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# Integrating Cognitive Mechanisms in the Relationship Between Trait Affect and Depressive Symptoms: The Role of Affect Amplification

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Integrating Cognitive Mechanisms in the Relationship Between Trait Affect and

Depressive Symptoms: The Role of Affect Amplification


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# Copyright

## Dedication

To clients I have met and clients who will touch my life in the future.

**Acknowledgements**

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Lastly, I am grateful to my family and friends who listen to my stories and prompt me to consider mental health from different perspectives. My husband is a constant support professionally and personally, and I would not be who I am without him.

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## Abstract

Trait levels of negative affect (NA) and positive affect (PA) are established risk factors for depressive symptoms (Clark & Watson, 1991), but the mechanisms through which high NA and low PA confer risk for depression are poorly understood.  Two proposed mechanisms in the transmission of affective vulnerabilities to depression are the cognitive responses of brooding and positive rumination.  Brooding and positive rumination may represent a common cognitive process that amplifies the intensity of affect and contributes to depressive symptoms.  Therefore, my dissertation purposes were to (a) determine whether brooding and positive rumination represent a shared cognitive process on distinct affective content and (b) examine brooding and positive rumination as cognitive mechanisms through which NA and PA predict depressive symptoms with an 8-week, prospective design among adults.  I hypothesized that brooding and positive rumination would be best modeled as distinct but related factors (Model 2).  I also hypothesized that greater brooding and less positive rumination would mediate the relationships between greater NA and less PA in predicting greater depressive symptoms. I first compared three confirmatory factor analysis models of the relationship between brooding and positive rumination as distinct constructs, as the same construct, and as distinct but related constructs to determine how these constructs relate.  Thereafter, I utilized structural equation modeling to examine whether brooding and positive rumination mediated the relationship between trait affect and depressive symptoms.

Participants were 321 (73.5% female) undergraduate students ($M$=19.03, $SD$=1.64). Participants completed online measures of trait affect, cognitive responses, and depressive symptoms at baseline and again completed an online measure of depressive symptoms seven weeks after baseline assessment. Results indicated that Model 2 best fit the data ($\chi$=195.07, $\Delta\chi$=8.78, $p$<.001, CFI=.91, RMSEA=.07), supporting a conceptualization of brooding and positive rumination as distinct but related constructs. Results further indicated that greater NA and less PA distinctly predicted greater depressive symptoms through greater brooding ($\beta_{NA}$=.08, $p$=.007; $\beta_{PA}$=-.02, $p$=.038), but positive rumination did not mediate either relationship ($\beta_{NA}$=.01, $p$=.443; ($\beta_{PA}$=.01, $p$=.441). Findings contribute to an integrated theoretical understanding of the joint contributions of brooding and positive rumination in the relationship between trait affect and depressive symptoms.

**CHAPTER I**

**Introduction and Literature Review**

**Purpose**

Depression is a pervasive mental health concern that markedly increases in adolescence and early adulthood, with 15.7% of Americans reporting at least one depressive episode by age 24 (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). Even subclinical depressive symptoms are associated with impairments in academic, occupational, and interpersonal functioning (Fergusson & Woodward, 2002; Kessler & Wang, 2009). Depression is a mood disorder characterized by an excess of negative affect (NA) and lack of positive affect (PA), and extensive research has identified individual differences in trait levels of NA and PA as risk factors for depressive symptoms and disorders (Clark & Watson, 1991; Kotov, Gámez, Schmidt, & Watson, 2010; Naragon-Gainey, Gallagher, & Brown, 2013; Verstraeten, Vasey, Raes, & Bijttebier, 2009). However, less is understood about the mechanisms through which affective vulnerabilities such as high trait NA and low trait PA confer risk for depression.

Cognitive responses to emotion-eliciting life events are proposed mechanisms in the transmission of affective vulnerabilities to the onset and maintenance of depression. Two cognitive responses that predict depressive symptoms are two forms of rumination: brooding and positive rumination. Rumination is "the process of thinking perseveratively about one's feelings and problems rather than in terms of the specific content of thoughts" (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Brooding describes responding to negative events and mood states with perseverative cognitive focus on negative content, which represents a maladaptive cognitive process that increases NA and predicts greater depressive symptoms (Nolen-Hoeksema, 1991; Treynor, Gonzales, &

Nolen-Hoeksema, 2003). As a counterpart to brooding, positive rumination describes responding to positive events and mood states with perseverative cognitive focus on positive content, which represents an adaptive cognitive process that increases PA and predicts fewer depressive symptoms (Feldman, Joormann, & Johnson, 2008). Brooding is a form of rumination on negative content while positive rumination is a form of rumination on positive content.

Trait affective vulnerabilities have been shown to predict depressive symptoms through ruminative cognitive responses to positive and negative life events. Research on affective vulnerabilities indicates that trait NA positively predicts greater brooding and greater depressive symptoms while trait PA positively predicts greater positive rumination and fewer depressive symptoms (Clark & Watson, 1991; Feldman et al., 2008; Naragon-Gainey et al., 2013; Treynor et al., 2003). Relatedly, research on cognitive vulnerabilities to depression, specifically, distinct from the role of trait NA or trait PA, also indicates that brooding is associated with greater depressive symptoms and positive rumination is associated with fewer depressive symptoms (Feldman et al., 2008; Treynor et al., 2003). Affective and cognitive pathways to depression have been studied separately, with literature supporting the relationship from trait NA to depressive symptoms as partially mediated by brooding (Mezulis, Simonson, McCauley, Vander Stoep, 2011) and the relationship from trait PA to depressive symptoms as partially mediated by positive rumination (Harding, Hudson, & Mezulis, 2014). In this way, theories that integrate cognitive-affective pathways that explain how trait affect may predict brooding and positive rumination, which in turn may predict depression combine multiple vulnerabilities to improve the prediction of depressive symptoms. However, an

empirical division still remains between negative and positive cognitive-affective pathways to depression – virtually all studies consider the NA-brooding-depression pathway as distinct and separate from the PA-positive rumination-depression pathway.

This distinction may mask important conceptual and statistical overlap between these pathways (Evans & Rothbart, 2007; Harding et al., 2014). First, the assumption that distinct affective vulnerabilities (i.e., trait NA and trait PA) predict distinct cognitive responses is untested and potentially inaccurate. Second, whereas brooding and positive rumination have been proposed as distinct cognitive responses to emotion-eliciting life events that may link affective vulnerabilities to depression (Fredrickson, 2001; Nolen-Hoeksema, 1991), both describe cognitive responses that amplify affective content. As a result, these two cognitive responses may represent constructs with shared conceptual overlap. Brooding and positive rumination may share a common process that is enacted on unique affective content, but no known research has examined these cognitive responses jointly in relation to either affective vulnerabilities or depressive symptoms.

Considering the considerable contributions of affective vulnerabilities (trait NA and trait PA) to depression, I sought to better understand the joint contributions of these affective and cognitive risk factors. The first purpose of my dissertation was to examine the relationship between the cognitive responses of brooding and positive rumination to determine whether they represent a partially or fully shared cognitive process of increasing affective content or two distinct cognitive processes that are specific to the valence of affective content. Thereafter, my second purpose was to examine brooding and positive rumination as potential cognitive mechanisms through which trait NA and trait PA may predict depressive symptoms. Research supports mediation relationships

for brooding and positive rumination independently, but the shared variance between these two cognitive responses may additionally explain the relationship between trait affect and depressive symptoms. If a shared cognitive process of affect amplification was supported in Part 1, Part 2 would examine the best fitting model of the relationship between brooding and positive rumination items as a mediator between trait affect and depressive symptoms. If a shared cognitive process was not supported, this second purpose would seek to corroborate previous research in predicting that the relationship between trait NA and depression would be mediated by brooding and the relationship between trait PA and depression would be mediated by positive rumination, controlling for the unique indirect effects of each mediator by controlling for the effect of the other mediator (i.e., brooding or positive rumination).

**Trait Predictors of Depressive Symptoms**

Temperament is a broad term describing individual differences in emotional and behavioral reactivity and regulation that are hypothesized to be genetically-based, present early in life, and associated with a broad range of psychological outcomes (Rothbart & Derryberry, 1981). Reactivity describes an individual's degree of emotional or behavioral arousal to events and may be represented as trait affectivity (Rothbart, 2004). While reactivity commonly refers to the expression of emotionality in response to stress, reactivity according to Rothbart refers to a core component of temperament that persists across situations and describes global degrees of trait affect. More specifically, trait NA and trait PA describe an individual's emotional reactivity across situations, and these traits are posited to predispose individuals to depressive symptoms (Evans & Rothbart, 2009; Rothbart, 2007).

**Trait NA.** Trait NA is a core dimension of temperament that represents a significant component of emotional reactivity throughout the lifespan. Trait NA describes an individual's tendency to experience intense and frequent negative emotions (Evans & Rothbart, 2007). As a result, high trait NA is consistently linked to a variety of negative psychological outcomes and is a particularly strong predictor of past, present, and future depressive symptoms (Hankin et al., 2009; Lengua & Long, 2002; Rothbart, 2004; Wetter & Hankin, 2009). Although all individuals exhibit some degree of trait NA, individuals with high trait NA generally experience negative emotions more frequently and more intensely in response to negative events (Hyde, Mezulis, & Abramson, 2008).

Although high trait NA significantly predicts greater rumination on negative events and greater depressive symptoms (Clark & Watson, 1991; Cox, Funasaki, Smith & Mezulis, 2012; Hankin, Fraley, & Abela, 2005), not every individual with high trait NA excessively ruminates or becomes significantly depressed and not all individuals with high trait NA report significant depressive symptoms. High trait NA is associated with a wide range of internalizing and externalizing difficulties, including greater anxiety and conduct disorder symptoms in adolescence and adulthood (Brown, Chorpita, & Barlow, 1998; Clark & Watson, 1991; Sanson & Prior, 1999). High trait NA is only one of the myriad vulnerabilities to depression. Consequently, examining how the affective vulnerability of high trait NA relates to other processes in the prediction of depressive symptoms may provide valuable insight into which temperamentally-predisposed individuals are likely to develop significant depressive symptoms. In light of this examination, another affective vulnerability to depression is the temperament dimension of trait PA.

**Trait PA.** Trait PA is a complementary but distinct component of temperament in comparison to trait NA, describing an individual's tendency to experience intense and frequently high activity, pleasure, and positive anticipation of the future (Rothbart, 2007). Similar to trait NA and other temperament dimensions, all individuals exhibit some degree of trait PA that remains relatively stable across time and situations. Low trait PA is associated with a variety of negative psychological outcomes, including depressive disorders (Naragon-Gainey, Watson, & Markon, 2009; Watson, Gamez, & Simms, 2005) and non-suicidal self-injury (Gratz, 2006). Furthermore, low trait PA is a unique predictor of depressive symptoms over and above the predictive value of high trait NA (Brown et al., 1998; Clark & Watson, 1991; Harding et al., 2014).

Trait PA has typically demonstrated a pattern of correlates opposite to those of trait NA. While high trait NA is considered an affective vulnerability to depressive symptoms, high trait PA may be considered a protective factor against depressive symptoms (Berkman, Glass, Brissette, & Seeman, 2000). Conversely, low trait PA may be considered an affective vulnerability that exerts unique effects in the prediction of depression (Brown et al., 1998; Olino et al., 2011). While high trait NA predicts both depressive and anxiety symptoms, low trait PA uniquely predicts depressive symptoms and thereby differentiates depressive symptoms from most forms of anxiety, since high trait NA is common for both depression and anxiety while low trait PA is unique to depression (Clark & Watson, 1991). Thus, trait NA and trait PA represent distinct constructs that remain relatively stable throughout an individual's lifespan and represent affective predictors of depressive symptoms (Caspi, 2000; Rothbart & Bates, 2006). Trait NA and trait PA are not simply opposing extremes of the same affective dimension.

Rather, high trait NA and low trait PA are distinct affective vulnerabilities to depression, with high trait NA and low trait PA predicting greater depressive symptoms (Riskind, Kleiman, & Schafer, 2013).

While research consistently demonstrates that high trait NA predicts greater depressive symptoms, research on affective vulnerabilities to depression often ignores the contributions of trait PA in the prediction of depression. As a result, less is known regarding the mechanisms by which low trait PA exerts its effect on depressive symptoms. Research has begun to investigate how event-specific NA and event-specific PA impact each other during depressive episodes (Wichers et al., 2012), but the mechanisms through which these affective vulnerabilities interrelate at a trait level to jointly predict depressive symptoms is less understood. However, to understand the development and course of depression, it is imperative that researchers examine potential mechanisms in the relationship between both high trait NA and low trait PA in relation to depressive symptoms. Based on cognitive theory and recent depression research, cognitive mediators of both trait NA and trait PA appear to be a likely pathway linking these affective vulnerabilities to depressive symptoms.

