Individual Facets of Effortful Control and Symptoms of General Distress and Depression

Thesis

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By

Adam Gregg Buffington, B.S, B.S.

Clinical Psychology Graduate Program

The Ohio State University

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Thesis Committee:

Michael W. Vasey, Ph.D., Advisor

Julian F. Thayer, Ph.D.

Jennifer S. Cheavens, Ph.D.

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Abstract

The present study explored the relationship between positive and negative reactivity, effortful control (EC), and symptoms of both general distress and depression in a sample of 1242 undergraduate students. Participant responses to self-report questionnaire measures of temperament and emotional symptoms were analyzed using multiple linear regression analyses. EC was divided into three facets of attentional control, inhibitory control, and activation control to examine the different relationships between the individual components of EC and emotional problems. Attentional control and inhibitory control were related to symptoms of general distress and depression that were associated with negative reactivity. There was also evidence that attentional control moderated the association between negative reactivity and symptoms of general distress and depression. Conversely, activation control was related to symptoms specific to depression, which are most strongly related to low positive reactivity. Activation control also moderated the association between positive reactivity and anhedonic symptoms such that low positive reactivity was more weakly related to depressive symptoms at higher levels of activation control. Sex differences were found indicating that men were more likely to report symptoms of depression not related to negative reactivity than women. The results also showed that low activation control was related to more depressive symptoms in men than women. There was evidence of an interactive relationship

between Behavioral Inhibition (BIS) and Behavioral Activation (BAS) for general distress such that the at low levels of BIS, low BAS was associated with higher reports of general distress, and at high levels of BIS, reports of general distress were similar for both high and low levels of BAS. There was also an interaction between negative affectivity (NA) and positive affectivity (PA) for symptoms of depression such that the relationship between NA and depressive symptoms was reduced at higher levels of PA.

Dedication

For Paige and my family, thank you for your love and support.

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Vita

1998	Thomas Worthington High School.
2002	.B.S. Government, US Coast Guard
	Academy
2006	.B.S. Psychology, Old Dominion University
2007 to 2008	.University Fellow, The Ohio State
	University
2008 to 2009	.Graduate Research Assistant, Department of
	Psychology, The Ohio State University
2009 to Present	.Graduate Teaching Assistant, Department of Psychology, The Ohio State University

Fields of Study

Major Field: Clinical Psychology

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Chapter 1: Introduction

A large research literature exists exploring the association between temperament and a wide range of psychological symptoms and disorders. There is strong evidence to suggest that temperament affects the etiology and course of psychopathology as well as treatment response. Rothbart and Bates (1998) broadly define temperament as biologically-based, individual differences in reactivity and self-regulation. Although there are numerous conceptualizations, temperament models generally include components of positive and negative reactivity. Broadly, negative reactivity incorporates negative emotionality, negative affect, neuroticism, and a tendency toward behavioral withdrawal or inhibition. High levels of negative reactivity are characterized by a propensity to experience sadness, fear, anger, distress, or agitation. Conversely, low levels are marked by a propensity for calm and relaxed feelings. Positive reactivity broadly incorporates positive emotionality, positive affect, extraversion, and a tendency toward behavioral approach (Clark, 2005; Clark & Watson, 1999; Lonigan, Vasey, Phillips, & Hazen, 2004; Nigg, 2006). High levels of positive reactivity are characterized by a propensity toward engagement, enthusiasm, and activity. Conversely, low positive reactivity is characterized by a lack of positive mood, fatigue, and general sluggishness, and at very low levels, anhedonia.

The integrative hierarchical model of anxiety and depression (Mineka, Watson, & Clark, 1998), an elaboration on the influential tripartite model (Clark & Watson, 1991),

describes both common and distinct temperament associations with anxiety and depression. The model proposes that anxiety and depression share a common general affective temperamental distress component characterized by high negative affect (NA). In addition, the model identifies an association between lower levels of positive affect (PA) and depression unique from anxiety. That is, anxiety is characterized by high NA, while depression is characterized by both high levels of NA and low levels of PA. The original tripartite model also included a component of physiological hyperarousal (PH), defined by autonomic hyperactivity, that is thought to be specific to anxiety. However, subsequent studies of the tripartite model have not found clear associations between PH and different anxiety disorders and have generally concluded that the anxiety spectrum may be too heterogeneous to identify a single distinguishing factor to represent all anxiety disorders (Clark & Watson, 1991; Mineka et al., 1998). Despite the lack of consensus on specific factors for various anxiety disorders, numerous studies have shown support for the general model of association between NA and anxiety and between NA and PA and depression (e.g., Brown, Chorpita, & Barlow, 1998; Joiner, 1996; Watson, Clark et al., 1995; Watson, Weber et al., 1995).

Negative reactivity has been shown to be a broad predictor of psychopathology. A recent meta-analysis by Malouff, Thorsteinsson, and Schutte (2005) of 33 studies from the past two decades examining the association between the Five-Factor Model of personality and symptoms of psychological disorders found that neuroticism, or negative reactivity, was associated with a wide array of psychological conditions. The disorders associated with negative reactivity included mood, anxiety, child internalizing, somatoform, substance abuse, schizophrenia, eating, and dissociative identity disorders.

The analysis also found associations between extraversion, or positive reactivity, and mood disorders, anxiety disorders, schizophrenia, and eating disorders. A review by Mineka et al. (1998) also found examples of associations in the literature between negative reactivity and substance abuse disorders, somatoform disorders, eating disorders, personality disorders, conduct disorders, and schizophrenia. That review also found associations between low levels of positive reactivity and depression, schizophrenia, and social phobia. Watson, Gamez, and Simms (2005) present evidence showing that although negative reactivity is related to nearly all forms of psychopathology, it is most strongly related to disorders characterized by high levels of individual distress. The authors reviewed several data sets showing negative reactivity was most strongly correlated with disorders characterized by chronic, pervasive distress such as depression and generalized anxiety disorder. Low levels of positive reactivity were most strongly correlated with features of depression and social phobia.

In a community sample of almost 500 mostly college-aged participants, Trull and Sher (1994) found differences in positive and negative reactivity between groups meeting criteria for various clinical disorders and non-disordered groups. The authors found that groups with substance abuse disorders, post traumatic stress disorder, social phobia, agoraphobia, anxiety disorders, and major depressive disorder all showed higher negative reactivity and lower positive reactivity than the control group.

Despite consistent evidence showing associations between positive and negative reactivity and psychological disorders, the specific nature of the association between temperament and psychopathology is difficult to identify as they may be related in a variety of different ways. For example, temperament may affect etiology or vulnerability to particular disorders. Clark, Watson, and Mineka (1994; see also Mineka et al., 1998) reviewed a series of longitudinal studies showing that low negative reactivity was associated with reduced risk of developing later depression. Kendler, Neale, Kessler, Heath, and Eaves (1993) also found that high negative reactivity measured at an initial assessment increased the probability of developing major depression 15 months later in an analysis of more than 1,500 individual female-female monozygotic and dizygotic twins. However, the authors did not find evidence for predictive value of positive reactivity across the 15-month interval. Conversely, although fewer studies have explored the role of positive reactivity on vulnerability to depression, Clark et al. (1994) presented preliminary indications that lower positive reactivity was longitudinally predictive of development of depression. Testing a vulnerability model is difficult because it requires long-term assessment of a community sample large enough to include enough participants who develop the disorder to allow for sufficient statistical power to find statistical effects of premorbid temperament. In one such extended longitudinal study of a large sample of almost 3,000 Swiss men, Clayton, Ernst, and Angst (1994) found that men who developed depression between age 19 and 36 reported characteristics consistent with high negative reactivity prior to the onset of depression.

Krueger, Caspi, Moffitt, Silva, and McGee (1996) also found that negative reactivity was correlated with multiple categories of diagnosed psychological disorders including affect, anxiety, substance, and conduct disorders in a sample of about 900 members of an unselected, New Zealand birth cohort. Further, the data showed that negative reactivity assessed at age 18 was correlated with four disorder categories assessed at age 15, 18, and 21. Further analysis of the same data by Krueger (1999)

showed that negative reactivity at age 18 was also predictive of the presence of a psychological disorder in all four categories at age 21 when controlling for the presence of disorders at age 18. These results extended previous cross-sectional associations and demonstrated that temperament can also predict the development of psychological disorders over time.

Similar results have been observed in younger samples. Joiner and Lonigan (2000) found that a combination of high negative reactivity and low positive reactivity was associated with depressive disorder diagnoses in a psychiatric inpatient sample of children and adolescents. In a separate inpatient child sample, the authors found that the combination of high negative reactivity and low positive reactivity was associated with changes in depressive symptoms, but not anxious symptoms, over a 2-month period. Similarly, in a sample of 4th through 11th graders, Lonigan, Phillips, and Hooe (2003) found that high negative reactivity, but not positive reactivity, was associated with increases in anxious symptoms over a 7-month period. Consistent with expectations, the results also showed that both high negative reactivity and low positive reactivity were associated with increases in depressive symptoms over the same period. Further, although positive reactivity accounted for unique variance in changes in depressive symptoms, the reverse was not true. That is, low positive reactivity was associated with an increase in depressive symptoms, but symptom level did not account for changes in positive reactivity. This pattern supports conceptualizations of positive reactivity as a factor in the development of depressive symptoms.

Clark et al. (1994) also reviewed evidence of a pathoplasty model in which temperament affects the course of a disorder. The evidence showed higher negative

reactivity is associated with poorer prognosis for depression including protracted symptom episodes, a more chronic course, and a slower treatment response. The reviewed evidence also suggested lower positive reactivity is associated with a more chronic course of depression.

Further, psychological disorders may affect temperament. This association is generally referred to as a scar model. Indeed, there is some evidence of increases in negative reactivity and decreases in positive reactivity following depressive episodes. However, it is not clear how long these changes last and the limited evidence available suggests that levels of reactivity return toward premorbid levels over time (Clark et al., 1994; Mineka et al., 1998). In a two-year longitudinal study of a clinical population, Brown (2007) found that levels of depression did not predict changes in temperament over time. Like a vulnerability model, the required time and sample size make it difficult to adequately test a scar model. State differences affecting temperament measurement also add to the difficulty of determining how much change in temperament is caused by a psychological disorder and how much is accounted for by state variability (Clark, Vittengl, Kraft, & Jarrett, 2003).

Finally, temperament and psychopathology may both be outcomes of a common underlying factor. In the large twin study previously discussed, Kendler et al. (1993) reported variance accounted for by a common genetic diathesis for both negative reactivity and depression. In a review of research designs and available information on links between genes, temperament, and psychological disorders, Carey and DiLalla (1994) similarly concluded that common genes may have effects on both temperament and psychological disorders. A spectrum model is an extension of the common cause model, which argues that psychological disorders are manifestations of extreme levels of temperament or personality. This association is supported by a dimensional model of psychological disorders as opposed to current categorical conceptualizations (e.g., Widiger & Samuel, 2005).

Each of the various models suggests different casual effects, but they are not mutually exclusive. Based on available evidence supporting the various models, the association between temperament and psychopathology is likely made up of a combination of these different models. Temperament likely predisposes an individual to psychological disorders and also affects the course of the disorder. Additionally, psychopathology may, in turn, cause changes in temperament. Finally, these associations may also be explained in part by a common diathesis for both temperament and psychopathology.

The majority of research into links between temperament and psychopathology has focused on reactive, involuntary aspects of temperament such as positive and negative reactivity. Less attention has been paid to temperamental capacity for voluntary control of reactive impulses. Rothbart and colleagues have proposed a separate temperament factor, effortful control (EC), to describe the capacity for regulation of reactive responses in circumstances where the prepotent response would be detrimental to the individual. In addition to regulating external behavioral actions, EC also plays an important role in regulating internal cognitive processes (Rothbart, 1989; Rothbart & Bates, 2006).

A temperamental capacity for regulation of cognitive processes is an important consideration in sorting out the association between temperament and psychological

disorders. The cognitive regulation function of EC suggests it may have a connection to psychological disorders unique from the established association between psychological disorders and reactive temperament. High levels of EC are likely to be associated with fewer psychological symptoms. In line with expectations, research exploring associations between EC and psychological problems has largely supported an inverse association between them independent of positive and negative reactivity. This research has included a focus on links between temperament and internalizing problems such as anxious and depressive symptoms. Empirical studies have found lower levels of EC in groups with internalizing problems (e.g., Eisenberg et al., 2001; Oldehinkel, Hartman, De Winter, Veenstra, & Ormel, 2004) and associations between EC and internalizing problems unique from associations between problems and reactive temperament (e.g., Muris, 2006; Verstraeten, Vasey, Raes, & Bijttebier, 2009). Although an association between EC and psychological problems independent of reactive temperament has been well documented in the developmental literature, it has not been extensively studied in adults.

Several studies comparing groups with internalizing problems to control groups have found increased negative reactivity and lower EC in problem groups. In a largesample study of over 2,000 Dutch preadolescent schoolchildren, Oldehinkel et al. (2004) found that groups with internalizing problems had higher negative reactivity and lower positive reactivity than a control group. In addition to the expected finding with regards to reactive temperament, the internalizing problem group had lower EC than the control group, although the difference was small. In a sample of 4th grade boys, John, Caspi, Robins, Moffitt, and Stouthamer-Loeber (1994) found higher negative reactivity and lower conscientiousness, a personality trait similar to EC (Evans & Rothbart, 2007; Nigg, 2006), in a group with internalizing problems compared to a control group.

Eisenberg et al. (2001) found similar results in a study exploring the association between temperament and problem behaviors in children. The authors reported both increased negative reactivity and lower attentional control, a central component of EC, in an internalizing problem group compared to a group without problems. At a two-year follow up for the sample, Eisenberg et al. (2005) again found higher negative reactivity in the internalizing problem group compared to a control group. However, despite finding lower EC in children with internalizing disorders at the initial assessment, this group no longer showed lower levels of attentional control than children with no psychological problems at the follow up assessment. However, using structural equation modeling with the same follow up data, Eisenberg et al. (2004) found that EC indirectly predicted internalizing problems and that the association was mediated by resiliency, a personality trait similar to EC that describes how an individual responds and adapts to stress.

