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Brooding, Avoidance, and Suppression as Mechanisms Linking Shame-Proneness with Depressive Symptoms

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Abstract

Depression is a significant mental health concern. Cognitive-affective models of depression identify that negative emotions and cognitive strategies for responding to negative emotions contribute to the development and maintenance of depressive symptoms. Shame has been identified as a problematic negative emotion and is associated with multiple mental health concerns including depression. Research has begun to examine cognitive emotion regulation strategies individuals use when experiencing shame and how these contribute to depressive symptoms. This study examined three strategies jointly (avoidance, brooding, and suppression) in a three-part prospective design. In a sample of 137 young adults, three hypotheses were tested. Participants ranged from 18 to 29-years-old (M = 19.29, SD = 1.56), 83.2% of the participants were female, and 74.5% were Caucasian. In cross-sectional analyses, shame-proneness predicted depressive symptoms (B = .029, 95% CI = .010 to .048, p = .003) and brooding mediated this relationship as hypothesized (B = .010, 95% CI = .003 to .019, p = .005). In prospective analyses shame-proneness marginally predicted depressive symptoms (B = .016, 95% CI = -.002 to .033, p = .074) and only suppression mediated the relationship when controlling for guilt-proneness (B = .012, 95% CI = .004 to .024, p = .002). Post hoc analyses of each mediator examined separately supported avoidance (B = .018, Z = 3.251, p = .001), brooding (B = .020, Z = 3.501, p = .001), and suppression (B = .022, Z = 3.602, p < .001) as cognitive strategies in the relationship between shame-proneness and depressive symptoms prospectively. State shame was

predicted to mediate the relationship between shame-proneness and state brooding, avoidance, and suppression. The shame induction did induce a significant change in shame [t (114) = -2.814, p = .006] but a small effect (r = .25). Therefore, hypothesis 3 was not supported. However, shame-proneness did predict use of avoidance (B = .003, p = .048) and brooding in the moment (B = .003, p = .071). These findings suggest that shame-proneness and avoidance, brooding, and suppression are significant factors to consider in treating depression. Future directions of research and clinical implications are discussed.

CHAPTER I

Introduction and Literature Review

Purpose

Depression is a significant mental health concern, increasing substantially in adolescence and often continuing into young adulthood, causing impaired functioning (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Depression is a mood disorder characterized by excessive negative emotions and the absence of positive emotions (Clark & Watson, 1991). Research shows that onset of depressive symptoms in late adolescence and early adulthood increases the risk for depressive disorders later in life (Bardone, Moffitt, Caspi, Dickson, & Silva, 1996). Increasing our understanding of the risk factors for depressive symptoms, especially among adolescents and young adults, is important.

While much research has focused on the link between broad negative emotionality and depressive symptoms, less research has examined the relationship between individual negative emotions and depressive symptoms. One emotion of interest, shame, has been implicated as a significant risk factor for psychopathology. The emotional state of shame is defined by intense pain felt in response to perceived personal characterological flaws or negative evaluation of the global self (Lewis, 1971). Pervasive feelings of shame at a state or trait level (*shame proneness*; Tangney, 1996) can lead to a number of psychological problems, including depression (Andrews, Qian, & Valentine, 2002; De Rubeis, & Hollenstein, 2009; Kim, Thibodeau, & Jorgensen, 2011; Tangney & Dearing, 2002). Shame is to be distinguished from the emotion of guilt. Guilt has previously been studied as largely indistinguishable from shame but is currently viewed as a less painful and damaging emotional state elicited by the negative evaluation of a specific behavior (Tangney, Wagner, & Gramzow, 1992). Research comparing shame and guilt

in relation to depression has found that shame-proneness, tendency to experience shame, is more strongly linked to depression than guilt-proneness, the tendency to experience guilt (Fontaine, Luyten, De Boeck, & Corveleyn, 2001; Harder, Cutler, & Rockart, 1992; Kim et al., 2011; Pineles, Street, & Koenen, 2006).

