Table 9: Factor Loadings of Seven-Factor Exploratory Factor Analysis

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<th>Factor 4</th>
<th>Factor 5</th>
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<td>0.126</td>
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Table 10: Factor Intercorrelations of Seven-Factor Exploratory Factor Analysis

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Notes: RMSEA = 0.055, 90% CI [0.050, 0.060], n = 575. Comm = Communality, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and R = reversed item.
Table 11: Factor Loadings of Confirmatory Factor Analysis

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<td></td>
<td></td>
<td></td>
<td>0.548</td>
<td></td>
<td>0.300</td>
</tr>
<tr>
<td>BADS 17R</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.816</td>
<td></td>
<td>0.666</td>
</tr>
<tr>
<td>BADS 18R</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.798</td>
<td></td>
<td>0.637</td>
</tr>
<tr>
<td>BADS 19R</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.735</td>
<td></td>
<td>0.540</td>
</tr>
<tr>
<td>BADS 20R</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.848</td>
<td></td>
<td>0.719</td>
</tr>
</tbody>
</table>

Notes: RMSEA = 0.066, 90% CI [0.062, 0.069], n = 623. Comm = Communality, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and R = reversed item.

Table 12: Factor Intercorrelations of Confirmatory Factor Analysis

<table>
<thead>
<tr>
<th></th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Factor 3</th>
<th>Factor 4</th>
<th>Factor 5</th>
<th>Factor 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor 1</td>
<td></td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor 2</td>
<td>0.668</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor 3</td>
<td>0.561</td>
<td>0.657</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor 4</td>
<td>0.327</td>
<td>0.415</td>
<td>0.144</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor 5</td>
<td>0.701</td>
<td>0.754</td>
<td>0.414</td>
<td>0.537</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor 6</td>
<td>0.192</td>
<td>0.389</td>
<td>0.095</td>
<td>0.614</td>
<td>0.412</td>
<td></td>
</tr>
</tbody>
</table>
Table 13: Multiple Regression Analysis with Composite Scales in Study 1

<table>
<thead>
<tr>
<th></th>
<th>Regression on</th>
<th>B</th>
<th>SE</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BADS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Composite PE</td>
<td>0.27</td>
<td>0.03</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>Act Ctrl</td>
<td>0.31</td>
<td>0.03</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>Composite PE x Act Ctrl</td>
<td>-0.02</td>
<td>0.02</td>
<td>.441</td>
<td></td>
</tr>
<tr>
<td>Composite NE</td>
<td>-0.31</td>
<td>0.03</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>Attn Ctrl</td>
<td>0.10</td>
<td>0.03</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Inhib Ctrl</td>
<td>0.11</td>
<td>0.03</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>Composite NE x Attn Ctrl</td>
<td>0.04</td>
<td>0.02</td>
<td>.067</td>
<td></td>
</tr>
<tr>
<td>Composite NE x Composite PE</td>
<td>0.03</td>
<td>0.02</td>
<td>.260</td>
<td></td>
</tr>
<tr>
<td>Composite NE x Act Ctrl*</td>
<td>-0.03</td>
<td>0.03</td>
<td>.369</td>
<td></td>
</tr>
<tr>
<td>Composite NE x Composite PE x Act Ctrl*</td>
<td>-0.01</td>
<td>0.02</td>
<td>.614</td>
<td></td>
</tr>
</tbody>
</table>

*The Composite NE x Act Ctrl and Composite NE x Composite PE x Act Ctrl interactions were added in an exploratory step. All other values are reported for the model without these interactions.

Notes: n = 979. NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, and BADS = Behavioral Activation for Depression Scale.