**Cognitive-Affective Theories on Trait Affect and Depression**

Cognitive responses to stressful negative and positive events are a suspected mechanism in the transmission of affective vulnerability to depression (Beck, 1967; Compas, Connor-Smith, & Jaser, 2004; Hyde et al., 2008; Lengua & Long, 2002; Nolen-Hoeksema, 1998). Individual differences in cognitive responses to stress may predispose individuals to becoming depressed, and individuals with high trait NA and low trait PA are more likely to deploy cognitive responses such as brooding in response to stress.

Relatedly, there is also growing evidence that trait NA may predict greater state NA through ruminating in response to stressful events, which in turn predicts depressive symptoms (Mezulis, Hyde, & Abramson, 2006; Simonson, Sanchez, Arger, & Mezulis, 2012). Consequently, the affective vulnerability of high trait NA may predict depressive symptoms by exacerbating the impact of stressors on subsequent negative mood through cognitive responses such as brooding in response to stressful events, which over time can lead to depressive symptoms (Hankin, Fraley, & Abela, 2005; Hyde, et al., 2008). In sum, individual differences in affective vulnerability to depression predict individual differences in cognitive vulnerability to depression (Mezulis, Priess, & Hyde, 2011).

The affective vulnerabilities of high trait NA and low trait PA are known predictors of both depressive symptoms and cognitive responses that themselves predict depressive symptoms. Brooding and positive rumination are two such cognitive responses, with trait NA predicting brooding and trait PA predicting positive rumination (Feldman et al., 2008; Hankin et al., 2009). Greater brooding and less positive rumination then predict greater depressive symptoms by translating trait affective vulnerability into cognitive vulnerability. The prospective relationships from trait affect to depression and ruminative responses to depression are supported, but the cognitive-affective pathways to depressive symptoms for trait NA and trait PA are largely examined as distinct relationships rather than shared pathways to common depressive outcomes. Therefore, a review of theories on depression is provided to better integrate current understanding of vulnerabilities to depression.

**Cognitive theory of depression.** Cognitive-affective theories of depression typically describe trait NA and trait PA as separate affective vulnerabilities to depression

that are mediated by distinct cognitive processes. A prominent theory on cognitive processes in the relationship between trait affect and depressive symptoms is Beck's cognitive theory of depression (1967). Beck states that certain maladaptive cognitive responses to NA-eliciting events may increase vulnerability to depression. This theory assumes that events naturally elicit positive and negative emotional responses, and individuals' cognitive responses to events and their resultant emotions may enhance or diminish their affective responses to those events (Abramson et al., 1999; Nolen-Hoeksema, 1991). For example, Beck (1967) states that individuals typically experience NA when faced with stressors such as a failing exam grade or the loss of a romantic relationship; however, if the individual responds to those stressors with maladaptive cognitive responses, that NA will become exacerbated and intensified over time, predisposing that individual to depression.

Although Beck's cognitive theory primarily applies to the cognitive processing of negative events, Beck and colleagues state in a more recent articulation of this theory that generating positive cognitions additionally may reduce vulnerability to depression (Beck, Rush, Shaw, & Emery, 1979, p. 299). Just as NA may be diminished or enhanced by cognitive processing, PA similarly may be diminished or enhanced by cognitive processing. Hence, cognitive responses to negative and positive events are acknowledged as meaningful actors in the relationship between affect, cognition and depression.

**Response styles theory.** As an extension of Beck's cognitive theory, the response styles theory (Nolen-Hoeksema, 1991) discusses maladaptive cognitive responses to NA and NA-eliciting events that predict depressive symptoms. In particular,

response styles theory emphasizes the maladaptive cognitive response of rumination. Rumination involves a perseverative process of thinking (typically about past or current events), but the content of this perseverative process is independent of the affective valence or details of cognitive content. Rumination describes a cognitive process of repetitive and focused attention that is independent of particular content. Two examples of rumination are brooding and positive rumination, which demonstrate a common perseverative process on negative versus positive content, respectively. Since greater brooding predicts greater depression symptoms and greater positive rumination predicts fewer depressive symptoms, the content of rumination may determine whether perseverative thinking confers vulnerability to depression.

Certain ruminative responses to negative events have been shown to increase vulnerability to depression. Specifically, brooding is a dimension of rumination that is concurrently and prospectively associated with depressive symptoms (Treynor et al., 2003; Siegle, Moore, & Thase, 2004). Brooding describes self-focus on the causes and consequences of NA and negative events. As a result, brooding augments NA by focusing attention on negative mood states and the sources of those mood states. Brooding is associated with significantly greater attentional biases toward negative content, such as sad faces (Joormann, Dkane, & Gotlib, 2006), which devotes cognitive resources to the processing of negative content to exacerbate and sustain the excessive NA that pervades depression. Rumination is a maladaptive cognitive process that predicts depressive symptoms, and brooding is a form of rumination specifically in response to negative emotions. To extend the example provided above, an individual who responded to their exam failure or lost romantic relationship by brooding on that

event and their resultant negative emotions would be more likely to experience longer and more intense NA that is characteristic of depression.

**Broaden-and-build theory of positive emotion.** The broaden-and-build theory is a contemporary cognitive theory that links trait and state PA to a wide range of behaviors and cognitions. This theory suggests that PA may be regulated through cognitive responses to PA-eliciting events. Specifically, this theory asserts that PA promotes building mental and physical resources to widen the range of thoughts and behaviors in which an individual is willing to engage, which may improve resiliency against the development of depressive moods and behaviors. The broaden-and-build theory assumes that depressive symptoms deplete mental and physical resources and encourage rigid cognitive patterns that are maintained by NA (Peterson & Seligman, 1984). Based on this view of depression, low PA may increase depressive symptoms by narrowing attentional biases and reducing access to alternative cognitions. Over time, these cognitive patterns are proposed to alter the experience of both NA and PA. While not limited to depressive symptoms, the broaden-and-build theory predicts that low PA limits the resources available to adaptively endure everyday challenges or appreciate positive events, which may lead to depressive symptoms over time. Consistently high PA may reduce depressive symptoms by widening narrow attentional biases and introducing alternative perspectives to the pessimism that is characteristic of depression. In this way, the relationship between trait PA and depressive behaviors is impacted by cognition.

Taken together, these three cognitive-affective theories of depression posit that the relationship between affect and depressive symptoms is mediated by the cognitive

processing of negative and positive affective content. Beck's cognitive theory of depression (1967) asserts that maladaptive cognitive responses to NA-eliciting events may increase vulnerability to depression over time by intensifying and prolonging NA. This theory is elaborated by response styles theory (Nolen-Hoeksema, 1991), which specifically proposes rumination on negative content as a central maladaptive cognitive response that predicts depression. Similar to theories on NA, the broaden-and-build theory states that cognitive responses to PA-eliciting events predict depression by intensifying or diminishing an individual's experience of PA over time. Although the combined interpretation of these theories highlights important vulnerabilities to depression, each theory largely describes one form of affective vulnerability but neglects to integrate the joint contributions of NA and PA, even though their shared predictions of depressive symptoms are reliably demonstrated from childhood through adulthood (Clark & Watson, 1991; Compas et al., 2004; Wetter & Hankin, 2009). As the literature is presently organized, the isolation of each affective vulnerability into separate models may constrain the utility of cognitive-affective models of depression in understanding how trait affect contributes to depressive symptomatology.

Integrating cognitive-affective models of depression for both NA and PA would likely provide a more complete theoretical paradigm within which to prevent and treat depressive symptoms. In particular, a more integrated cognitive-affective model of the relationship between trait affect and depressive symptoms may reveal partially or fully shared cognitive processes that mediate this relationship. Integrating theoretical explanations of trait NA and trait PA in the transmission of affective vulnerability to depression may provide a more comprehensive picture of how an individual's trait affect

may predict depressive symptoms as mediated by the shared cognitive processing of the affective content of events.

**Cognitive Responses May Mediate the Effect of Trait Affect on Depression**

  **Brooding.** As previously noted, a cognitive response to negative events that is consistently shown to predict depressive symptoms is brooding. Brooding describes a form of rumination that amplifies NA content in response to negative mood states. An example of brooding may include thinking, "I feel so sad. Why can't I be happy like everyone else?" Brooding concurrently and prospectively predicts depressive symptoms in clinical and nonclinical adolescent and adult populations across Western and Eastern cultures (Arger, Sánchez, Simonson, & Mezulis, 2012; Burwell & Shirk, 2007; Ito, Takenaka, Tomita, & Agari, 2006; Mezulis et al., 2011). Brooding is also related to other internalizing symptoms, including suicidal ideation (Miranda & Nolen-Hoeksema, 2007) and anxiety symptoms (Olatunji, Naragon-Gainey & Wolitzsky-Taylor, 2013). Extensive research has also shown that trait NA predicts brooding, such that individuals already high in NA may be particularly likely to employ this maladaptive cognitive response to negative events and emotions (Arger et al., 2012; Mezulis et al., 2011).

  **Positive rumination.** As a more adaptive counterpart to brooding, positive rumination may be considered a form of rumination that amplifies positive affective content to predict fewer depressive symptoms. Positive rumination involves perseverative self-focused attention to one's own thoughts, feelings, and behaviors in reference to positive events (Feldman et al., 2008). Examples of positive rumination include focusing attention on personal strengths and current experiences of positive emotion (Martin & Tesser, 1996), such as thinking, "I did well on that project because I

worked so hard!  It feels great to finish such a large task."  Individuals who respond to positive events through positive rumination report greater PA compared to individuals who do not positively ruminate (Quoidbach, Berry, Hansenne, Mikolajczak, 2010).  Positive rumination is proposed to alter PA, but the likelihood of an individual engaging in positive rumination may be predicted by trait PA.  Specifically, high trait PA predicts greater positive rumination, such that individuals already high in trait PA may be particularly likely to employ this adaptive cognitive response to positive events while individuals low in trait PA may be less likely to utilize positive rumination (Harding et al., 2014).

In contrast to brooding, positive rumination predicts *fewer* depressive symptoms due to a tendency to increase event-specific PA.  In fact, positive rumination is concurrently associated with fewer depressive symptoms over and above brooding and is negatively correlated with brooding across multiple studies of depression (Bijttebier, Raes, Vasey, & Feldman, 2012; Feldman et al., 2008; Raes, Daems, Feldman, Johnson, & Van Gucht, 2010).  Despite the seemingly adaptive finding of positive rumination predicting fewer depressive symptoms, positive rumination is also associated with the maladaptive outcome of predicting greater hypomanic symptoms among young adults (Feldman et al., 2008; Johnson, McKenzie, & McMurrich, 2008; Raes et al., 2010).  Consequently, positive rumination may predict mood symptoms within the broader spectrum of depression to mania.  Although an excess of NA and lack of PA predict unipolar mood disorder, an excess of PA can predict a fewer depressive symptoms or greater bipolar mood symptoms.  However, the focus of the current study was limited to depressive symptoms.

Whereas positive rumination has been investigated in only a handful of recent studies, it provides an adaptive cognitive response to positive events that predicts fewer depressive symptoms. Positive rumination is shown to amplify state PA, which in turn predicts fewer depressive symptoms by reducing anhedonia and potentially offering resiliency against the experience of state NA (Fredrickson, 2001; Quoidbach et al., 2010). Whereas brooding predicts greater depressive symptoms through amplifying event-specific NA, positive rumination may predict fewer depressive symptoms through amplifying event-specific PA.

**Brooding and Positive Rumination May Represent Similar Forms of Affect Amplification**

Brooding and positive rumination are forms of ruminative cognitive processing that amplify affective content and significantly contribute to depressive symptomatology. As such, both cognitive responses may be described as cognitive processes of affect amplification (Weitzman, McHugh, & Otto, 2011). Although brooding is defined as exclusively pertaining to NA and positive rumination is defined as exclusively pertaining to PA, the underlying process of affect amplification may jointly mediate trait NA and trait PA in the prediction of depressive symptoms. That is, the amplification of affective content may be a cognitive process that is common to both trait NA and trait PA in the prediction of depression.

Affect amplification refers to the process of directing attention to the affective content of events, which results in increasing the intensity of an individual's affective experience regardless of the valence of the affect. The amplification of NA is often described as brooding, while the amplification of PA is often described as positive

rumination.  However, distinguishing brooding and positive rumination may impose unnecessary distinctions in the relationship between trait affect and depression.  This is because they may represent the same cognitive process operating on distinct affective content, with brooding increasing negative emotions and positive rumination increasing positive emotions.  Brooding and positive rumination both represent the perseverative processing of affective content that may be described as a common cognitive process of affect amplification. Alternatively, these constructs may represent unique cognitive processes with different downstream effects on affect.

