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Internalizing disorders are marked by negative emotion toward the self. Past research has highlighted early adversity (EA), that is, treatment which deviates from a healthy environment, as a risk factor for later internalizing behaviors. What is unclear is why some, but not all, who experience early adversity go on to experience internalizing symptoms. Prior research further suggests that negative emotionality, the propensity to experience and exhibit negative affect, may help to explain these individual differences in response to EA, although it is unclear whether these two factors function primarily independently (via main effects) or conjointly (in a multiplicative interaction). Furthermore, three constructs related to negative emotionality—neuroticism, dysfunctional attitudes, and brooding rumination—were previously thought to represent distinct constructs, but recent research suggests that dysfunctional attitudes is best modeled as part of latent negative emotionality. The present study first tests whether brooding rumination, like dysfunctional attitudes, is better modeled as part of negative emotionality or instead as its own construct. Second, it tests whether negative emotionality and EA interact to predict latent internalizing, or instead operate independently. In a majority minority sample of emerging adults (n = 768), latent moderated structural equations (LMS) were used to assess potential multiplicative or additive effects of EA and negative emotionality on the pathway to internalizing in emerging adulthood. Results indicated that brooding rumination was best modeled as a latent factor separate from but highly correlated with latent negative emotionality. Latent trait vulnerabilities and EA did not significantly interact to predict internalizing symptoms, but there were significant main effects of latent brooding rumination and negative emotionality. Although EA predicted internalizing alone, its effects were negligible once trait

vulnerabilities were included in models. Implications for future methodological and etiological research were discussed.

RELATIONSHIP OF EARLY ADVERSITY AND NEGATIVE EMOTIONALITY TO EMERGING ADULT LATENT INTERNALIZING

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CHAPTER I: INTRODUCTION

Internalizing disorders are emotional syndromes marked primarily by internal processes, such as depression, anxiety, and somatization (Willner et al., 2016). Past epidemiological samples have reported that internalizing problems increase in prevalence during the developmental period of older adolescence and emerging adulthood (Rohde et al., 2013; Lunetti et al., 2021). The increase in prevalence highlights the importance of identifying risk factors that may contribute to internalizing symptoms in emerging adulthood.

Past research identifies early adversity as one risk factor for internalizing symptomatology. Early adversity (EA) has been defined as exposure to environmental conditions during childhood and/or adolescence that (a) represents a deviation from an expected healthy environment and (b) requires adaptation from the average child psychologically, behaviorally, and/or neurobiologically (Duffy et al., 2018). EA is associated with later poorer physical and mental health outcomes, including internalizing symptoms (McLaughlin, 2018). Some, but not all, who experience early adversity will go on to develop psychiatric disorders. I argue that these individual differences in response to EA result in part from differing levels of maladaptive vulnerabilities, such as latent neuroticism, dysfunctional attitudes, and trait rumination.

Widiger and Oltmanns (2017) define neuroticism as the trait disposition for experiencing negative emotionality, including irritability, unstable emotions, anger, anxiety, and depression. Similarly, dysfunctional attitudes cause negative thinking about oneself, others, and the future (Horiuchi et al., 2017). Trait rumination is the tendency to focus attentional resources on negative cognitions and emotions about the self (Nolen-Hoeksema, 2011). Because past research suggests brooding rumination, but not reflective rumination, predicted the onset of depressive symptoms (Treynor et al., 2003), the present study measures rumination using the brooding

rumination subscale. Previously, researchers believed that constructs such as dysfunctional attitudes and trait rumination were distinct from neuroticism; however, a recent latent variable analysis suggests that, instead, a measure of dysfunctional attitudes loaded highly on latent neuroticism rather than forming its own distinct construct (Zinbarg et al., 2016). What is unclear is whether in fact trait rumination is distinct from latent neuroticism, or instead, like dysfunctional attitudes, loads very highly on latent neuroticism. Thus, as a first aim, the present study probed whether trait rumination and negative emotionality are best modeled as one latent construct or two.

Moreover, these vulnerabilities such as neuroticism are theorized to work together with environmental exposures like EA. Specifically, diathesis-stress models indicate that both individual vulnerabilities and stress exposure, potentially including early adversity, are linked to the emergence of psychopathology (Monroe and Simons, 1991). Within this larger class of models, additive diathesis-stress models suggest that diatheses and stressors have significant individual effects on psychopathology (i.e., dual main effects only; Mineka et al., 2020), whereas multiplicative diathesis-stress models postulate that the combined effect of the diathesis and the stressor is greater than their multiplicative individual effects (i.e., a traditional multiplicative interaction effect; Colodro-Conde, 2018). While the field has long favored multiplicative models, new prospective evidence suggests that a multiplicative effect, while probably present, is significantly smaller than the main effects of recent chronic stress and stressful life events in predicting major depressive episode onsets (Mineka et al., 2020). However, this has not been examined with EA, only with recent chronic stress and stressful life events. Thus, as a second aim, the present study examined whether EA and negative emotionality had primarily additive effects on latent internalizing in emerging adulthood.

Latent Internalizing

Achenbach (1978) suggested a clear distinction in psychopathology between externalizing and internalizing disorders. Externalizing behaviors are manifested outwardly, marked by negative actions toward the physical environment (Eisenberg et al., 2001). Distinctively, externalizing behaviors are associated with poor-impulse control exemplified through aggression and deviant behavior (Samek and Hicks, 2014). By contrast, internalizing symptoms present themselves inwardly, such that the person's internal psychological state is negatively affected which manifests in an imbalance of their emotional state (Willner et al., 2016). Past research suggests that major depressive disorder and generalized anxiety disorder, common internalizing disorders, have similar etiologies (Kendler, 1996), while panic disorder and phobias do not have the same genetic diathesis as MDD and GAD (Kendler et al., 1993). Thus, Krueger's hierarchical internalizing model (Krueger, 1999), later refined by Watson (2005) (and here referred to as the Krueger-Watson model for brevity), suggested that internalizing symptoms should be divided into two clusters of disorders: fear and anxious misery.

In the Kreuger-Watson model, fear disorders include social phobia, simple phobia, agoraphobia, and panic disorder. Broadly, fear is an emotion and fight-or-flight response to perceived or actual threat that often leads to physiological symptoms (e.g., tachycardia; Conway et al., 2019). For example, the phobias are characterized by irrational, persistent fear of a certain object, situation, or activity (e.g., everyday social interactions in social phobia, or animals, crowded spaces within specific phobias; Eaton et al., 2018). Panic disorder is an anxiety disorder marked by recurrent, unanticipated panic attacks associated with persistent worry about having another attack and potential consequences of such attacks, which results in behavioral adjustments (e.g., avoidance; Roy-Byrne, 2006).

The anxious-misery cluster, also referred to as distress disorders, is comprised of unipolar mood disorders (e.g., major depressive disorder and dysthymia), generalized anxiety disorder, and post-traumatic stress disorder (PTSD; Watson, 2009). These disorders are characterized by heightened negative affect, decreased positive affect, and cognitive impairment, including excessive rumination (Seok et al., 2020). Depressive disorders are characterized by sadness, irritability, empty mood, and/or anhedonia along with a prolonged experience of somatic (e.g., sleep disturbance) and cognitive abnormalities (for a review, see Roiser et al., 2012). PTSD is the experience of fear, hyperarousal, reexperiencing of the traumatic event, and/or terror resulting from witnessing or experiencing threatening life events (Ehlers and Clark, 2000). Generalized anxiety disorder (GAD) is an anxiety disorder distinguished by excessive anxiety, worry that is difficult to control, and somatic symptoms (e.g., disturbed sleep; Hoge et al., 2012). Excluded from the Kreuger-Watson model is somatization, which is the expression of psychological distress through physical symptoms (e.g., conversions; Lipowski, 1998). While not included in the Kruger-Watson model of internalizing disorders, which focused only on the most common and well-studied phenomena, there is longstanding evidence of a robust correlation between somatic symptoms and internalizing mental illnesses, particularly depression and anxiety, (Tingstedt et al., 2018). Therefore, to accurately assess latent internalizing symptomatology, I also measured experiences of somatization in the study described here.

More recent research on the structure of psychopathology has examined whether a higher order factor accounts for commonalities in internalizing and externalizing, called p-factor, short for psychopathology-factor (Caspi et al., 2014; Lahey et al., 2012). Other research has suggested that the content of the p-factor depends largely on the sample studied and measures used (Levin-Aspenson et al., 2020). Therefore, given the limited consistency of the p-factor, the present study

focuses solely on the internalizing spectrum rather than attempting to model p-factor. Furthermore, due to the range of acceptable approaches to modeling internalizing in prior work (e.g., unidimensional internalizing or a hierarchical model with fear and distress) and comorbidity between unipolar and anxiety disorders, I will focus on a general unidimensional factor for internalizing, though I will also preliminarily assess fit of a hierarchical model.

Early Adversity

Early life stress, which I refer to here as early adversity, is chronic or severe environmental conditions that diverge from a healthy environment and that create a need for significant adaptation (McLaughlin, 2018). Deviation from the healthy environment is evident by (a) deprivation, the absence of expected environmental factors (e.g., absence of caregiver) or (b) threat, the presence of unexpected threatening environmental factors (e.g., instances of physical abuse; McLaughlin, 2018). Previous research reported an estimated 50% prevalence of exposure to at least mild early adversity in the United States (Green et al., 2010). Generally, those who experience early adversity are more susceptible to psychopathology, including both internalizing and externalizing disorders, as compared to those who do not experience substantial early life stress (Kessler et al., 2010).