Differences between groups with and without psychological problems in levels of both reactive and effortful temperament are consistent with an association between EC and psychological problems independent of reactive temperament. Regression analyses showing that reactive and effortful temperament account for unique variability in psychological symptoms have also shown support for this independent association. In study of 600 students from 4th to 11th grade, Lonigan, Phillips, and Hooe (1999 in Lonigan et al., 2004) found that negative reactivity and EC were each associated with anxious and depressive symptoms. In line with the tripartite model of anxiety and depression, the authors also found that positive reactivity was associated with depressive problems but not anxiety problems. In a similar test, Loukas and Robinson (2004) also found that EC was related to depressive symptoms. Although their findings offer support for an association between EC and depressive symptoms, it is important to note that reactive temperament variables were not considered in the regression equation. Thus, the results do not address the association between EC and depressive symptoms distinct from the influence of reactive temperament.

Contrary to expectations, at a one-year follow up assessment of a portion of the sample used by Loukas and Robinson (2004), Loukas and Roalson (2006) found that EC at the initial assessment did not predict depressive symptoms at the second assessment when controlling for initial levels of depressive symptoms. Similarly, Verstraeten et al. (2009) found that EC was associated with depressive symptoms in a cross-sectional assessment, but was predictive at only a trend level in a longitudinal analysis when controlling for symptoms measured at the initial assessment one year earlier.

In contrast to these unexpected longitudinal results, 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. A measure of lack of control, a self-regulation construct similar to EC including inability to control impulsive expression and lack of persistence in solving problems, was included along with reactive measures of approach and sluggishness. Similar to positive reactivity, approach reflected willingness and eagerness to explore new situations and similar to negative reactivity, sluggishness indicated passivity, withdrawal from novel stimuli, fearfulness, and shyness. The results revealed that 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. Reactive measures also predicted later symptoms in accordance with expectations. Boys with high approach in early childhood were less likely to suffer from anxiety or distress problems later on and girls with high sluggishness were more likely to develop problems with anxiety and distress, especially during adolescence. Additionally, 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, even when controlling for symptoms at the initial assessment. It is difficult to interpret these mixed results, but longitudinal evidence at least partially supports theoretical links between EC and depressive symptoms. Future studies with large enough samples to detect what may be a small effect size are necessary to adequately determine the predictive effect of EC for depression across time.

Expanding on the association between EC and psychological problems, several authors have introduced models proposing that high levels of EC can have a protective effect against psychological problems. The models suggest that individuals with reactive temperaments associated with psychological problems may not experience symptoms if they are high in EC because they are able to control or compensate for their reactive tendencies (Derryberry & Rothbart, 1997; Lonigan & Phillips, 2001; Lonigan et al., 2004; Nigg, 2006). In terms of the tripartite model, for example, this suggests an individual who is predisposed to depression due to high negative reactivity and low positive reactivity may experience fewer or no depressive symptoms if EC is high. The protective model highlights the importance of including EC in conceptualizations of the

link between temperament and psychological problems because levels of positive and negative reactivity alone may not be sufficient to predict psychological symptoms.

In addition to finding unique associations between psychological problems and both EC and reactive temperament, multiple studies have found support for a protective model of EC by showing that EC moderates the association between reactive temperament and psychological problems. In a study including an examination of the association between temperament and depressive symptoms in 304 7th through 10th graders in Belgium, Verstraeten et al. (2009) found that negative reactivity, positive reactivity, and EC were each independently related to depressive symptoms. However, as previously mentioned, EC did not predict depressive symptoms one year later when controlling for initial levels of depressive symptoms. Negative reactivity also did not predict symptoms in the longitudinal analysis, although positive reactivity did. In addition to individual associations between temperament and depressive symptoms, the authors found that EC moderated the association between negative reactivity and depressive symptoms. There was also an interaction between positive reactivity and EC, although only for girls. That is, adolescents with high negative reactivity were most likely to report depressive symptoms when EC was low, as were girls with low positive reactivity. Additionally, Vasey et al. (2002 in Lonigan et al., 2004) also found that EC and reactive temperament had unique associations with symptoms of depression and anxiety and that EC moderated relationships between reactivity and symptoms in a sample of approximately 200 adolescents. The results showed interactions both between negative reactivity and EC and between positive reactivity and EC for depressive symptoms. Consistent with expectations, the study also found a significant interaction

between negative reactivity and EC for anxious symptoms, but not between positive reactivity and EC. These moderating effects support a protective model of EC.

Similar studies have also found a moderating effect of EC on the association between negative reactivity and internalizing problems. In a sample of Dutch adolescents, Muris (2006) found unique associations with levels of emotional problems for both negative reactivity and EC. The results also showed an interaction between negative reactivity and EC consistent with the protective model of EC. Similarly, Muris, Meesters, and Blijlevens (2007) also found that both negative reactivity and effortful control accounted for unique variability in internalizing problems. The results also showed a moderating effect of EC on the associations between negative reactivity and symptoms of anxiety and depression in a sample of about 200 Dutch children age 9 to 13.

In study of almost 2,000 Dutch adolescents, Oldehinkel et al. (2007) found that both negative reactivity and EC predicted internalizing problems two to three years later and that EC moderated the longitudinal association between negative reactivity and internalizing problems, albeit with a small effect size ($R^2 < 0.01$). As previously mentioned, EC also predicted internalizing problems after controlling for the initial assessment. However, only one of the two constructs used to measure negative reactivity retained significance and the interaction between EC and negative reactivity was nonsignificant when controlling for initial levels of internalizing problems. Evidence has also shown that some observational measures of EC moderate the association between adjustment and risk factors such as SES, education, maternal depression, so that risk for adjustment problems is higher at low levels of EC (Lengua, 2002). Although research has shown support for a protective model of EC, null results have also been reported for a moderating effect of EC on the association between reactivity and psychological problems. In a study of Dutch children, de Boo and Kolk (2007) found unique associations for negative reactivity, positive reactivity, and EC with depressive mood, but the interactions between negative reactivity and EC and between positive reactivity and EC were not statistically significant. The authors were careful to point out though, that depressive mood was measured as opposed to depressive symptoms as in most other studies reviewed here. The null results may have also been due to the small effect size of the interaction. Among studies that found a significant interaction, R^2 values range from less than 0.01 to 0.06 (Muris, 2006; Oldehinkel et al., 2007). Due to the small effect size, large samples are necessary to obtain the statistical power necessary to obtain significant results (Oldehinkel et al., 2007).

The small effect size may be explained by unselected samples, which are not likely to equally represent the four combinations of high and low negative reactivity and high and low EC. Negative reactivity has been shown to be negatively correlated with EC in both adults (Derryberry & Rothbart, 1988) and children (Ahadi, Rothbart, & Ye, 1993; Rothbart, Ahadi, Hershey, & Fisher, 2001). Thus, an unselected sample would likely over-represent high negative reactivity/low EC and low negative reactivity/high EC groups, and especially under-represent a high negative reactivity/high EC group because they are negatively correlated. As the underrepresented high negative reactivity/high EC group is the focus of the moderating effect, large numbers of participants are needed to achieve the power necessary to detect it. In addition to unequal group distributions, the interaction likely occurs primarily at the tails of the distribution of individual differences in negative reactivity and EC (Lengua & Long, 2002). The relative scarcity of the affected group requires large samples to find a statistical effect because the majority of an unselected sample will be unaffected by an interaction (McClelland & Judd, 1993). In a study of associations between temperament and children's attentional bias, Lonigan and Vasey (2009) selected participants based on extreme scores on the joint distribution of negative reactivity and EC and found a significant interaction. Larger or selected samples are likely necessary for consistent significant interactions.

The few null results from various child and adolescent studies might be influenced by increasing development of EC in children as they age. Unfortunately, the stability of EC in children has not been the focus of a large amount research (Derryberry & Rothbart, 1997). Capacity for voluntary regulation begins to develop during the first year of infancy and increases dramatically during early development (Rothbart & Bates, 2006). EC is reliably measureable at a young age (Ahadi et al., 1993) but it continues to develop until at least age 12 (Murphy, Eisenberg, Fabes, Shepard, & Guthrie, 1999). Several authors have suggested that null results may be due to inadequately developed EC among child participants (Eisenberg et al., 2004, 2007; Muris, de Jong, & Engelen, 2004; Oldehinkel et al., 2007). If fully developed EC can be more reliably measured, reduced measurement error in adolescent and adult samples would increase statistical power.

Despite these suggestions, preliminary empirical tests have not found evidence for differences in the association between EC and psychological problems across development. As previously mentioned, Eisenberg et al. (2001) found that children with internalizing problems had lower attentional control, a central aspect of EC, than a control group. However, at a two year follow up, Eisenberg et al. (2005) found no difference in attentional control between internalizing and control groups. Based on these findings, the authors suggested that attentional control may actually play less of a role in processing negative emotions as children mature. Oldehinkel et al. (2007) did not find a difference in the association between EC and internalizing problems at age 11 and at a follow-up sample 2 to 3 years later. Meesters, Muris, and Van Rooijen (2007) tested whether the association between attentional control and anxious symptoms or the interaction between negative reactivity and attentional control were moderated by age and found null results. Similarly, Lonigan and Vasey (2009) found that age did not moderate an interaction between negative reactivity and EC for anxious symptoms. Although these non-significant tests do not support changes in the effect of EC on psychological symptoms as children age, high power is necessary to detect two- and three-way interactions and the null results do not preclude the possibility of an effect. Tests of an increasingly stable association between EC and psychological problems across development have been cursory to date and the potential effect on tests of the protective model in children remains unclear. Studies of adult populations should be free from this concern and may show improved consistency of positive results.

Research exploring the effects of effortful temperament on psychopathology has been nearly exclusively focused on children, reflecting the developmental origins of the addition of EC to temperament models. Studies of the association between EC and psychological problems have only recently been conducted using adult samples. Dinovo and Vasey (2009) found that EC was associated with measures of both symptoms of general distress and anhedonia in a cross-sectional analysis for a sample of 477 undergraduate students. The authors also found evidence of a prospective association between EC and symptoms across a 6-week interval in a separate sample of 44 undergraduates. Additionally, the results demonstrated support for protective effects of EC both cross-sectionally and prospectively. The evidence was strongest for moderation by EC of the association between negative reactivity and symptoms of both general distress and anhedonia, although this interaction was not found for one measure of negative reactivity in the cross-sectional analysis. Evidence of EC moderating the association between positive reactivity and symptoms was more inconsistent, emerging in the cross-sectional analysis but not the prospective analysis.

Taken together, available evidence supports an association between EC and psychological problems independent of reactive temperament. These findings demonstrate the importance of EC in temperamental models of psychopathology. 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 negative reactivity and emotional problems such as anxiety and depression. Evidence has also supported a moderating effect of EC on the association between positive reactivity and depressive problems. These results show that the association between high negative reactivity, low positive reactivity, and internalizing psychological problems is diminished when EC is high. Although the majority of research has been done with child and adolescent samples, preliminary findings among adult populations suggest a similar pattern. These findings will be strengthened by future replication.

Based on empirical support for a moderating effect of EC on the association between negative reactivity and depressive symptoms, Vasey, Harbaugh, Buffington, Bills, and Dinovo (2008) tested rumination as a potential mechanism of the protective effect of EC. Specifically, the authors tested whether EC moderated how rumination mediated the association between negative reactivity and depressive symptoms. Rumination, the tendency to repetitively dwell on negative emotions, is characteristic of depression and has been linked to increased severity and duration of depressive episodes (Nolen-Hoeksema, 1991, 2000). Using a prospective design with three assessments of 210 undergraduate participants over eight weeks, the results showed that Time 2 (T2) rumination mediated the association between Time 1 (T1) negative reactivity and Time 3 (T3) depressive symptoms when controlling for T1 depressive symptoms. Further, the results showed that this mediation effect was in turn moderated by T1 EC so that the mediation effect of T2 rumination only occurred at low levels of T1 EC. That is, the level of T1 EC determined whether T2 rumination mediated the association between T1 negative reactivity and T3 depressive symptoms. This finding not only supports a moderating effect of EC, but also offers a preliminary direction for identifying a potential mechanism of this effect.

Verstraeten et al. (2009) also tested whether EC moderates how rumination mediates the association between negative reactivity and depressive symptoms. In crosssectional data, rumination mediated the association between negative reactivity and depressive symptoms. Consistent with expectations, the mediation effect was moderated by EC so that mediation by rumination did not occur when EC was high. The authors also found evidence of moderated mediation in a prospective design including measures of depressive symptoms one year later and controlling for initial symptoms, although with a different pattern. In the cross-sectional data, EC moderated the path between EC and rumination, while in the prospective data EC moderated the path between rumination and depressive symptoms. More research is needed to determine how rumination relates to the protective effect of EC, but evidence from both cross-sectional and longitudinal analyses suggests an important role.

Three Facets of Effortful Control

There is a large body of evidence supporting an association between EC and psychological problems, but results have not been entirely consistent. Research has shown that EC is higher in groups without psychological problems and that EC is uniquely related to anxiety and depression problems independent of positive and negative reactivity in cross-sectional analyses. The results from longitudinal studies though, are less conclusive. Some studies found null or only trend level main effects for EC over time when controlling for initial levels of psychological problems. Evidence for a protective effect of EC has also been somewhat mixed. Although multiple studies have found that EC moderates the association between reactivity and psychological problems, other studies have reported null results.

Distinguishing between distinct facets of EC may improve our understanding of the associations between reactive temperamental factors, EC, and psychological disorders. According to Rothbart and colleagues, EC can be further divided into components of attentional control, inhibitory control, and activation control in adults and adolescents (Evans & Rothbart, 2007; Putnam, Ellis, & Rothbart, 2001). The division of EC into these facets reflects a distinction between control of internal cognitive processes and control of behavior. These facets likely have distinct roles in their associations with various psychological problems. Consideration of EC as a single broad construct has been partially successful in the past, but it is potentially limiting. Exploration of the associations between individual facets of EC and psychological disorders may provide a better understanding of the mechanisms of EC, potentially improve identification of risk factors, and ultimately inform advances in psychological therapy and prevention.