While the link between shame-proneness and depression has been well established, research has only begun to explore the mechanisms driving this relationship. Current theory and research suggests that the relationship between shame-proneness and depression may be mediated by maladaptive emotion regulation strategies designed to reduce, escape, or avoid shame. In the early stages, studies have examined this relationship with cross-sectional designs, finding that rumination, defined as perseverative thought on negative emotions or events (Nolen-Hoeksema, 1991; Treynor, Gonzales, & Nolen-Hoeksema, 2003), mediates the relationship between state shame and depression (Cheung, Gilbert, & Irons, 2004). In addition, De Rubeis and Hollenstein (2009) found that the relationship between shame-proneness and depressive symptoms was fully mediated by avoidant coping strategies in an early adolescent sample. Aldao, Nolen-Hoeksema, and Schweizer (2010) found that rumination and avoidance are two emotion regulation strategies with the strongest association with depression. In addition, suppression is a maladaptive emotion regulation strategy associated with depression (Campbell-Sills, Barlow, Brown & Hofmann, 2006); however, no studies have examined suppression as a potential mediator between shame-proneness and depression. Thus, one purpose of the current study is to examine the maladaptive trait emotion regulation strategies of rumination, avoidance, and suppression as potential mediators of the relationship between trait shame-proneness and depressive symptoms among young adults. By learning what emotion regulation strategies

increase likelihood of developing depressive symptoms when faced with the emotion of shame, therapeutic strategies that intervene on this process can be developed and implemented.

In addition to research focused on self-report of trait shame-proneness and trait emotion regulation strategies, some research has explored state shame and state emotion regulation of shame. Orth, Berking, and Burkhardt (2006) examined the state emotion of shame in parents who were recently separated, state rumination, and current depressive symptoms. They found a pattern consistent with the proposition that state rumination mediated the relationship between state shame and depression. A second purpose of the current study is to expand the current literature by examining these relationships prospectively to support causal inferences. Matos, Pinto-Gouveia, and Costa (2013) examined the brooding, thought suppression and dissociation in response to shame-related traumatic memories and found that each mediated the relationship between shame traumatic memories and depression, suggesting that state efforts to escape or reduce shame may be a key mechanism linking shame with depression. Thus, a third purpose of the current study is to examine the relationship between state shame and the state deployment of emotion regulation strategies of brooding, avoidance, and suppression.

Conceptualization of Depression

Depressive symptoms are a significant mental health concern worldwide. In addition to increased negative emotions and decreased positive emotions, depressive symptoms include feelings of worthlessness or guilt, difficulties concentrating, thoughts of death, significant changes in weight or appetite, sleep difficulties, and fatigue or low energy (DSM-5, American Psychological Association, 2013). When a number of these symptoms persist over time and impair daily functioning, a diagnosable depressive disorder may develop. The variation in presentation and severity fall on a continuum of depression that is influenced by the number of

symptoms and the frequency of occurrence (Hankin, Fraley, Lahey, & Waldman, 2005). The onset of depression is often in adolescence and early adulthood and research has found that 20% of individuals will experience a major depressive episode during this period of development (Hankin & Abramson, 1999). By the age of 24, estimates are that 1 in 4 adults have experienced a depressive episode (Kessler & Walters, 1998). Studies indicate that even subclinical levels of depressive symptoms are a risk factor for future depression (Bardone, Moffitt, Caspi, Dickson, and Silva, 1996) and other negative consequences, including increased suicidality (Andrews & Lewinsohn, 1992), increased substance use (Lewinsohn, Solomon, Seeley, & Zeiss, 2000), and difficulties with daily functioning and academic performance (Gotlib, Lewinsohn, & Seeley, 1995; Rothon, et al., 2008). Subclinical levels of depression have been defined in various ways according to duration, number of symptoms present, or scores on depression assessment measures. Therefore, the research shows variations in prevalence rates. For instance, in a young adult sample, Rosenthal and Schreiner (2000) found that 42% of participants experienced moderate to high levels of subthreshold depressive symptoms. Knowing the pervasiveness of this disorder and the negative consequences that result for individuals with depressive symptoms, it is important to explore and understand mechanisms driving this outcome in order to ultimately prevent the onset or reduce the impairment of depressive symptoms in society.

Theoretical Framework

Affective models of depression. Depression is recognized as a disorder of emotion dysregulation that results in high levels of negative emotions and low levels of positive emotions. From a vulnerability perspective, affective models of depression stress the importance of acknowledging affective vulnerabilities in the relationship between life events and depressive outcomes (Compas, Connor-Smith, & Jaser, 2004), such that how individuals respond

emotionally to life events explains the development of depressive symptoms. Present in infancy, temperament is an enduring pattern of interaction with the world through emotions, behaviors, and attention (Rothbart, 2007). Temperament varies across individuals, but remains fairly stable over time, impacting the development of adaptive and maladaptive responses to the world that carry on into adulthood. Research suggests that temperament is a vulnerability factor for depression (Muris & Ollendick, 2005). One component of temperament, trait negative affect, captures the pattern of affective reactions to life events, including one's capacity to experience negative emotions and sensitivity to negative events (Belsky, Hsieh, & Crnic, 1996; Rothbart & Bates, 2006).