Those exposed to early adversity are more sensitive to perceive threat, and though temporarily adaptative, chronic hypervigilance and increased emotional reactivity is linked with a heightened risk for developing internalizing disorders (e.g., mood and anxiety disorders) in adulthood (McLaughlin et al., 2010). This heightened vulnerability to experience mental health disorders is thought to persist across the individuals' lifetime (McLaughlin, 2018). For example, Clark et al. (2010) found that experiences of early adversity were correlated with increased propensity for onset of depressive and anxiety disorders in adolescence, emerging adulthood, and

middle adulthood. Therefore, early adversity exposure is a predictor for onset of mental health disorders, with an estimated 30% of lifetime psychopathology being due to early life stress (McLaughlin, 2018).

Early adversity as a determinant of both poorer physical and mental health outcomes was the motivation for the Adverse Childhood Experiences (ACEs) study conducted by the CDC and Kaiser Permanente (1995 to 1997). Research prior to the ACE study focused on stressors individually (e.g., parental divorce; Amato & Keith, 1991); by contrast, the ACE study was the first to conceptualize distinct stressors as a general factor, adverse childhood experiences. The ten distinct stressors from the ACE study are: physical abuse, emotional abuse, sexual abuse, physical neglect, emotional neglect, witnessed domestic violence, substance abuse in household, mental illness in household, family member in prison, and parents divorced or separated. Researchers reported the commonality of early adversity exposure, with two-thirds of participants in the original ACE study reporting at least one adverse childhood experience and more than one-fifth experiencing three or more (Felitti et al., 1998). Previous findings suggest that cumulative adversity, the accumulation of adverse experiences, is associated with an increased risk of later onset of mental health issues, especially depressive symptoms (Turner and Lloyd, 1995; McLaughlin, 2018).

Despite its numerous contributions, one limitation of the original ACE study is that the sample was predominantly white individuals of middle/higher socioeconomic status, with a focus on in-home experiences of early adversity (Cronholm et al., 2015). Adversity exposure is notably higher in minority and lower SES individuals, and these minoritized groups are prone to specific adversities (e.g., experiencing discrimination) that were omitted from the original ACE study (Cronholm et al., 2015). Therefore, to increase sociodemographic diversity and consider

exposure beyond the home environment, Cronholm and colleagues developed the Revised ACEs measure. Additionally, this revision would allow for proper examination of the differential effects of specific ACEs on certain demographic groups (Cronholm et al., 2015). Their research suggested that to best assess adversity exposure across sociodemographic groups beyond White, high SES groups, the original ACEs needed to be extended to include other types of adversity exposure such as discrimination and witnessing violence. Therefore, to best capture early adverse experiences most adequately with research participants enrolled at a minority-serving institution, I used the Revised ACEs measure, which considers both the distinctiveness and co-occurrence of exposure to early adversity.

Negative Emotionality

Some, but not all, who experience early adversity go on to develop psychiatric disorders. Therefore, the present study considers other etiological factors of internalizing symptoms, such as trait diatheses (e.g., negative emotionality). Negative emotionality (NE) refers to the tendency for some individuals to experience and exhibit negative affect (Kann et al., 2017); here I will review three constructs related to negative emotionality—neuroticism, dysfunctional attitudes, and rumination—and their potential roles in contributing variance to emerging adult internalizing symptoms.

Neuroticism has been suggested as a potential vulnerability for both unipolar mood disorders, such as major depressive disorder, and anxiety disorders (Griffith et al., 2010). Specifically, Khan et al. (2005) found large effect sizes between neuroticism and internalizing disorders, such as major depressive disorder, generalized anxiety disorder, and panic disorder and medium effect sizes between neuroticism and phobia disorders. Past research highlighted a robust correlation (r = 0.98) between latent neuroticism and internalizing psychopathology,

especially in comparison to externalizing behaviors, such as substance use (r = 0.29; Griffith et al., 2010). Given the strong association between N and internalizing symptoms, it is understandable that neuroticism accounts for a significant portion of the comorbidity between internalizing disorders (Clark & Watson, 1991). Specifically, neuroticism has been noted to explain 16% of the variance shared between GAD and MDD (Khan et al., 2005). It is evident that neuroticism will have its own main effect on internalizing.

Dysfunctional attitudes are beliefs and attitudes that foster negative cognitions about oneself, the world, and one's future (Haeffel et al., 2005; Horiuchi et al., 2017), thought to arise because of underlying latent cognitive schemas. Diathesis-stress models predict that when activated by stressors, dysfunctional attitudes rise to the forefront and increase affective disruptions (Conway et al., 2015). In past research, Beck (1967) suggested that dysfunctional attitudes increase the susceptibility for depressive, but not anxiety disorders. More recent research suggests that these maladaptive beliefs predispose individuals not only to depressive (Felix and Hooker, 2016) but also anxiety symptoms (Conway et al., 2015) and creates risk for recurrence of psychological problems (e.g., anxiety and depression) across their lifetime (Conway et al., 2015).

Previously, it was assumed that dysfunctional attitudes and trait neuroticism captured different constructs due to face valid differences in their question content and moderate conventional correlations between the two constructs (e.g., r = 0.58; Sutton et al., 2011). However, latent variable approaches provide the opportunity to isolate shared variance and separate out statistical noise, known as error variance, revealing previously undetected relationships (Cooper et al., 2019). Critically, Zinbarg and colleagues (2016) addressed a gap in the theoretical understanding of the relationship between neuroticism and dysfunctional attitudes,

which suggests that dysfunctional attitudes are best modeled as part of neuroticism, as opposed to a distinct latent construct, yielding support for a general neuroticism factor. This general latent neuroticism factor incorporating dysfunctional attitudes together with more traditional measures of neuroticism was found to be a significant predictor of unipolar mood disorders (UMDs), anxiety disorders (ADs), and co-occurring UMDs and ADs.

The relationship of a third construct to latent negative emotionality remains to be fully characterized. Trait rumination, the inclination to focus attentional resources toward negative cognitions and emotions (McLaughlin & Nolen-Hoeksema, 2011), is a cognitive vulnerability associated with internalizing symptomatology and is most often conceptualized as a form of maladaptive emotion regulation. Rumination has been shown to both maintain and worsen current depressive symptoms (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1991), as well as predict the onset of depressive symptoms (Just & Alloy, 1997). Ruminative cognitions may activate when experiencing internalizing symptoms, which can interfere with effective coping (Shapero et al., 2013). Additional research shows that rumination may in fact increase symptoms of depression and anxiety, especially in adolescents (Shapero et al., 2013), and therefore, rumination is thought to contribute to the maintenance of internalizing problems (McLaughlin & Nolen-Hoeksema, 2011).

There are two subtypes of rumination: reflective and brooding. Reflection rumination is defined as intentionally pondering techniques to understand and alleviate one's depressive symptoms (Treynor et al., 2003). Brooding rumination is less intentional, in that individuals passively consider their mood and its relation to an "unachieved standard" (Treynor et al., 2003, p. 256). Research suggests that brooding rumination predicts the risk of depressive episodes (Young & Dietrich, 2014). When examining associations between depression and the subtypes of

rumination longitudinally, brooding rumination predicted the onset of depressive symptoms, but this relationship was not evident with reflective rumination (Burwell & Shirk, 2007; Krause et al., 2018; Treynor et al., 2003). Additionally, brooding rumination was associated with higher general anxiety symptoms (Jose & Weir, 2013; Lopez et al., 2014; Olatunji et al., 2013; Verstraeten et al., 2011) and mediated the relationship between early maladaptive vulnerabilities and social anxiety (Orue et al., 2014). Considering this, I focused solely on modeling brooding rumination as a predictor of internalizing disorders.

It has not yet been tested whether other trait measures related to the processing of negative emotion, namely trait rumination, also load onto latent neuroticism rather than creating its own construct. Previous research reports a robust correlation between rumination and neuroticism (r = 0.64; Lu et al., 2017) like preliminary evidence on DAS and neuroticism (r = 0.58; Sutton et al., 2011). This suggests that rumination may behave like DAS, which has been shown to fit best modeled as part of N and load onto latent neuroticism as opposed to being its own distinct construct. I hypothesize that trait brooding rumination will load together with dysfunctional attitudes and traditional measures of neuroticism, forming a general negative emotionality factor. Whether trait brooding rumination will load onto neuroticism is an important question because, without this clarification, it is not possible to know how to best test diathesis-stress models—that is, using separate vulnerability risk factors or a single trait vulnerability.

Diathesis-Stress Theories in the Context of Early Adversity

Considering the prevalence of depressive symptoms, much work has focused on identifying risk factors for onset of symptoms. Traditional theories of diathesis-stress effects focused largely on biological effects, such that stress was thought to activate biological predispositions, leading to the onset of psychopathology (Meehl, 1962). Later work suggested

that other vulnerabilities, such as cognitive and social diatheses may also interact with stress to elicit an onset of depression symptoms (Abramson et al., 1989; Beck, 1987; Brown & Harris, 1978). Therefore, those low in diathesis and high in stress would have lower rates of depression than those high in both diathesis and stress. As a critique of traditional approaches, Monroe and Simons (1991) suggested psychological research move beyond assessing their interactive effects with comprehensive theories and correlations, but instead, should test diathesis-stress models using more precise association modeling that can better assess their effects on depression. Thus, vulnerabilities and stressors can have additive, as well as multiplicative effects on major depression.