Attentional control is the capacity to flexibly focus and shift attention between stimuli as well as to focus thought and resist distraction. The capacity to shift between mental concepts can shape cognitive activity by enhancing or minimizing focus on positive and negative thoughts. Attentional control is an important coping mechanism and is thought to play a key role in controlling emotion (Derryberry & Reed, 1996, 2002). In addition to control of internal processes, EC also includes components of deliberate management of behavior. Inhibitory control is the capacity to intentionally suppress 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. However, despite the behavioral focus of inhibitory control in this context, there is some evidence that cognitive and behavioral inhibition are not be separable (Aron, 2007) suggesting cognitive inhibition may also be strongly associated with behavioral inhibitory control. It is also important to distinguish inhibitory control from inhibition due to negative reactivity, such as suppression of responding due to fear. 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

temperament may cause suppression of desired behavior as well as undesired behavior (Derryberry & Rothbart, 1997). Activation control is the capacity to perform an action despite motivation not to act (Evans & Rothbart, 2007; Putnam et al., 2001). 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.

The division of EC into facets of attentional control, inhibitory control, and activation control captures the flexible control of both internal cognitive processes and behavior included in the broad theoretical conceptualization of EC. 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 et al., 1993; Evans & Rothbart, 2007; Putnam et al., 2001; Rothbart, Ahadi, & Evans, 2000; Rothbart et al., 2001).

Attentional control. Attentional control is the capacity to focus and shift attention to and from positive or negative stimuli. It is the basis for EC because voluntary deployment of attention enables individuals to control reactive tendencies and optimally manage behavior (Rothbart & Rueda, 2005). It is also the most commonly researched facet of EC. In fact, several studies of EC have used measures of attentional control in place of EC measures (Meesters et al., 2007; Muris et al., 2004; Muris, Mayer, van Lint, & Hofman, 2008; Muris, Meesters, & Rompelberg, 2006) or operationalized EC as attentional control (Vasey et al., 2002 in Lonigan et al., 2004). Finally, 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 significantly in one factor analysis with a child sample (Ahadi et al., 1993).

Attentional control is likely to be especially important as a protective factor for anxiety and depression. High negative reactivity is a likely vulnerability for both disorders and attentional control is thought to enable individuals to regulate negative emotion by limiting attention toward negative thoughts or stimuli (Derryberry & Reed, 1994, 1996, 2002; Eisenberg, Fabes, Guthrie, & Reiser, 2000; Posner & Rothbart, 2000). Analyses of self-report measures, attentional task studies, and neural imaging data all largely support theoretical expectations that attentional control has a protective effect against psychological disorders such as anxiety and depression. However, it is important to note that much of this research is preliminary and further exploration is necessary.

As with EC, an association between attentional control and psychological problems has been found in multiple studies. Research has found that attentional control is negatively correlated with symptoms of anxiety and depression (Healy & Kulig, 2006; Lengua, West, & Sandler, 1998; Muris et al., 2008; Muris et al., 2006) and that children with internalizing problems demonstrate lower attentional control than non-disordered children (Eisenberg et al., 2001). However, as previously mentioned, Eisenberg et al. (2005) found that children with internalizing problems no longer demonstrated lower attentional control than non-disordered children as they had at an initial assessment two years earlier (Eisenberg et al., 2001).

Attentional control has also been shown to be uniquely related to symptoms of anxiety and depression independent of reactive temperament. Muris et al. (2007) found that subscale measures of negative reactivity and attentional control all accounted for unique variability in internalizing problems in a sample of 200 children. Attentional control has also been shown to moderate the association between temperament and psychological disorders, although not without exception. Vasey et al. (2002 in Lonigan et al., 2004) found that negative reactivity and attentional control were each uniquely related to anxiety problems. Consistent with expectations of a protective effect of attentional control against anxiety, the data also showed an interaction between negative reactivity and attentional control. Additionally, the authors found that positive reactivity, negative reactivity, and attentional control were all uniquely associated with depressive symptoms. There was also a moderating effect of attentional control on the association both between negative reactivity and depressive symptoms, and between positive reactivity and symptoms. In a sample of 400 children and adolescents, Meesters et al. (2007) also found that both negative reactivity and attentional control were uniquely associated with anxiety problems. The results also showed the expected interaction between negative reactivity and attentional control. Muris et al. (2004) similarly found unique associations between anxiety problems and both negative reactivity and attentional control in a sample of 300 children and early adolescents. However, the data did not support the expected interaction between negative reactivity and attentional control.

Analyses of self-report data have shown general support for a protective effect against anxiety and depression, but additional research is still necessary. Research to date has been entirely cross-sectional and attentional control has not been clearly distinguished from inhibitory control and activation control. Unless all three facets of EC are concurrently measured and considered, associations between attentional control and psychological problems cannot be assumed to be independent from the other facets of EC.

Research using performance tasks to measure attentional bias also provides support for a protective effect of attentional control that moderates the association between negative reactivity and anxiety. Multiple reviews have presented extensive empirical support for a robust attentional bias toward threat in anxious individuals in both adults (e.g., Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007; Mathews & MacLeod, 1994; Mogg & Bradley, 1998, 2005) and children (Vasey & MacLeod, 2001). The link between anxiety and attention suggests that effortful control of a reactive attentional bias toward threat may reduce the emergence of anxious symptoms.

The emotional Stroop task is a widely used measure of cognitive inhibition in which participants are asked to name the text color of a neutral or emotional word. Studies have consistently found a larger increase in response time between negative and neutral words among individuals with a range of anxious disorders and symptoms than for control groups (for a review, see Williams, Mathews, & MacLeod, 1996). The slowed response is regularly interpreted as a measure of enhanced attention toward the negative word causing a disruption in cognitive processing. This interpretation supports a negative attentional bias, but it is unclear that the interference is caused solely by attention and is not affected by cognitive processing (MacLeod, 1991, 2005).
Findings using a visual probe task have further indicated that attentional bias toward threat is a feature of anxiety. The visual probe task is a more direct measure of attention than the emotional Stroop task. The task begins with a simultaneous presentation of neutral and negative stimuli on a computer screen followed by a target dot in the location of one of the stimuli. Variation in reaction times for identifying targets in different locations is an indicator of visual attention to the initial stimuli because participants can react faster to targets that appear in the area they are attending to faster than targets that appear in other areas (Navon & Margalit, 1983; Posner, Snyder, & Davidson, 1980). Studies have found that anxious individuals demonstrate a faster response time when the target dot appears in the position previously occupied by a negative or threatening word than non-anxious individuals, indicating that anxious individuals were more focused on negative cues than neutral cues. This effect has been shown in clinical samples of generalized anxiety disorder (MacLeod, Mathews, & Tata, 1986; Mathews, Ridgeway, & Williamson, 1996; Mogg, Bradley, & Williams, 1995; Mogg, Mathews, & Eysenck, 1992) as well as nonclinical samples (Broadbent & Broadbent, 1988; Fox, 1993; MacLeod & Mathews, 1988; Mogg, Bradley, & Hallowell, 1994). The same effect is observed when threatening facial images are used as cues instead of words (Bradley, Mogg, Falla, & Hamilton, 1998; Bradley, Mogg, & Millar, 2000; Mogg & Bradley, 1999) although not at longer cue presentation intervals of 1000 ms (Gotlib, Krasnoperova, Yue, & Joormann, 2004; Mogg, Millar, & Bradley, 2000).

Based on findings demonstrating a link between a negative attentional bias and anxiety, several authors have suggested that individuals may be able to override a reactive bias toward threat (MacLeod & Hagan, 1992; Mathews & Mackintosh, 1998; Mathews & MacLeod, 2005; Mogg, Kentish, & Bradley, 1993). Additionally, in a metaanalysis of studies exploring threat bias in anxiety, Bar-Haim et al. (2007) reported that subliminal trials of visual probe tasks had a larger effect size than supraliminal trials. Lonigan and Vasey (2009) noted that the lower effect size in supraliminal trials may be due to the capacity for some individuals to effortfully override a negative bias when given sufficient time.

Consistent with these suggestions from the attention bias literature, research measuring attentional control has shown that individuals with high attentional control can indeed reduce or eliminate the expected attentional bias associated with anxiety. Derryberry and Reed (2002) compared performance on a visual probe task between adult groups with high and low trait anxiety, and high and low attentional control. The authors measured attentional control with the Attentional Control Scale (ACS), a self-report questionnaire developed by the authors. Their results confirmed that the anxious group demonstrated a bias toward threat cues when there was a short delay between the cue and the target. However, at a longer interval, the bias in the anxious group was moderated by attentional control so that among anxious participants, those with low attentional control continued to display the bias, but those with high attentional control did not. The authors concluded that given sufficient time, high anxious individuals with high attentional control can reduce the reactive bias toward threat. In a similar study, Lonigan and Vasey (2009) also found that EC moderated the association between negative reactivity and attentional bias using a measure of EC similar to the ACS that emphasized low distractibility and persistence. The study used a visual probe task with an extended cue presentation to allow for sufficient time to permit effortful control of attention.

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Participants in the study were selected to represent the extremes of combinations of high and low negative reactivity, and high and low EC. As expected, high negative reactivity was related to increased attentional bias toward threat words, but only for participants with high negative reactivity and low EC. Participants with high negative reactivity and high EC did not demonstrate a negative attentional bias.

Neurologic research has identified several brain areas that appear to be associated with attentional control and found that activation in these areas are negatively correlated with anxiety. Imaging studies indicate increased brain activity in areas of the frontal lobe during tasks requiring active attention. Additionally, patients with frontal lobe injuries often demonstrate reduced capacity for attentional control (Posner & Raichle, 1994). Using non-clinical samples, Bishop and colleagues demonstrated that neural activity in brain areas thought to be part of the effortful attention system correlated negatively with anxiety and positively with attentional control (S. Bishop, Duncan, Brett, & Lawrence, 2004; S. J. Bishop, Jenkins, & Lawrence, 2007). Mathews, Yiend, and Lawrence (2004) found a similar pattern for attentional control as well as a negative correlation between inhibition of behavior and neural activity in areas associated with attentional control during a threat attention task.

Like anxiety, research exploring attentional biases in depression also indicates attentional patterns typical of the disorder, suggesting attentional control may have a moderating effect on the association between negative reactivity and depressive symptoms. This protective effect against depressive problems is likely linked to rumination, a major aspect of depression. A low capacity for attentional control would make it difficult to prevent the intrusion of ruminative thoughts or to shift attention away from negative thoughts. Indeed, as previously discussed, Vasey, Harbaugh et al. (2008) and Verstraeten et al. (2009) found evidence that rumination mediates the association between negative reactivity and depressive symptoms and that EC in turn moderates the meditational effect. Depressed individuals are more likely to focus attention on negative emotional information and also have difficulty disengaging from it. This pattern of attentional bias is especially conducive to the repetitive negative thinking characteristic of rumination (Carver, Johnson, & Joormann, 2008). Attentional control of cognitive thought is the facet of EC most likely to be related to rumination and depression.

Research using performance tasks to measure attention suggests a negative attentional bias associated with depression consistent with a tendency toward negative, ruminative thoughts. However, evidence indicates a different pattern of attentional bias for depression than for anxiety. Unlike anxiety, no study of an emotional Stroop or visual probe task using subliminal presentation lengths has found an attentional bias toward threat in a clinically depressed sample (Bradley, Mogg, Millar, & White, 1995; Mathews et al., 1996; Mogg et al., 1995; Mogg, Bradley, Williams, & Mathews, 1993). Studies using supraliminal cues have found generally mixed evidence for an attentional bias, but results are most reliable in studies using longer cue presentations (Mogg & Bradley, 1998, 2005). Consistent with this pattern, Mogg et al. (1995) found a significant attentional bias in a clinical sample of depressed participants compared to normal controls for a 1000 ms cue presentation in a visual probe task, but not in a subliminal presentation condition. The authors did not control for comorbid anxiety disorders in the depressed group. Gotlib and colleagues (Gotlib, Kasch et al., 2004; Gotlib, Krasnoperova et al., 2004) replicated this result with a 1000 ms cue presentation among depressed

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individuals using sad faces instead of negative words as cues. Further, Donaldson, Lam, and Mathews (2007) also found a negative bias for a 1000 ms exposure condition using word cues in a clinical sample of depressed participants without comorbid anxiety disorders, but did not find a bias in a 500 ms condition.

Although findings of an attentional bias in depression using visual probe tasks with longer presentations are more reliable than shorter presentation lengths, they are not without exceptions. Two studies of performance on visual probe tasks in clinical samples of mixed anxiety and depression or depression did not find an attentional bias toward negative words compared to a control group despite a presentation time of 1500 ms, although the study used an adolescent sample (Neshat-Doost, Moradi, Taghavi, Yule, & Dalgleish, 2000; Taghavi, Neshat-Doost, Moradi, Yule, & Dalgleish, 1999). Mogg et al. (2000) also found null results for a negative attentional bias in a visual probe task with a presentation of 1000 ms, although 13 of 15 participants in the depressed sample also met criteria for generalized anxiety disorder. In a test of the difference in attentional bias between anxiety and depression, the previously discussed study by Gotlib, Krasnoperova et al. (2004) found a bias toward negative faces using a 1000 ms presentation in a group of depressed participants without comorbid generalized anxiety disorder but not in an anxious group without comorbid depressive disorders. As future studies further explore attentional bias in depression, the nature of the bias will become clearer. To date, available evidence seems to generally support a negative attentional bias in depression for longer interval presentations.

In addition to support for a negative attentional bias, emerging evidence indicates that depression is also characterized by deficits in cognitive inhibitory control of negative emotional information. The cognitive nature of this deficit suggests it is closely associated with attentional control, but as cognitive and behavioral inhibition may not be separable (Aron, 2007), it may also be associated with behavioral inhibitory control.