Cognitive processes in the relationship between trait affect and depressive symptoms have been separately considered for trait NA and trait PA, but investigation of a shared affect amplification process is yet unexplored.  Both cognitive responses may represent the same underlying cognitive process, which could help unify depression literature in understanding how trait affect predicts depression outcomes through shared language.  However, the common cognitive process of affect amplification is distinct from the valence of the affective content amplified, meaning that individuals may be predisposed to depressive symptoms by cognitively focusing on negative content instead of positive content.  For example, an individual who is temperamentally high in trait NA but low in trait PA may exhibit a shared amplification process, but their temperament may bias cognitive processing toward negative events and provide fewer opportunities to amplify affect. If a shared cognitive process does exist across affective content, distinct constructs for NA and PA may pose unnecessary divisions in the discussion of affective vulnerability to depression.  The similar perseverative cognitive process (i.e., rumination) that describes brooding and positive rumination is one reason to consider shared variance

between constructs. In other words, it may be the individuals are prone to perseverative

thought regardless of the valence of content. Another reason is that the correlation

between brooding and positive rumination ($r$ = .19-.34) after controlling for depressive

symptoms in a previous study (Feldman et al., 2008) indicates a degree of overlap.

However, the opposing effects of brooding and positive rumination in predicting

depressive symptoms in previous literature and the fact that the correlation between

brooding and positive rumination has not been of large magnitude alternatively suggest

that these constructs represent distinct variance in the prediction of depressive symptoms.

As a result, affect amplification may offer a unifying cognitive process to jointly describe

overlapping but distinct cognitive mechanisms in the transmission of affective

vulnerability to depression.

**The Current Study**

The present study consists of two parts. My hypotheses were examined in a

short-term prospective study among young adults with trait affect and cognitive

responses measured at baseline and depressive symptoms measured seven weeks after

baseline assessment. Part 1 compared three structural equation models (SEM)—

specifically, measurement models—to examine whether brooding and positive

rumination, which were measured at baseline assessment, are best conceptualized by a

single latent construct of amplification. Model 1 assumed that five brooding items would

load onto the latent construct of negative amplification while nine positive rumination

items would load onto the separate latent construct of positive amplification, with no

covariance between these factors (see Figure 1). In contrast, Model 2 (see Figure 2)

proposed that five brooding items would load onto the latent construct of negative

amplification while nine positive rumination items would load onto the separate but *related* latent construct of positive amplification (permitting covariance). Finally, Model 3 proposed that the latent constructs of negative amplification and positive amplification would be best represented by a single latent construct of affect amplification (see Figure 3). In summary, I compared three models: Model 1 assumed separate, uncorrelated factors, Model 2 assumed separate factors that were allowed to covary, and Model 3 assumed that both factors represented the same, shared construct. I hypothesized that Model 2 would provide the best fit to the data, supporting the idea of a partially shared cognitive process of affect amplification that is further specified into amplification of positive or negative content. Model 2 is consistent with the above rationales for both shared variance and distinct variance in considering the relationship between brooding and positive rumination. In contrast, Model 1 represented distinct cognitive processes while Model 3 represented fully shared cognitive processes.

Thereafter, Part 2 of my dissertation examined the cognitive responses of brooding and positive rumination as potential mediators of the relationship between trait affect and depressive symptoms through a SEM mediation model. The best-fitting measurement model from Part 1 was included as the mediation model (see Figure 4). I hypothesized that (a) high trait NA would predict greater brooding and less positive rumination, (b) low trait PA would predict greater brooding and less positive rumination, (c) greater brooding would predict greater depressive symptoms, and (d) less positive rumination would predict greater depressive symptoms. I predicted that trait NA and trait PA would distinctly predict both forms of rumination given my hypothesis that brooding and positive rumination represent distinct but related constructs. Given the shared

variance between trait NA and trait PA as well as the shared variance between brooding and positive rumination, I predicted that the distinctions between negative and positive cognitive-affective pathways to depression ignore the shared variance of vulnerability and resilience pathways. I additionally hypothesized that brooding and positive rumination would mediate the relationships between both trait NA and trait PA in predicting depressive symptoms, representing a shared affect amplification process in the prediction of depression. Given the hypothesized shared variance between brooding and positive rumination in predicting depressive symptoms, we similarly hypothesized that both forms of rumination would be predicted by trait NA and trait PA distinctly.
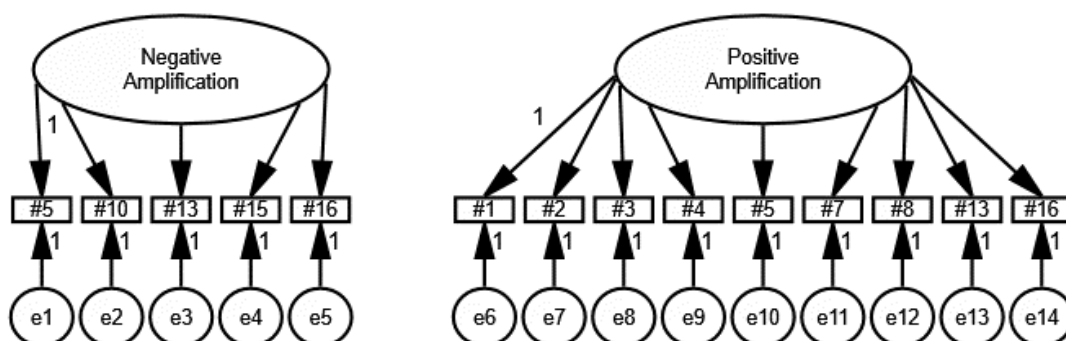


*Figure 1.* Model 1 of brooding and positive rumination representing the distinct processes of negative amplification and positive amplification.
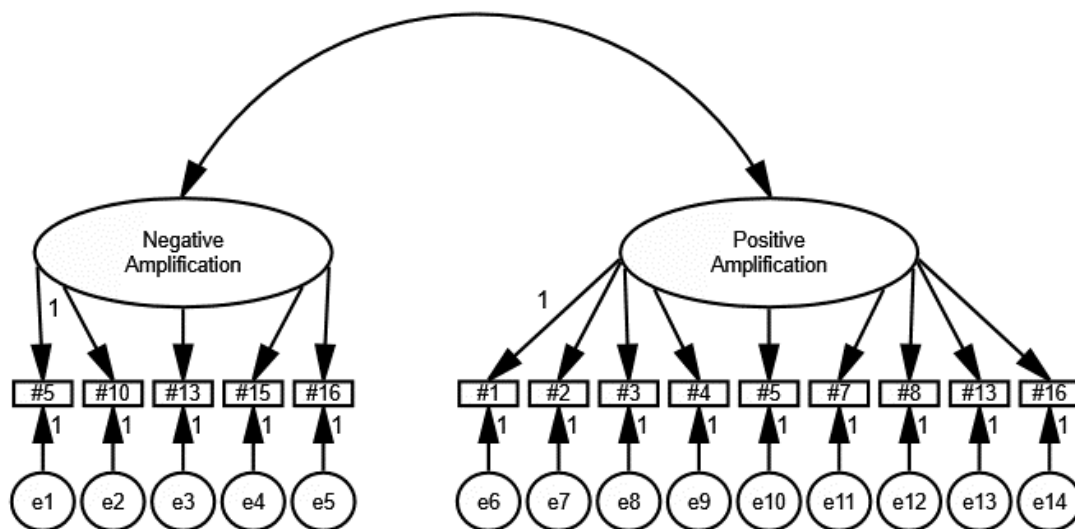
*Figure 2.* Model 2 of brooding and positive rumination representing the distinct but related processes of negative amplification and positive amplification.



*Figure 3.* Model 3 of brooding and positive rumination representing the common process of amplification.

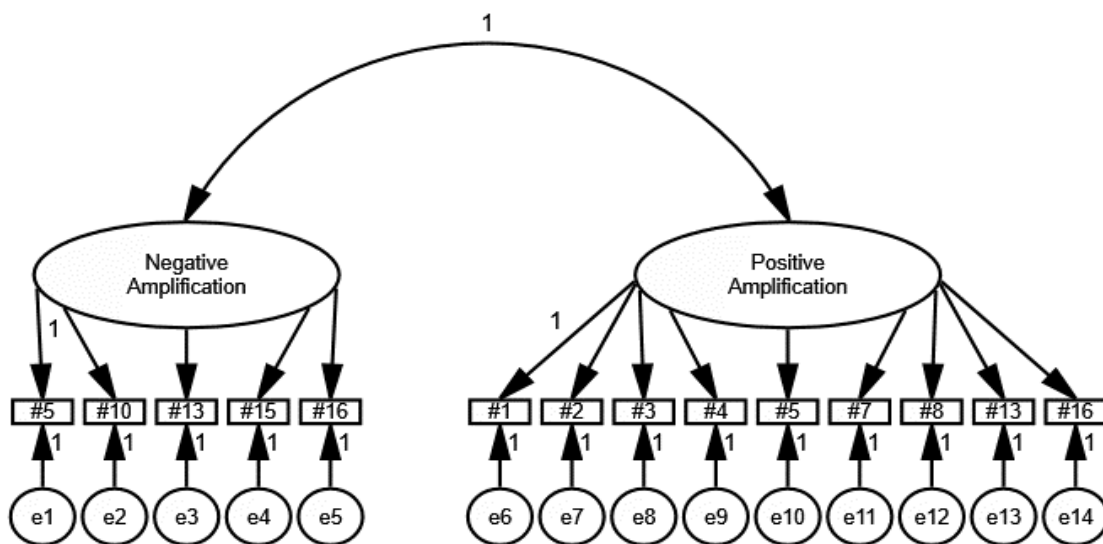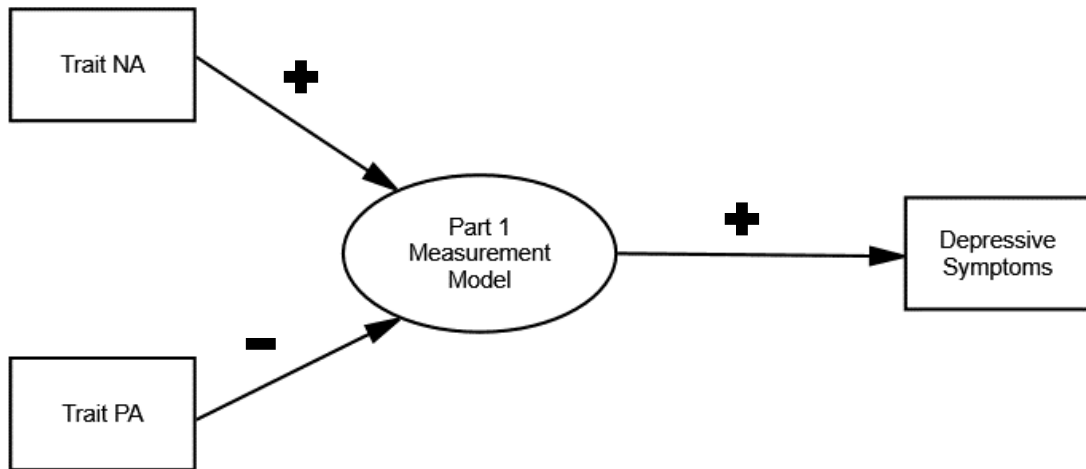*Figure 4.* Model 4 of brooding and positive rumination mediating the relationship between trait affect and depressive symptoms through the distinct but related processes of negative amplification and positive amplification.

## CHAPTER II

## Method

### Participants and Sampling

**Participants.**  I recruited undergraduate students from Seattle Pacific University to participate in the PACE (Positive Affect and College Events) Study.  Participants were at least 18 years old to exclude the need for parental consent and ensure that the sample represents a young adult population.  Participants were compensated through five research participation credits, which was a university course requirement.  No upper age cut-offs or exclusion criteria were imposed for participation.

Based on feasibility and power analysis, I proposed a sample size of at least 300 participants.  Referencing my most complex model, a power analysis for a SEM with two latent variables (brooding and positive rumination) and 18 observed predictors (five brooding items, nine positive rumination items, trait NA, trait PA, and depressive symptoms at weeks 1 and 8) calculated that a minimum sample size of 150 participants would be required to detect a small effect size (.10) with a power of .80 and probability level of .05 (Soper, 2013).  This calculation is derived from the recommendations of Westland (2010) on the acceptable minimum sample size requirements for SEM.

Participants were 321 (73.5% female) undergraduate students from Seattle Pacific University (SPU) with an age range of 18-29 ($M = 19.03$, $SD = 1.64$).  Approximately 70.40% of participants were Caucasian American, 2.80% were African American, 16.5% were Asian American, 0.60% were Native American, 4.70% were Hispanic/Latino American, and 5.00% identified as another or multiple cultural backgrounds.  SPU reported the Autumn 2013 student population as consisting of 68% female students with

an average of 21 years and predominantly Caucasian (68%) ethnicity, so the collected sample is representative of the larger SPU population.