Past research has noted chronic and episodic stressors as risk factors for depression. Specifically, pathways from stressful life events to major depressive episodes are often moderated by preexisting diatheses, such as personality traits. Notably, in a study on diathesis-stress effects of neuroticism and stressful life events (SLEs) on depression, Kendler et al. (2004) found support for both main effects and interaction effects of neuroticism and SLEs on depression. First, neuroticism increased risk of depression at all levels of stressful events. Second, neuroticism amplified the effects between SLEs and depression, such that individuals high in neuroticism were at increased risk for depression in response to adversity when compared to less neurotic individuals. One recent critique of their findings, however, is that while Kendler et al.'s multiplicative interaction effect was statistically significant in their very large sample (n = 7517), the additive main effect sizes were markedly larger than their interaction effects (Mineka et al., 2020). This raises the question of the real-world significance, as opposed to statistical significance, of Kendler et al.'s multiplicative interaction effects.

To further improve understanding of neuroticism-SLE relationships on depression, Mineka et al. (2020) assessed the additive and multiplicative effects of diatheses and stress on major depressive episodes (MDEs) using a hierarchical CFA model of neuroticism and its related constructs. Recent stressful life events, chronic stressors, and neuroticism had moderate to large significant effects on MDEs, and the interaction effects were similar in magnitude to those noted in the Kendler et al. study but were small relative to main effects and not significant, owing in part to Mineka et al.'s relatively smaller sample size (n = 559). Additionally, the multiplicative effects were significantly smaller than the additive effect sizes in the majority of analyses (Mineka et al., 2020). Thus, the neuroticism-stress model of depressive symptoms is primarily the result of their additive effects.

While new research suggests that a multiplicative effect of diathesis and stress is significantly smaller than the main effects (Mineka et al., 2020), this has not to my knowledge been examined with early adversity, only with recent chronic and episodic stress. Thus, the present study seeks to extend the Mineka et al. (2020) findings to include early adversity, and to extend the Zinbarg et al. (2016) findings to include another potential facet of negative emotionality, trait rumination. I anticipated that EA and negative emotionality, modeled as a single trait vulnerability, would each have significant main effects on latent internalizing psychopathology, and we did not anticipate significant, robust effects of their potential interaction effects.

Present Study

The aim of the present study was to examine how early adverse experiences and trait vulnerabilities act individually versus together to predict latent internalizing in emerging adulthood, by focusing on defining, first, the boundaries of latent neuroticism and, second, the

nature of its action with EA (additive or multiplicative) in the pathway to latent internalizing. Two primary hypotheses were tested. I hypothesized that trait brooding rumination would load with traditional neuroticism measures on latent negative emotionality (which will include dysfunctional attitudes) rather than forming its own construct (Hypothesis 1). Specifically, I expected that a model in which both constructs load on neuroticism would demonstrate significantly better fit than a model in which rumination is not forced to load on latent neuroticism. I also hypothesized that early adversity and this latent vulnerability would have a primarily additive effect on latent internalizing in emerging adulthood (Hypothesis 2). Specifically, I tested whether a model with an interaction effect between EA and latent negative emotionality demonstrated significantly better fit than a model with no such interaction effect, but I did not expect the interactive model to improve fit compared to the additive model. I examined these questions in a cross-sectional sample of 768 emerging adults self-reporting retrospective early adversity, trait vulnerability, and past-year internalizing symptoms, using structural equation modeling methods.

CHAPTER II: METHOD

Participants

Data were drawn from a larger investigation on early adverse experiences, internalizing symptomatology, and chronic stress exposure. Participants were recruited from an undergraduate research pool at a highly diverse institution in the southeast and received course credit for participation. After consenting, all participants (n = 886) were prompted to complete the computerized Qualtrics survey. Participants were excluded from primary analyses for any one or more of the following reasons: being over 24 years of age (n = 37), failure to report age (n = 13), failure to respond to the final measure (the Childhood Trauma Questionnaire, which was central to Hypothesis 2) with at least 80% completion (n = 49), and failing ARS-33 attention checks or non-response to ARS items (Maniaci and Rogge, 2014; n = 59), totaling 118 excluded participants. Participants over the age of 24 were *a priori* excluded because: (1) emerging adulthood (i.e., age 18 to 24) is characterized by elevations in internalizing psychopathology (Rohde et al., 2013), and (2) to ensure that the reference period between early adversity and current symptom reports is similar across all participants.

Of the 768 remaining participants, the sample was 73.3% female (n = 563) with a mean age of 18.87 (SD = 1.28). In addition, the sample was majority minority race/ethnicity. Self-reported race was Black or African American, 37.0%; White, 28.9%; Hispanic, Latino, or Spanish Origin, 15.5%; Multiple races/ethnicities, 9.6%; Asian or Asian American, 5.7%; Middle Eastern, Arab, or North African, 1.4%; Native Hawaiian or Other Pacific Islander, 0.5%; American Indian or Alaskan Native, 0.4%, and Other, 0.9%.

Measures

Attentive Response Scale

I used the Attentive Response Scale (ARS-33; Maniaci and Rogge, 2014) to measure for inattentiveness and inconsistency in responses. The ARS-33 includes 33 scale items, 22 inconsistency items and 11 infrequency items. Several items (n = 18) from the ARS scale were inadvertently placed in sections of the questionnaire battery with skip-out programming in questionnaires collected for aims beyond the scope of the current project (e.g., in a romantic chronic stress measure, a participant indicating having no current romantic relationship would advance them past questions about the quality of their dating relationship). For this reason, not all participants were administered these items and therefore these could not be used in our calculation of the ARS score. I used other items from the ARS-33 to substitute for these items, with the final scale including 12 inconsistency items and 6 infrequency items and used the established cutoffs from the ARS-18 short form to exclude participants with evidence of inattentive responding.

Early Adversity

I collected two measures of EA, with a total of 6 subscales, to attempt latent variable analyses with multiple manifest indicators. To adequately estimate latent variables in structural equation modeling, at least three indicator variables are typically needed (Byrne, 2013). However, as described below, preliminary modeling did not support treating EA as a latent variable, and I therefore created a single composite score for use as a manifest variable.

First, the Childhood Trauma Questionnaire Short-Form (CTQ-SF; Bernstein et al., 2003) was used to measure experiences of childhood trauma prior to the age of 17 years old in college students. The questionnaire consists of 25 Likert-scale retrospective questions that form five

subscales: emotional abuse, physical abuse, sexual abuse, physical neglect, and emotional neglect. The participants selected their agreement with the questions on a 5-point scale that ranges from "never true" to "very often true" responding to items such as, "I believe that I was emotionally abused." In the present sample, internal consistency for the CTQ and its' subscales was adequate with estimates ranging from McDonald's ω_t = .71 to .94.

The Revised Adverse Childhood Experiences Measure (Revised ACEs; Cronholm et al., 2015) was used to assess levels of childhood adversity prior to the age of 17 years old in college students. This revised version of the "Conventional" ACE Survey (Felitti et al., 1998) consists of 21 Likert-scale questions. The participants indicated the most accurate answer to questions such as, "While you were growing up, did you live with anyone who was depressed or mentally ill?" on a 3-point scale including "more than once," "once," or "never." I first followed the recommended scoring, and then I scaled the score (by dividing total score by 3) to be similar to the range of other measures. Internal consistency of the Revised ACEs was slightly below the acceptable range (McDonald's ω_t = .64), but this is not particularly surprising given that people who experience one form of early adversity do not necessarily also experience other forms.

Internalizing Symptoms

I collected six measures of internalizing to facilitate latent variable analysis with multiple indicators and cover the breadth of this construct. I also initially collected the PCL-5 (Blevins et al., 2015) but on further consultation prior to analyses, I determined not to use it because we did not evaluate for PTSD Criterion A trauma exposure per se. To maximize variability on these measures, all questionnaires asked participants to report about the most pronounced week of symptoms within the past year.

Self-reported depression symptoms were measured using the Diagnostic Inventory for Depression (DID; Zimmerman et al., 2004). This questionnaire was adapted to measure depressive symptomatology during the most pronounced week over the past year and excluded questions regarding suicidality. Participants answered four background questions prior to completion of the 17 Likert-scale questions relating to diagnostic inventory for depression symptoms, such as "During that week in the past year when you felt the most down, were you feeling sad or depressed?" Response options were on a 5-point scale and specific to each symptom, with 0 typically indicating "not at all" and 4 indicating "extreme depressive symptoms." Internal consistency was acceptable (McDonald's $\omega_t = .91$).

The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) is a 16-item internalizing measure used to assess the week in the past year that participants were the most worried. The questionnaire measured the occurrence and intensity of worry symptoms. The participants rated statements, such as "My worries overwhelm me" on a Likert-scale of 1, "not typical of me" to 5, "very typical of me." Higher scores indicate greater endorsement of worry. McDonald's ω_t for the PSWQ was .94; therefore, internal consistency was excellent.