Cognitive inhibitory control may cause a vulnerability to depression through increased processing of negative information and rumination, which could lead to prolonged periods of negative affect typical of depressive episodes (Joormann, 2005; Joormann, Yoon, & Zetsche, 2007). Linville (1996) first proposed that cognitive inhibitory deficits may be a major cause of ruminative thought and presented data from a negative priming task showing deficits in the ability to inhibit distracter cues in a depressed group. In the task, participants were asked to respond to a target while ignoring a distracter stimulus presented simultaneously. For most people, reaction time increases in a following trial when the target is identical to the distracter from the previous trial. This delay is thought to be due to residual inhibition of the distracter in the first trial. The increase in response time from the first trial to the second is used as an indication of the strength of inhibition of the original distracter.

Joormann (2004) extended these findings by testing differences in inhibitory deficits between negative and positive distracters. The results showed low inhibition of negative, but not positive, distracters in a dysphoric group and a group with a history of depressive episodes. Frings, Wentura, and Holtz (2007) found similar results in a sample of undergraduate students separated by a median level of depressive symptoms. Goeleven, De Raedt, Baert, and Koster (2006) replicated these results in a clinically depressed sample using emotional faces as cues and distracters, although formerly depressed participants exhibited inhibitory deficits for both positive and negative information. Similarly, Joormann (2006) found inhibitory deficits for both positive and negative words in a group of participants with high scores on a measure of rumination compared to a group with low scores even when controlling for current depressive symptoms. These results support a deficit in cognitive inhibition for at least negative information consistent with an increased vulnerability to rumination and depression.

Similar to results from studies using the negative affective priming tasks, Joormann and Gotlib (2008) found that depressed individuals exhibited higher interference from negative information than a control group. Participants were instructed to memorize two lists of words presented simultaneously. Then, a cue identified one list as relevant for a subsequent recognition task. In the recognition task, participants were presented with words and asked to identify whether or not the words were from the relevant list. Words from the non-relevant list as well as new words were used as distracters. Consistent with previous findings demonstrating a deficit in cognitive inhibition for negative words, the depressed group demonstrated greater response time delays to negative words, but not positive words, from the non-relevant list. That is, the depressed group had more difficulty inhibiting negative words from the non-relevant list than the control group in the recognition task.

Research has also shown support for prolonged attention toward negative information in depressed individuals. This tendency is also consistent with links between attentional control and rumination as it may cause difficulty shifting attention away from negative cognitions. Recent research confirms Bradley, Mogg, and Lee's (1997) suggestion that depressed individuals may have difficulty disengaging from negative information. Using eye-movement monitoring to track attentional focus, Eizenman et al. (2003) presented participants with groups of images with dysphoric, threatening, social, and neutral themes. Each presentation had one image from each category. Results showed that the depressed group spent more time attending to the dysphoric images than a control group. There were no group differences for the other types of images. Similarly, Caseras, Garner, Bradley, and Mogg (2007) used eye-movement monitoring to compare time spent attending to negative and neutral images presented in pairs in a sample separated into high and low depressive symptoms groups. Consistent with expectations, the high symptom group demonstrated a greater attentional bias toward negative images than the low symptom group. There was no difference between the groups in time spent looking at positive images in pairs of positive and neutral images.

In a recent imaging study, depressed participants demonstrated prolonged activation of the amygdala, an area thought to be responsible for identifying emotional aspects of information, compared to a control group when presented with negative words. Sustained processing of negative information was also correlated with self-report levels of rumination (Siegle, Steinhauer, Thase, Stenger, & Carter, 2002). These imaging data offer further support to indications of prolonged cognitive processing of negative information in depression.

Inhibitory control. Inhibitory control is the capacity to purposefully restrain behavior. Deliberate control of behavior is a theoretically important factor of EC, but measures of inhibitory control have not consistently performed as expected. In factor analyses, it 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 for adults has demonstrated a low internal reliability of $\alpha = 0.66$ (Evans & Rothbart, 2007).

Inhibition is a commonly used term that can have several meanings. In an effort to clarify the various definitions, Nigg (2000) developed a taxonomy of inhibition including a separation between effortful and reactive inhibition as well as between internal and external types of inhibition. Internal inhibition is the capacity to suppress processing of distracting stimuli and aversive thoughts and external inhibition is the capacity to suppress behavioral reactions. Internal inhibition processes are thought to be an important precursor to behavioral control (Barkley, 1997), but they appear to be most similar to attentional control in terms of the facets of EC. However, it is not entirely clear how cognitive inhibition fits into this framework. Inhibitory control as a facet of EC refers to effortful suppression of external behavior and does not include reactive processes or inhibition of internal processes such as cognition or attention. Despite these theoretical distinctions, there is evidence suggesting that areas of the brain responsible for motor control are also involved in cognitive tasks traditionally used to test cognitive inhibition (Aron, 2007).

Inhibitory control of behavior is not likely to be an important factor for anxiety or depression. High reactive control is common to emotional disorders. 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 nondisordered children in inhibitory control. Additionally, internalizing disorders are characterized by high reactive control (Eisenberg et al., 2001; Eisenberg et al., 2005).

Even if behavioral inhibition is not theoretically an important factor itself, if inhibitory control of behavior and cognition are not separable as suggested by Aron (2007), measures of inhibitory control of behavior may account for aspects of cognitive control not otherwise accounted for by attentional control. For example, behavioral inhibition items might be able to account for variability in inhibitory control of attention as studied by Joormann and colleagues better than attentional control items. Indeed, in a recent study of adolescents, Raes, Verstraeten, Bijttebier, Vasey, & Dagleish (in press) used inhibitory control as a measure of executive function in relation to depressive symptoms. The results showed that inhibitory control partially mediated the association between overgeneral memory recall of autobiographical memories and depressive symptoms. Inhibitory control was measured as the score of the inhibitory control subscale score of the Revised Early Adolescent Temperament Questionnaire (EATQ-R; Ellis & Rothbart, 2001; Putnam et al., 2001), which was developed by the same group who created the Adult Temperament Questionnaire (ATQ; Evans & Rothbart, 2007; Rothbart et al., 2000) used in the present study.

Activation control. Activation control is the capacity to perform an action despite motivation not to act or in the absence of immediate reward. Just as suppression of unwanted behavior is important to proper adjustment, so is activation of behavior when there is a natural tendency not to act. 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 the least attention in the literature of the three facets of EC, although reliable measures for activation control have not been available for as long as for the other facets. The first questionnaire measure of activation control failed to show adequate internal reliability ($\alpha = .51$; Derryberry & Rothbart, 1988). 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). Although published measures are available, they are designed for adolescents and adults and most EC research uses child samples, so activation control continues to receive the least consideration of the three facets.

Activation control is likely important to regulation of avoidance in depression. Depressed individuals often engage in avoidance behaviors to escape from potential stressors. Avoidance can include reduced socialization, low attendance at work, or remaining in bed throughout the day. In addition to causing life problems associated with failing to meet obligations, overly applied avoidance can filter out positive, antidepressant stimuli in the environment (Jacobson, Martell, & Dimidjian, 2001). Behavioral avoidance and anhedonia are marks of depression related to low positive reactivity (Clark & Watson, 1991; Mineka et al., 1998). The association between low positive reactivity, low activity or anhedonia, and depressive symptoms suggests that a potential protective effect of activation control may operate by moderating the association between positive reactivity and depression. That is, individuals with low positive reactivity and a tendency toward low activity may not experience depressive symptoms if they are also high in activation control and continue to engage with the environment despite reactive tendencies. An extension of the definition of activation

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control to include action in the absence of immediate rewards offers further theoretical support for a protective effect against general malaise associated with depression.

Based on these considerations, Behavioral Activation treatment was designed as a psychological treatment to help depressed individuals suppress their avoidant tendencies, increase contact with their environment, and eventually find natural reinforcement to maintain normal levels of activity (Ferster, 1973; Jacobson et al., 2001). A high capacity for activation control may have a protective effect against depressive symptoms by naturally facilitating the same behaviors identified by Behavioral Activation treatment in spite of low positive reactivity.

Avoidance is also a characteristic of several other anxious disorders. For example, avoidance of social situations is an element of social phobia, which like depression, is associated with low positive reactivity (Brown et al., 1998; Mineka et al., 1998; Watson, Clark, & Carey, 1988). Behavioral avoidance is also an aspect of general negative reactivity (Watson et al., 2005), which as previously discussed, is associated with a litany of psychological disorders. Specifically, agoraphobia, post traumatic stress disorder, and specific phobias also include elements of behavioral avoidance (American Psychiatric Association, 2000), suggesting activation control may associated with symptoms of these disorders, too.

Few studies of have considered positive reactivity in relation to internalizing disorders, but the available evidence is encouraging. As previously discussed, Verstraeten et al. (2009) found a moderating effect of EC on the association between positive reactivity and depressive symptoms, although only for girls. Similarly, Vasey et al. (2002 in Lonigan et al., 2004) found the same interaction operationalizing EC as attentional

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control (although see de Boo & Kolk, 2007). Based on association between low activity and positive reactivity, activation control is the facet of EC most likely responsible for the observed moderation of the association between positive reactivity and depressive symptoms by EC. Consistent with this logic, activation control has been shown to negatively correlate with depressive symptoms (Moriya & Tanno, 2008).

A recent initial investigation of the protective effects of individual facets of EC against depressive symptoms found support for expectations that individual facets of EC moderate different links between reactive temperament and depressive symptoms (Vasey, Buffington, & Dinovo, 2008). In a reanalysis of data collected by Dinovo and Vasey (2009), attentional focusing and persistence scales were developed using exploratory and confirmatory factor analyses of items on the ACS (Derryberry & Reed, 2002) and the persistence/low distractibility subscale of the Effortful Control Scale (ECS; Lonigan, 1998 & Lonigan, Phillips, & Hooe, 1999 in Lonigan et al., 2004). Although specific measures of attentional control and activation control were included in the original data set, the attentional focusing and persistence scales were used as alternatives as they are theoretically similar constructs. The authors found unique associations with depressive symptoms for negative reactivity, positive reactivity, and attentional focusing, although surprisingly not for persistence. As predicted, attentional focusing, but not persistence, moderated the association between negative reactivity and depressive symptoms. That is, high levels of negative reactivity were more strongly associated with depressive symptoms among individuals reporting low versus high capacity for attentional control. Similarly, as predicted persistence moderated the association between positive reactivity and depressive symptoms, but attentional focusing did not. The different moderating

effects of attentional focusing and persistence are a preliminary indication of the importance of considering individual facets of EC in the association between EC, reactive temperament, and depressive symptoms.

The Present Study

The present study was designed to extend current knowledge about the association between reactive temperament, EC, and symptoms of psychological problems by analyzing EC as the three individual facets, including attentional control, inhibitory control, and activation control. General distress symptoms common to anxiety and depression and symptoms unique to depression were considered. The study was designed to test the protective effects of the different facets of EC against high negative reactivity for symptoms of both general distress and depression. Based on the association between low positive reactivity and depression in the tripartite model, the study also considered the protective effect of the facets of EC against low positive reactivity for depressive symptoms. Due to past mixed findings for the moderating effect of EC and its small effect size, the current study utilized a large sample of over one thousand participants. Differentiating EC into more specific facets was expected improve the ability to find effects instead of using a broad measure of EC. Finally, the study was designed to test whether current information about EC derived from child studies generalize to adult populations as expected by using a sample of undergraduate students.

I hypothesized that negative reactivity and attentional control would account for unique variability in general distress such that high negative reactivity and low attentional control would be associated with increases in symptoms of general distress. I also predicted that the protective model of EC would be supported by a moderation effect. Specifically, I expected that attentional control would moderate the association between negative reactivity and general distress in line with previous research as well as theoretical links between attention and emotion regulation.

I also hypothesized that negative reactivity, positive reactivity, and the facets of EC would all account for unique variability in depressive symptoms. Specifically, high negative reactivity, low positive reactivity, and low attentional control and activation control were all expected to be associated with higher levels of depressive symptoms. I also predicted that the protective model of EC would be supported by a moderation effect but by different facets for different reactive temperaments. I expected that attentional control would moderate the association between negative reactivity and depressive symptoms based on past research findings and the role of attention in regulating negative emotions. I also expected activation control to moderate the association between positive reactivity and depressive symptoms as low activity and anhedonia are characteristic of low positive reactivity.

Chapter 2: Methods

Participants

1277 undergraduate students enrolled in an introductory psychology class completed a series of self-report measures. Students were recruited through announcements posted to an online register of research participation options to fulfill a course requirement. Participants in this unselected sample were not prescreened or selected based on any individual characteristics. Response packets with 25% or more missing items were assumed to represent unreliable responding and were dropped from the analysis. The remaining 1242 participants who were included in the analysis were between 18 and 52 years-old with a mean age of 19.1. The sample was 55.3% female. *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 on a 5-point Likert scale where 1 is "very slightly" and 5 is "extremely." The scales have shown high internal reliability and test-retest reliability with undergraduates. Each scale also correlates highly with other similar measures, they are not highly correlated with each other (Watson, Clark, & Tellegen, 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 items are rated on a 4-point Likert scale with 1 indicating "strongly agree" and 4 indicating "strongly disagree." The scale includes 7 BIS items as well as 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).

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. Respondents are instructed to rate how well each statement describes them on a 7-point Likert scale where 1 is "extremely untrue" and 7 is "extremely true." Although the attentional control and activation control subscales have shown good internal reliability and load on an expected EC factor in factor analyses of the items, the internal consistency of the inhibitory control subscale is somewhat low ($\alpha = .66$) and the items have not consistently loaded exclusively an EC factor (Evans & Rothbart, 2007).

Effortful Control Scale (ECS). The ECS (Lonigan, 1998 & Lonigan, Phillips, & Hooe, 1999 in Lonigan et al., 2004) is a 24-item self-report measure designed to measure

EC. The items are scored on a 5-point Likert scale where 1 is "not at all" and 5 is "very much" based on how much each statement describes the respondent. The measure includes a persistence/low distractibility subscale similar to the activation control subscale of the ATQ as well as an impulsivity subscale. The ECS was originally developed for school-aged children, including adolescents, and although many of the items addressing schoolwork may not be appropriate for some adult samples, they are suitable for the undergraduate sample in this study.