Trait negative affect as a vulnerability to depressive symptoms. A strong association has been demonstrated between trait NA and depression (Anthony, Lonigan, Hooe, & Phillips, 2002; Clark & Watson, 1991; Lengua, West, & Sandler, 1998; Mineka, Watson, & Clark, 1998). In addition, research has found that in adolescent samples, trait NA predicts variations in depressive symptoms over the subsequent eight weeks (Mezulis & Rudolph, 2012). In addition, trait NA was found to predict depressive symptoms in adolescents five (Verstraeten, Vasey, Raes, & Bijttebier, 2009) and 12 months later (Wetter & Hankin, 2009). Like adolescence, young adulthood is also a time of increased risk of developing depression. Research has found that trait NA predicts depressive symptoms in young adults three months later (Loh & Schutte, 2014). Ormel, Oldehinkel, and Vollebergh (2004), in a three-wave study, found that neuroticism (another name for trait NA) positively predicted a major depressive episode one to three years later. In addition, Parrish, Cohen, and Laurenceau (2011) examined the reverse relationship to determine if NA was a symptom of or developed along with depressive symptoms and found

support that NA represents an affective vulnerability present prior to and affecting the development of later depressive symptoms.

Trait NA is hypothesized to influence state NA in response to life events and research has suggested that when faced with stressful life events, individuals with high trait NA are more likely to respond to life events with high levels of state NA (Simonson, Sanchez, Arger, & Mezulis, 2012). However, state NA may still vary depending upon the degree of stress and context of the life event encountered; therefore, it is possible that state NA may predict depressive symptoms above and beyond trait levels of NA. State NA, such as sadness, fear, anger or shame, is the current experience and intensity of negative emotions occurring in response to a life events (Watson, Clark, & Tellegen, 1988). Research found that state NA in response to life events predicted depressive symptoms two months later (O'Neill, Cohen, Tolpin, & Gunthert, 2004; Parrish, Cohen, & Laurenceau, 2011). In addition to studying how NA predicts depression, research also examines the role of individual negative emotions in the development and maintenance of depression.

Shame-proneness is an Affective Vulnerability to Psychopathology. The emotion of shame is a self-conscious, social, and moral emotion pervasive and unique in humans. While not currently recognized as a predictor of depression according to the DSM-5 (APA, 2013), research has found that a pattern of responding with shame (*shame-proneness*) is associated with negative psychological outcomes over time, including PTSD, anxiety disorders, eating disorders, and depression (Mills, 2005; Tangney & Dearing, 2002).

Cognitive theories view shame as an emotion elicited by appraisal or cognitive evaluation of an event that results in the conclusion that the self is bad or unacceptable (Mills, 2005). Experiencing multiple and profound negative life events over time and concluding from those

events that the self is bad elicits a disposition toward shame that is accompanied by shame-related cognitive biases, emotional states, and behaviors, shame proneness (Bosson & Prewitt-Freilino, 2007). Research has also examined shame-proneness and guilt-proneness in relation to attributional style, which is a pattern a person develops over time to determine the cause of an event. In an undergraduate population, Tangney, Wagner, and Gramzow (1992) found that shame-proneness, which is conceptualized as the moral emotion that elicits a self-focus rather than a focus on specific behaviors, was positively associated with internal, stable and global attributions for negative and positive events. This means that a person prone to experience shame is likely to attribute the cause of an event to something about themselves, that is enduring (ex. a trait), and consistent across multiple contexts. In contrast, guilt-proneness was not consistently correlated with a specific attributional style.

Shame-proneness is Distinct from Guilt-proneness. Historically, shame was studied as a synonym of guilt. Even today, the difference between guilt and shame is largely unknown in the general population. Both shame and guilt are moral and self-conscious, negatively-valenced emotions elicited by negative events and involve direct cognitive attention to the self and the self's role in an event that often involves moral failures or transgressions (Tangney & Dearing, 2002). Lewis suggested that guilt represents an emotional response to the appraisal that one's own specific behavior physically or emotionally harmed another person whom one values. In contrast, shame is an emotional response to the appraisal that others view the self as worthless, bad, or defective. Whereas the emotion of guilt is experienced when the focus is on the behavior of the event, shame is the emotion experienced when the focus is on the whole core self (Lewis, 1971). Research has found that shame-proneness is positively associated with maladaptive

interpersonal outcomes including less empathy and more anger and hostility (Tangney, 1991; Tangney, Wagner, Fletcher, & Gramzow, 1992).