The Somatic Symptom Scale-8 (SSS-8; Gierk et al., 2014) was used to measure occurrence and severity of somatic symptoms, typical physical symptoms associated with many medical diseases (e.g., a cold), during the single worst week in the past year. The scale consists of 8 items where participants rated their agreement with the items, such as "back pain" and "headaches" on a Likert-scale ranging from 0, "not at all" to 4, "very much." The SSS-8 had adequate internal consistency, with a McDonald's ω_t of .83.

The Albany Panic and Phobia Questionnaire (APPQ; Rapee et al., 1994) is a 27-item questionnaire of panic and phobia symptoms experienced in the single worst week in the past

year. Participants rated the extent to which they feared each item, such as "meeting strangers" during the previously indicated worst week. Items were scored on a fear scale of 0, "no fear" to 8, "extreme fear," with middle scores indicating slight fear to increasingly moderate fear. Measured internal consistency was high (McDonald's $\omega_t = .94$).

The Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007) was used to measure levels of panic-disorder-related anxiety sensitivity during the week in the past year where such fears were most pronounced. The index consists of 18 self-report questions with 6 items per each of the three subscales: physical concerns, cognitive concerns, and social concerns. Participants selected their agreement to items, such as "it is important for me not to appear nervous" on a Likert-scale with selections 0, "very little," 1, "a little," 2, "some," 3, "much," and 4, "very much." Internal consistency for the ASI-3 was McDonald's $\omega_t = .90$.

The Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998) was used to measure social fears during the week in the past year when most pronounced. The SIAS consists of 20 items in which participants rated the degree to which the statement was characteristic of their experience on a 5-point Likert-scale. Items such as "I am worried people will think my behavior is odd" were rated on a scale of 0, "not at all" to 4, "extremely." The SIAS had good internal consistency (McDonald's $\omega_t = .89$). The Social Phobia Scale (SPS; Mattick & Clarke, 1998) was used to measure social fears. Participants indicated the degree to which they agreed with 20 statements during the week in the past year that their social fears were most pronounced. Items were scored on a Likert-scale of 0, "not at all" to 4, "extremely." Sample items include, "I become anxious if I have to write in front of other people." Internal consistency was high (McDonald's $\omega_t = .95$). Recent evidence suggests that when both the SIAS and the SPS are available, they should be scored as a single measure (here McDonald's $\omega_t = .96$) because they do

not constitute two separate constructs (Gomez and Watson, 2017). Thus, the present study scored the SIAS and SPS as one measure.

Negative Emotionality-Related Constructs

Neuroticism. Two scales were collected to measure neuroticism; the present research questions are primarily concerned with a global conceptualization of neuroticism and not with subscales (e.g., anger, self-consciousness), and as such, general indicators were used rather than calculating subscales. First, the Eysenck Personality Questionnaire Revised N Scale (EPQ-R-N; Eysenck & Eysenck, 1975) was used to measure neuroticism. The revised EPQ-R-N consists of 23 items. Participants selected their agreement or disagreement with items, such as "does your mood often go up and down?" by choosing either (A) = yes or (B) = no. The EPQR Revised N Scale had good internal consistency with a McDonald's ω_t of .87. Second, the 10-item International Personality Item Pool (IPIP; Goldberg, 1999) scales targeting NEO-PI-R was also used to measure neuroticism. Participants rated their agreement with statements such as "often feel blue" on a scale of 1, "very inaccurate" to 5 "very accurate." The NEO-PI-R neuroticism scale had adequate internal consistency demonstrated with a McDonald's ω_t of .81.

Trait Brooding Rumination. The Ruminative Response Scale (RRS; Treynor et al., 2003) was used to measure ruminative behaviors. Participants were asked how often they have thought about or carried out one of the listed behaviors when they felt down, sad, or depressed. The RRS includes 22 items, and participants scored their agreement with the items, such as "Think about how alone you feel" on a scale of 1, "almost never" to 4, "almost always." Higher scores indicate higher degrees of rumination. In the present analyses, I only used the brooding rumination items (Items 5, 10, 13, 15, 16) to fit a latent variable at the item-level to facilitate a

stringent test of its independence from latent negative emotionality. Internal consistency for the brooding items of the RRS was adequate (McDonald's ω_t = .81).

Dysfunctional Attitudes. The Dysfunctional Attitudes Scale Short Form (DAS-SF1; Beevers et al., 2007) was used to measure participants' attitudes. The DAS-SF1 consists of 9 items, such as "It is best to give up your own interests in order to please other people," that are rated on a scale of 1, "totally agree" to 4, "totally disagree." Dysfunctional attitudes have been shown to load strongly on latent neuroticism rather than forming its own latent variable (Zinbarg, 2016), and thus were modeled as a manifest indicator of latent neuroticism. The DAS-SF1 had good internal consistency (McDonald's ω_t = .86).

Assessment Procedure

All study procedures were approved by the institutional review board at the university. Participants were recruited from the research pool in psychology. After obtaining informed consent, demographic information was collected. Participants completed a series of self-report questionnaires on Qualtrics. The questionnaires measured experiences of early adversity, past-year internalizing symptoms, and negative emotionality as a vulnerability (neuroticism, trait rumination, and dysfunctional attitudes). Some participants went on to complete additional measures to facilitate testing of other research questions that are not a part of the current project, but since the questionnaires relevant to my study precede the additional measures, I do not suspect this affected attentiveness.

Data Analytic Plan

Preliminary Analyses

As a preliminary step, I inspected each variable's descriptive statistics including mean, standard deviation, and skew. Results are displayed in a bivariate correlation table (Table A1). I

used natural log transformation to adjust select subscales (physical abuse, sexual abuse, and physical neglect) and global item-mean in the Childhood Trauma Questionnaire (CTQ) that had a skewness greater than 1 because this is known to influence the results of structural equation models (Bulmer, 1979).

Missing Data

To minimize the amount of data missing at the indicator level, questionnaire scales were computed as item means (excluding the Revised ACEs which is scored using a count of experiences endorsed) and permitted up to 20 percent missing data within a scale. Any remaining missing data was estimated with maximum likelihood estimation with robust standard errors in structural equation modeling (Myung, 2003). I conducted Little's MCAR test (1988) to assess whether missing data was missing completely at random, a necessary assumption for maximum likelihood estimation, which was fulfilled. As noted earlier, if participants consented to the study but did not complete at least 80% of the final measure (CTQ) and/or failed the attention checks, their data were excluded from analysis.

Primary Analyses

Methodological advancements in estimating interaction effects between latent variables using structural equation modeling (SEM) are appropriate for primary analysis of the dataset; strengths of SEM include the ability to estimate missing data and the ability to extract statistical error from the effects of interest. I estimated latent interactions using the latent moderated structural equations (LMS) method built into the M*plus* software using the XWITH command (Muthén & Muthén, 1998-2017; Maslowksky et al., 2015). In M*plus*, the loading of the first item was set to 1.0 for all latent variables by default. For each model, I generated standardized betas in M*plus* using the "standardize" command, as M*plus* will not standardize regression coefficients

for planned models. Additionally, I used maximum likelihood estimator with robust errors (MLR) to help account for minor skew remaining in the data (Yuan & Bentler, 2000).

I also examined data for potentially overly influential multivariate outliers in all primary models using Cook's distance; participants with a Cook's distance of 0.5 or greater should be examined, and participants with a Cook's distance of 1.0 or greater should be excluded (Cook & Weisberg, 1982). Cook's Distance was < 0.5 for all participants in the primary models, and thus, there were no overly influential outliers to be excluded. Across models, adequate fit was supported if the comparative fit index (CFI) and Tuckers-Lewis index (TLI) values were greater than .95 and root mean square error of approximation (RMSEA) values were below .08 (Little, 2013).

As a first step, I estimated the measurement model prior to testing the entire structural model to ensure fit. At this step, I inspected models for indicators that failed to properly load on their hypothesized latent variable; none were identified. I anticipated that early adversity may not produce a good fit as a latent variable because adverse experiences may have heterogeneous rather than consistent sources—e.g., sexual abuse by a stranger would be unrelated to emotional abuse by caregivers. The fit of latent early adversity was not acceptable (χ^2 (8, N = 768) = 69.174, p < .001, RMSEA = 0.100, CFI = 0.953, TLI = 0.911), so to achieve adequate model fit, the latent variable for early adversity was replaced with a manifest composite score.

As part of this first step, I estimated the measurement model for latent neuroticism, dysfunctional attitudes, and trait brooding rumination by itself. For step one, I modeled brooding rumination at the item-level to permit it to form its own latent variable. This allowed me to analyze whether trait rumination loaded on latent neuroticism or created its own latent construct (Hypothesis 1). I compared model fit of confirmatory factor analysis (CFA) using a log

likelihood-ratio test between two competing models where either (a) manifest variables are forced to load on a single latent variable for negative emotionality or (b) brooding rumination is forced to load onto a separate and distinct but correlated latent variable. The log-likelihood ratio test statistic, designated as *D*, was calculated using this equation: $D = -2[(\text{log-likelihood for Model 0}) - [(\text{log-likelihood for Model 1})]/cd$. The correction factor, *cd*, was calculated using this equation: $CD = [(p0 * c0) - (p1 * c1)] / (p0 - p1)$, where p0 and p1 represent the number of parameters for Model 0 and Model 1a/1b, and c0 and c1 represents the scaling correction factor for Model 0 and Model 1a/1b respectively. Because I used MLR, the test statistic was scaled using this scaling correction factor (Satorra & Bentler, 2010). If rumination failed to fit best loading on latent negative emotionality, I planned to carry out remaining analyses using two separate latent variables to test Hypothesis 2.