Attentional Control Scale (ACS). The ACS (Derryberry & Reed, 2002) is a standalone 20-item self-report measure of attentional control with high internal reliability. Respondents are instructed to indicate how often each item is true for them on a 4-point Likert scale where 1 is "almost never" and 4 is "always." The measure was developed by colleagues of Rothbart and the items are very similar to the attentional control subscale of the ATQ.

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 using 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 on a Likert scale ranging from 0 to 3 with increasing applicability to their emotions. 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).

The DASS anxiety subscale is made up of items measuring acute, physiological fear responses to anxiety. The DASS stress subscale consists of items measuring chronic non-specific arousal measuring what Lovibond and Lovibond (1995) defined as a coherent set of symptoms distinct from anxiety and depression based on a factor analysis. Multiple factor analyses have confirmed a three factor model of DASS items (Antony et al., 1998; Brown et al., 1997; Clara et al., 2001; Crawford & Henry, 2003; Norton, 2007), but evidence also suggests that the stress subscale measures symptoms common to anxiety and depression consistent with the common factor of the tripartite model (Tully, Zajac, & Venning, 2009 although see Henry & Crawford, 2005). The stress subscale items include symptoms common to generalized anxiety and depression such as consistent arousal, nonspecific distress, and a tendency to easily become upset or frustrated. In clinical samples, participants with both mood and general anxiety disorders score higher on the stress subscale than other clinical groups, while participants with

other anxiety disorders such as panic disorder scored highest on the anxiety subscale. Consistent with the tripartite model, these clinical responses patterns suggest that the stress subscale measures a cluster of symptoms common to high negative reactivity and the anxiety subscale measures symptoms of physiological hyperarousal (Antony et al., 1998; Brown et al., 1997). Also consistent with conceptualization of the stress subscale as a measure of general distress, negative reactivity has been shown to have a stronger association with the stress subscale than to the anxiety or depression subscales (Crawford & Henry, 2003; Norton, 2007).

Procedure

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 questionnaires in the battery was included in the present study to measure positive and negative reactivity, the three facets of EC including attentional control, inhibitory control, and activation control, and symptoms of general distress and depression. 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 high for both inhibitory control (r = .97) and activation control (r = .98). Therefore the unintentionally shortened scales were used for the analyses.

Missing Data

There were 260 participants with at least one missing item among the measures included in the present study. A complete case analysis (i.e., casewise or listwise

deletion) would have resulted in excluding 20.9% of the sample, which would have created an unacceptable loss of power. Instead, a multiple imputation (MI) procedure was used to compensate for missing data (Allison, 2002; Graham, 2009; Rubin, 1987). MI estimates values for missing data based on the associations between all of the variables included in the imputation model. Variation is added to each imputed value to correct for biased variance estimates. Missing value estimates derived from a statistical model underestimate variance because they do not account for random error associated with a random sample. Adding random variation to the each imputed value adjusts for this bias. Each missing value is estimated multiple times to create multiple full data sets comprised of observed data and imputed values. In addition to variation in the amount of error added to different estimates of a given data point, variation is also added to the model parameters used to estimate each set of imputed values. Although the imputed values in each data set will be slightly different, each data set is an equally probable estimation of the complete data. Using only a single imputation underestimates variability because it is based on the parameters of a given sample, not of the population. Using multiple estimates of missing values helps correct for this bias by creating variability across imputations and improving estimates of standard error. Each data set can be analyzed by a variety of data methods, including linear regression. The results from the analyses on each full data set are then pooled to obtain aggregate estimates of results and standard errors for the analysis.

MI assumes missing data are ignorable. That is, the pattern of missing data should be random or influenced only by variables included in the model. When the pattern of missing data is random, the data are said to be Missing Completely At Random (MCAR). When the pattern of missing data is influenced only by other variables in the data set, the data are said to be Missing At Random (MAR, Schafer & Graham, 2002). Missing data in the present sample may be have been influenced by individual differences in EC that affected participants' decisions to skip items. This is an example of MAR because measurements of EC, which is the potential source of influence, are included in the MI model. No direct test of the MAR assumption is possible because it would require unavailable data, although most data sets likely included some departure from MAR (Schafer & Olsen, 1998). Despite these reservations, failing to consider unforeseen sources of influence on missingness will likely have only a minor impact on results (Collins, Schafer, & Kam, 2001). In the present sample, there were no obvious potential sources of bias in the pattern of missingness that were not included in the study, and the MAR assumption was assumed to be valid.

Due to the number of items and interactions included in the analyses, the MI procedure could not be applied to impute values for individual item scores and so was used to impute total scale means (Graham, 2009; Schafer & Graham, 2002). To best preserve the data collected, the mean of available items was used as the scale mean for scales with at least 50% of non-missing items (Graham, 2009) and a reliability of $\alpha \ge$ 0.70 (Schafer & Graham, 2002). Higher percentages of non-missing items were required for scales with lower alpha levels so that for $\alpha \ge 0.90$, 50% of items were required, for $\alpha \ge 0.80$, 67% were required, and for $\alpha \ge 0.70$, 80% of items were required. 50 participants had one or more scales without the required minimum number of items. Total scores for these scales were estimated using MI.

All of the scale scores used in the various regression analyses included in this study were included in the imputation model. The DASS depression subscale was positively skewed (skew = 1.68), as expected. However, no transformation was used to account for the skew before imputation as minor departures from normality do not have a meaningful effect on the MI results, especially when the amount of missing information is low as it was in the present study (Graham & Schafer, 1999; Schafer & Graham, 2002).

The imputation model is based on linear associations between variables, so nonlinear transformations of imputed data are not valid (Allison, 2002; Graham, 2009). Therefore, nonlinear transformations necessary for an analysis such as standardization must be done prior to imputation. For this reason, standardized values for all independent variables used in the regression analyses in the present study were entered into the imputation model as standardized variables.

All two-way interactions tested in the analyses were included in the model because interactions are also nonlinear combinations of variables. The imputation model must include all interaction terms that will be tested because the procedure is based on an assumption that the correlation between variables included in the model and variables omitted from the model is zero. Excluding variables will bias their correlation with other variables, including the outcome variable, toward zero in any analyses using the imputed dataset. Including the interaction terms in the imputation model allows the model to account for this nonlinear combination and reliably estimate missing interaction values (Graham, 2009).

Analyses were based on pooled information from 20 imputations. Although past guidelines for the ideal number of imputations with even large amounts of missing information were as low as 3 to 5, these estimates did not account for loss of power. More recent investigations suggest that at least 20 imputations are necessary to achieve a loss of power less than 1% when the amount of missing information is 0.30 or lower (Graham, Olchowski, & Gilreath, 2007) as it was in the present study.

Chapter 3: Results

Two measures of positive and negative reactivity were entered into separate multiple linear regression analyses with measures of attentional control, inhibitory control, and activation control to determine the amount of variability accounted for by each variable in symptoms of general distress and depression. Sex was also entered into each analysis as findings that females are more likely to experience both depression and anxiety are well established (Craske, 2003; Nolen-Hoeksema, 2001). Males were coded 0 and females were coded 1. The mean item scores for the NA and BIS subscales of the PANAS and BIS/BAS scales were used to measure negative reactivity, and PA and BAS were used to measure positive reactivity. A composite score of the ACS and the attentional control subscale of the ATQ was used to measure attentional control due to the similarity of the content of the items from each scale as well as the high statistical correlation between the scales (see Table 1). The composite score was measured as the average of the standardized mean item score of each scale. The mean item score of the inhibitory control subscale of the ATQ was used to measure inhibitory control. A composite score of the activation control subscale of the ATQ and the persistence/low distractibility subscale of the ECS was used to measure activation control due to the similarity of the content of the items from each scale as well as the moderately high statistical correlation between the scales (see Table 1). The composite score was measured as the average of the standardized mean item score of each scale. General

distress and depressive symptoms were measured using the total scores of the DASS stress and depression subscales. Each of the variables was normal (skew < 1) with the exception of the DASS depression subscale (skew = 1.68). No transformation was used to account for the skew as slight to moderate violations of normality in linear regression analyses do not bias results for large samples (Cohen, Cohen, West, & Aiken, 2003). Descriptive statistics including scale reliabilities are listed in Table 2 and correlations are listed in Table 3.

In addition to the main effects of the temperamental variables, predicted moderating influences of the facets of EC were tested by entering relevant interaction terms into the regression models. Standardized main effect variables were used to compute the product terms to reduce multicollinearity between the main effect variables and the interaction terms (Aiken & West, 1991).

Main effect variables and interaction terms were added to each model in successive steps to measure variability accounted for by each step over and above variability accounted for by earlier steps. In a final step, the effects of the other possible interactions between reactive temperament and the facets of EC were tested to examine any unanticipated moderating effects. The interactions between reactive temperament variables (i.e., NAxPA and BISxBAS) were also tested in the exploratory step because significant interactions have been found in previous studies exploring emotional problems (e.g., Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapil, 2007; Joiner & Lonigan, 2000; Loney, Lima, & Butler, 2006), although these studies did not include EC in their analyses. Results have generally shown that the association between high negative reactivity and emotional symptoms is attenuated at high levels of positive reactivity, although not without exception.

All predicted interactions were retained regardless of their statistical significance, while all exploratory interactions where p > .100 were dropped from the final model using a step-down procedure (Aiken & West, 1991). Significant interactions were probed by calculating the significance of simple slopes at conditional values of each variable and by determining regions of significance (Aiken & West, 1991).

No large differences affecting interpretations were found between the results of the analyses using the MI data and the raw data entered into the imputation model. A few results had different levels of significance prior to the MI procedure and after MI and are noted throughout this section. It is important to note that although some differences crossed conventional standards for significance and trend level effects, the differences themselves were quite small. Further, none of the differences involved robust findings from the other analyses or were inconsistent with the patterns of results across the other analyses. All reported statistics are from the MI data except where noted.

General Distress

PANAS scales. Sex, NA, PA, Attentional Control, Inhibitory Control, and Activation Control were entered into Step 1 of a multiple regression analysis. In Step 2, the predicted NAxAttentional Control interaction was added. NAxPA and the other possible two-way interactions between reactive temperament and the facets of EC were tested in an exploratory Step 3 using a step-down procedure. The outcome variable for the analysis was the DASS stress subscale. The results of the analysis are summarized in Table 4. Consistent with expectations, NA and Attentional Control both accounted for a statically significant amount of variability for General Distress. PA also accounted for significant variability. Sex and Inhibitory Control were significant, while Activation Control was not. Contrary to predictions, the NAxAttentional Control interaction was not significant. In the exploratory step, NAxPA had a trend level effect. The remaining exploratory interaction terms, NAxInhibitory Control, NAxActivation Control, PAxAttentional Control, PAxInhibitory Control, and PAxActivation Control, all failed to account for additional significant variability in General Distress and were dropped from the model.

An additional exploratory regression analysis was carried out including the DASS depression subscale to control for common variability between symptoms of depression and general distress to determine whether this would account for the unexpected significance of PA. The results of the analysis are in Table 5. As before, Sex, NA, Attentional Control, and Inhibitory Control all accounted for statically significant amounts of variability, while Activation Control and NAxAttentional Control did not. PA remained significant after controlling for Depressive Symptoms, but the sign of the beta weight changed from negative to positive. Further exploration showed that PA was negatively related to Depressive Symptoms and Depressive Symptoms was positively related to General Distress. Thus, the indirect path from PA to Depressive Symptoms to General Distress was negative, while the direct path from PA to General Distress was positive. A Sobel test of the indirect path was significant (z = -8.47, p < .001). The opposite signs of the direct and indirect paths indicate a suppressor relation between PA and Depressive Symptoms in relation to General Distress (MacKinnon, Krull, &

Lockwood, 2000; Shrout & Bolger, 2002). Depressive Symptoms was also significant. In the final step, none of the exploratory interaction terms were significant and so were not included in the model.

BIS/BAS scales. Sex, BIS, BAS, Attentional Control, Inhibitory Control, and Activation Control were entered into Step 1 of a multiple regression analysis. In Step 2, the predicted BISxAttentional Control interaction was added. BISxBAS and the other possible two-way interactions between reactive temperament and the facets of EC were also tested in an exploratory Step 3 using a step-down procedure. The outcome variable for the analysis was the DASS stress subscale.

The results of the analysis are summarized in Table 6. Consistent with predictions, BIS and Attentional Control accounted for a statistically significant amount of variability in General Distress. BAS and Activation Control also accounted for significant variability. Inhibitory Control was also significant, but Sex was not. Contrary to prediction, the BISxAttentional Control interaction was not significant. In the exploratory step, BISxBAS and BASxAttentional Control accounted for a statistically significant amount of variability. Additionally, BISxAttentional Control was significant after the exploratory interaction terms were added to the model. The remaining exploratory interactions, BISxInhibitory Control, BISxActivation Control, BASxInhibitory Control, and BASxActivation Control, all failed to account for additional significant variability in General Distress and were dropped from the model.

A graph of the BISxAttentional Control interaction is shown in Figure 1. Consistent with expectations, the graph shows the association between BIS and General Distress was reduced when Attentional Control was high. At high BIS (+1 SD), the predicted total score on the DASS stress subscale was 3.6 points lower for high Attentional Control (+1 SD) than for low Attentional Control (-1 SD). The simple slope of BIS at low Attentional Control (-1 SD) was 3.33, t = 10.23, p < 0.001. At high Attentional Control (+1 SD), the simple slope was 2.47, t = 8.12, p < 0.001. The region of significance showed that the simple slope of BIS was not significant for values of Attentional Control > 3.53 SDs. Higher levels of Attentional Control significantly attenuated the association between BIS and General Distress, although the association remained significant for realistic values of Attentional Control.