**Measures**

**Depressive symptoms.** Depressive symptoms were measured at baseline as a covariate and seven weeks later as the dependent variable with the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The CES-D is a 20 item measure of depressive symptoms that is intended for both clinical and nonclinical adult populations. Participants responded to items that are written in the first person by rating how they felt and behaved in the past week. Responses range from 0 *(rarely or none of the time)* to 3 *(most or all of the time)* for items such as, "I was bothered by things that usually don't bother me" and "I felt hopeful about the future." A mean score was calculated by averaging all 20 item ratings, with four items requiring reverse-scoring. A cutoff total score of 16 for the CES-D is suggested to indicate the presence of clinically significant depressive symptoms, with higher scores shown to significantly relate to a greater severity of depressive symptoms (Ensel, 1986).

The CES-D was fielded on three samples: a nonclinical adult sample in Kansas City, Missouri and Washington County, Maryland ($N = 2514$), a nonclinical adult sample in Washington County, Maryland only ($N = 1060$) and an adult sample of psychiatric patients in Kansas City, Missouri and New Haven, Connecticut ($N = 70$). Results were only provided for Caucasian individuals, since one of the samples contained 3% non-Caucasian individuals and the authors stated that they wanted the sample to be more demographically comparable. Additionally, demographic information was provided in a separate publication and did not provide distinct demographic information for each

sample.  All four samples (*N* = 3845) were primarily of Caucasian ethnicity (92.33%) and

female gender (58.70%).  However, the CES-D is also psychometrically validated cross-

culturally (Cheung & Bagley, 1998; Privado & Garrido, 2013).  No means or standard

deviations were provided for the samples (Comstock & Helsing, 1976).

Cronbach's alpha coefficients for the CES-D were reported as .85 in the

nonclinical samples and .90 in the psychiatric sample.  Additionally, test-retest reliability

tested from two weeks to 12 months ranged from .45-.70 (Radloff, 1977).  The CES-D

showed convergent validity with other depression measures, including the Depression

Adjective Checklist (*r* = .37-.70, *p* < .01; Lubin, 1981) and Bradburn Affect Balance

Scale (*r* = .61-.72, *p* < .01; Bradburn, 1969).  Confirmatory factor analyses (CFA) also

supported strong structural validity (Orme, Reis, & Herz, 1986) and a stronger mean item

correlation (*r*[114] = .52, *p* < .01) with the total score in comparison to measures of self-

esteem (*r*[114] = .34, *p* < .01; Rosenberg, 1965), state anxiety (*r*[114] = .41, *p* < .01;

Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), and trait anxiety (*r*[114] = .25, *p*

< .01; Spielberger et al., 1983).  In my study, the internal consistency for depressive

symptoms was α = .88 at baseline and .86 at follow-up seven weeks later.

**Trait NA.**  Trait NA was measured at baseline with the NA subscale of the Adult

Temperament Questionnaire (ATQ; Evans & Rothbart, 2007), which is a 51 item

measure of trait negative emotion.  Participants responded to items that are written in the

first person by rating how well each statement describes them.  Responses range from 1

*(extremely untrue of you)* to 7 *(extremely true of you)* for items such as, "I become easily

frightened" and "I often get irritated when I'm trying to make an important phone call and

get a busy signal."  A mean score was calculated for this subscale, ranging from 1 to 7,

by reverse-scoring certain items and averaging all 51 item ratings, with higher scores representing higher trait NA.

The Cronbach's alpha coefficient for the four subscales that comprise the larger NA subscale ranged from .76 to .86 in a nonclinical university sample similar to my sample (Evans & Rothbart, 2007). In the same study, the NA subscale also demonstrated strong convergent validity with the Big Five personality factor of Neuroticism ($r = .74$). Overall, CFA supported strong structural validity for the NA subscale as a distinct trait. In the study, the internal consistency for trait NA was $\alpha = .88$.

**Trait PA.** Trait PA was measured at baseline with the PA subscale of the Adult Temperament Questionnaire (ATQ; Evans & Rothbart, 2007), which is an 11 item subscale measuring PA within the larger dimension of Extraversion/Surgency. Participants responded to items that are written in the first person by rating how well each statement describes them. Responses range from 1 *(extremely untrue of you)* to 7 *(extremely true of you)* for items such as, "I rarely feel happy" and "When I don't feel unhappy, I usually feel happy instead of neutral." A mean score was calculated for this subscale, ranging from 1 to 7, by reverse-scoring certain items and averaging all 11 item ratings, with higher scores representing higher trait PA.

The Cronbach's alpha coefficient for the Extraversion/Surgency subscale that includes the PA subscale was reported as .84 in a nonclinical university sample (Evans & Rothbart, 2007), similar to the present study ($\alpha = .81$). In the same study, the PA subscale demonstrated discriminant validity with the NA subscale ($r = -.20$) and the Extraversion/Surgency scale that contains the PA subscale demonstrated strong

convergent validity with the Big Five personality factor of Extraversion ($r = .67$).

Overall, CFA supported strong structural validity for the PA subscale as a distinct trait.

**Brooding.** Brooding was measured at baseline with the five item brooding subscale of the Ruminative Responses Scale (RRS; Nolen Hoeksema, 1991), which is part of a 22 item measure of ruminative responses to negative emotions. The RRS additionally includes items for a reflection subscale and a depressive-related subscale, but neither subscale was included since reflection items are inconsistently predictive of depressive symptoms and depression-related items share considerable variance with the outcome of depressive symptoms. Participants responded to five items that are written in the first person by rating how often they generally think or do each statement when they feel down, depressed, or sad. Responses range from 1 *(never)* to 4 *(always)* for items such as, "Think 'What am I doing to deserve this?'" and "Think about a recent situation, wishing it had gone better." A mean score of all five brooding items was calculated, ranging from 1 to 4, with higher scores reflecting greater brooding.

The Cronbach's alpha coefficient for the brooding subscale ranged from .72 to .78 across university samples (Olson & Kwon, 2008; Surrence, Miranda, Marroquín, & Chan, 2009; Treynor et al., 2003), similar to the present study ($\alpha = .80$). The brooding subscale also demonstrated test-retest reliability of .62, which indicated a weaker internal consistency compared to the RRS that the authors attributed to the smaller item pool (Treynor et al., 2003). The brooding subscale demonstrated concurrent ($r = .56$) and predictive ($r = .49$) validity with the Beck Depression Inventory, Second Edition (BDI-II; Beck, Steer, & Brown, 1996), which supports a strong relationship with depressive symptoms (Olson & Kwon, 2008).

**Positive rumination.** Positive rumination was measured at baseline with the nine positive rumination items of the Response to Positive Affect Scale (RPA; Feldman et al., 2008), which is a 17 item scale on ruminative responses to positive emotions. Although the RPA contains two separate subscales on positive rumination (self-focused and emotion-focused positive rumination), they were combined in analyses due to their strong correlation ($r = .50$) and my theoretical interest in the construct of overall positive rumination. Self-focused positive rumination describes "rumination on aspects of self and pursuit of personally relevant goals," while emotion-focused positive rumination describes rumination on mood and somatic experiences" (Feldman et al., 2008, p. 5). Participants responded to nine items that are written in the first person by rating how often they generally think or do each statement when they are feeling happy. Responses range from 1 *(almost never)* to 4 *(almost always)* for items such as, "When you are feeling happy, how often do you think about how happy you feel?" and "When you are feeling happy, how often do you think 'I am living up to my potential'?" A mean score of all nine positive rumination items were calculated, ranging from 9 to 36, by reverse-scoring certain items and summing all item ratings, with higher scores reflecting greater positive rumination.

The Cronbach's alpha coefficients for positive rumination were .73 to .76. In convergent and discriminant validity analyses, emotion-focused and self-focused positive rumination scores were associated with brooding ($r = .10$, $p > .05$, $r = .27$ $p < .05$) and depressive symptoms (r = -.15, $p < .05$; r = -.07, $p > .05$) respectively (Feldman et al., 2008). According to incremental validity analyses, responses on the RPA predicted 10% of depressive symptom variability above and beyond brooding, with positive rumination

predicting significantly fewer depressive symptoms (Feldman et al., 2008).  In my study, the internal consistency for positive rumination was $\alpha = .83$.

**Procedure**

Participants were recruited via in-class presentations and contacted via email with more information about the study and a link to the baseline questionnaire.  Eligible participants provided consent and completed an online baseline questionnaire administered through SurveyMonkey or Qualtrics.  The baseline questionnaire included measures of trait NA, trait PA, brooding, positive rumination, and depressive symptoms.

Participants who completed the baseline questionnaire on trait affect, brooding, positive rumination, and depressive symptoms were invited to complete a second online assessment of their depressive symptoms seven weeks later via SurveyMonkey or Qualtrics.  Data collection occurred in the fall quarter of 2012, winter quarter of 2013, fall quarter of 2013, winter quarter of 2014 and spring quarter of 2014 for a total of five 8-week quarters of data collection as part of a larger data collection collaboration across research labs.  Most data from this larger collection was not used, although relevant data on demographics, trait affect, rumination, and depressive symptoms were included across all five quarters.  Surveymonkey was utilized for the first and second collection quarters while Qualtrics was used in the third, fourth and fifth collection quarters.  Measurement items were identical across all data collection waves.  See Appendix A for the 2012-2013 Institutional Review Board (IRB) Application and Appendix B for the 2013-2014 IRB Application.

# CHAPTER III

## Results

**Data Analytic Plan**

Data analyses were conducted with AMOS 21.0. Part 1 conducted a CFA of baseline measures of brooding (five items) and positive rumination (nine items), sequentially testing three nested models as proposed. The CFA compared the hypothesized models to determine the best model fit to the data. Model fit was evaluated through comparing chi square difference tests and model fit indices between models to determine which model demonstrated significantly improved fit based on relative changes in chi square difference tests and model fit indices within recommended limits (Byrne, 2010, pp. 53-95).

Thereafter, Part 2 examined the relationship between baseline trait NA and trait PA as well as depressive symptoms seven weeks after baseline assessment as mediated by the best fitting model in Part 1. Part 1 examined baseline brooding and positive rumination while Part 2 examined baseline trait affect, brooding, and positive rumination in the prediction of depressive symptoms seven weeks later. In Part 2, I generated 1,000 bootstrap samples with 95% bias-corrected confidence intervals and bootstrap estimates of indirect, direct, and total effects, which maximizes statistical power by computing non-symmetric confidence intervals and reducing Type II error (Mallinckrodt, Abraham, Wei, & Russell, 2006). The Part 2 design allowed prospective predictions that invite causal inferences by establishing temporal precedence between baseline trait affect, baseline cognitive responses, and subsequent depressive symptoms at week eight. I controlled for baseline depressive symptoms as a covariate in Part 2 to ensure that depressive symptoms at baseline assessment were not accounting for the prediction of depressive symptoms

seven weeks later (see Table 1). In Part 2 I also included trait NA, trait PA, positive

rumination, and brooding in the same SEM model to control for their combined effects

and ensure that I measured the distinct contributions of each variable in predicting

depressive symptoms at week eight.

Table 1

*Data Analytic Plan*

| Hypotheses | Statistical Techniques | Independent Variables | Dependent Variable | Covariates |
|---|---|---|---|---|
| **Part One** | | | | |
| (1) Model two will provide the best fit to the data. | SEM CFA with 3 models and parceling | Brooding (week 1; RRS 5 items) Positive rumination (week 1; RPA 9 items) | None | None |
| **Part Two** | | | | |
| (1) Greater trait NA will predict greater brooding and less positive rumination. (2) Less trait PA will predict greater brooding and less positive rumination. (3) Greater brooding will predict greater depressive symptoms. (4) Less positive rumination will predict greater depressive symptoms. (5) Brooding and positive rumination will mediate the relationship between trait affect and depressive symptoms. | SEM Mediation | Brooding (week 1; RRS 5 items) Positive rumination (week 1; RPA 9 items) | Depressive symptoms (week 8; CES-D, 20 items) | Depressive symptoms (week 1; CES-D, 20 items) |

*Note.* SEM = Structural Equation Modeling; CFA = Confirmatory Factor Analysis; RRS

= Ruminative Responses Scale; RPA = Response to Positive Affect Scale; CES-D =

Center for Epidemiologic Studies Depression Scale.

**Data Preparation and Descriptive Analyses**

Data first were examined to ensure that all parametric assumptions were met. In support of the normality assumption, variable skewness and kurtosis were all within recommended ranges (Kline, 2005) and histogram examination corroborated normally distributed data. In support of the homogeneity of variance assumption, scatterplots of standardized variables with corresponding residuals supported homogeneously distributed variance. Two participants were excluded from analyses due to their ages of 31 and 41 exceeding the young adult age range of the remaining sample. Scaled scores were then computed for all variables, resulting in a final sample size of 321 participants. Variable correlations, means, and standard deviations for study variables are presented in Table 2.