Shame-proneness is Associated with Greater Depressive Symptoms. In the DSM-IV (APA, 2000), guilt, not shame, is indicated as a symptom of a major depressive episode. Previous clinical research focused heavily on guilt as a maladaptive emotion associated with mental illness. This was due in part because the distinction between guilt and shame was not established. Research, following the establishment of a distinction between shame and guilt in 1971, has shown that shame and shame-proneness are more strongly associated than guilt with psychopathology (Tangney & Dearing, 2002) and specifically, depression (Andrews, Qian, & Valentine, 2002; Cheung, Gilbert, & Irons, 2004; Kim, Thibodeau, & Jorgensen, 2011; Pineles, Street, & Koenen, 2006; Tangney, Wagner, & Gramzow, 1992).

Tangney, Wagner, and Gramzow (1992) examined shame-proneness and guilt-proneness and their relation to psychological maladjustment and depressive symptoms in an undergraduate population. In their correlational analyses, they found that shame-proneness was strongly associated with psychological maladjustment while guilt-proneness was only moderately associated. In turn, given the high correlation between guilt and shame in studies 1 and 2 (.46 and .63), they conducted part-correlation analyses controlling independently for shame and guilt. They found that shame was significantly correlated with all indices of psychopathology and guilt was not. This suggests that guilt-proneness, independent of factors that correlate with shame, has little association with psychological maladjustment and predicts better outcomes in some studies (Mascolo, 1995; Strelan, 2007; Tangney & Dearing, 2002).

In regards to depression specifically, research has found that the depressogenic attribution style, attributing the cause of negative life events to internal, stable and global factors, accounts

for a significant amount of variance in depression (Alloy, et al., 1999). However, shame-proneness has been shown to account for a significant amount of variance in depression even when controlling for this attribution style (Tangney, Wagner, & Gramzow, 1992).

More recently, Cheung, Gilbert, and Irons (2004) examined the relationship between shame and depression and found that shame was associated with depressive symptoms concurrently. De Rubeis and Hollenstein (2009) conducted a longitudinal study examining the relationship between shame-proneness and depressive symptoms and found that shame-proneness predicted depressive symptoms concurrently and prospectively a year later. Recognizing the relationship between shame-proneness and depressive symptoms is a first step. Understanding mechanisms driving this relationship is the next step that also may lay the groundwork for future novel interventions.

Cognitive models of depression. Cognitive models of depression posit that when faced with life events, how individuals perceive and cognitively respond will predict the development of depressive symptoms. Below I summarize cognitive theories of depression and how they may inform the relationship between shame-proneness and depressive symptoms.

Beck (1967) developed a cognitive model of depression that centered on the presence of negative cognitive schemas. The term "negative cognitive schema" refers to the cognitive processing structure that develops as a result of negative early life experiences and becomes activated to facilitate the evaluation, organization and interpretation of current life events (Abramson & Alloy, 1990; Beck, 1987; Haaga, Dyck, & Ernst, 1991). Negative schemas map onto one of three themes of the negative cognitive triad which includes negative views of the self, the environment, or the future (Wright & Beck, 1983). Ultimately, the negative schemas and the attributed attitudes increase the risk of developing depressive symptoms over time.

While Beck was developing this cognitive model of depression, Seligman was also developing a cognitive model of depression, the learned helplessness theory of depression (1975). Seligman hypothesized that individuals learn a lack of control over time from experiences without control in life events. He described learned helplessness as a failure to respond effectively in the face of uncontrollable life events. This theory was later reformulated (Abramson, Seligman, & Teasdale, 1978) to posit that when individuals believe that an aversive outcome is likely and that they have no control to change this outcome, they feel helpless and become vulnerable to the development of depression. Learned helplessness includes four deficits of lowered response initiation, slower learning that a response has an outcome, increased emotional distress in an uncontrolled event, and lower self-esteem. This learned helplessness could be viewed as situational or global depending on what the individual believes caused the event and how similar the situation is to previous uncontrollable life events (Alloy, Peterson, Abramson, & Seligman, 1984). Making global attributions about life events increases the likelihood of generalizing the events and developing a pattern of learned helplessness.

The hopelessness theory of depression developed by Abramson, Metalsky, and Alloy (1989) hypothesized that depression develops and is maintained by the negative attributions and self-inferences an individual has about negative life events. Focused on the content of cognitions, this theory described three factors that influence the impact negative life events have on depressive symptoms including the inferences individuals make about the cause of the event, the consequences of the event, and the self, which are collectively called negative cognitive style. The hopelessness theory of depression posits that negative cognitive style and the importance an individual assigns to the life event influences the impact a life event will have on depressive outcomes.