Table 14: Multiple Regression Analysis with the PANAS in Study 1

<table>
<thead>
<tr>
<th></th>
<th>Regression on</th>
<th>B</th>
<th>SE</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BADS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PANAS PA</td>
<td>0.24</td>
<td>0.02</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>Act Ctrl</td>
<td>0.25</td>
<td>0.03</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>PANAS PA x Act Ctrl</td>
<td>-0.01</td>
<td>0.02</td>
<td>.711</td>
<td></td>
</tr>
<tr>
<td>PANAS NA</td>
<td>-0.38</td>
<td>0.02</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>Attn Ctrl</td>
<td>0.10</td>
<td>0.03</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>Inhib Ctrl</td>
<td>0.05</td>
<td>0.02</td>
<td>.034</td>
<td></td>
</tr>
<tr>
<td>PANAS NA x Attn Ctrl</td>
<td>0.01</td>
<td>0.02</td>
<td>.615</td>
<td></td>
</tr>
<tr>
<td>PANAS NA x PANAS PA</td>
<td>-0.04</td>
<td>0.02</td>
<td>.058</td>
<td></td>
</tr>
<tr>
<td>PANAS NA x Act Ctrl*</td>
<td>-0.02</td>
<td>0.03</td>
<td>.454</td>
<td></td>
</tr>
<tr>
<td>PANAS NA x PANAS PA x Act Ctrl*</td>
<td>0.01</td>
<td>0.02</td>
<td>.482</td>
<td></td>
</tr>
</tbody>
</table>

*The PANAS NA x Act Ctrl and PANAS NA x PANAS PA x Act Ctrl interactions were added in an exploratory step. All other values are reported for the model without these interactions.

Notes: n = 1,126. PANAS = Positive and Negative Affect Schedule, NA = Negative Affect, PA = Positive Affect, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, and BADS = Behavioral Activation for Depression Scale.
Table 15: Multiple Regression Analysis with the BIS/BAS in Study 1

<table>
<thead>
<tr>
<th></th>
<th>Regression on BADS</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE</td>
<td>p</td>
</tr>
<tr>
<td>BAS</td>
<td>0.12</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Act Ctrl</td>
<td>0.35</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>BAS x Act Ctrl</td>
<td>0.01</td>
<td>0.02</td>
<td>.735</td>
</tr>
<tr>
<td>BIS</td>
<td>-0.15</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Attn Ctrl</td>
<td>0.21</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Inhib Ctrl</td>
<td>0.08</td>
<td>0.03</td>
<td>.007</td>
</tr>
<tr>
<td>BIS x Attn Ctrl</td>
<td>0.03</td>
<td>0.02</td>
<td>.168</td>
</tr>
<tr>
<td>BIS x BAS</td>
<td>-0.02</td>
<td>0.02</td>
<td>.315</td>
</tr>
<tr>
<td>BIS x Act Ctrl*</td>
<td>-0.02</td>
<td>0.03</td>
<td>.412</td>
</tr>
<tr>
<td>BIS x BAS x Act Ctrl*</td>
<td>-0.02</td>
<td>0.02</td>
<td>.292</td>
</tr>
</tbody>
</table>

*The BIS x Act Ctrl and BIS x BAS x Act Ctrl interactions were added in an exploratory step. All other values are reported for the model without these interactions.

Notes: n = 1,061. BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, and BADS = Behavioral Activation for Depression Scale.

Table 16: Multiple Regression Analysis with the AIM in Study 1

<table>
<thead>
<tr>
<th></th>
<th>Regression on BADS</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE</td>
<td>p</td>
</tr>
<tr>
<td>AIM PA</td>
<td>0.18</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Act Ctrl</td>
<td>0.36</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>AIM PA x Act Ctrl</td>
<td>-0.02</td>
<td>0.03</td>
<td>.366</td>
</tr>
<tr>
<td>AIM NR</td>
<td>-0.15</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Attn Ctrl</td>
<td>0.22</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Inhib Ctrl</td>
<td>0.09</td>
<td>0.03</td>
<td>.002</td>
</tr>
<tr>
<td>AIM NR x Attn Ctrl</td>
<td>0.01</td>
<td>0.02</td>
<td>.595</td>
</tr>
<tr>
<td>AIM NR x AIM PA</td>
<td>0.00</td>
<td>0.02</td>
<td>.996</td>
</tr>
<tr>
<td>AIM NR x Act Ctrl*</td>
<td>-0.01</td>
<td>0.03</td>
<td>.688</td>
</tr>
<tr>
<td>AIM NR x AIM PA x Act Ctrl*</td>
<td>0.03</td>
<td>0.02</td>
<td>.225</td>
</tr>
</tbody>
</table>

*The AIM NR x Act Ctrl and AIM NR x AIM PA x Act Ctrl interactions were added in an exploratory step. All other values are reported for the model without these interactions.