Missing data were handled through multiple imputation in the Statistical Package for the Social Sciences (SPSS) 21.0 for participants who completed at least 80% of a given measure (Eekhout et al., 2014). I conducted five multiple imputations and selected one to import into AMOS. Missing data analyses in SPSS 21.0 indicated that 0.61% of data were missing at week 1 and 0.33% of data were missing at week 8 for a combined 0.56% of missingness across weeks. Data were missing completely at random (MCAR) for both weeks as indicated by a non-significant Little's MCAR tests (week 1 $\chi^2$[3808] = 3699.08, $p$ = .895; week 8 $\chi^2$[139] = 162.977, $p$ = .080). There was a subset of 20 participants for whom the short form of the CES-D (9 of the original 20 items) was administered, but there were no missing data for this subset. The mean score for this short form was weighted to be comparable with the full form CES-D scores of the

remaining sample.  Data therefore were considered MCAR overall and multiply imputed

to maximize available power to detect significant effects.

Table 2

*Variable Correlations, Means, and Standard Deviations*

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | *M* | *SD* |
|---|---|---|---|---|---|---|---|---|
| 1. Week 1 CES-D | | | | | | | 14.41 | 9.09 |
| 2. Week 1 NA | .55** | | | | | | 4.00 | 0.65 |
| 3. Week 1 PA | -.47** | -.41** | | | | | 5.09 | 0.88 |
| 4. Week 1 BR | .44** | .51** | -.30** | | | | 9.70 | 2.98 |
| 5. Week 1 PR | -.05 | .03 | .27** | .17** | | | 21.87 | 4.70 |
| 6. Gender | .17** | .34** | .08 | .14* | .13* | | 1.74 | 0.44 |
| 7. Week 8 CES-D | .49** | .40** | -.33** | .35** | .02 | .10 | 12.84 | 9.93 |

*Note.* CES-D = Center for Epidemiologic Studies Depression Scale; NA = Negative

Affect; PA = Positive Affect; BR = Brooding; PR = Positive Rumination; Gender = 1 is

male, 2 is female. * $p < .05$, ** $p < .01$, *** $p < .001$.

**Part 1: Confirmatory Factor Analyses**

CFA in AMOS 21.0 compared three SEM models to determine whether baseline

brooding and positive rumination were best conceptualized as two distinct factors (Model

1), two related factors (Model 2), or a single factor (Model 3).  I first examined chi square

values between models, which indicated that Model 2 best fit the data (i.e., closest to 0)

and fit the data significantly better than models 1 or 3 (i.e., the chi square difference tests

comparing models 2 vs. 1 and models 3 vs. 2 were statistically significant in favor of

model 2). See Table 3 for fit indices.  I then examined the Comparative Fit Index (CFI)

for each model, which also indicated that Model 2 best fit the data but was below the

recommended cutoff for superior model fit (i.e., CFI of .95 or above indicates superior

model fit; Byrne, 2010).  Similarly, the Root Mean Square Error of Approximation

(RMSEA) supported mediocre model fit and was above the recommended cutoff of .05

for below to support superior model fit (<.05 is superior model fit, .05-.10 indicates

mediocre model fit, Byrne, 2010). However, Model 2 (RMSEA = .070) demonstrated

slightly better model fit compared to Model 1 (RMSEA = .072) and demonstrated

considerably better fit compared to Model 3 (RMSEA = .12). Lastly, Model 2 best fit the

data based on the Akaike information criterion (AIC) and Bayesian Information Criterion

(BIC), which demonstrated slightly lower values than Model 1 and considerably lower

values than Model 3 (lower values indicate better fit). Model 2 fit significantly better

than Model 1, whereas constraining the latent factors to equality in Model 3 led to a

significant loss of fit relative to Model 2. Across model fit indices, Model 2 was

supported as the best fitting model to the data, which was consistent with my initial
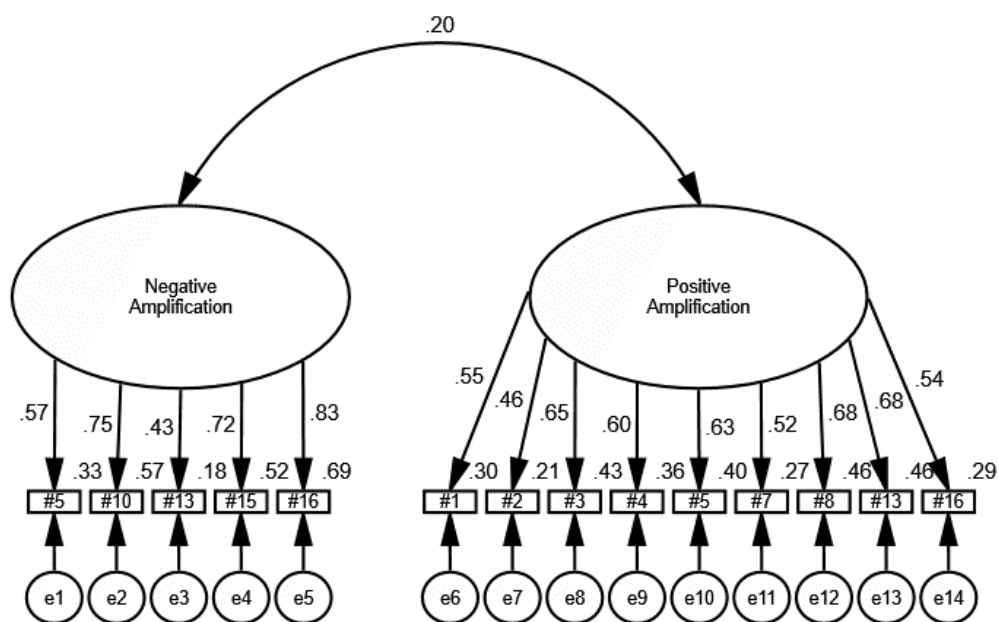
hypothesis (see Table 3).



*Figure 5.* Model 2 loadings of brooding and positive rumination representing the distinct

but related processes of negative amplification and positive amplification.

Table 3

*Part 1 Model Comparisons*

| | $\chi^2$ | *df* | Models | $\Delta\chi^2$ | $\Delta df$ | CFI | RMSEA | AIC/BIC |
|---|---|---|---|---|---|---|---|---|
| Model 1 | 203.85** | 77 | | | | .90 | .07 | 259.85/365.45 |
| Model 2 | 195.07** | 76 | 1 vs. 2 | -8.78* | -1 | .91 | .07 | 253.07/362.44 |
| Model 3 | 422.26** | 77 | 1 vs. 3 | 218.41** | 0 | .73 | .12 | 478.26/583.86 |

\* $p < .001$, \*\* $p < .0001$.  Cutoff for 1 $df = 3.841$.

Due to the marginal but comparatively best fit of Model 2, I examined the modification indices for this model to determine the potential for theory-consistent modifications.  Modification indices displayed the greatest improvements in model fit if the error terms of certain items within the RRS and the residual terms of items within the RPA were allowed to covary.  In addition, allowing covariance between the error terms of RPA item 7 (Think about how happy you feel) and item 8 (Think about how strong you feel) as well as covariance between the error terms of RPA item 13 (Think "I am achieving everything") and item 16 (Think about how proud you are of yourself) suggested improved model fit.  Lastly, allowing covariance between RRS item 15 (Why do I have problems other people don't have) and RPA item 1 (Think about how full of energy you feel) suggested improved model fit.  While allowing error covariances within the RRS and within the RPA slightly improved model fit (CFI=.97 and RMSEA=.04 with all modification indices added), there was no theoretical justification for adding error covariances between these items but not adding error covariances for all remaining items.  As a result, no modification indices were added to Model 2.

**Part 2: Mediation Analyses**

After establishing Model 2 as the best fitting conceptualization of brooding and positive rumination in Part 1, consistent with the idea of distinct but covarying latent

brooding and positive rumination factors, Part 2 examined baseline measures of trait NA, trait PA, brooding, and positive rumination in the prediction of depressive symptoms seven weeks later through an SEM mediation model (see Figure 6). The indirect effects of each mediator were calculated and the contributions of the opposite mediator were controlled in analyses. Baseline depressive symptoms were controlled and all variables were simultaneously entered into one SEM. Phantom variables were modeled to accommodate multiple predictor and mediator variables in the same model (see Figure 7). Phantom variables allowed the calculation of distinct indirect effects for each mediator in the relationships between trait NA and depressive symptoms and between trait PA and depressive symptoms. Their pathways are constrained to equal the variable pathways of the observed model, so they do not require additional degrees of freedom (Macho & Ledermann, 2011). Part 2 results are in provided Table 4.

If Model 1 had demonstrated the best model fit, SEM analyses would have consisted of two distinct SEM models with one mediator in each model and no covariances between the disturbance terms for each latent mediator variable. There would be separate models for each mediator because no correlation between brooding and positive rumination would be assumed. If Model 3 had demonstrated the best fitting model, SEM analyses would have consisted of a combined model that is identical to Model 2, but Model 3 would have examined the shared contributions of brooding and positive rumination rather than the distinct contributions of each mediator controlling for the effects of the other provided in Model 2. Models 2 and 3 would be structured identically in SEM, but Model 2 would interpret the distinct effects of brooding and positive rumination while Model 3 would interpret the effects of brooding and positive

rumination with their covariance constrained to 1. In summary, Model 1 as a mediator would be represented as two distinct models with one mediator in each model, Model 2 would be represented as a combined model to examine the distinct effects of negative and positive amplification, and Model 3 would be represented as a combined model but would examine the shared effects of both mediators as a global process of affect amplification. All three versions of the Part 2 mediation analyses would include phantom variables due to the presence of two independent variables (i.e., trait NA and trait PA).

We examined mediation relationships through a bias-corrected bootstrapping approach (Shrout & Bolger, 2002) to test for the significance of SEM indirect effects in Model 4. Based on this approach, I generated 1,000 bootstrap samples with 95% bias-corrected confidence intervals and bootstrap estimates of indirect, direct, and total effects. I also examined the confidence intervals surrounding each effect to ensure that the interval range did not include zero and thus indicate non-significance. The relationships between trait affect and depressive symptoms were determined to be mediated by brooding and positive rumination if (a) the direct effect value decreased compared to the total effect value, (b) the indirect effect was statistically significant, and (c) the confidence intervals for the indirect effect did not contain zero (Hayes, 2013; Shrout & Bolger, 2002). My hypotheses proposed four separate mediation relationships, with baseline trait NA and trait PA predicting depressive symptoms at week 8 as mediated by baseline brooding and positive rumination.
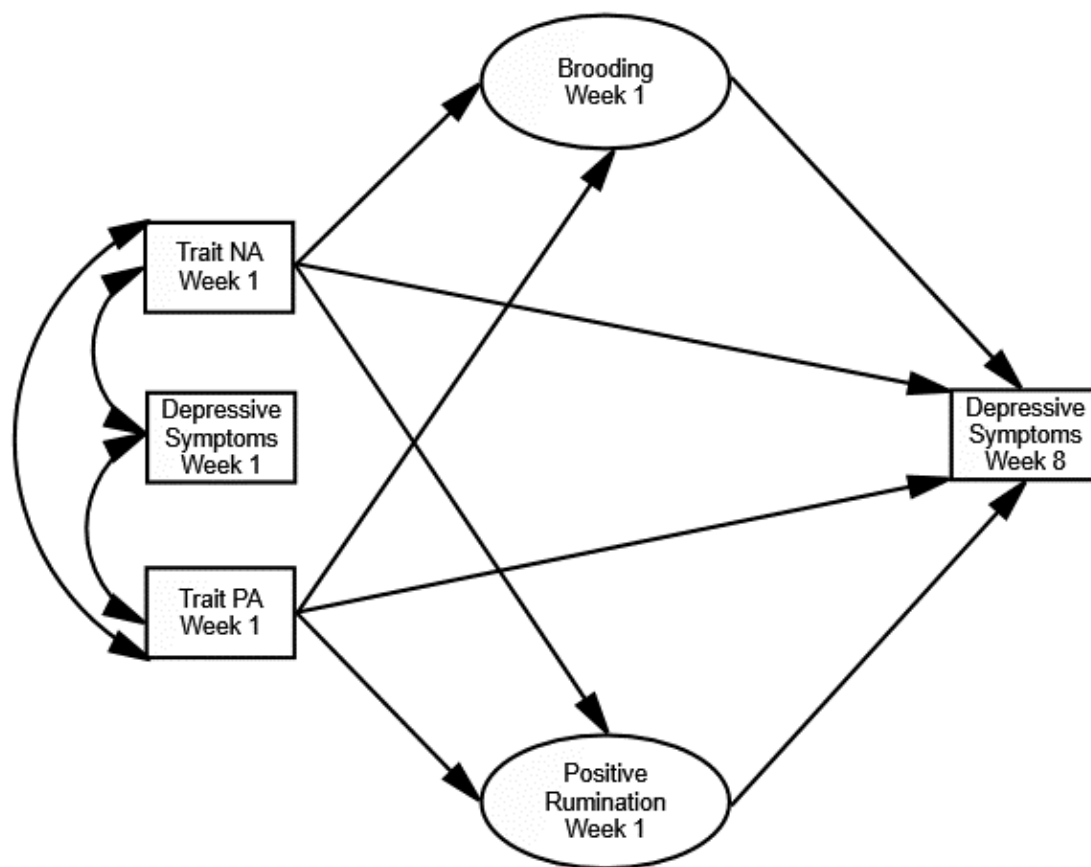
*Figure 6.* Model 4 of brooding and positive rumination mediating the relationship between trait affect and depressive symptoms with Model 2 as the mediator.