Nolen-Hoeksema and Morrow (1991) developed the response styles theory of depression that focused on cognitive processes. This model posits that how individuals respond to life events impacts their emotions and the development and maintenance of depressive symptoms. One response, rumination, is the repetitive focus on negative emotions in response to life events. In contrast, distraction is the response of actively engaging in positive activities or positive thinking that redirects the mind from negative emotions (Nolen-Hoeksema, 1991). Research has found that engagement in rumination maintains and exacerbates depressed mood and increases risk of developing depressive symptoms (Nolen-Hoeksema, 2000; Nolen Hoeksema & Morrow, 1991; 1993; Nolen- Hoeksema, Morrow, & Fredrickson, 1993) and that engagement in distraction has been found to reduce the negative emotions (Nolen-Hoeksema & Morrow, 1993). The response styles theory of depression made an important distinction between cognitive content and cognitive processes in the development of depression and highlighted the importance of considering emotion regulation cognitive strategies in the relationship between life events and depression.

Maladaptive Emotion Regulation Cognitive Strategies May Mediate Between Shame-Proneness and Depressive Symptoms

Cognitive affective theories of depression emphasize that underlying the relationship between affect and depression are emotion regulation cognitive mechanisms. Emotion regulation refers to the mechanisms people use to influence the emotions they have, as well as how the emotions are experienced and expressed (Gross, 1998). In relation to depression, responses to the emotional reactions individuals have when exposed to stressful events influence the relationship between those emotional reactions and the depression outcomes (Gentzler, Kerns, & Keener, 2010; Johnson, McKenzie, & McMurrich, 2008). Therefore, in order to

understand the relationship between shame and the development of depressive symptoms, the logical next step is to examine emotion regulation cognitive responses to negative emotions. While the relationship between shame-proneness and depressive symptoms have been explored, research has only recently begun to explore the emotion regulation cognitive mechanisms driving this relationship.

Brooding May Mediate the Relationship between Shame and Depressive Symptoms. Brooding as previously described is a perseverative focus on negative emotions and thoughts resulting from negative events (Nolen-Hoeksema, 1991). Brooding has been well established as a cognitive emotion regulation strategy predictive of onset and maintenance of depression (Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1991; 1993; Nolen- Hoeksema, et al., 1993). Researchers have also begun to explore brooding and/or the larger construct of rumination in relation to shame. In a young adult sample, Cheung, Gilbert, and Irons (2004) found that shame-proneness was associated with depressive symptoms and this relationship was partially mediated by trait rumination. Candea, Matu, and Szentagotai (2014) found trait rumination was a significant moderator of the relationship between shame-proneness and depressive symptoms, such that 21% to 27% of the variance in depressive symptoms was accounted for by the interaction between shame-proneness and trait rumination.

In addition to understanding to the relationship between shame-proneness and depressive symptoms, it is important to examine the variable responses occurring in response to negative life events at a state level. In a sample of parents surveyed following their family breakup, Orth, Berking, and Burkhardt (2006) found support for state rumination as a mediator in the relationship between state shame and depressive symptoms. Together, these findings suggest that the use of brooding in an attempt to regulate shame emotions may be one of the mechanisms that

explains the impact of shame-proneness on depressive symptoms. However, additional studies that move beyond the cross-sectional design are necessary to strengthen the causal inferences that can be made about these relationships in the young adult population.

Avoidance May Mediate the Relationship between Shame and Depressive Symptoms. Avoidance has been implicated as an emotion regulation strategy associated with a number of maladaptive outcomes including general maladjustment (Ebata & Moos, 1991), binge eating (Heatherton & Baumeister, 1991; McCarthy, 1990; Polivy & Herman, 2002), anxiety disorders (Barlow, Craske, Cemy, Klosko, 1989; Foa & Kosak, 1986; Lissek et al, 2009; Merckelbach, de Jong, Muris, & van Den Hout, 1996; Rachman, 1993) and depressive symptoms (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Blalock & Joiner, 2000; Holahan, Moos, Holahan, Brennan, & Schutte, 2005; Seiffge-Krenke & Klessinger, 2000). Avoidance is an emotion regulation strategy stemming from an unwillingness to face or experience an emotion or event. Using avoidance, an individual attempts to down-regulate distressing emotions, thoughts, memories, and physical sensations (Hayes, Strosahl, & Wilson, 2011). By definition, avoidance represents a natural response to shame emotions as described by Tangney and Wagner (1992). Seiffge and Klessinger (2000) examined avoidance as a coping style in an adolescent sample. They found that avoidant strategies predicted greater depressive symptoms up to two years later. In an adult sample, greater avoidance predicted greater depressive symptoms ten years later (Holahan et al., 2005). In a small, early adolescent sample, De Rubeis and Hollenstein (2009) found support for avoidance as a mediator in the relationship between shame-proneness and depressive symptoms one year later. This study assessed these variables at two time points and therefore, more research is needed. When using avoidance to regulate emotions of shame, the literature suggests that vulnerability to developing depressive symptoms may increase.