The graph of the BISxBAS interaction shown in Figure 2 illustrates a pattern in which individuals with lower levels of BIS and higher levels of BAS reported the lowest levels of General Distress, which is consistent with expectations. At low BIS (-1 SD), the predicted total score on the DASS stress subscale was 3.2 points lower for high BAS (+1 SD) than for low BAS (-1 SD). At higher levels of BIS, symptom reports were similar for high and low BAS. The simple slope of BIS at high BAS (+1 SD) was 3.73, t = 11.01, *p* < 0.001. At low BAS (-1 SD), the slope was 2.07, t = 7.02, *p* < 0.001. The region of significance showed that the simple slope of BIS was no longer significant at very low, not high, levels of BAS < -2.33 SDs.

A graph of the BASxAttentional Control interaction is shown in Figure 3. At higher levels of Attentional Control, low BAS was not associated with reports of General Distress. At low BAS (-1 SD), the predicted total score on the DASS stress subscale was 4.1 points lower for high Attentional Control (+1 SD) than for low Attentional Control (-1 SD). The simple slope of BAS at low Attentional Control (-1 SD) was -1.41, t = -4.31, p < 0.001. At high Attentional Control (+1 SD), the simple slope was -0.11, t = -0.32, p = 0.751. The region of significance showed that the simple slope of BAS was no longer significant for values of Attentional Control > 0.37 SDs.

An additional exploratory regression analysis was carried out including the DASS depression subscale to control for common variability between symptoms of depression and general distress to determine whether this would account for the unexpected significance of BAS and Activation Control. The results of the analysis are in Table 7. As predicted, BAS and Activation Control were no longer significant after controlling for Depressive Symptoms. Consistent with the initial analysis, BIS, Attentional Control, and Inhibitory Control all accounted for a statically significant amount of variability, while BISxAttentional Control did not. In the final step, BISxBAS was again significant, but BASxAttentional Control was not. Unlike the initial analysis, Sex was significant. None of the remaining exploratory interaction terms were significant and so were not included in the model.

Depressive Symptoms

PANAS scales. Sex, NA, PA, Attentional Control, Inhibitory Control, and Activation Control were entered into Step 1 of a multiple regression analysis. In Step 2, the predicted NAxAttentional Control and PAxActivation Control interactions were added. NAxPA and the other possible two-way interactions between reactive temperament and the facets of EC were tested in an exploratory Step 3 using a step-down procedure. The outcome variable for the analysis was the DASS depression subscale.

The results of the analysis are summarized in Table 8. Consistent with expectations, NA, PA, Activation Control, and PAxActivation Control all accounted for significant variability in Depressive Symptoms. Contrary to predictions, neither

Attentional Control nor NAxAttentional Control was significant. Inhibitory Control also did not account for significant variability. Sex was significant, but the beta weight was negative. When excluding NA from the model, Sex was no longer significant. Further exploration showed that Sex was positively related to NA and NA was positively related to Depressive Symptoms. Thus, the indirect path from Sex to NA to Depressive Symptoms was positive, while the direct path from Sex to Depressive Symptoms was negative. A Sobel test of the indirect path was significant (z = 2.54, p = .011). The opposite signs of the direct and indirect paths indicate a suppressor relation between Sex and NA in relation to Depressive Symptoms (MacKinnon et al., 2000; Shrout & Bolger, 2002). In the exploratory step, only NAxPA accounted for a statistically significant amount of variability. Additionally, PAxActivation Control was no longer significant in the model including NAxPA. The remaining exploratory interaction terms, NAxInhibitory Control, NAxActivation Control, PAxAttentional Control, and PAxInhibitory Control, all failed to account for additional significant variability in Depressive Symptoms and were dropped from the model.

A graph of the PAxActivation Control interaction is shown in Figure 4. Consistent with expectations, the graph shows the association between low PA and Depressive Symptoms was attenuated at higher levels of Activation Control. At low PA (-1 SD), the predicted total score on the DASS depression subscale was 2.7 points lower for high Activation Control (+1 SD) than for low Activation Control (-1 SD). The simple slope of PA at low Activation Control (-1 SD) was -2.20, t = -9.17, p < 0.001. At high Activation Control (+1 SD), the simple slope was -1.37, t = -4.99, p < 0.001. The region of significance showed that the simple slope of PA was not significant for values of Activation Control > 2.26 SDs.

The graph of the NAxPA interaction is shown in Figure 5. The unique effects of NA and PA show that high NA and low PA were associated with Depressive Symptoms as predicted. Consistent with this pattern, the graph of the interaction shows that the combination of high NA and low PA was most strongly predictive of higher reports of Depressive Symptoms and that this association was not merely additive. That is, the association between low PA and Depressive Symptoms was strongest at high levels of NA. At high NA (+1 SDs), the predicted total score on the DASS depression subscale was 5.1 points higher for low PA (-1 SD) than for high PA (+1 SD). The simple slope of NA at high PA (+1 SD) was 2.65, t = 8.67, *p* < 0.001. At low PA (-1 SD), the simple slope of NA was not significant for values of PA > 2.92 SDs.

An additional exploratory regression analysis was carried out including the DASS stress subscale to control for common variability between symptoms of depression and general distress to better examine depression specific (e.g., anhedonic) symptoms. This was done to determine if the expected PAxActivation Control interaction would be clearer as it was not robust to including additional terms in the initial analysis. The results of the analysis are in Table 9. Unlike the previous analysis, the predicted PAxActivation Control interaction followed the same pattern as the initial analysis. As before, Sex, NA, PA, and Activation Control all accounted for significant variability in Depressive Symptoms, although NA accounted for less variability than in the initial analysis. Unlike the initial regression, Attentional

Control had a trend level effect, although not prior to MI (p = .127). NAxAttentional Control also had a trend level effect before adding the exploratory interaction terms, though it was not significant in the initial model that did not control for General Distress. Also in contrast to the initial analysis, Inhibitory Control was significant after the addition of General Distress to the model, but the beta weight was positive. Further exploration showed that Inhibitory Control was negatively related to General Distress and General Distress was positively related to Depressive Symptoms. Thus, the indirect path from Inhibitory Control to General Distress to Depressive Symptoms was negative, while the direct path from Inhibitory Control to Depressive Symptoms was positive. A Sobel test of the indirect path was significant (z = -3.29, p < .001). The opposite signs of the direct and indirect paths indicate a suppressor relation between Inhibitory Control and General Distress in relation to Depressive Symptoms (MacKinnon et al., 2000; Shrout & Bolger, 2002). General Distress was also significant. In the final exploratory interaction step, NAxPA remained significant and retained the same pattern as in the initial analysis and PAxInhibitory Control had a trend level effect. NAxAttentional Control was no longer significant in the final step.

BIS/BAS scales. Sex, BIS, BAS, Attentional Control, Inhibitory Control, and Activation Control were entered into Step 1 of a multiple regression analysis. In Step 2, the predicted BISxAttentional Control and BASxActivation Control interactions were added. BISxBAS and the other possible two-way interactions between reactive temperament and the facets of EC were tested in an exploratory Step 3 using a step-down procedure. The outcome variable for the analysis was the DASS depression subscale.

The results of the analysis are in Table 10. Consistent with predictions, BIS, BAS, Attentional Control, Activation Control, and BASxActivation Control all accounted for significant variability in Depressive Symptoms. Inhibitory Control did not account for significant variability. Contrary to predictions, BISxAttentional Control was not significant. Sex had a trend level effect and was significant prior to MI (p = .049), but the beta weight was negative. When excluding BIS from the model, Sex was no longer significant. Further exploration showed that Sex was positively related to BIS and BIS was positively related to Depressive Symptoms. Thus, the indirect path from Sex to BIS to Depressive Symptoms was positive, while the direct path from Sex to Depressive Symptoms was negative. A Sobel test of the indirect path was significant (z = 6.34, p < 100.001). The opposite signs of the direct and indirect paths indicate a suppressor relation between Sex and NA in relation to Depressive Symptoms (MacKinnon et al., 2000; Shrout & Bolger, 2002). In the exploratory step, BASxAttentional Control accounted for a statistically significant amount of variability, although this was a trend level effect (p =.078) prior to MI. BISxBAS had a trend level effect, although not prior to MI (p = .132). Additionally, BASxActivation Control was no longer significant in the model including the exploratory interaction terms. Although it was not significant in the step prior to the addition of the exploratory interactions, BISxAttentional Control had a trend level effect in the final step, but not prior to MI (p = .123). The remaining exploratory interaction terms, BISxInhibitory Control, BISxActivation Control, and BASxInhibitory Control, all failed to account for additional significant variability in Depressive Symptoms and were dropped from the model.

A graph of the BASxActivation Control interaction is shown in Figure 6. Consistent with expectations, the graph shows the association between low BAS and Depressive Symptoms was reduced at high levels of Activation Control. At low BAS (-1 SD), the predicted total score on the DASS depression subscale was 5.2 points lower for high Activation Control (+1 SD) than for low Activation Control (-1 SD). The simple slope of BAS at low Activation Control (-1 SD) was -1.67, t = -6.12, p < 0.001. At high Activation Control (+1 SD) the slope was -0.74, t = -2.46, p = 0.014. The region of significance showed that the simple slope of BAS was no longer significant for values of Activation Control > 1.20 SDs.

A graph of the BASxAttentional Control interaction is shown in Figure 7. At higher levels of Attentional Control, low BAS was not associated with reports of depressive symptoms. At low BAS (-1 SD), the predicted total score on the DASS depression subscale was 2.7 points lower for high Attentional Control (+1 SD) than for low Attentional Control (-1 SD). The simple slope of BAS at low Attentional Control (-1 SD) was -1.53, t = -4.88, p < 0.001. At high Attentional Control (+1 SD) the slope was -0.65, t = -2.06, p = 0.039. The region of significance showed that the simple slope of BAS was no longer significant for values of Attentional Control > 1.04 SDs.

An additional exploratory regression analysis was carried out including the DASS stress subscale to control for common variability between symptoms of depression and general distress. This was done to determine of this would make the expected BASxActivation Control interaction more clear as it was not robust to including additional terms in the initial analysis. The results of the analysis are shown in Table 11. Contrary to expectations, the predicted BASxActivation Control interaction, which would
be expected to be most related to anhedonic aspects of depression, was not significant even when controlling for General Distress. As before, Sex, BAS, and Activation Control accounted for significant variability in Depressive Symptoms. BIS and Attentional Control no longer accounted for significant variability in Depressive Symptoms after controlling for General Distress. BISxAttentional Control was also not significant. In contrast to the initial analysis, Inhibitory Control was significant after the addition of General Distress to the model, but the beta weight was positive. Further exploration showed that Inhibitory Control was negatively related to General Distress and General Distress was positively related to Depressive Symptoms. Thus, the indirect path from Inhibitory Control to General Distress to Depressive Symptoms was negative, while the direct path from Inhibitory Control to Depressive Symptoms was positive. A Sobel test of the indirect path was significant (z = -4.37, p < .001). The opposite signs of the direct and indirect paths indicate a suppressor relation between Inhibitory Control and General Distress in relation to Depressive Symptoms (MacKinnon et al., 2000; Shrout & Bolger, 2002). General Distress was also significant. Finally, the exploratory interactions included in the initial regression were included in a final step, but none accounted for significant variability and all were dropped from the model.

Composite Symptoms

The BISxAttentional Control interaction was significantly related to General Distress and had a trend effect for Depressive symptoms, but was nonsignificant for both outcome variables when controlling for the other. This pattern suggests the BISxAttentional Control interaction is related to variability common to the DASS stress and depression subscales, but not unique variability for each subscale. To test this interpretation, an additional analysis was done to test the BISxAttentional Control interaction using a composite score of the DASS stress and depression subscales. The composite was calculated by averaging the totals of each subscale. Sex, BIS, BAS, Attentional Control, Inhibitory Control, and Activation Control were entered into Step 1 of a multiple regression analysis. In Step 2, the BISxAttentional Control and BASxActivation Control interactions were added. In Step 3, the BISxBAS and BASxAttentional Control interactions were added.

The results of the analysis are in Table 12. BIS, BAS, Attentional Control, Inhibitory Control, and Activation Control all accounted for significant variability in the composite symptoms measure. BISxAttentional Control was also significant in the final model, although it was not significant prior to the final step. Conversely, BASxActivation Control was significant in Step 2, but not in Step 3. Both the BISxBAS and BASxAttentional Control interactions were significant.

Moderation by Sex

A set of exploratory multiple regression analyses was carried out to test whether effects found the previous analyses were moderated by Sex. A second MI model including Sex interaction terms was created for this analysis. There were no major differences between the analyses from this and the original MI models.

There were no statistically significant moderation effects for Sex with General Distress as the outcome variable. For Depressive Symptoms, only the Activation ControlxSex interaction was significant. The interaction was significant for both the analyses with the PANAS and with the BIS/BAS scales. Sex, NA, PA, Attentional Control, Inhibitory Control, Activation Control, and General Distress were entered into Step 1 of a multiple regression analysis. In Step 2, the NAxAttentional Control and PAxActivation Control interactions were added. In Step 3, NAxPA and the interactions between Sex and all other variables and interactions were added.

The results of the analysis are in Table 13. Sex, NA, PA, Inhibitory Control, Activation Control, and General Distress accounted for significant variability. The NAxPA and Activation ControlxSex interactions were also significant. PAxActivation control had a trend effect, although it was significant in step 2. Attentional Control and the NAxActivation Control interaction were not significant in the final model, but both had a trend effect in Step 2. All other interactions with Sex were not significant and were dropped from the model.

Sex, BIS, BAS, Attentional Control, Inhibitory Control, Activation Control, and General Distress were entered into Step 1 of a multiple regression analysis. In Step 2, the BISxAttentional Control and BASxActivation Control interactions were added. In Step 3, the interactions between Sex and all other variables and interactions were added.

The results of the analysis are in Table 14. Sex, BAS, Inhibitory Control, Activation Control, and General Distress accounted for significant variability, while BIS did not. The BISxAttentional Control and BASxActivation Control interactions were also not significant. The Activation ControlxSex interaction was significant. All other interactions with Sex were not significant and were dropped from the model.