**Model fit**

Model fit indices for model 4 indicated adequate to good overall fit to the data ($\chi^2$ [127] = 313.04, $p < .001$, CFI = .90, RMSEA = .07). Specifically, the CFI for Model 4 was below acceptable limits to support superior model fit (Byrne, 2010). The RMSEA indicated mediocre model fit (Byrne, 2010). Taken together, Model 4 fit to the data marginally supported interpretation of the structural model and subsequent mediation analyses.

**Do greater trait NA and less trait PA predict greater depressive symptoms?**

Before examining the accuracy of my mediation hypotheses, I first established whether greater trait NA and less trait PA predicted greater depressive symptoms at week eight. In support of my hypotheses, greater trait NA and less trait PA uniquely predicted greater depressive symptoms. Findings additionally support significant predictive relationships between trait affect and depressive symptoms, since (a) trait affect and depressive symptoms statistically correlated, (b) trait affect temporally preceded depressive symptoms, and (c) third variable explanations were mitigated through the covariates of baseline depressive symptoms and the alternative form of trait affect in analyses (i.e., trait NA was controlled in trait PA analyses and trait PA was controlled in trait NA analyses; Field, 2009, pp. 173-174).

**Do greater trait NA and less trait PA predict greater brooding and less positive rumination?**

Consistent with hypotheses, greater trait NA predicted greater brooding and less positive rumination. Also consistent with hypotheses, less trait PA predicted greater brooding and less positive rumination (see Table 4).

**Do greater brooding and less positive rumination predict greater depressive symptoms?**

Partially consistent with hypotheses, greater brooding predicted greater depressive symptoms, but positive rumination did not significantly predict depressive symptoms in either direction (see Table 4).

**Do greater brooding and less positive rumination mediate the relationship between trait affect and depressive symptoms?**

Positive rumination did not significantly mediate the relationships between trait affect and depressive symptoms based on the criteria that (a) the direct effect did not significantly decrease in value compared to the total effect value, (b) the indirect effects were non-significant, and (c) the confidence intervals of the indirect effects contained zero (see Table 4). However, brooding did partially mediate the relationship between trait NA and depressive symptoms based on the above criteria. Greater trait NA predicted greater depressive symptoms through greater brooding at week eight, and the effect of baseline trait NA on depressive symptoms at week 8 decreased by 28.56% when brooding was in the model. Based on the above criteria, brooding also mediated the relationship between trait PA and depressive symptoms such that less trait PA predicted greater depressive symptoms through greater brooding. In support of mediation, the effect of baseline trait PA on depressive symptoms at week 8 decreased by 14.79% when brooding was in the model. The mediation of brooding in the relationship between trait NA and depressive symptoms was stronger than the mediation between trait PA and depressive symptoms, but both mediation pathways occurred in the hypothesized directions (see Table 4).
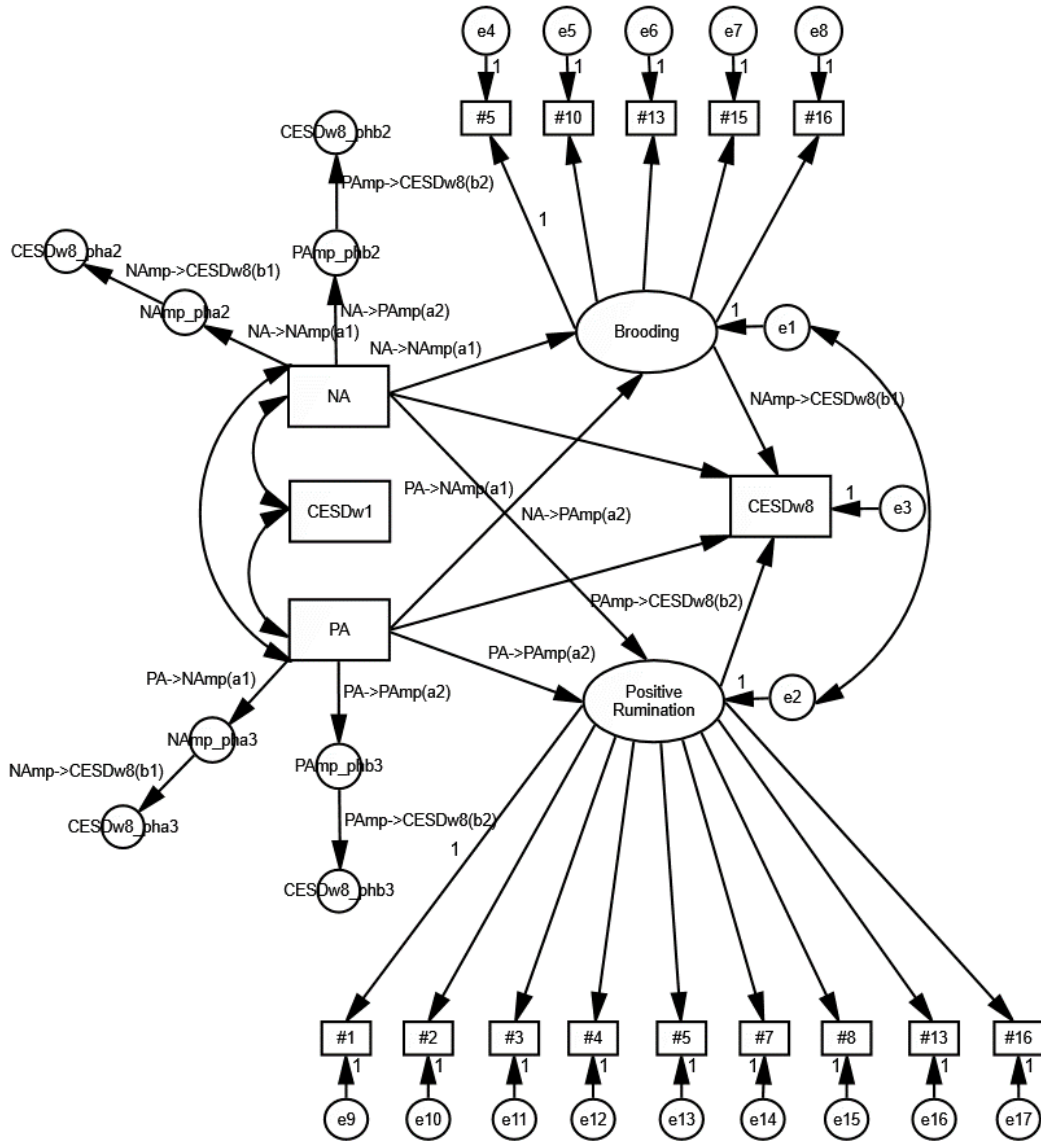
*Figure 7.* Model 4 with phantom variables of brooding and positive rumination mediating the relationship between trait affect and depressive symptoms with Model 2 as the mediator.
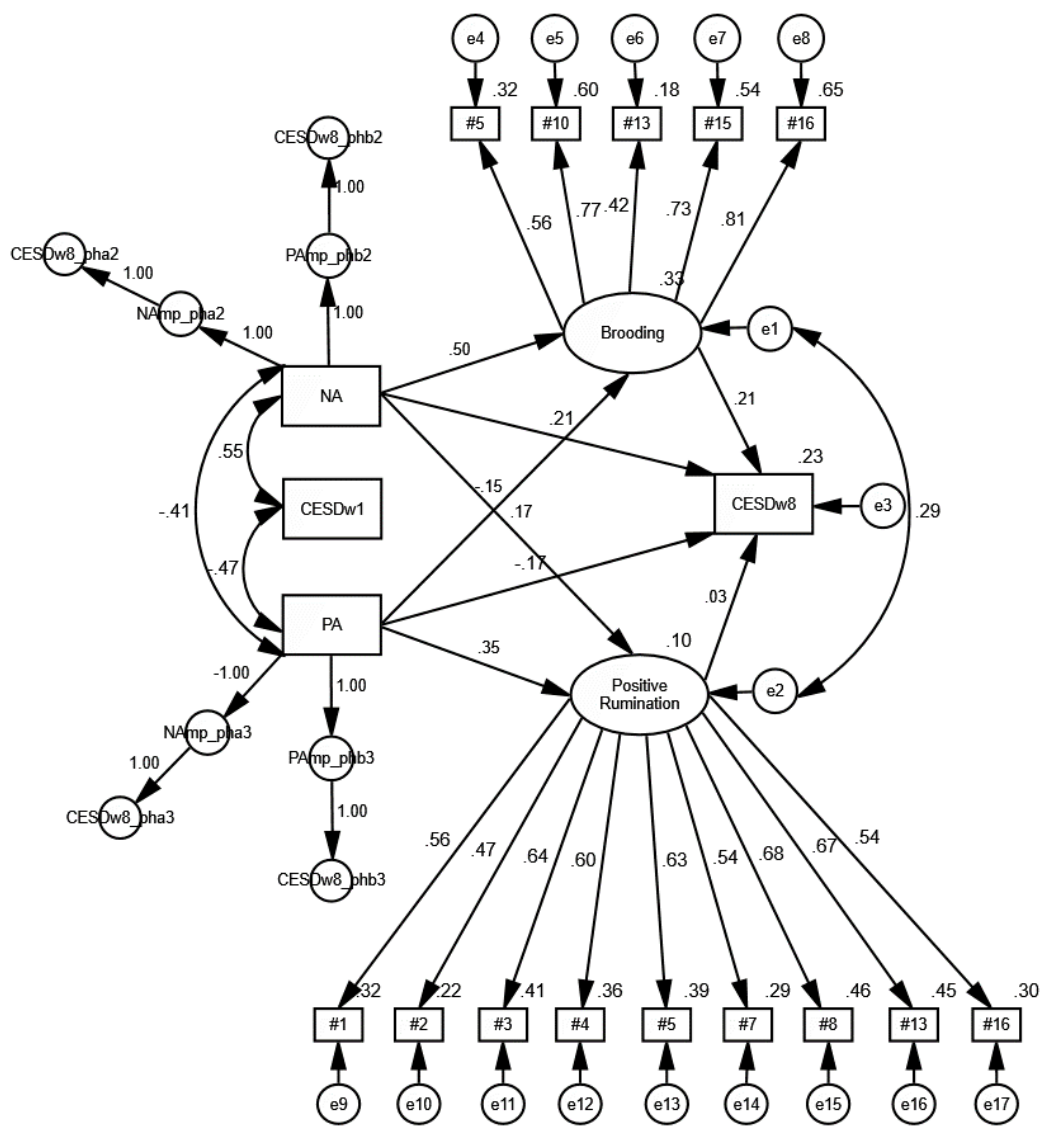
*Figure 8.* Model 4 of brooding and positive rumination mediating the relationship between trait affect and depressive symptoms with Model 2 as the mediator with standardized parameter estimates.

Table 4

*Model 4 Bootstrap Analysis of Brooding and Positive Rumination Mediating the Relationships Between Trait Affect and Depressive Symptoms*

| Pathway | β | *SE* | Standardized 95% CI | | *p* |
| --- | --- | --- | --- | --- | --- |
| | | | Lower | Upper | |
| *c* Trait NA→Depressive | .32 | .06 | .21 | .44 | .002 |
| *c* Trait PA→Depressive | -.20 | .06 | -.31 | -.09 | .002 |
| *c'* Trait NA→Depressive | .21 | .07 | .10 | .35 | .001 |
| *c'* Trait PA→Depressive | -.17 | .06 | -.29 | -.05 | .003 |
| **Brooding** | | | | | |
| *α* Trait NA→Brooding | .50 | .05 | .39 | .59 | .002 |
| *α* Trait PA→Brooding | -.15 | .06 | -.27 | -.04 | .014 |
| β Brooding→Depressive | .21 | .07 | .07 | .36 | .005 |
| Trait NA→Depressive *α* x β (*c* - *c'*) | .10 | .04 | .04 | .19 | .004 |
| Trait PA→Depressive *α* x β (*c* - *c'*) | -.03 | .11 | -.08 | -.06 | .012 |
| **Positive Rumination** | | | | | |
| *α* Trait NA→Positive Rumination | .17 | .07 | .04 | .31 | .010 |
| *α* Trait PA→Positive Rumination | .35 | .07 | .21 | .47 | .002 |
| β Positive Rumination→Depressive | .03 | .06 | -.10 | .15 | .684 |
| Trait NA→Depressive *α x* β (*c* - *c'*) | .01 | .14 | -.01 | .03 | .505 |
| Trait PA→Depressive *α x* β (*c* - *c'*) | .01 | .11 | -.03 | .06 | .619 |

*Note.* NA = Negative Affect; PA = Positive Affect.