Suppression May Mediate the Relationship between Shame and Depressive Symptoms. Wenzlaff and Wegner (2000) define suppression as inhibition of unwanted thoughts. Suppression has been found to lead to greater access to the unwanted thoughts (Wegner & Erber, 1992; Wegner, Schneider, Carter, & White, 1987), greater emotional arousal (Wegner, Broome, & Blumberg, 1997), and hypersensitivity to depressive thoughts and symptoms (Wegner & Zanakos, 1994; Wenzlaff & Wegner, 2000). Prospectively, research has found that suppression predicted depressive symptoms over eleven days later (Borton & Casey, 2006), seven weeks later (Beevers & Meyer, 2004) and over ten weeks later (Wenzlaff & Luxton, 2003) in college samples. Dalgleish and Yiend (2006) examined the effects of intentional suppression in a sample of young adults. Those individuals asked to suppress the memory of a negative event reported stronger levels of depressed mood following the task during the lab visit than participants not asked to suppress their memories. Similarly, Borton, Markowitz, and Dieterich (2005) found that individuals asked to suppress their negative self-referent thoughts experienced more depressive and anxious mood. Regardless of current depressive symptoms, individuals who suppress their negative thoughts are more vulnerable to developing depressed mood, suggesting suppression may represent a maladaptive strategy for regulating emotions around negative events.

Researchers exploring the relationship between shame-proneness and depressive symptoms have found that shame-proneness is also correlated with suppression. Specifically, children and adolescents reporting higher levels of depressive symptoms reported greater expressive suppression and greater shame-proneness than those reporting low levels of depressive symptoms (Hughes, Gullone, & Watson, 2011). The directional relationships between these constructs are important to understanding the development and maintenance of

depressive symptoms. Suppression may serve as a mediator between shame-proneness and depressive symptoms, such that using suppression to regulate emotions of shame will increase the shame and increase vulnerability to depressive symptoms.

Researchers have begun exploring emotion regulation strategies implicated in the development and maintenance of depressive symptoms and have identified three maladaptive strategies, rumination (brooding), avoidance, and suppression. In addition, research on shame is now beginning to explore mechanisms driving the relationship between shame-proneness and depressive symptoms, as well as, state responses to event-specific shame. Aldao, Nolen-Hoeksema, and Schweizer (2010) emphasized that more research is needed on the relationships between emotion regulation strategies and their joint effects. For example, Gross and John (2003) found that suppression was correlated with rumination and more avoidance in close relationships. Also, more research is needed to understand how these cognitive emotion regulation strategies work at the state level when people are responding to life events (Berkman & Lieberman, 2009; Egloff, Schmukle, Burns, & Schwerdtfeger, 2006).

The Current Study

Previous research has firmly established a link between shame-proneness and depressive symptoms. Previous research has also supported the hypothesis that individuals who regulate emotions using brooding, suppression, and avoidance are more vulnerable to developing depressive symptoms. Current theory and research suggests that the relationship between shame-proneness and depression may be mediated by maladaptive emotion regulation strategies that function to reduce, escape, or avoid shame. While initial progress has been made in investigating the explanatory mechanisms driving the effect of shame-proneness on depressive symptoms, we are in need of stronger research designs that will allow for more confident

conclusions about causality. Studies have explored this topic from correlational self-report data but to this point, less has been done to explore this relationship longitudinally and through lab-based inductions. In the current study I investigated the relationship between shame-proneness and depressive symptoms, by examining possible mediators of this relationship (brooding, suppression, and avoidance), in both prospective and lab-based inductions.

Specifically, I planned to examine the following hypotheses in two parts in a short-term prospective study of young adults through a baseline questionnaire, lab visit, and follow up questionnaires administered across about two weeks. First, I hypothesized that the relationship between baseline trait shame-proneness and depressive symptoms would be concurrently mediated by the trait emotion regulation strategies of brooding, avoidance, and suppression, controlling for trait guilt-proneness (see Figure 1). The first hypothesis was examined using baseline measures of shame-proneness and guilt-proneness, trait emotion regulation strategies, and concurrent depressive symptoms. Second, I hypothesized that when controlling for guilt-proneness and baseline depressive symptoms, brooding, avoidance, and suppression would each simultaneously mediate the relationship between trait shame-proneness and depressive symptoms prospectively (see Figure 1). The second hypothesis was examined using a baseline measure of trait shame-proneness and follow up measures of daily emotion regulation strategies and depressive symptoms assessed prospectively across a 2-week period.