Additionally, prior to estimating the full measurement model, I assessed whether internalizing fit best as a general unidimensional internalizing factor or hierarchical model.

After assessing fit of the measurement model, I estimated the structural models. First, I estimated the model—termed Model 0—without the latent interaction term. Second, I estimated the structural models (for latent negative emotionality and rumination separately) including the latent interaction effect between EA and latent vulnerability, termed Model 1, with separate iterations of this model if two latent vulnerabilities were identified in testing Hypothesis 1. The relative fit of Model 0 versus Model 1a and 1b respectively was analyzed using a log-likelihood ratio test.

Power Considerations

As a preliminary step, I conducted an a priori power analysis using R Studio. For sample size estimation needed to reach 80% statistical power at a significance criterion of $\alpha = .05$, I used

observed effect sizes from past research (Henry et al., 2021; Liu et al., 2021; Jeronimus, 2015). Results indicated that to reach adequate statistical power to detect the expected additive effect sizes for EA (r = .29) and neuroticism (r = .55) on internalizing, a sample size of n = 340 was needed. Additionally, Zinbarg et al., 2016 achieved adequate power with a sample size of n = 547. Considering their findings are the foundation for Aim 1, the present study, with a sample size of 768, is expected to have the necessary statistical power for the primary analyses. Further, while there is no consensus regarding the number of participants necessary for SEM, one rule of thumb is a minimum of 10 participants per indicator variable (Nunnally, 1967), and the present study anticipated up to a maximum of 16 indicators (6 early adversity, 4 negative emotionality, and 6 internalizing symptoms) in the analyses, with the exact number of indicators pending several outcomes in model fitting.

CHAPTER III: RESULTS

Preliminary Analyses

As a first step prior to testing Hypothesis 1, I fit a measurement model (Figure A1) and adapted the initial model based on a priori predictions that were theory-consistent, first, adding positive correlations between the error terms of the two neuroticism scales (EPQR-N and NEO-IPIP-N), and anxiety sensitivity (ASI-3) and social phobia scales. Their positive association reflects that they are more closely related than the latent variable can account for, which in both cases is consistent with theory. Second, I added a negative correlation between the error terms of social phobia and depression based on modification indices that were theory consistent. Their negative association reflects that, after the latent variable accounted for commonality across scales, their remaining variance was negatively correlated; if we had used a hierarchical model, we would expect them to load on different dimensions of internalizing, and thus, this is understandable.

In preparation for testing Hypothesis 2, I examined several preliminary tests. First, to examine the effect of EA prior to accounting for trait vulnerabilities, I conducted a structural model with only early adversity predicting latent internalizing. As anticipated, in this model, results show that EA significantly predicted internalizing ($\beta = 0.369$, SE = 0.036, $p < .001$). Second, results indicated that modeling internalizing as a unidimensional factor had excellent model fit (RMSEA = .078, CFI = .983, TLI = .955) as opposed to a hierarchical model which was poor fitting in this sample (RMSEA = .108, CFI = .954, TLI = .914). Therefore, for the structural models, latent internalizing is modeled as a single latent factor. Third, I estimated the full measurement model including only the latent variables: rumination, negative emotionality (latent neuroticism and dysfunctional attitudes) and internalizing (Figure A1). I permitted these

latent variables to correlate. The full measurement model achieved adequate model fit: χ^2 (70, N = 768) = 260.773, p < .001, RMSEA = 0.060, CFI = 0.960, TLI = 0.948.

Primary Analyses

Hypothesis 1.

Results indicated that a model where brooding rumination loads onto a separate but correlated latent negative emotionality variable has better model fit than a model where brooding rumination is forced to load on latent negative emotionality, together with neuroticism and dysfunctional attitudes. The fit was determined based on the log-likelihood difference test (D = 40.62), which was significant at p < .001. Therefore, Hypothesis 1 was not supported, and although the two latent variables for trait vulnerability were highly correlated (r = .82), in this study's sample, these behave as separate constructs. Since rumination failed to load onto latent negative emotionality, these were treated as two latent variables in further analyses, and I tested whether each interacted with EA to predict latent internalizing.

Hypothesis 2.

As conducted in the preliminary analyses, CFI, TLI, RMSEA, and a chi-squared test was obtained from Model 0. Model 0 fit the data adequately: χ^2 (81, N = 768) = 332.829, p < .001, RMSEA = 0.064, CFI = 0.949, TLI = 0.933. Both negative emotionality and rumination (Figure A2) significantly predicted internalizing symptoms (β = .797, SE = 0.075, p < .001 and β = .174, SE = 0.072, p = .016, respectively). In contrast to this effect, and in contrast to preliminary analyses without the trait vulnerabilities, early adversity did not significantly predict internalizing symptoms in this sample when examined together with latent negative emotionality and latent brooding rumination (β = .006, SE = 0.029, p = .828). Model 0 explained 88.5% of the variance in latent internalizing symptomatology.

Next, I assessed the structural models, Model 1a (Figure A3) and Model 1b (Figure A4), including both vulnerabilities in the model when testing interaction effects. Results yielded a likelihood difference value of D = 0.80 for Model 1a (EA x Latent Negative Emotionality interaction) and D = 1.08 for Model 1b (EA x Latent Rumination interaction). The log-likelihood difference test was not significant for Model 1a compared to Model 0 (p = .370), or for Model 1b compared to Model 0 (p = .300), indicating that the models with the interaction effect did not significantly improve model fit relative to the alternative model, Model 0. The EA x Latent Negative Emotionality interaction effect was not significant (β = 0.020, SE = 0.023, p = 0.370), and the EA x Latent Brooding Rumination interaction effect was also not significant (β = 0.024, SE = 0.023, p = 0.298).

Model 1a yielded an R^2 of .888, suggesting it explained 88.8% of the variance for internalizing symptoms. Subtracting the R^2 of Model 0 from Model 1a yields a value of .003, which means the interaction of EA and latent negative emotionality only explained an additional .3% of the variance in internalizing symptoms in this sample. Model 1b yielded an R^2 of .885, suggesting it explained 88.5% of the variance for internalizing symptoms. Subtracting the R^2 of Model 0 from Model 1a yields a value of .000, which means the interaction effect of EA and latent brooding rumination did not offer any further explanation for the variance in internalizing symptoms. This was expected since Model 1a and Model 1b did not significantly improve model fit in comparison to Model 0. These findings yield partial support for Hypothesis 2, suggesting that early adversity and rumination, as well as EA and negative emotionality, do not have multiplicative effects on latent internalizing. However, in contrast to a preliminary model where EA predicted latent internalizing when included by itself, in the best fitting model including trait vulnerabilities (Model 0), EA was not a significant predictor, and trait vulnerabilities provided

the only significant main effects. Thus, we also do not support an additive model with dual main effects of diatheses and stressors.

CHAPTER IV: DISCUSSION

The present study provided evidence in a diverse sample of emerging adults, first, that trait brooding rumination and negative emotionality can be conceptualized and modeled as distinct yet correlated constructs, and second, that both of these vulnerabilities acted independently of early adversity (rather than primarily in interaction with it) to predict latent past-year internalizing symptoms. Contrary to expectations, however, despite evidence of an association between early adversity and internalizing when examined alone, in the final model, early adversity's effects on latent internalizing were negligible in the present sample after accounting for trait vulnerabilities. This discussion also considers the role of sample characteristics and study design in these findings.

Latent Rumination and Negative Emotionality

Contrary to prediction in Hypothesis 1, rumination did not fit best as part of a latent negative emotionality variable, together with traditional neuroticism measures and dysfunctional attitudes. Instead, trait rumination, specifically, brooding rumination, loaded onto its own distinct construct that is separate, but highly correlated with latent negative emotionality in this sample. Past research about the structure of latent neuroticism and related vulnerabilities found that dysfunctional attitudes and other latent vulnerabilities loaded onto latent neuroticism (Zinbarg et al., 2016). The present study sought to extend Zinbarg et al. to include trait brooding rumination because past research had suggested rumination and neuroticism are highly correlated (Lu et al., 2017). The current findings suggest that brooding rumination is distinct from, if still highly correlated with, latent neuroticism in this group of emerging adults. Therefore, while the current study did not yield support for a general neuroticism factor with the inclusion of trait rumination, at the very least, the two constructs are closely related. This result could be due in part to

measuring rumination using only the brooding rumination items; I selected brooding rumination to apply the most stringent test possible, to avoid the potential for depression-related items in the full rumination construct (Treynor et al., 2003) to artificially inflate its relatedness with trait negative emotionality. Modeling rumination with only brooding rumination allowed for a more stringent test of the role of ruminative behaviors most associated with internalizing symptoms, but rumination is not always measured using the brooding rumination subscale only. Future research should replicate whether the constructs are better modeled as one latent variable or two separate constructs in other types of sample demographics, giving consideration to how rumination might be best measured and modeled, because there may be variability across samples.