Notes: n = 1,072. AIM = Affect Intensity Measure, NR = Negative Reactivity, PA = Positive Affectivity, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, and BADS = Behavioral Activation for Depression Scale.
Table 17: Multiple Regression Analysis with Composite Scales in Study 2

<table>
<thead>
<tr>
<th></th>
<th>Regression on</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T2 BADS</td>
<td>B</td>
<td>SE</td>
<td>p</td>
</tr>
<tr>
<td>T1 Composite PE</td>
<td>0.20</td>
<td>0.08</td>
<td>.008</td>
<td></td>
</tr>
<tr>
<td>T1 Act Ctrl</td>
<td>0.18</td>
<td>0.08</td>
<td>.023</td>
<td></td>
</tr>
<tr>
<td>T1 Composite PE x T1 Act Ctrl</td>
<td>0.05</td>
<td>0.06</td>
<td>.439</td>
<td></td>
</tr>
<tr>
<td>T1 Composite NE</td>
<td>-0.12</td>
<td>0.08</td>
<td>.132</td>
<td></td>
</tr>
<tr>
<td>T1 Attn Ctrl</td>
<td>0.15</td>
<td>0.08</td>
<td>.073</td>
<td></td>
</tr>
<tr>
<td>T1 Inhib Ctrl</td>
<td>0.19</td>
<td>0.09</td>
<td>.030</td>
<td></td>
</tr>
<tr>
<td>T1 Composite NE x T1 Attn Ctrl</td>
<td>0.06</td>
<td>0.07</td>
<td>.370</td>
<td></td>
</tr>
<tr>
<td>T1 Composite NE x T1 Composite PE</td>
<td>0.04</td>
<td>0.07</td>
<td>.554</td>
<td></td>
</tr>
<tr>
<td>T1 DASS Depression</td>
<td>-0.06</td>
<td>0.01</td>
<td>&lt;.001</td>
<td></td>
</tr>
</tbody>
</table>

Notes: n = 160. T1 = Time 1, T2 = Time 2, NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

Table 18: Multiple Regression Analysis with the PANAS in Study 2

<table>
<thead>
<tr>
<th></th>
<th>Regression on</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T2 BADS</td>
<td>B</td>
<td>SE</td>
<td>p</td>
</tr>
<tr>
<td>T1 PANAS PA</td>
<td>0.12</td>
<td>0.07</td>
<td>.068</td>
<td></td>
</tr>
<tr>
<td>T1 Act Ctrl</td>
<td>0.20</td>
<td>0.07</td>
<td>.006</td>
<td></td>
</tr>
<tr>
<td>T1 PANAS PA x T1 Act Ctrl</td>
<td>0.05</td>
<td>0.05</td>
<td>.321</td>
<td></td>
</tr>
<tr>
<td>T1 PANAS NA</td>
<td>-0.26</td>
<td>0.07</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>T1 Attn Ctrl</td>
<td>0.06</td>
<td>0.07</td>
<td>.398</td>
<td></td>
</tr>
<tr>
<td>T1 Inhib Ctrl</td>
<td>0.09</td>
<td>0.07</td>
<td>.190</td>
<td></td>
</tr>
<tr>
<td>T1 PANAS NA x T1 Attn Ctrl</td>
<td>-0.02</td>
<td>0.05</td>
<td>.659</td>
<td></td>
</tr>
<tr>
<td>T1 PANAS NA x T1 PANAS PA</td>
<td>0.10</td>
<td>0.05</td>
<td>.061</td>
<td></td>
</tr>
<tr>
<td>T1 DASS Depression</td>
<td>-0.04</td>
<td>0.01</td>
<td>&lt;.001</td>
<td></td>
</tr>
</tbody>
</table>

Notes: n = 179. T1 = Time 1, T2 = Time 2, PANAS = Positive and Negative Affect Schedule, NA = Negative Affect, PA = Positive Affect, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Table 19: Multiple Regression Analysis with the BIS/BAS in Study 2