Graphs of the Activation ControlxSex interaction are shown in Figure 8 and Figure 9. Both graphs show that males and females report similar levels of Depressive Symptoms at higher levels of Activation Control, but that males report more Depressive Symptoms than females at low levels of Activation Control. In the PANAS analysis, the predicted total score on the DASS depression subscale was 2.1 points higher for males than females at low Activation Control (-1 SDs). The simple slope of Activation Control for males was -1.27, t = -5.45, p < 0.001. For females, the simple slope was -0.49, t = -2.18, p = 0.030. In the BIS/BAS scales analysis, the predicted total score on the DASS depression subscale was 1.7 points higher for males than females at low Activation Control (-1 SDs). The simple slope of Activation Control for males was -1.71, t = -7.10, p< 0.001. For females, the simple slope was -1.04, t = -4.65, p = 0.02.

Chapter 4: Discussion

Four regression analyses were performed to test a priori hypotheses concerning the variability in symptoms of general distress and depression accounted for by positive and negative reactivity, the three facets of EC, and product terms representing interactions between these variables. Two measures of positive and negative reactivity, the PANAS and BIS/BAS scales, were tested in interaction with the facets of EC in separate models. These models were tested for two dependent variables: symptoms of general distress and depression.

Consistent with expectations based on past research, negative reactivity was robustly, positively associated with symptoms of general distress in both the PANAS and BIS/BAS models. Negative reactivity was also positively associated with depressive symptoms, although less strongly than with general distress. This is consistent with expectations based on the tripartite model that symptoms unique to depression would be less strongly related to negative affectivity. When controlling for general distress, BIS was no longer related to depressive symptoms and the association with NA dropped substantially.

Also consistent with expectations based on past research, positive reactivity was robustly, negatively associated with depressive symptoms in both the PANAS and BIS/BAS models. Positive reactivity was also negatively associated with general distress, although less strongly. After controlling for depressive symptoms, BAS was no longer related to general distress and the association between PA and general distress changed from negative to positive. That is, after partialling out symptoms of depression, high PA was actually related to higher reports of general distress. One possible explanation for this finding is that low PA is inconsistent with the chronic nonspecific arousal of general distress. This pattern suggests that low positive reactivity is strongly related to symptoms specific to depression.

Separating EC into three facets and exploring the unique associations between each facet and symptoms of general distress and depression revealed several important patterns that advanced our understanding of EC. Generally, the results were consistent with expectations. Attentional control was negatively associated with symptoms associated with negative reactivity. There was also evidence of a moderating effect of attentional control on the relationship between negative reactivity and these symptoms. Conversely, activation control was related to symptoms specific to depression, which are most strongly related to low positive reactivity. Further, activation control moderated the association between positive reactivity and depressive symptoms. Finally, like attentional control, inhibitory control was negatively associated with symptoms associated with negative reactivity.

Attentional control was robustly related to general distress and was also related depressive symptoms before controlling for general distress. As predicted, high attentional control was related to reports of fewer symptoms of general distress. This was true for both the PANAS and BIS/BAS models, suggesting a robust association between attentional control and general distress. Attentional control was related to depressive symptoms in the BIS/BAS model, but not the PANAS model. When controlling for general distress, attentional control was no longer related to depressive symptoms in either model. This pattern is consistent with a stronger association between attentional control and general distress than with symptoms specific to depression.

As hypothesized, there was also evidence of a moderating effect of attentional control on the association between negative reactivity and general distress. The interaction was significant in the BIS/BAS model for general distress, but not after controlling for depressive symptoms. The interaction also had a trend level effect in the BIS/BAS model for depressive symptoms, but also only before controlling for general distress. This pattern of results suggested the interaction was most strongly related to commonalities between the symptom measures. Indeed, the interaction was significantly related to a composite of the symptom measures. Additionally, there was a trend effect in the PANAS model for depressive symptoms when controlling for general distress, although the interaction was not significant after adding the NAxPA interaction. The moderation effect of PA on the association between NA and depressive symptoms (see below for more discussion of this interaction) raises the possibility that PA and attentional control overlap somewhat, which may have contributed to null findings. The remaining analyses did not show a moderation effect, although some of the null findings may have been due to insufficient power despite the large sample size because only a small percentage of the sample was high in both negative reactivity and attentional control (e.g., less than 15% of the sample was above the mean for both NA and attentional control and less than 1% was one standard deviation above the mean for both variables). A more consistent pattern was expected, but the results do support a moderating effect of attentional control. Future research with larger samples of

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individuals high in both negative reactivity and attentional control will be able to further clarify this effect.

As predicted, high levels of activation control were associated with lower reports of depressive symptoms across all of the analyses. Also as expected, activation control was not related to general distress. These results support the expected association between activation control and symptoms related to low positive reactivity. Although activation control was related to lower reports of general distress in the BIS/BAS model, this was no longer true after controlling for depressive symptoms, further suggesting activation control is most strongly associated with depressive symptoms.

Consistent with expectations, activation control moderated the association between low positive reactivity and depressive symptoms. The interaction between positive reactivity and activation control was significant for both the PANAS and BIS/BAS models. The results were not robust to the inclusion of exploratory interaction terms in the initial models, but were robust in the PANAS model after controlling for general distress. This pattern is consistent with expectations that the moderating effect would be most strongly related to depressive symptoms associated with low positive reactivity (e.g., anhedonia). However, the moderating effect was not found in the BIS/BAS model after controlling for general distress, although the effect was in the expected direction. In sum, although there were some exceptions, the results support the predicted moderating effect of activation control on the association between low levels of positive reactivity and depressive symptoms.

Like attentional control, high inhibitory control was also related to lower reports of general distress across the analyses. There were no specific predictions for inhibitory control, but this finding is consistent with expectations that an increased capacity for cognitive control should be related to fewer symptoms of general distress based on past research demonstrating a link between cognitive inhibitory deficits and general symptoms of depression (e.g., Frings et al., 2007; Joormann, 2004, 2006; Raes et al., in press). The results also suggested that attentional control alone does not entirely account for all aspects of cognitive control related to general distress. The inhibitory control subscale of the ATQ is designed to assess inhibition of behavioral responses, but it is not clear that cognitive and behavioral inhibition can be meaningfully separated or measured independently (Aron, 2007). The present findings suggest the scale may indeed tap cognitive control as well as behavioral control. Future research on the EC model should include a focus on inhibitory control as a potential moderator of reactive risk using additional measures of cognitive inhibitory control.

Inhibitory control was not related to symptoms specific to depression in either the PANAS or BIS/BAS models. This is consistent with both cognitive and behavioral components of inhibitory control. Cognitive control should be related to symptoms most strongly associated with high negative reactivity (e.g., rumination; Davis & Nolen-Hoeksema, 2000), but less related to symptoms more specific to depression, which are associated with low positive reactivity (e.g., low approach and anhedonia). Behavioral inhibitory control would also not be expected relate to depressive symptoms. Although these findings fit the general patterns of results in the analyses, it is important to note that the null results may also be due to the low reliability of the inhibitory control measure.

One additional finding involving inhibitory control also warrants discussion. When controlling for general distress, inhibitory control was related to depressive symptoms in both the PANAS and BIS/BAS models, but the relation changed from negative to positive. That is, after partialling out symptoms of general distress, higher levels of inhibitory control were associated with higher reports of depressive symptoms. This is surprising because inhibitory control certainly would not be expected to be associated with increased symptoms. However, low levels of positive reactivity are associated with reduced responding and the inhibitory control items on the ATQ may tap this reduced responding somewhat. That is, the inhibitory control measure may be confounded to some extent with positive reactivity. Given the low internal consistency of this scale and its lack of items clearly pertaining to cognitive inhibitory control, future research should include alternative measures of this construct.

Tests of possible moderating effects of sex generally suggested the observed effects held for men and women. However, several sex differences were observed that warrant discussion. Whereas the expected sex difference was observed for symptoms of general distress, with women reporting more symptoms than men on average, the opposite pattern emerged for symptoms unique to depression. In this sample, men were more likely to report symptoms of depression than women on average. This effect was strongest when symptoms of general distress were partialled out of the model. Although at first glance this appears to be contrary to past research indicating that depressive symptoms are more common for females, Craske (2003) suggests that the sex difference in anxiety and depression may be due to a general proneness to negative reactivity. Partialling out the association between sex and negative reactivity and related symptoms (i.e., general distress) revealed that men in this sample reported experiencing more symptoms specific to depression than women. Related to this, sex was found to significantly moderate the association between activation control and depressive symptoms such that low activation control was associated with higher reports of depressive symptoms in men than women. One potential explanation for this finding is that low levels of activation control may be more detrimental to men than women because of masculine gender norms that encourage action. Failure to meet responsibilities due to a low capacity to effortfully overcome anhedonic feelings and low approach motivation may be more distressing and debilitating for men than for women and contribute to higher reports of depressive symptoms. Men are less likely than women ruminate in response to depression (Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Instead, social gender norms may lead men respond to negative emotions with avoidance or distraction, which may include positive coping behaviors or negative behaviors, such as alcohol abuse (Addis, 2008). Another potential explanation for the observed moderation is that low activation control may impede an individual's ability to cope with negative emotion behaviorally, which would be more detrimental to men.

The NAxPA interaction was significant for depressive symptoms. The interaction also had a trend effect for general distress, although not when controlling for depressive symptoms. In all cases, high levels of PA were associated with reductions in the positive association between high levels of NA and depressive symptoms. However, low levels of PA combined with high levels of NA were associated with higher reports of depressive symptoms than would be expected by a linear combination of their individual effects. That is, the effects were more than additive. These results show that the relative levels of positive and negative reactivity are important to consider in accounting for variance in depressive symptoms as well as, to a lesser degree, general distress.

The BISxBAS interaction was significant for general distress such that at low levels of BIS, low BAS was related to higher reports of general distress. This is consistent with expectations of stronger associations between low BAS and general distress. At high levels of BIS, reports of general distress were similar for both high and low levels of BAS. That is, high levels of BAS were not associated with a reduction in the association between high levels of BIS and general distress. The BISxBAS interaction also had a trend effect for depressive symptoms, but was no longer significant after controlling for general distress. These results are similar to findings by Hundt et al. (2007) where at high levels of BIS, high levels of BAS were 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 approachavoidance conflicts likely experienced by individuals high in both BIS and BAS. These findings are also consistent with the positive association between PA and general distress when controlling for depressive symptoms observed in the present study. See Harbaugh (2009) for a more detailed discussion of the NAxPA and BISxBAS interactions in this sample.

Advantages and Limitations of the Present Study

Given the expected small effect size for interactions between the temperament variables, the greatest strength of the present study was its large sample size relative to other studies. However, the sample size was not so large that power was not a problem because the sample was unselected and so was not equally representative of all combinations of high and low reactivity and high and low levels of the facets of EC. Specifically, groups with high negative reactivity and high EC and those with low positive reactivity and high EC were underrepresented. This distribution makes it more difficult to find moderating effects as the underrepresented groups are the focus of these effects.

In addition to the large sample, considering individual facets of EC allowed tests of more specific components of EC and may have also improved statistical power. Another advantage was that the study considered symptoms of general distress and depression separately and also included tests controlling for each. This approach allowed a more throughout examination of variance unique to each symptom type as well as shared variance between them.

The study also included some limitations. First, it was based on self-report questionnaires to assess levels of temperament and symptoms. Questionnaires with the best available psychometric properties for each variable were used, but the results still necessarily relied on each participant's personal assessment of themselves. The study was also unable able to satisfactorily test the association between inhibitory control and the symptom variables. Future replication may help clarify the association between inhibitory control and emotional symptoms by including other measures of inhibitory control with higher reliability that included more direct cognitive items.

The study was correlational in nature and although the results were consistent with theoretical expectations, no causal interpretations can be made. The cross-sectional design also makes it impossible to rule out the possibility that the observed effects of temperament variables and their interactions are consequences of elevations in symptoms of general distress and depression rather than contributors to elevations in such

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symptoms. It is important to explore the direction of these effects in prospective studies in the future.

Another potential limitation of the study design was potential item overlap between the predictor and outcome variables. Some items from the DASS scales were similar in content to items from the temperament scales, but this was true for only a small minority of items. Past research on the association between temperament and psychological symptoms has shown that the expected associations are generally preserved after removing confounding items (Lemery, Essex, & Smider, 2002; Lengua et al., 1998; Oldehinkel et al., 2004). Although further exploration would be required to determine the impact of the item overlap in the present sample, the high reliabilities ($\alpha \ge$.93) of the DASS scales suggest that removing potential confounding items would have a minimal impact on the results. Further, to the extent that item overlap occurred it would affect main effects and would not likely contribute to spurious interaction terms. *Conclusions*

In summary, the results generally supported expectations about the association of different facets of EC to emotional symptoms associated with positive and negative reactivity. Attentional control and inhibitory control were most strongly related to symptoms of general distress, but not depression. Attentional control was also found to moderate the association between negative reactivity and symptoms of general distress and depression. Conversely, activation control was strongly related to symptoms of depression, but not general distress. There was also evidence for a moderation effect of activation control reducing the association between low positive reactivity and depressive symptoms. Findings also suggested that men may be more likely to report depressive

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symptoms related to low positive reactivity than women and that low activation control is associated with higher reports of depressive symptoms in men than women. Finally, the results revealed significant interactions between BIS and BAS for general distress and NA and PA for depressive symptoms.

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Appendix A: Tables

Table 1: Correlations Between Components of Composite Scores

		1	2	3	4
1.	ATQ-Attentional Control	-			
2.	ACS	$.75^{*}$	-		
3.	ATQ-Activation Control	$.48^{*}$	$.39^{*}$	-	
4.	ECS-Persistence/Low Distractibility	.41*	.34*	.64*	-

* *p* < 0.05

Notes: Values reported are based on dataset prior to missing data procedures. ATQ = Adult Temperament Questionnaire, ACS = Attentional Control Scale, ECS = Effortful Control Scale.