Additionally, trait rumination and latent negative emotionality, though separate constructs, were very highly correlated in the present study. Despite this, the relative effect size of brooding rumination on internalizing was descriptively smaller in comparison to latent negative emotionality. Past research found significant small effect sizes between brooding rumination and depression ($\beta = .10$, $p = .004$; Jose & Weir, 2013) and anxiety ($\beta = .09$, $p = .016$; Jose & Weir, 2013). Furthermore, Mineka et al. found moderate main effects of the general neuroticism factor on major depressive episodes. In the present study however, the effects of negatively emotionality on internalizing were descriptively much larger ($\beta = .797$, SE = 0.075), especially in comparison to effect sizes of brooding rumination ($\beta = .174$, SE = 0.072). Taken together, this suggests that while brooding rumination significantly predicts internalizing symptomatology, its effects are descriptively somewhat less robust than are those of negative emotionality, and thus negative emotionality offers a more definitive implication in the etiology

of internalizing symptoms. Therefore, intervention efforts should target reductions in negative emotionality and to a lesser extent, brooding rumination.

Effects of Early Adversity and Trait Vulnerabilities on Latent Internalizing

As hypothesized, brooding rumination and negative emotionality each had significant main effects on internalizing symptomatology above and beyond any observed multiplicative effects in this sample (Hypothesis 2). Interaction effects were not significant, suggesting that primarily the trait vulnerabilities conferred risk of internalizing symptoms alone, with descriptively larger effects for negative emotionality than for brooding rumination.

Past research on diathesis-stress relationships has largely concluded that their effects were primarily multiplicative rather than additive for depressive disorders (Brown & Rosellini, 2011). On the contrary, more recent research supports additive depressogenic effects of diatheses and stressors. Specifically, results suggest that chronic stress has a main effect on depressive symptoms, independent of any diathesis effects (Vrshek-Schallhorn et al., 2015). The present study suggests that there are significant main effects rather than interactive effects of diatheses and stressors on internalizing etiology. These findings extend Mineka et al.'s (2020) finding on the additive effects of neuroticism and stress on MDD to consider early adversity and other trait vulnerabilities, specifically trait rumination. The current findings suggest that there was no evidence of multiplicative effects, and instead, internalizing was predominantly predicted by trait vulnerabilities alone, whereas the unique contributions of EA in the final model were negligible and not significant. An implication of the current research is that the lack of multiplicative effects between EA and trait vulnerabilities may offer a clearer explanation on how risk is conferred for internalizing disorders—and therefore also clues to novel early or preventative interventions. Specifically, the current work suggests that intervention efforts should focus

primarily on reducing negative emotionality tendencies (e.g., the unified protocol for transdiagnostic treatment of emotional disorders; see Barlow et al., 2011, for a review).

Beyond this, Griffith et al.'s latent variable analyses suggested latent neuroticism and internalizing symptoms were so highly correlated (latent variable $r = .98$) that they represent the same concept in a sample of adolescents. Similar studies in an adult sample (Krueger et al., 2001), like the present study, found relatively smaller yet still robust associations between latent negative emotionality and latent internalizing symptoms, suggesting that while neuroticism significantly predicts internalizing, it indeed represented its own distinct construct in the present sample. Furthermore, in the Zinbarg et al. study (2016), neuroticism was used as a prospective determinant of first onset of unipolar mood (UMDs) and anxiety disorders (ADs). Their findings suggest that participants who scored high on neuroticism at the baseline assessment did not have the presence of UMDs or ADs until later in the 3-year follow-up sessions. Thus, these results yield support for neuroticism as its own distinct construct from internalizing. The present study challenges prior results on neuroticism-stress relationships because testing early adversity, as opposed to current stress, leaves potential for mediation effects of negative emotionality on internalizing that are not as plausible with recent adversity.

One useful future direction could be a meta-analysis that compares the effect sizes of interactive effects versus main effects in diathesis-stress models of internalizing. It may be that there are conditions under which interactions account for a similar amount of variance as main effects, and one of the potential factors that may influence this in the present study could be that the relatively weak main effects of EA on internalizing (after addition of trait vulnerabilities) left very little variance for an interaction effect to parse out. As such, it may be that interaction effects in this sample were smaller than would be observed in a sample that found similar EA

effect sizes to past research. Testing this in a meta-analysis would allow researchers to decipher whether the interaction effects were as robust as the main effects across an array of samples, and the conditions and methodology—if any—under which the interaction effects are both significant and comparable in effect size to main effects.

Structural Modeling of Early Adversity and Latent Internalizing

Several aspects of model fitting have implications for future research. As anticipated, early adversity did not have adequate fit as a latent variable and as such, I included it as a manifest variable. Early adversity often has heterogenous sources rather than similar sources, which likely impacted my ability to model it as a latent variable. In the future, researchers attempting to model EA as a latent variable could focus on more homogeneous specific aspects of EA (e.g., emotional abuse) that are likely to be consistent and caused by a single latent factor (e.g., caregiver's behaviors).

In addition, a general internalizing model fit better than a hierarchical model in this sample. Previous research has found support for a hierarchical model of internalizing (Kreuger, 1999; Watson, 2009), whereas other work suggested modeling internalizing as a unidimensional factor (e.g., Eaton et al., 2011; Fergusson et al., 2006). Some research has noted robust correlations between fear and distress disorders, supporting the use of a single internalizing factor in the present study (Kotov et al., 2011). Therefore, future work should continue to assess which model of internalizing is best fitting in each given sample.

Early Adversity Predicting Internalizing

Although preliminary models before testing Hypothesis 2 showed a significant effect of EA on internalizing of the magnitude anticipated when examining it as the only predictor, in the primary models for Hypothesis 2, unexpectedly, early adversity did not significantly predict past-

year internalizing symptoms in the structural models after the addition of trait vulnerabilities to models. Past research consistently suggests that those who experience early adversity are at risk of mental health symptoms compared to those without EA (Kessler et al., 2010; McLaughlin, 2018; Tackett et al., 2013), and thus, my results are surprising. Given that I obtained significant effects of EA in preliminary models before adding latent negative emotionality to my model, one explanation for this is that negative emotionality accounts for so much variance that it may be "absorbing" the variance between EA and internalizing in the full structural models. Specifically, Zhou et al. (2019) suggested that neuroticism mediates the relationship between childhood emotional abuse, a facet of early adversity, and later depression symptoms—something the present cross-sectional study was not designed to test.

In addition, although EA is a well-documented risk factor for internalizing, research on stress recency theory suggests that the influence of recent adversity is more influential on mental health outcomes than distal events, like early adversity (Shanahan et al., 2011). Similarly, Hazel et al. (2008) found that when accounting for recent stress experiences, the main effects of EA diminish. Therefore, negative emotionality, a trait vulnerability capturing *current* rather than *distal* tendencies (rather than experience per se), may be performing similarly in the current study by decreasing the potency of early adversity effects. Nonetheless, it is evident with further investigation that early adversity does have significant main effects on internalizing risk even in this sample when examined as a predictor by itself, but this effect was eliminated by effects of negative emotionality.

One additional possible explanation for the diminished effects of EA on past-year internalizing is the composition of the present sample. Unlike most samples in psychosocial research, this sample was made up of a majority-minority participants, with over seventy percent

of the participants endorsing minoritized racial/ethnic status. Furthermore, the largest represented racial group was Black/African Americans, who are disproportionately affected by adverse childhood experiences (ACEs). Previous research on the effects of ACEs and racial/ethnic differences found that the types of early adversity differ and/or are experienced to a much larger degree in Black respondents than White respondents. For example, Black respondents reported witnessing serious violence or murder at a rate twice as high as White participants did (Schilling et al., 2007). Therefore, because the types of adversity experienced are different in racial/ethnic minorities, the relationship between EA and internalizing symptoms may also plausibly be different as well. Results indicated that the effects of early adversity on psychopathology are stronger in Whites than in Blacks or Hispanics despite them being exposed to more adverse experiences (Schilling et al., 2007). Considering the present sample makeup, the effects of EA on internalizing may be diminished beyond what would be observed in more representative samples of psychosocial research.

Though past research has shown that ACEs, especially cumulative adversity, confers risk for internalizing symptoms, less is understood about this relationship in racially diverse samples. This study makes a unique contribution to highlighting these potential effects, but the current study was not suited to test this using a multi-group analysis since the subsample sizes were not large enough. This is an area for future research.

Limitations

Although the sample size was moderately large for psychosocial research and racially/ethnically diverse, the present study is not without limitations. First, while I initially collected a measure of PTSD, because we did not evaluate for PTSD Criterion A trauma exposure, it was removed from the planned primary analyses. Second, I was only able to assess

peak past year internalizing symptoms and did not have access to any longitudinal data because this was not within the scope of the current project. In addition to being preferable to increase variation across individuals and potentially reduce skew, choosing to measure peak internalizing symptoms in the past year as opposed to current symptoms allowed the present study to capture episodic symptoms (common in depression) that would have been overlooked if I had examined a shorter timespan such as the past two weeks or the past month. Third, my sampling strategy included a narrow age range including only emerging adults. While the current project sought to understand such relationships in emerging adults, future studies could assess the relationship between EA and negative emotionality to latent internalizing in adolescents or older adults. Fourth, the present sample size may be too small to have adequate statistical power to detect small effects of an EA-negative emotionality interaction. Kendler et al. (2004) reported significant interaction effects between neuroticism and stress, and the sample size used to detect this effect was 10 times larger than that of the present study. Nonetheless, even the significant interaction effects found in Kendler et al. were so small in magnitude as to lack real-world significance, which suggests that the present sample size did not meaningfully alter the conclusions I draw here.