**CHAPTER IV**

**Discussion**

The aim of the present study was to integrate understanding of cognitive processes common to trait NA and trait PA in the prediction of depressive symptoms. Specifically, I sought to better understand how ruminative cognitive processes (i.e., brooding and positive rumination) may be best conceptualized as distinct cognitive constructs with overlapping variance as forms of rumination. Furthermore, I sought to examined whether brooding and positive rumination may uniquely predict depressive symptoms, with greater brooding as a risk factor predicting greater depressive symptoms and greater positive rumination as a protective factor predicting fewer depressive symptoms.. How might different forms of rumination represent a partially shared cognitive process on distinct negative and positive affective content, and how might this understanding of rumination enhance the future prevention and treatment of depressive symptoms?

My dissertation consisted of two sets of distinct but related analyses with 321 young adults. Part 1 conducted a CFA of three models to examine whether brooding and positive rumination represented a partially shared cognitive process of affect amplification on distinct negative and positive affective content that in turn predicts depressive symptoms (Model 2). I compared this hypothesized model to a model representing both constructs as distinct (Model 1) and another model representing both constructs with their covariance constrained to 1 (Model 3). I hypothesized that brooding would amplify negative emotions and positive rumination would amplify positive emotions. In addition, I hypothesized that brooding and positive rumination would share a higher-order factor structure that represents a partially shared cognitive process of

affect amplification. My hypotheses were supported by Model 2 of the three compared CFA models, which proposed that brooding and positive rumination items would load onto distinct but correlated constructs (see Figure 2). Part 2 then inserted the best-fitting CFA model into an SEM mediation model in AMOS 21.0 to evaluate the accuracy of my hypotheses that greater brooding and less positive rumination at baseline would mediate the relationships between greater trait NA and less trait PA at baseline in predicting greater depressive symptoms seven weeks later. I controlled for baseline depressive symptoms, the alternative form of trait affect, and the alternative form of rumination in analyses to isolate the distinct effects of trait NA and trait PA as independent variables as well as brooding and positive rumination as mediators.

**A Partially Shared Cognitive Process on Distinct Affective Content**

My Part 1 hypothesis was supported that Model 2 would provide the best fit to the data. While Model 2 differed from Model 3 only in the constraint of the covariance pathway between negative amplification (i.e., brooding) and positive amplification (i.e., positive rumination), allowing this path to freely vary considerably improved model fit (see Table 3). Due to the constraint of this pathway in Model 3, Models 1 and 3 contained equal degrees of freedom and therefore could not be directly compared. Instead, Models 1 and 3 both were compared to Model 2 through a stepwise set of tests that examined whether changing paths between constructs resulted in a loss or gain in model fit to the data. Model 2 supported a conceptualization of brooding and positive rumination as a partially shared cognitive process on distinct affective content.

The joint examination of brooding and positive rumination within the same model represents a novel endeavor to integrate rumination in response to negative and positive

affective content. Whereas the psychometric validation of the RPA, the instrument that measures positive rumination, examined its statistical distinction from brooding (Feldman et al., 2008), no research has examined similarities between constructs or considered how process similarities may contribute to clinical interventions that may redirect ruminative tendencies from negative content to positive content. An early examination of rumination types began when Treynor et al. (2003) divided rumination in response to negative events into reflection, depressive rumination, and brooding. Depressive rumination represented items that shared considerable variance with the construct of depressive symptoms, so they were controlled to support that reflection and brooding represented distinct constructs from depressive symptoms. The potential for rumination on non-negative content was examined as "reflection," which is defined as rumination on neutrally valenced affective content. However, no research previously bridged the gap between rumination in response to negative versus positive affect.

Affect amplification occurs across depressive and bipolar mood disorder symptoms (Gilbert, Nolen-Hoeksema, & Gruber, 2013), although research has primarily focused on positive events *or* negative events rather than a combination of both. One notable exception is a study that I co-authored from the larger data set that includes my dissertation, which investigated whether weekly brooding and dampening distinctly mediated the relationships between trait NA and trait PA in predicting subsequent depressive symptoms (Hudson, Harding, & Mezulis, 2015). Brooding uniquely mediated the relationship between trait NA and depressive symptoms, while dampening uniquely mediated the relationship between trait PA and depressive symptoms. However, this study examined vulnerabilities across negative and positive emotions rather than

broadening focus to vulnerabilities and protections against depressive symptoms. Brooding and dampening also appeared to represent distinct cognitive processes with similar affective consequences, meaning that they both increased negative emotions and decreased positive emotions. My dissertation extended this study to additionally examine positive rumination as a proposed protective factor against depressive symptoms and compare brooding and positive rumination as overlapping forms of rumination that exert distinct effects in predicting depressive symptoms.

In further support that brooding and positive rumination represented a partially shared cognitive process on negative and positive affective content, a recent study demonstrated that rumination in response to an imagined future goal increases an individual's emotional reactivity to that event by amplifying their experience of negative *and* positive emotions (Gilbert & Gruber, 2014). While this study did not distinguish between rumination in response to positive versus negative content, rumination was applied to a negative to positive emotional spectrum similar to my dissertation. As previously mentioned, positive rumination also may exert stronger predictive effects within the spectrum of bipolar mood symptoms and be more predictive of hypomanic and manic symptoms than depressive symptoms (Gilbert et al., 2013). Insufficient research exists to strongly support either relationship between positive rumination and unipolar or bipolar mood symptoms, but both hypotheses offer directions for future research. Specifically, both hypotheses may corroborate brooding and positive rumination as a similar process of affect amplification that exerts distinct effects on negative versus positive affective content, respectively.

**Trait Affect, Cognitive Responses, and Depressive Symptoms**

In Part 2, I demonstrated that greater trait NA and less trait PA predicted greater depressive symptoms. This finding was consistent with existing literature on the relationships between trait affect and depression (Clark & Watson, 1991; Kotov et al., 2010; Naragon-Gainey et al., 2013; Verstraeten et al., 2009). In partial support of my hypothesis, greater trait NA predicted greater brooding. Earlier research is consistent with this result (Mezulis et al., 2011), although the present study is the first demonstration of this relationship after controlling for the effect of positive rumination. In contrast to my hypothesis, greater trait NA predicted *greater* positive rumination. This finding contrasted a similar manuscript from this data set (Harding, DeSimone, Willey, Kuhn, & Mezulis, 2015), which found no significant effect of weekly positive rumination in mediating the relationships between trait NA in predicting negative or positive emotions in response to positive events. Hence, event-anchored positive rumination appears to demonstrate contrasting effects with the full-form RPA. One potential explanation for the unanticipated relationship between trait NA and positive rumination is that trait NA broadly may predict greater rumination across events, and controlling for the effect of brooding may isolate a relationship with positive rumination that is confounded with more general ruminative tendencies. However, another potential explanation is that this relationship represented a spurious finding that is not representative of positive rumination across adult samples. This latter explanation is supported by the absence of a significant correlation between trait NA and positive rumination at week 1 yet a significant relationship when trait PA was entered into the model, which suggests a suppression effect (Friedman & Wall, 2005).

My hypothesis that less trait PA predicted greater brooding and less positive rumination was supported. The finding that less trait PA predicted less positive rumination is consistent with my previous work (Harding et al., 2014), although independent research is needed to replicate this relationship. The finding that less trait PA predicted greater brooding differed from a previous study indicating that brooding was only predicted by greater trait NA, although this study differs from my dissertation in that it controlled for dampening and examined event-anchored measures of brooding and dampening (Hudson et al., 2015). Either difference may partially account for the significance of trait PA predicting brooding in my dissertation, and replication through independent researchers is needed to more firmly support this relationship.

**Affect Amplification and Depressive Symptoms**

My hypothesis also was supported that greater brooding predicted greater depressive symptoms. This relationship was consistent with previous research (Nolen-Hoeksema, 1991; Treynor et al., 2003) and supported the role of brooding as a cognitive vulnerability to depression. My fourth hypothesis that less positive rumination would predict greater depressive symptoms was not supported, which contrasted with my previous work examining greater weekly positive rumination predicting fewer depressive symptoms (Harding et al., 2014), although the work of multiple studies similarly demonstrated no effect between positive rumination and depressive symptoms (Gilbert et al., 2013; Raes, Smets, Nelis, & Schoofs, 2012). Hence, literature on positive rumination and depressive symptoms is mixed. One research group recently acknowledged this literature discrepancy and specifically investigated the effect of positive rumination on anhedonic depressive symptoms (Nelis, Holmes, & Raes, 2015). They demonstrated that

less positive rumination predicted greater anhedonic symptoms, despite a lack of overall significance predicting depressive symptoms or depressive symptoms predicting positive rumination. This finding suggested that the impact of positive rumination may be specific to anhedonia, but further research is needed to disentangle the effects of positive rumination in the prediction of mood disorder symptoms.

Lastly, my hypothesis was supported that brooding would significantly mediate the relationships between greater trait NA and less trait PA in predicting greater depressive symptoms, but no significant mediation of positive rumination was present for trait NA or trait PA. Specifically, greater trait NA predicted greater depressive symptoms through greater brooding. This relationship was demonstrated among 12 to 15-year-old youth by Mezulis et al. (2011), but my dissertation represented the first examination among young adults. In addition, less trait PA predicted greater depressive symptoms through greater brooding in response to negative events. Mezulis et al. (2011) did not find a significant relationship between trait PA and greater brooding among youth, which may be partially explained by the distinct age group examined, their lack of a relationship between youth trait PA and depressive symptoms, and their inclusion of reflection as a covariate. Despite the significant relationships between trait affect and positive rumination, positive rumination did not significantly predict depressive symptoms.

In sum, findings from Part 1 supported the idea that brooding and positive rumination represent a partially shared cognitive process on distinct negative and positive affective content. Part 2 further supported brooding as a cognitive vulnerability between trait affect and depressive symptoms, but no significant mediation was present for positive rumination. Brooding more strongly mediated the relationship between trait NA

and depressive symptoms compared to the mediation of trait PA and depressive symptoms, which reinforces its designation as a cognitive response to negative events that primarily amplifies negative emotions to those events.

**Positive Rumination and Depressive Symptoms**

The finding that positive rumination did not predict depressive symptoms reflects a larger literature on the inconsistent relationship of positive rumination predicting depressive symptoms (Gilbert et al., 2013; Harding et al., 2014; Raes et al., 2010; Raes et al., 2012) and may be due to a combination of measurement inaccuracy, theoretical inaccuracy, and the unique characteristics of the undergraduate population sampled. Regarding measurement inaccuracy, discrepant findings regarding the ability of positive rumination to predict depressive symptoms may be partially explained by the role of less positive rumination in specifically predicting greater anhedonia rather than fewer overall depressive symptoms (Nelis et al., 2015). The CES-D does not contain items that specifically reference anhedonia, so the ability for a study to demonstrate that greater positive rumination predicts fewer depressive symptoms may depend on the emphasis of anhedonia within a depressive symptoms measure.

Related to measurement inaccuracy, the theoretical foundation underlying measures of depressive symptoms may represent similar imprecision in how depressive symptoms are conceptualized. While depressive disorders are diagnostically characterized by the prominence of persistent sadness or anhedonia, depressive symptoms are more commonly associated with sadness despite the reality that many depressive disorders do not require sadness as a diagnostic criterion if anhedonia is significantly present (American Psychiatric Association, 2013, pp. 155-188). This over-

emphasis on excessive negative emotions and under-emphasis on limited positive emotions in measuring depressive symptoms reflects existing literature on vulnerability models of depression that similarly focus on negative thoughts and negative emotions that predict depressive symptoms (Beck, 1967; Nolen-Hoeksema, 1991). While I sought to counterbalance this focus by including trait PA and positive rumination, I did not utilize a measure of depressive symptoms that equitably emphasized negative and positive emotional symptoms. Consequently, the lack of a significant relationship between positive rumination and depressive symptoms may reflect a measurement inaccuracy that is rooted in a theoretical inaccuracy regarding how depressive symptoms are conceptualized.