<table>
<thead>
<tr>
<th>Regression on T2 BADS</th>
<th>B</th>
<th>SE</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 BAS</td>
<td>0.15</td>
<td>0.07</td>
<td>.035</td>
</tr>
<tr>
<td>T1 Act Ctrl</td>
<td>0.21</td>
<td>0.07</td>
<td>.006</td>
</tr>
<tr>
<td>T1 BAS x T1 Act Ctrl</td>
<td>0.01</td>
<td>0.06</td>
<td>.906</td>
</tr>
<tr>
<td>T1 BIS</td>
<td>-0.13</td>
<td>0.07</td>
<td>.073</td>
</tr>
<tr>
<td>T1 Attn Ctrl</td>
<td>0.11</td>
<td>0.08</td>
<td>.161</td>
</tr>
<tr>
<td>T1 Inhib Ctrl</td>
<td>0.17</td>
<td>0.08</td>
<td>.036</td>
</tr>
<tr>
<td>T1 BIS x Attn Ctrl</td>
<td>0.10</td>
<td>0.07</td>
<td>.139</td>
</tr>
<tr>
<td>T1 BIS x T1 BAS</td>
<td>0.05</td>
<td>0.07</td>
<td>.450</td>
</tr>
<tr>
<td>T1 DASS Depression</td>
<td>-0.06</td>
<td>0.01</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

Notes: n = 168. T1 = Time 1, T2 = Time 2, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

Table 20: Multiple Regression Analysis with the AIM in Study 2

<table>
<thead>
<tr>
<th>Regression on T2 BADS</th>
<th>B</th>
<th>SE</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 AIM PA</td>
<td>0.11</td>
<td>0.07</td>
<td>.112</td>
</tr>
<tr>
<td>T1 Act Ctrl</td>
<td>0.20</td>
<td>0.08</td>
<td>.008</td>
</tr>
<tr>
<td>T1 AIM PA x T1 Act Ctrl</td>
<td>0.02</td>
<td>0.07</td>
<td>.799</td>
</tr>
<tr>
<td>T1 AIM NR</td>
<td>0.07</td>
<td>0.07</td>
<td>.304</td>
</tr>
<tr>
<td>T1 Attn Ctrl</td>
<td>0.15</td>
<td>0.08</td>
<td>.045</td>
</tr>
<tr>
<td>T1 Inhib Ctrl</td>
<td>0.21</td>
<td>0.08</td>
<td>.009</td>
</tr>
<tr>
<td>T1 AIM NR x T1 Attn Ctrl</td>
<td>-0.02</td>
<td>0.07</td>
<td>.788</td>
</tr>
<tr>
<td>T1 AIM NR x T1 AIM PA</td>
<td>-0.05</td>
<td>0.05</td>
<td>.317</td>
</tr>
<tr>
<td>T1 DASS Depression</td>
<td>-0.06</td>
<td>0.01</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

Notes: n = 172. T1 = Time 1, T2 = Time 2, AIM = Affect Intensity Measure, NR = Negative Reactivity, PA = Positive Affectivity, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Appendix B: Figures

Figure 1: Simple Mediation

\[
\begin{align*}
X & \rightarrow M \\
M & \rightarrow Y
\end{align*}
\]

Figure 2: Model of Moderated Mediation

\[
\begin{align*}
W & \rightarrow M \\
M & \rightarrow Y
\end{align*}
\]

Figure 3: Hypothesized Model of Moderated Mediation

\[
\begin{align*}
\text{Activation Control} & \rightarrow \text{Behavioral Activation} \\
\text{Behavioral Activation} & \rightarrow \text{Depressive Symptoms}
\end{align*}
\]
Notes: n = 1,129. PANAS NA, Attentional Control, Inhibitory Control, Activation Control, PANAS NA x Attentional Control, PANAS PA x Activation Control, and PANAS NA x PANAS PA were included as covariates. PANAS = Positive and Negative Affect Schedule, NA = Negative Affect, PA = Positive Affect, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Figure 6: Indirect Path of BAS to BADS to DASS Depression in Study 1

\[ ab = -0.60, 95\% \text{ CI} [-0.89, -0.30], \kappa^2 = 0.11 \]

A similar figure is shown for AIM PA and Composite PE to BADS to DASS Depression in Study 1.