Conclusion

In conclusion, the present study extended previous findings on the relationship between latent neuroticism and other related factors to show that brooding rumination, though similar to negative emotionality, was best model as its own construct in this sample. Also, I observed main effects of early adversity only when examined in isolation in preliminary models, but it did not significantly predict internalizing once trait vulnerabilities were included in models; by contrast, trait vulnerabilities (latent negative emotionality and latent brooding rumination) had robust

main effects on latent internalizing in emerging adults in the final model in my sample. These findings aid in the conceptualization of etiological factors of internalizing symptoms, which can help clinicians more accurately determine who is deemed “at risk” for internalizing symptoms and targeting with preventive interventions.

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APPENDIX A: EA, NEGATIVE EMOTIONALITY, AND LATENT INTERNALIZING

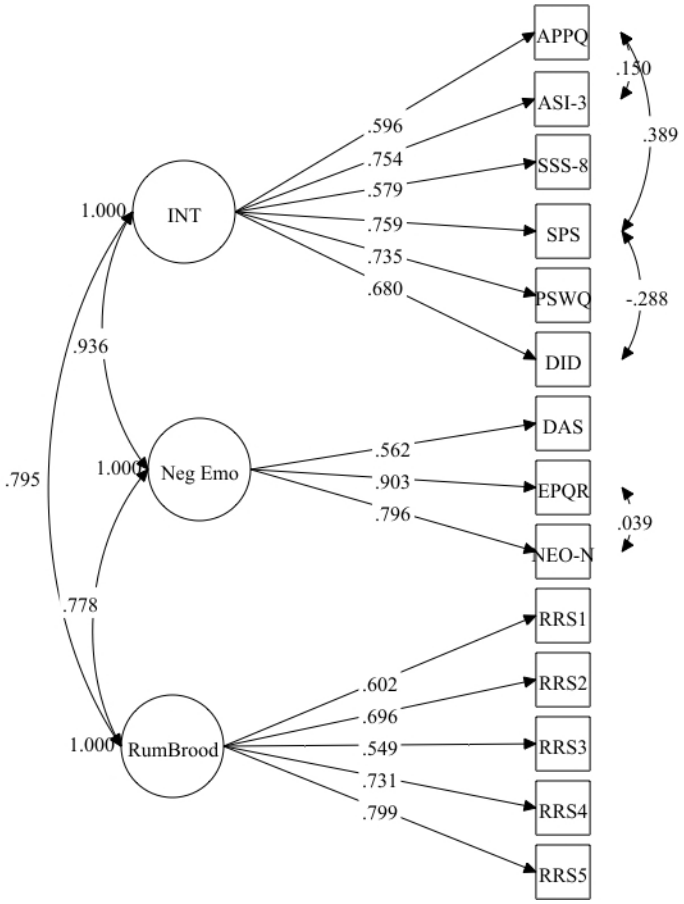
Table A1. Bivariate Correlations Among Variables

		Variable number																		
Variable	*M (SD)*	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. ACE	4.97 (3.52)	—																		
2. SSS-8	2.67 (0.91)	.230**	—																	
3. APPQ	3.78 (1.84)	.158**	.373**	—																
4. ASI-3	1.39 (0.77)	.193**	.458**	.555**	—															
5. SPS	2.76 (0.75)	.144**	.405**	.664**	.637**	—														
6. CTQ EA	2.24 (1.17)	.599**	.316**	.246**	.344**	.283**	—													
7. CTQ PA	1.52 (0.74)	.551**	.138**	.162**	.153**	.092*	.554**	—												
8. CTQ SA	1.47 (0.95)	.428**	.153**	.190**	.130**	.108**	.374**	.462**	—											
9. CTQ EN	2.13 (1.02)	.427**	.158**	.160**	.218**	.167**	.551**	.342**	.262**	—										
10. CTQ PN	1.46 (0.59)	.531**	.129**	.188**	.196**	.096**	.451**	.482**	.350**	.592**	—									
11. CTQ Tot	1.76 (0.67)	.679**	.255**	.258**	.293**	.218**	.828**	.737**	.660**	.754**	.723**	—								
12. PSWQ	3.54 (0.87)	.133**	.410**	.414**	.494**	.557**	.247**	.044	.079*	.078*	.003	.143**	—							
13. RRS Tot	2.36 (0.65)	.251**	.462**	.448**	.626**	.558**	.384**	.135**	.134**	.222**	.105**	.289**	.569**	—						
14. RRS-Brood	2.44 (0.75)	.239**	.387**	.454**	.578**	.542**	.365**	.137**	.126**	.171**	.084*	.261**	.533**	.880**	—					
15. RRS-Refl	2.22 (0.70)	.200**	.318**	.288**	.417**	.331**	.253**	.076*	.089*	.143**	.068	.186**	.370**	.790**	.599**	—				
16. DAS-SF	2.17 (0.60)	.153**	.271**	.266**	.420**	.382**	.266*	.084*	.127**	.178**	.140**	.227**	.375**	.446**	.435**	.214**	—			
17. DID	4.35 (0.80)	.335**	.495**	.396**	.479**	.379**	.425**	.220**	.244**	.235**	.216**	.377**	.470**	.613**	.491**	.451**	.330**	—		
18. EPQR-N	1.62 (0.23)	.256**	.472**	.493**	.627**	.638**	.376**	.133**	.161**	.238**	.145**	.305**	.682**	.683**	.647**	.442**	.485**	.563**	—	
19. NEO-IPIP	2.92 (0.69)	.274**	.423**	.403**	.526**	.515**	.395**	.160**	.214**	.296**	.194**	.359**	.593**	.598**	.533**	.338**	.499**	.557**	.729**	—

Note. ** correlation is significant at the .01 level, two-tailed; * correlation is significant at the .05 level, two-tailed.

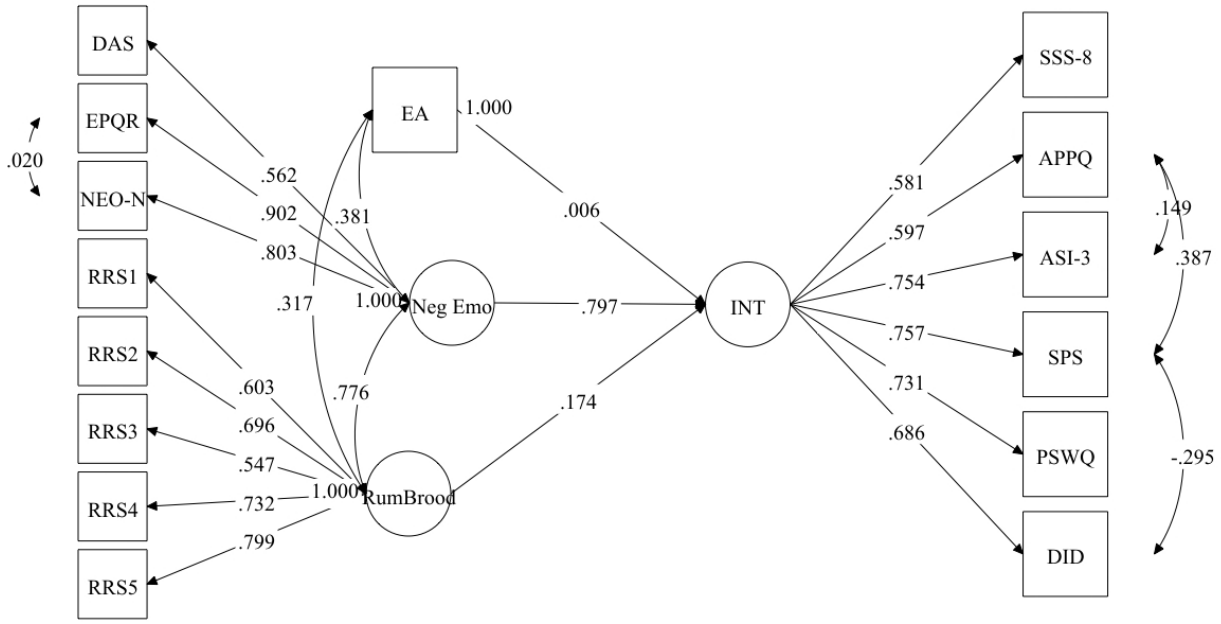
[α] *M* = Mean; *SD* = Standard Deviation; ACE = Revised ACEs; SSS-8 = Somatic Symptom Scale-8; APPQ = Albany Panic and Phobia Questionnaire; ASI-3 = Anxiety Sensitivity Index-3; SPS = Social Interaction Anxiety and Social Phobia Scales; CTQ EA = Childhood Trauma Questionnaire Emotional Abuse; CTQ PA = Childhood Trauma Questionnaire Physical Abuse; CTQ SA = Childhood Trauma Questionnaire Sexual Abuse; CTQ EN = Childhood Trauma Questionnaire Emotional Neglect; CTQ PN = Childhood Trauma Questionnaire Physical Neglect); CTQ Tot = Childhood Trauma Questionnaire Total Scale; PSWQ = Penn State Worry Questionnaire; RRS Total = Ruminative Response Scale Total Scale; RRS- Brood = RRS Brooding Rumination Subscale; RRS- Refl = RRS Reflective Rumination Subscale; DAS-SF = Dysfunctional Attitudes Scale Short Form; DID = Shortened Diagnostic Inventory for Depression; EPQR-N = Eysenck Personality Questionnaire Revised N Scale; NEO-IPIP =10-item International Personality Item Pool NEO-PI-R Scale.