Lastly, the non-significant relationship between positive rumination and depressive symptoms may be attributable to the unique characteristics of my undergraduate population. Young adults in my university community may report patterns of thoughts and emotional experiences in response to positive events that are distinct from the experiences of other university communities or more demographically diverse populations. For example, a previous study involving an undergraduate sample supported a significant negative relationship between positive rumination and depressive symptoms (Feldman et al., 2008) while another study involving another undergraduate sample did not support a significant relationship between positive rumination and depressive symptoms (Raes et al., 2012). Hence, young adults in undergraduate populations may exhibit distinct and variable vulnerabilities and protections to depressive symptoms that partially explain literature disagreement on these constructs and do not accurately generalize to all adult populations.

**Theoretical Implications**

Depression literature presently separates theories on symptom development and maintenance into vulnerability and protective mechanisms. While no unified theory exists to explain the joint roles and similarities between brooding and positive rumination, I seek to integrate theories on positive emotions and negative emotions in the prediction of depressive symptoms (Fredrickson 2001; Beck et al., 1979; Nolen-Hoeksema, 1991). Despite the non-significant mediation of positive rumination that was demonstrated in previous studies, statistical modeling of brooding and positive rumination supported a factor structure that modeled the covariance of brooding and positive rumination as a distinct, but covaried constructs. That is, brooding and positive rumination are best conceptualized as related constructs, despite their impact on differently valenced affective experiences. The impact of trait NA and trait PA in predicting brooding further blurs these distinctions between vulnerabilities and protections, since brooding may impact negative and positive affective experiences to negative events.

Cognitive theories on depression and resiliency against depression are increasingly acknowledging the joint roles of positive emotions and negative emotions (Fredrickson, 2001; 2004; Beck et al., 1979). While a vast literature is present on cognitive and emotional responses to negative events (Liu et al., 2014; Peng et al., 2012; Siedlecka, Capper, & Denson, 2015), the prospective relationships between cognitive and emotional responses to positive events remain largely unexamined. Appraisal theories assert that events are positive or negative based on cognitive interpretations of specific events and resultant emotions elicited by those events (Ellsworth & Scherer, 2003).

Consequently, all events have the potential to elicit negative emotions and positive emotions, with the valence of a given emotional experience impacted by how the event is interpreted. For example, an event that is appraised as negative may elicit a range of negative and positive emotions, which may explain why brooding is distinctly impacted by trait NA and trait PA *and* how brooding potentially exerts its effects on depressive symptoms through increasing state negative emotions and decreasing state positive emotions in response to negative events.

My dissertation results were consistent with Beck's cognitive theory of depression (Beck et al., 1979) and response styles theory's extension of this theory to rumination in response to negative events (Nolen-Hoeksema, 1991). I demonstrated that greater trait NA predicted greater depressive symptoms through greater brooding, and the predictive power of brooding is distinct from positive rumination. Beck also referenced cognitive responses to positive events as a potential protection against depression (p. 179), but in this study positive rumination was not supported as a cognitive mechanism between trait affect and depressive symptoms. Cognitive theories of depression acknowledge cognitive responses to negative and positive events as meaningful actors in generation and maintenance of depression. My dissertation supported this idea and delved into the differential effects of rumination in response to negative and positive events.

My dissertation specifically supported the broaden-and-build theory of positive emotion, which asserts that trait PA predicts cognitive responses to positive events that amplify event-specific experiences of positive emotion (Fredrickson, 2001; 2004). My findings demonstrated that less trait PA predicted less positive rumination, which I further extended through recent work demonstrating that less event-specific positive

rumination predicts fewer event-specific positive emotions (Harding et al., 2015). This extended study did not find a significant relationship between positive rumination and event-specific negative emotions, which suggests that positive rumination may selectively predict the low positive emotions characteristic of depression (i.e., anhedonia) rather than the global construct of depressive symptoms (Nelis et al., 2015). Findings extend emerging literature by identifying positive rumination as an adaptive cognitive response that is predicted by trait PA, despite its uncertain role in the prediction of depressive symptoms.

My dissertation also was consistent with appraisal theories on the relationship between cognition and emotion. As previously mentioned, appraisal theories assert that events elicit a combination of negative and positive emotions (Ellsworth & Scherer, 2003). The appraisal theory of Ortony, Clore, and Collins (1988) discusses the cognitive appraisal of events as an ongoing process that involves negative and positive emotions that shift in valance and intensity based on how an event is interpreted (pp. 1-25). While negative events may predominantly involve negative emotional experiences, events that are interpreted as negative may comprise a spectrum of negative and positive emotions. As a result, how individuals appraise events as negative or positive additionally may invite opportunities to redirect rumination on negative content to rumination on positive content.

Redirecting rumination on negative content to rumination on positive content may be considered a form of reappraisal, which is defined as "changing how we think about a situation in order to decrease its emotional impact" (Gross, 2001, p. 214). Reappraisal may allow an event to be re-interpreted and thereby orient cognitive focus on negative

content to cognitive focus on positive content. Reappraisal is shown to reduce experiences of negative and positive emotions in response to events (Kalokerinos, Greenaway, & Denson, 2015). Given the broaden-and-build theory, reappraisal may broaden attentional focus during negative events to positive content, which may transition the individual from brooding to positive rumination. If any event may elicit a range of emotions depending on how the event is appraised (Ellsworth & Scherer, 2003; Ortony et al., 1988, pp. 1-25), then every negative event may transition into a more positive event based on the individual's cognitive focus and emotional valence of their ruminative content. Reappraisal from negative to positive content over time may build resources that increase that individual's capacity for future positive emotions and resilience against depression (Fredrickson 2001; 2004).

**Clinical Implications**

Findings highlighted important clinical implications in the prevention and treatment of depressive symptoms. Findings suggested that brooding may exert a stronger and more distinct effect in the relationship between trait affect and depressive symptoms compared to positive rumination. Considering the continued significance of brooding even controlling for the effects of positive rumination, brooding's amplification of negative emotions and related diminishing of positive emotions appears to hold distinct predictive value above and beyond the shared variance explained by similarities to positive rumination. The emotional amplification of brooding was a unique contributor to depressive symptoms above and beyond the shared contribution of a ruminative cognitive process. Consequently, decreasing brooding may be a more

effective intervention in the prevention and treatment of depressive symptoms than increasing positive rumination.

Due to the limited literature examining brooding and positive rumination as related constructs, it is unknown whether clinical interventions that aim to decrease brooding also may decrease positive rumination. This is an important clinical consideration, since targeting rumination as a blanket construct may limit a protective factor in therapy and inaccurately communicate to individuals that rumination is maladaptive across contexts. To address this potential concern, future research may investigate whether clinical interventions that decrease brooding also impact positive rumination and subsequent vulnerability or resilience to depressive symptoms. This question is partially addressed through research by Kiken and Shook (2014), which found that trait mindfulness was negatively associated with negative rumination but not associated with positive rumination, which suggests that mindfulness interventions may decrease brooding without discouraging positive rumination. Additionally, interventions that target both brooding and positive rumination may present a greater therapeutic benefit for individuals with bipolar mood symptoms, since greater brooding is demonstrated to predict greater depressive symptoms and greater positive rumination is demonstrated to predict greater hypomanic symptoms (Feldman et al., 2008; Johnson et al., 2008; Raes et al., 2010). Since increasing positive rumination may risk the onset of a hypomanic or manic episode in individuals with bipolar disorders, interventions that increase positive rumination should be monitored and moderated in such individuals.

Clinical interventions that promote directed attention to positive events and reappraisal of events from negative to positive content are important future directions in

the study of depression and resilience against depression (Quoidbach et al., 2015).  If redirecting brooding to positive rumination may be considered a form of reappraisal, this shift in ruminative focus may improve control over an individual's event-specific experience of positive emotions.  While Gilbert and Gruber (2014) framed rumination as a maladaptive cognitive response and contrasted its effects on event-specific emotions with mindfulness as an adaptive cognitive response, future research may consider examining similarities between rumination and mindfulness as common pathways to amplifying positive emotions despite the idea that mindfulness seeks to discontinue rumination and to emphasize present-moment focus without the repetitive thought characteristic of rumination.  Instead of considering mindfulness as a cognitive intervention to combat ruminative tendencies, mindfulness instead may be clinically applied as a similar or complementary approach to rumination.  For example, a mindfulness exercise involving a full sensory experience of an enjoyable meal may pair mindful attention to the sensory experience of eating with positive rumination on thoughts related to how enjoyable the meal is.  Comparing the emotional experience of this activity when engaging in rumination only, mindfulness only, or a combination of rumination and mindfulness may provide valuable insight into the contributions of rumination as a context-specific vulnerability or protective factor in positive emotional experiences.  Clinical literature commonly assumes that rumination and mindfulness are contradictory cognitive responses (Ietsugu et al., 2015; Snippe et al., 2015), but this unexamined assumption may limit our capacity to clinically utilize the potential strengths inherent in both cognitive responses.

**Limitations and Future Directions**

My dissertation involved design limitations that should be considered when interpreting findings.  First, the current study applied a non-experimental design that is consistent with causality but cannot directly test for causal relationships due to the presence of only two time points and lack of direct experimental manipulation.  Second, the similar nature of both weekly measures of depressive symptoms may have biased participant responses by increasing their familiarity with the measures.  Third, my sample of university students represented a convenience population that may not generalize to all adult populations.  Fourth, the gender imbalance of my sample may have biased the measurement of depressive symptoms, since adult females report a higher prevalence of depressive symptoms compared to adult males (Kessler et al., 1993).

My dissertation also involved several theoretical limitations that warrant consideration and invite future research.  First, trait affect and cognitive responses were all measured at baseline assessment, which limited the prospective nature of my causal model and prevented us from statistically demonstrating the temporal precedence of trait affect before cognitive responses to weekly events.  However, the nature of trait affect does theoretically support the temporal precedence of trait affect due to its description as a stable dimension of temperament across time and situations.  In contrast, brooding and positive rumination represent more malleable and event-specific responses.  Second, an overall lack of research on the integration of cognitive mechanisms in the relationship between trait affect and depressive symptoms limits available literature to support my proposed relationships.  Therefore, study limitations underscore a need to further examine the empirical basis upon which my dissertation was proposed.

Future directions may seek to replicate the supported relationships, since the presented findings represent the only known examination of the joint contributions of brooding and positive rumination. Specifically, the growing literature on positive emotions and depression would benefit from an integrated theoretical understanding on the joint contributions of cognitive responses in the relationship between trait affect and depressive symptoms. To date, theories on depression offer largely distinct conceptualizations of affective and cognitive vulnerabilities and protections. Future research also could investigate whether the shared process of affect amplification extends to co-rumination in response to both negative and positive events, since co-rumination may represent a partially shared cognitive process that extends perseverative self-focused processing to a dyadic, social context. Finally, future efforts to clinically translate research on affective vulnerabilities to depression could develop cognitive interventions that both decrease negative emotions and increase positive emotions, since the limited experiences of positive emotions characteristic of depression is often underemphasized. While brooding represents a vulnerability to depression, positive rumination may be encouraged as a protective cognitive response that adaptively harnesses the perseverative tendencies of many individuals at risk for depression.

**Conclusions**

Rumination likely is not the only cognitive mechanism that straddles the emotional continuum between negative and positive emotional experiences that predict depressive symptoms. However, I hope that my identification of rumination as a shared mechanism of vulnerability and resilience offers a more comprehensive understanding of depression and invites future investigations into shared cognitive processes that predict

and protect against depressive symptoms. I conducted this study to identify cognitive mechanisms through which affective vulnerabilities such as high trait NA and low trait PA confer risk for greater depressive symptoms. Based on current findings, brooding is an important mechanism that shapes the impact of trait NA and trait PA in predicting depressive symptoms and is best considered as a related but distinct cognitive process compared to positive rumination.

Brooding is a central maladaptive cognitive response that predicts depressive symptoms and predisposes individuals to continued symptoms (Nolen-Hoeksema, 1991; Treynor et al., 2003). Furthermore, the degree of brooding that an individual reports may be more predictive of future depressive symptoms than their degree of positive rumination. This implication suggests that interventions that decrease brooding may more effectively prevent and treat depressive symptoms than interventions that increase positive rumination. However, the potential efficacy of interventions that simultaneously decrease brooding and increase positive rumination are unexamined and may be considered a form of ruminative reappraisal that transitions individuals with ruminative tendencies from negative to positive emotional experiences.

Depression is a complex symptom category that is most accurately conceptualized as an excess of negative emotion and a lack of positive emotion. Vulnerability and resiliency factors in the prediction of depressive symptoms are evident at a trait level and may be translated across events through cognitive responses that amplify or diminish emotional responses to those events. Broadening understanding of depression broadens possibilities for mitigating vulnerability and building resilience. Integrating

understanding of vulnerabilities and protections may better enable individuals to think

adaptively in addition to or in spite of their trait tendencies.

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