In part 2 of the study, I hypothesized that high trait shame-proneness would predict greater use of brooding, avoidance and suppression strategies through individuals' experienced state shame (see Figure 2). The third hypothesis is that these relationships would hold when shame was induced in the lab task. Specifically, participants were introduced to a brief shame-inducing manipulation in which they are asked to write for 5 minutes about a previous shameful

experience (De Hooge, Breugelmans, & Zeelenberg, 2008; Ketelaar & Au, 2003). The participants then completed short questionnaires measuring state shame and current emotion regulation strategies.

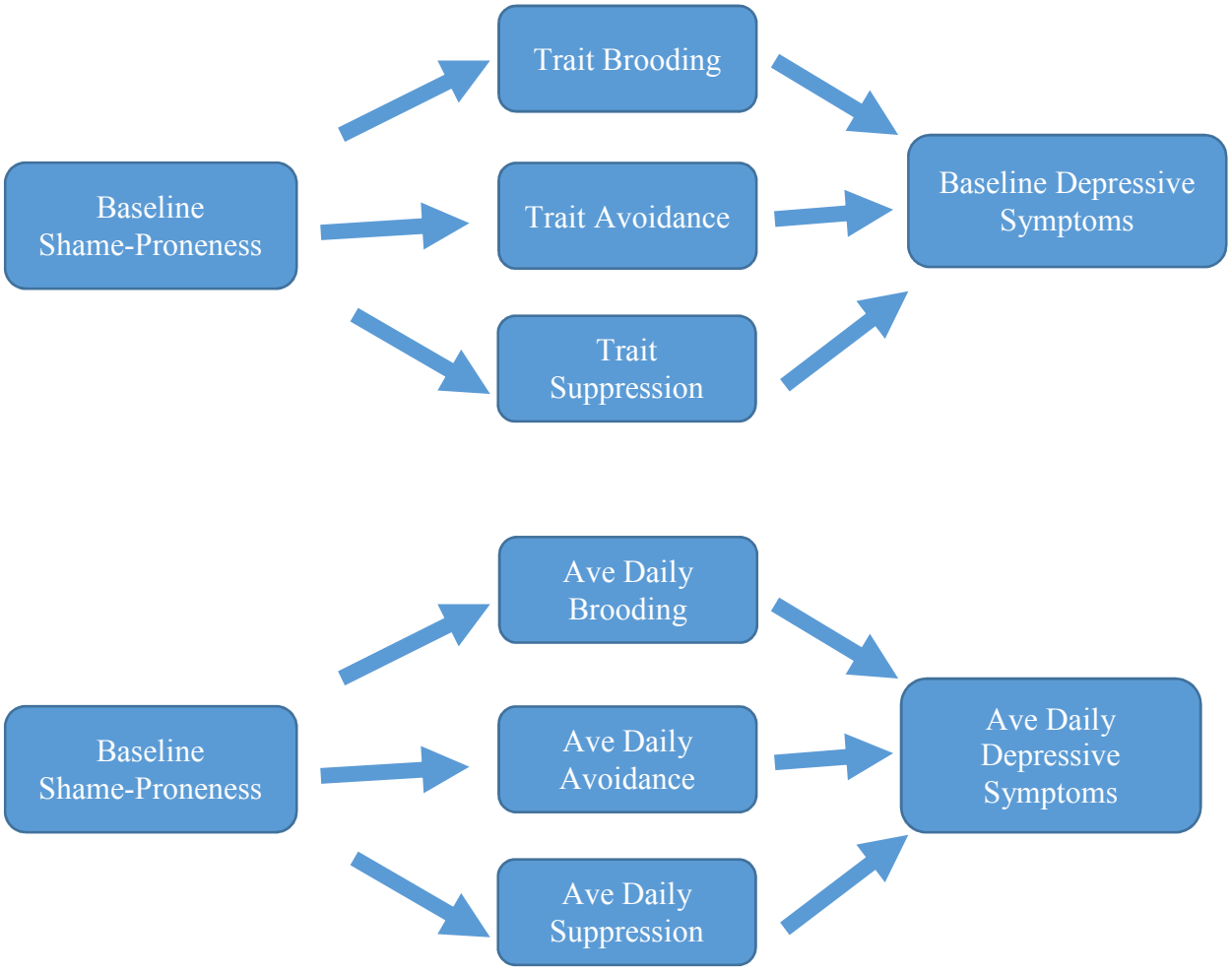


Figure 1. Mediation model of shame-proneness predicting depressive symptoms and mediated through emotion regulation strategies of brooding, avoidance, and suppression responses to shame for concurrent and prospective analyses.