Figure A1. Full Measurement Model for EA and Trait Vulnerabilities on Internalizing



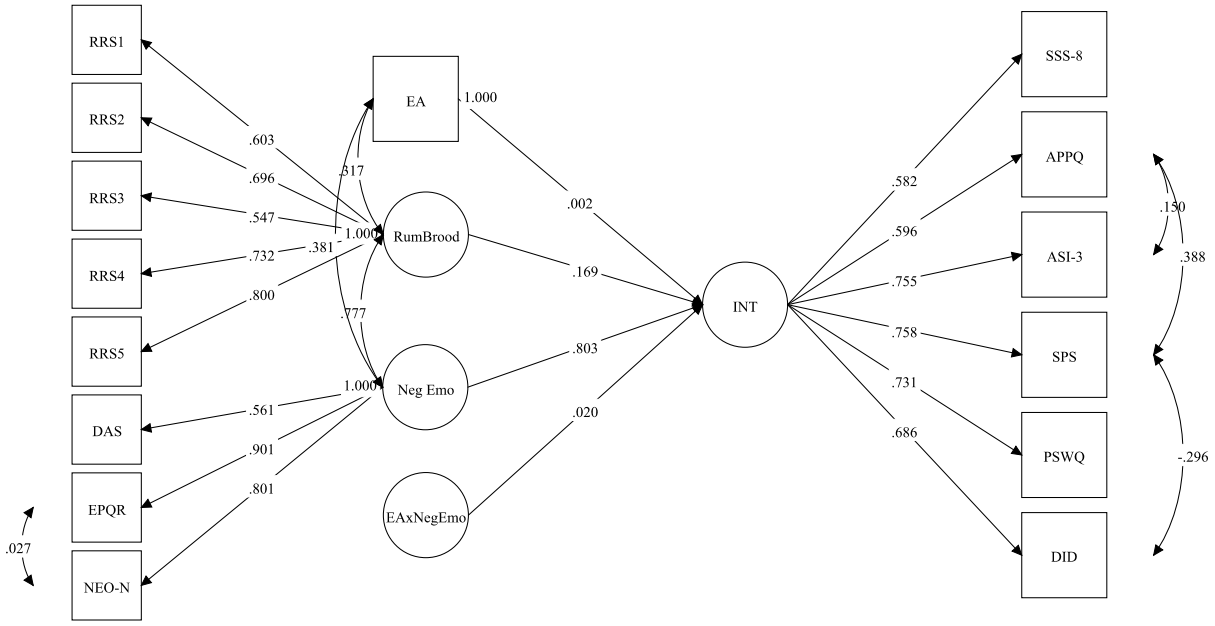
Note. In this figure, INT = Internalizing; Neg Emo = Negative Emotionality; RumBrood = Brooding Rumination; APPQ = Albany Panic and Phobia Questionnaire; ASI-3 = Anxiety Sensitivity Index-3; SSS-8 = Somatic Symptom Scale-8; SPS = Social Interaction Anxiety and Social Phobia Scales; PSWQ = Penn State Worry Questionnaire; DID = Shortened Diagnostic Inventory for Depression; DAS = Dysfunctional Attitudes Scale Short Form; EPQR = Eysenck Personality Questionnaire Revised N Scale; NEO-N =10-item International Personality Item Pool NEO-PI-R Scale; RRS1 = Ruminative Response Scale Item 5; RRS 2 = Ruminative Response Scale Item 10; RRS 3 = Ruminative Response Scale Item 13; RRS 4 = Ruminative Response Scale Item 15; RRS5 = Ruminative Response Scale Item 16.

Figure A2. Structural Model 0 for EA and Negative Emotionality on Internalizing



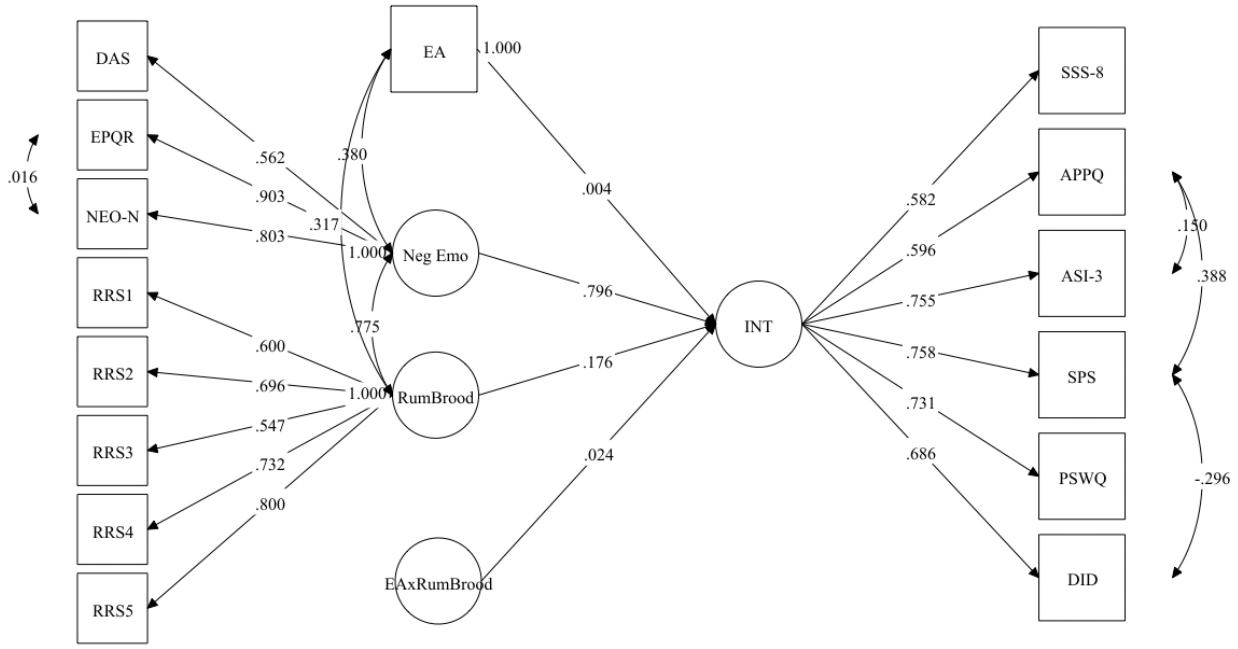
Note. In this figure, INT = Internalizing; Neg Emo = Negative Emotionality; RumBrood = Brooding Rumination; DAS = Dysfunctional Attitudes Scale Short Form; EPQR = Eysenck Personality Questionnaire Revised N Scale; NEO-N =10-item International Personality Item Pool NEO-PI-R Scale; RRS1 = Ruminative Response Scale Item 5; RRS 2 = Ruminative Response Scale Item 10; RRS 3 = Ruminative Response Scale Item 13; RRS 4 = Ruminative Response Scale Item 15; RRS5 = Ruminative Response Scale Item 16; SSS-8 = Somatic Symptom Scale-8; APPQ = Albany Panic and Phobia Questionnaire; ASI-3 = Anxiety Sensitivity Index-3; SPS = Social Interaction Anxiety and Social Phobia Scales; PSWQ = Penn State Worry Questionnaire; DID = Shortened Diagnostic Inventory for Depression.

Figure A3. Structural Model 1a for EA and Negative Emotionality on Internalizing



Note. In this figure, INT = Internalizing; Neg Emo = Negative Emotionality; RumBrood = Brooding Rumination; EAxNegEmo = EA interaction with Negative Emotionality; RRS1 = Ruminative Response Scale Item 5; RRS 2 = Ruminative Response Scale Item 10; RRS 3 = Ruminative Response Scale Item 13; RRS 4 = Ruminative Response Scale Item 15; RRS5 = Ruminative Response Scale Item 16; DAS = Dysfunctional Attitudes Scale Short Form; EPQR = Eysenck Personality Questionnaire Revised N Scale; NEO-N =10-item International Personality Item Pool NEO-PI-R Scale; SSS-8 = Somatic Symptom Scale-8; APPQ = Albany Panic and Phobia Questionnaire; ASI-3 = Anxiety Sensitivity Index-3; SPS = Social Interaction Anxiety and Social Phobia Scales; PSWQ = Penn State Worry Questionnaire; DID = Shortened Diagnostic Inventory for Depression.

Figure A4. Structural Model 1b for EA and Trait Rumination on Internalizing



Note. In this figure, INT = Internalizing; Neg Emo = Negative Emotionality; RumBrood = Brooding Rumination; EAxRumBrood = EA interaction with Brooding Rumination; DAS = Dysfunctional Attitudes Scale Short Form; EPQR = Eysenck Personality Questionnaire Revised N Scale; NEO-N =10-item International Personality Item Pool NEO-PI-R Scale; RRS1 = Ruminative Response Scale Item 5; RRS 2 = Ruminative Response Scale Item 10; RRS 3 = Ruminative Response Scale Item 13; RRS 4 = Ruminative Response Scale Item 15; RRS5 = Ruminative Response Scale Item 16; SSS-8 = Somatic Symptom Scale-8; APPQ = Albany Panic and Phobia Questionnaire; ASI-3 = Anxiety Sensitivity Index-3; SPS = Social Interaction Anxiety and Social Phobia Scales; PSWQ = Penn State Worry Questionnaire; DID = Shortened Diagnostic Inventory for Depression.