**Notes:** n = 1,061. BIS, Attentional Control, Inhibitory Control, Activation Control, BIS x Attentional Control, BAS x Activation Control, and BIS x BAS were included as covariates. BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Figure 9: Indirect Path of Composite NE to BADS to DASS Depression in Study 1

\[ ab = 1.26, \text{ 95\% CI } [1.03, \ 1.52], \ \kappa^2 = 0.24 \]

\[ a = -0.31, \ \text{ } p < .001 \]
\[ b = -4.14, \ \text{ } p < .001 \]

Notes: \( n = 979 \). Composite PE, Attentional Control, Inhibitory Control, Activation Control, Composite NE x Attentional Control, Composite PE x Activation Control, and Composite NE x Composite PE were included as covariates. NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

Figure 10: Indirect Path of Activation Control to BADS to DASS Depression in Study 1

\[ ab = -1.29, \text{ 95\% CI } [-1.59, -1.04], \ \kappa^2 = 0.32 \]

\[ a = 0.31, \ \text{ } p < .001 \]
\[ b = -4.13, \ \text{ } p < .001 \]

Notes: \( n = 979 \). Composite NE, Composite PE, Attentional Control, Inhibitory Control, Composite NE x Attentional Control, Composite PE x Activation Control, and Composite NE x Composite PE were included as covariates. NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

Figure 11: Indirect Path of Activation Control to Modified BADS to DASS Depression in Study 1

\[ ab = -0.98, \text{ 95\% CI } [-1.26, -0.73], \ \kappa^2 = 0.27 \]

\[ a = 0.25, \ \text{ } p < .001 \]
\[ b = -3.92, \ \text{ } p < .001 \]

Notes: \( n = 984 \). Composite NE, Composite PE, Attentional Control, Inhibitory Control, Composite NE x Attentional Control, Composite PE x Activation Control, and Composite NE x Composite PE were included as covariates. NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

*The BADS Activation subscale items were removed.

Notes: \( n = 984 \). Composite NE, Composite PE, Attentional Control, Inhibitory Control, Composite NE x Attentional Control, Composite PE x Activation Control, and Composite NE x Composite PE were included as covariates. NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Figure 12: PANAS NA x PANAS PA Interaction Predicting the BADS in Study 1

![Graph showing the interaction effect of PANAS NA and PANAS PA on BADS in Study 1.]

Figure 13: Indirect Path of T1 Composite PE to T2 BADS to T3 DASS Depression in Study 2

\[ ab = -0.52, \text{ 95% CI} [-1.42, -0.17], \kappa^2 = 0.16 \]

\[ a = 0.20, \quad p = 0.008 \]

\[ b = -2.76, \quad p < 0.001 \]

\[ c = -2.02, \quad p = .006 \]

\[ c' = -1.46, \quad p = .043 \]

Notes: n = 160. T1 Composite NE, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 Composite NE x T1 Attentional Control, T1 Composite PE x T1 Activation Control, T1 Composite NE x T1 Composite PE, and T1 DASS Depression were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Figure 14: Indirect Path of T1 Composite NE to T2 BADS to T3 DASS Depression in Study 2

\[ ab = 0.33, \text{ 95\% CI [-0.04, 0.98], } \kappa^2 = 0.22 \]

Notes: \( n = 160. \) T1 Composite PE, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 Composite NE x T1 Attentional Control, T1 Composite PE x T1 Activation Control, T1 Composite NE x T1 Composite PE, and T1 DASS Depression were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

Figure 15: Indirect Path of T1 Composite NE to T2 BADS to T3 DASS Depression Excluding T1 DASS Depression as a Covariate in Study 2

\[ ab = 0.90, \text{ 95\% CI [0.29, 1.83], } \kappa^2 = 0.22 \]

Notes: \( n = 165. \) T1 Composite PE, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 Composite NE x T1 Attentional Control, T1 Composite PE x T1 Activation Control, and T1 Composite NE x T1 Composite PE were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Figure 16: Indirect Path of T1 Activation Control to T2 BADS to T3 DASS Depression in Study 2

\[ ab = -0.51, 95\% \text{ CI } [-1.15, -0.04], \kappa^2 = 0.22 \]

\[ a = 0.18 \quad p = .023 \]
\[ b = -2.76 \quad p < .001 \]
\[ c = -0.07, p = .921 \]
\[ c' = 0.42, p = .568 \]

Notes: n = 160. T1 Composite NE, T1 Composite PE, T1 Attentional Control, T1 Inhibitory Control, T1 Composite NE x T1 Attentional Control, T1 Composite PE x T1 Activation Control, T1 Composite NE x T1 Composite PE, and T1 DASS Depression were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