Table 2: Means, Standard Deviations, and Reliabilities of All Variables

	М	SD	α	Ν
NA	19.08	6.66	0.86	1217
PA	35.28	6.69	0.87	1222
BIS	20.19	3.89	0.78	1227
BAS	40.85	5.92	0.86	1153
ATQ-Attentional Control	19.84	5.26	0.73	1219
ACS	50.79	8.68	0.85	1218
ATQ-Inhibitory Control	24.33	5.54	0.49	1217
ATQ-Activation Control	29.02	6.14	0.70	1220
ECS-Persistence/Low Distractibility	28.23	4.59	0.83	1178
General Distress	10.91	8.92	0.93	1217
Depressive Symptoms	6.71	7.90	0.94	1215

Notes: Values reported are based on dataset prior to missing data procedures. NA = Negative Affectivity, PA = Positive Affectivity, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, ATQ = Adult Temperament Questionnaire, ACS = Attentional Control Scale, ECS = Effortful Control Scale.

Table 3: Correlations Between All Variables

		1	2	3	4	5	6	7	8	9	10
1.	Sex	-									
2.	NA	$.12^{*}$	-								
3.	PA	08^{*}	33*	-							
4.	BIS	.33*	.42*	20*	-						
5.	BAS	$.06^{*}$	13*	$.29^{*}$.13*	-					
6.	Attentional Control	11*	37*	.38*	31*	$.06^{*}$	-				
7.	Inhibitory Control	11*	18*	.09*	10*	17*	$.40^{*}$	-			
8.	Activation Control	$.07^{*}$	28*	$.48^{*}$	06*	.16*	$.48^{*}$	$.28^{*}$	-		
9.	General Distress	$.15^{*}$	$.68^{*}$	32*	.36*	09*	38*	24*	29*	-	
10.	Depressive Symptoms	.01	$.57^{*}$	45*	$.22^{*}$	18*	32*	13*	37*	.73*	-

* *p* < 0.05

Notes: NA = Negative Affectivity, PA = Positive Affectivity, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System.

	В	SE	p	R ²	ΔR^2
Step 1			< .001	.489	.489
Step 2			.896	.489	< .001
Step 3			.060	.491	.002
Sex	0.48	0.19	.010		
NA	5.18	0.21	< .001		
PA	-0.60	0.22	.006		
Attentional Control	-0.73	0.23	.002		
Inhibitory Control	-0.68	0.21	.001		
Activation Control	-0.33	0.23	.149		
NAxAttentional Control	0.15	0.19	.445		
NAxPA	-0.36	0.19	.060		

Table 4: Multiple Regression Analysis Predicting General Distress with the PANAS

Notes: PAxInhibitory Control, PAxActivation Control, NAxActivation Control, PAxAttentional Control, and NAxInhibitory Control were also tested in an exploratory final step but were dropped from the model in that order due to *p*-values > .100 in a step-down procedure. NA = Negative Affectivity, PA = Positive Affectivity.

Table 5: Exploratory Multiple Regression Analysis Predicting General Distress with the PANAS Controlling for Depressive Symptoms

U	1		<i>7</i> 1		
	В	SE	р	R ²	ΔR^2
Step 1			< .001	.649	.649
Step 2			.156	.649	< .001
Sex	0.75	0.16	< .001		
NA	3.16	0.19	< .001		
PA	0.49	0.19	.010		
Attentional Control	-0.69	0.19	< .001		
Inhibitory Control	-0.70	0.17	< .001		
Activation Control	0.29	0.19	.130		
Depressive Symptoms	4.69	0.20	< .001		
NAx Attentional Control	0.20	0.14	156		

Notes: PAxAttentional Control, NAxActivation Control, NAxPA, NAxInhibitory Control, PAxActivation Control, and PAxInhibitory Control were also tested in an exploratory final step but were dropped from the model in that order due to p-values > .100 in a step-down procedure. NA = Negative Affectivity, PA = Positive Affectivity.

	В	SE	р	R ²	ΔR^2
Step 1			< .001	.253	.253
Step 2			.180	.254	.001
Step 3			<.001	.268	.014
Sex	0.18	0.24	.451		
BIS	2.90	0.25	<.001		
BAS	-0.76	0.25	.003		
Attentional Control	-1.39	0.28	<.001		
Inhibitory Control	-1.08	0.25	<.001		
Activation Control	-1.20	0.26	<.001		
BISxAttentional Control ¹	-0.43	0.19	.027		
BISxBAS	0.83	0.20	<.001		
BASxAttentional Control	0.65	0.22	.003		

 Table 6: Multiple Regression Analysis Predicting General Distress with the BIS/BAS

 Scales

Notes: BASxInhibitory Control, BISxActivation Control, BASxActivation Control, and BISxInhibitory Control were also tested in an exploratory final step but were dropped from the model in that order due to p-values > .100 in a step-down procedure. BIS = Behavioral Inhibition System, BAS = Behavioral Activation System.

¹ Not significant in step 2 (B = -0.26, SE = 0.19, p = .180)

	the
BIS/BAS Scales Controlling for Depressive Symptoms	

	В	SE	р	R ²	ΔR^2
Step 1			< .001	.592	.592
Step 2			.492	.592	< .001
Step 3			< .001	.597	.005
Sex	0.51	0.17	.004		
BIS	1.60	0.19	< .001		
BAS	0.05	0.19	.785		
Attentional Control	-0.70	0.21	.001		
Inhibitory Control	-0.93	0.19	< .001		
Activation Control	0.32	0.20	.100		
Depressive Symptoms	5.82	0.18	< .001		
BISxAttentional Control	-0.18	0.14	.214		
BISxBAS	0.54	0.14	<.001		

Notes: BASxActivation Control, BASxInhibitory Control, BISxActivation Control, BISxInhibitory Control, and BASxAttentional Control were also tested in an exploratory final step but were dropped from the model in that order due to p-values > .100 in a step-down procedure. BIS = Behavioral Inhibition System, BAS = Behavioral Activation System.

	В	SE	р	R ²	ΔR^2
Step 1			< .001	.417	.417
Step 2			.002	.423	.006
Step 3			< .001	.430	.007
Sex	-0.44	0.18	.011		
NA	3.39	0.20	< .001		
PA	-1.78	0.21	< .001		
Attentional Control	-0.08	0.22	.718		
Inhibitory Control	0.04	0.19	.821		
Activation Control	-1.02	0.21	< .001		
NAxAttentional Control	0.03	0.18	.869		
PAxActivation Control ¹	0.24	0.16	.142		
NAxPA	-0.74	0.19	< .001		

Table 8: Multiple Regression Analysis Predicting Depressive Symptoms with the PANAS

Notes: NAxActivation Control, PAxAttentional Control, NAxInhibitory Control, and PAxInhibitory Control were also tested in an exploratory final step but were dropped from the model in that order due to p-values > .100 in a step-down procedure. NA = Negative Affectivity, PA = Positive Affectivity. ¹ Significant in step 2 (B = 0.42, SE = 0.16, p = .009)

 Table 9: Exploratory Multiple Regression Analysis Predicting Depressive Symptoms

 with the PANAS Controlling for General Distress

	В	SE	р	R ²	ΔR^2
Step 1			< .001	.599	.599
Step 2			.001	.604	.005
Step 3			< .001	.609	.005
Sex	-0.68	0.15	< .001		
NA	0.68	0.21	.001		
PA	-1.45	0.17	< .001		
Attentional Control ¹	0.31	0.18	.092		
Inhibitory Control	0.42	0.16	.010		
Activation Control	-0.86	0.18	< .001		
General Distress	4.68	0.20	< .001		
NAxAttentional Control ²	-0.06	0.15	.684		
PAxActivation Control	0.30	0.14	.035		
NAxPA	-0.58	0.15	< .001		
PAxInhibitory Control	-0.28	0.15	.058		

Notes: NAxInhibitory Control, NAxActivation Control, and PAxAttentional Control were also tested in an exploratory final step but were dropped from the model in that order due to *p*-values > .100 in a step-down procedure. NA = Negative Affectivity, PA = Positive Affectivity.

¹ Not significant prior to MI (p = .126)

² Trend level effect in step 2 (B = -0.27, SE = 0.14, p = .055)

	В	SE	р	R²	ΔR^2
Step 1			< .001	.208	.208
Step 2			.023	.213	.005
Step 3			.054	.216	.003
Sex ¹	-0.42	0.22	.052		
BIS	1.73	0.23	<.001		
BAS	-1.09	0.23	<.001		
Attentional Control	-0.90	0.26	<.001		
Inhibitory Control	-0.18	0.23	.437		
Activation Control	-2.10	0.24	<.001		
BISxAttentional Control ²	-0.30	0.18	.091		
BASxActivation Control ³	0.27	0.21	.189		
BASxAttentional Control ⁴	0.44	0.22	.044		
BISxBAS ⁵	0.31	0.18	.085		

Table 10: Multiple Regression Analysis Predicting Depressive Symptoms with the BIS/BAS Scales

Notes: BISxActivation Control, BASxInhibitory Control, and BISxInhibitory Control were also tested in an exploratory final step but were dropped from the model in that order due to p-values > .100 in a step-down procedure. BIS = Behavioral Inhibition, System BAS = Behavioral Activation System.

¹ Significant prior to MI (p = .049)

² Not significant in step 2 (B = -0.22, SE = 0.17, p = .200) or in step 3 prior to MI (p = .123)

³ Significant in step 2 (B = 0.47, SE = 0.19, p = .014)

⁴ Trend level effect prior to MI (p = .078)

⁵ Not significant prior to MI (p = .132)

Table 11: Exploratory Multiple Regression Analysis Predicting Depressive Symptoms with the BIS/BAS Scales Controlling for General Distress

	В	SE	р	R ²	ΔR^2
Step 1			< .001	.567	.567
Step 2			.350	.568	.001
Sex	-0.53	0.16	.001		
BIS	-0.03	0.18	.854		
BAS	-0.54	0.16	.001		
Attentional Control	-0.03	0.19	.867		
Inhibitory Control	0.51	0.17	.003		
Activation Control	-1.36	0.18	<.001		
General Distress	5.47	0.17	<.001		
BISxAttentional Control	-0.06	0.13	.629		
BASxActivation Control	0.19	0.14	.170		

Notes: BASxAttentional Control, BASxInhibitory Control, BISxInhibitory Control, BISxActivation Control, and BISxBAS were also tested in an exploratory final step but were dropped from the model in that order due to *p*-values > .100 in a step-down procedure. BIS = Behavioral Inhibition System, BAS = Behavioral Activation System.

	В	SE	р	R ²	ΔR^2
Step 1			< .001	.254	.254
Step 2			.016	.259	.005
Step 3			.001	.267	.008
Sex	-0.24	0.41	.555		
BIS	4.65	0.44	<.001		
BAS	-1.84	0.44	<.001		
Attentional Control	-2.29	0.49	< .001		
Inhibitory Control	-1.26	0.45	.005		
Activation Control	-3.31	0.45	<.001		
BISxAttentional Control ¹	-0.72	0.34	.033		
BASxActivation Control ²	0.43	0.40	.284		
BISxBAS	1.12	0.35	.001		
BASxAttentional Control	1.03	0.42	.015		

Table 12: Exploratory Multiple Regression Analysis Predicting Composite Symptoms with the BIS/BAS Scales

Notes: BIS = Behavioral Inhibition System, BAS = Behavioral Activation System.

¹ Not significant in step 2 (B = -0.49, SE = 0.34, p = .145)

² Significant in step 2 (B = 0.92, SE = 0.37, p = .012)

Table 13: Exploratory Regression Analysis Testing Moderation by Sex Predictin	ng
Depressive Symptoms with the PANAS Controlling for General Distress	U

	В	SE	р	R ²	ΔR^2
Step 1			< .000	.768	.768
Step 2			.001	.771	.003
Step 3			< .000	.775	.004
Sex	-0.68	0.15	< .000		
NA	0.68	0.20	.001		
PA	-1.46	0.17	< .000		
Attentional Control ¹	0.28	0.18	.115		
Inhibitory Control	0.38	0.16	.021		
Activation Control	-0.88	0.18	< .000		
General Distress	4.66	0.20	< .000		
NAxAttentional Control ²	-0.10	0.15	.520		
PAxActivation Control ³	0.23	0.14	.088		
NAxPA	-0.57	0.15	< .000		
Activation ControlxSex	0 39	0.15	.007		

Notes: The remaining interaction terms between Sex and all other variables and interactions were also tested and dropped from the model for failing to account for significant symptom variability. NA = Negative Affectivity, PA = Positive Affectivity. ¹ Trend effect in step 2 (B = 0.32, SE = 0.18, p = .081) ² Trend effect in step 2 (B = -0.27, SE = 0.14, p = .057)

³ Significant in step 2 (B = 0.35, SE = 0.13, p = .008)

	В	SE	р	R ²	ΔR^2
Step 1			<.001	.750	.750
Step 2			.349	.750	< .001
Step 3			.028	.751	.001
Sex	-0.51	0.16	.001		
BIS	-0.04	0.18	.827		
BAS	-0.56	0.16	< .000		
Attentional Control	-0.04	0.19	.819		
Inhibitory Control	0.48	0.17	.005		
Activation Control	-1.38	0.18	< .000		
General Distress	5.46	0.17	< .000		
BISxAttentional Control	-0.12	0.13	.367		
BASxActivation Control	0.16	0.14	.256		
Activation ControlxSex	0.34	0.15	.028		

Table 14: Exploratory Regression Analysis Testing Moderation by Sex Predicting Depressive Symptoms with the BIS/BAS Scales Controlling for General Distress

Notes: The remaining interaction terms between Sex and all other variables and interactions were also tested and dropped from the model for failing to account for significant symptom variability. BIS = Behavioral Inhibition System, BAS = Behavioral Activation System.


Appendix B: Figures





Figure 2: BISxBAS Interaction Predicting General Distress

Figure 3: BASxAttentional Control Interaction Predicting General Distress





Figure 4: PAxActivation Control Interaction Predicting Depressive Symptoms

Figure 5: NAxPA Interaction Predicting Depressive Symptoms





Figure 6: BASxActivation Control Interaction Predicting Depressive Symptoms

Figure 7: BASxAttentional Control Interaction Predicting Depressive Symptoms





Figure 8: Activation ControlxSex Predicting Depressive Symptoms with the PANAS

Figure 9: Activation ControlxSex Predicting Depressive Symptoms with the BIS/BAS Scales