Figure 17: Indirect Path of T1 PANAS PA to T2 BADS to T3 DASS Depression in Study 2

\[ ab = -0.28, 95\% \text{ CI } [-0.82, -0.01], \kappa^2 = 0.21 \]

\[ a = 0.12 \quad p = .068 \]
\[ b = -2.23 \quad p = .002 \]
\[ c = -1.68, p = .011 \]
\[ c' = -1.40, p = .031 \]

Notes: n = 179. T1 PANAS NA, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 PANAS NA x T1 Attentional Control, T1 PANAS PA x T1 Activation Control, T1 PANAS NA x T1 PANAS PA, and T1 DASS Depression were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, PANAS = Positive and Negative Affect Schedule, NA = Negative Affect, PA = Positive Affect, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Figure 18: Indirect Path of T1 BAS to T2 BADS to T3 DASS Depression in Study 2

\[ ab = -0.42, 95\% CI [-1.08, -0.10], \kappa^2 = 0.07 \]

\[ a = 0.15 \quad p = .035 \]
\[ b = -3.00 \quad p < .001 \]
\[ c = -1.20, p = .074 \quad c' = 0.75, p = .244 \]

Notes: n = 168. T1 BIS, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 BIS x T1 Attentional Control, T1 BAS x T1 Activation Control, T1 BIS x T1 BAS, and T1 DASS Depression were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

Figure 19: Indirect Path of T1 AIM PA to T2 BADS to T3 DASS Depression in Study 2

\[ ab = -0.17, 95\% CI [-0.79, 0.14], \kappa^2 = 0.10 \]

\[ a = 0.08 \quad p = .294 \]
\[ b = -2.61 \quad p < .001 \]
\[ c = -1.16, p = .087 \quad c' = 0.97, p = .141 \]

Notes: n = 172. T1 AIM NR, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 AIM NR x T1 Attentional Control, T1 AIM PA x T1 Activation Control, T1 AIM NR x T1 AIM PA, and T1 DASS Depression were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, AIM = Affect Intensity Measure, NR = Negative Reactivity, PA = Positive Affectivity, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Figure 20: Indirect Path of T1 AIM PA to T2 BADS to T3 DASS Depression Excluding T1 DASS Depression as a Covariate in Study 2

\[ ab = -0.70, 95\% CI [-1.83, -0.08], \chi^2 = 0.10 \]

\[ a = 0.17, \quad p = .025 \]
\[ b = -4.29, \quad p < .001 \]
\[ c = -2.17, \quad p = .004 \]
\[ c' = -1.42, \quad p = .040 \]

Notes: n = 177. T1 AIM NR, T1 Attentional Control, T1 Inhibitory Control, T1 Activation Control, T1 AIM NR x T1 Attentional Control, T1 AIM PA x T1 Activation Control, and T1 AIM NR x T1 AIM PA were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, AIM = Affect Intensity Measure, NR = Negative Reactivity, PA = Positive Affectivity, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.

Figure 21: Indirect Path of T1 Activation Control to T2 Modified BADS to T3 DASS Depression in Study 2

\[ ab = -0.38, 95\% CI [-0.97, 0.09], \chi^2 = 0.19 \]

\[ a = 0.12, \quad p = .128 \]
\[ b = -3.01, \quad p < .001 \]
\[ c = -0.51, \quad p = .515 \]
\[ c' = -0.14, \quad p = .850 \]

*The BADS Activation subscale items were removed.

Notes: n = 165. T1 Composite NE, T1 Composite PE, T1 Attentional Control, T1 Inhibitory Control, T1 Composite NE x T1 Attentional Control, T1 Composite PE x T1 Activation Control, T1 Composite NE x T1 Composite PE, and T1 DASS Depression were included as covariates. T1 = Time 1, T2 = Time 2, T3 = Time 3, NE = Negative Emotionality, PE = Positive Emotionality, Attn Ctrl = Attentional Control, Inhib Ctrl = Inhibitory Control, Act Ctrl = Activation Control, BADS = Behavioral Activation for Depression Scale, and DASS = Depression Anxiety Stress Schedule.
Figure 22: T1 PANAS NA x T1 PANAS PA Interaction Predicting T2 BADS in Study 2