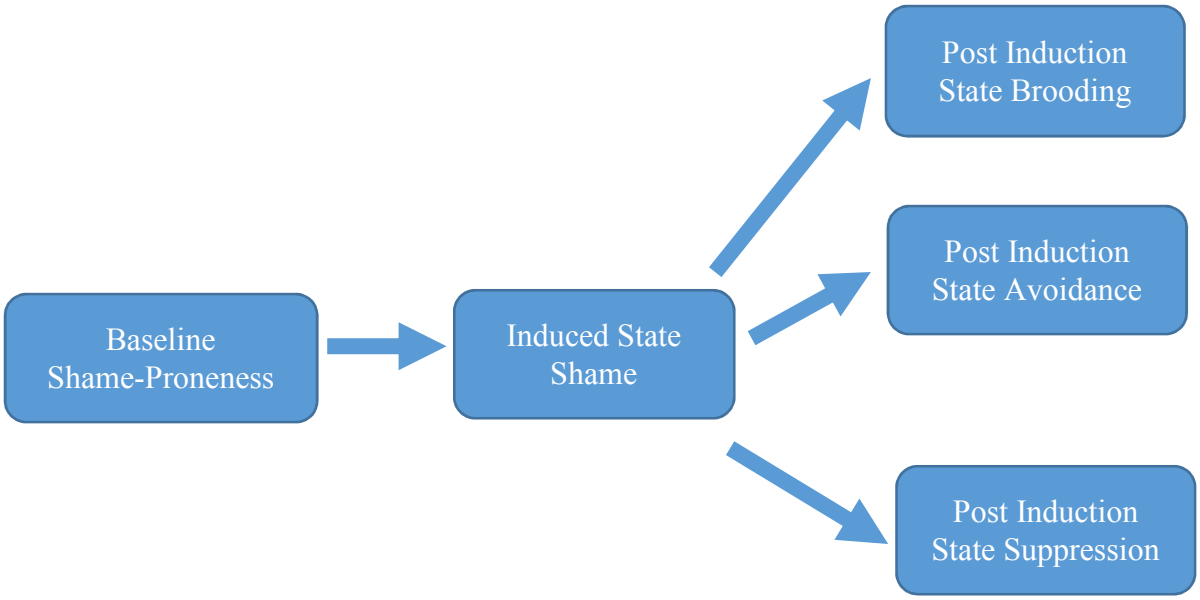


Figure 2. Mediated model of shame-proneness predicting cognitive responses of brooding, avoidance, and suppression through state shame.

CHAPTER II

Method

Sample and Participant Selection

Participants. I recruited undergraduate students from a Pacific Northwestern university. In order to determine my sample size, I referred to Fritz and MacKinnon (2007) and considered the alpha and beta pathway effect sizes from previous studies. A prior study examining shame-proneness predicting depressive symptoms a year later showed a significant effect size of .29 (De Rubeis & Hollenstein, 2009). In addition, that study found an effect size of .37 for shame-proneness predicting avoidance and an effect size of .38 for avoidance predicting depressive symptoms. Studies examining the effect of brooding on depressive symptoms have demonstrated an effect size of .23 to .26 (Burwell and Shirk, 2007; Mezulis, Simonson, McCauley, & Vander Stoep, 2011). Finally, examining thought suppression as a predictor of depressive symptoms, research has found effect sizes of .27 to .29 (Beevers & Meyer, 2004). With .8 power to obtain moderate to high effect sizes on the alpha and beta paths, Fritz and MacKinnon (2007) recommend a sample size between 71 and 148. Given all this previous research and the full model, I elected to aim for a conservative 100 participants.

Participant (N = 137) demographics per self-report are presented in Table 1. Participants ranged in age from 18 to 29 years old (M = 19.29, SD = 1.56). In addition to the racial categories noted in Table 1, participants identified additional races of African and Middle Eastern. For the 2015 Autumn Quarter, the most recent statistics available, Seattle Pacific University (SPU) reported a total population of 3,202 undergraduate students. SPU reports that the average age of these students is 21, females represent 69% of the undergraduate population, and 36% of the undergraduate students fall under the broad category of "ethnic minority." As such, the recruited

sample has higher female representation than the undergraduate population. The sample did have a comparable age range and racial and ethnic diversity of the whole undergraduate population sampled.

A total of 137 participants completed the baseline questionnaire for the present study. Of these, 115 participants completed the lab visit and 113 completed the lab visit and at least one daily questionnaire. I used the sample of 137 participants for concurrent analyses, the sample of 113 participants for prospective analyses (H2), and the sample of 115 participants for prospective analyses (H3). Utilizing simple t-tests, I compared the group of 115 participants who completed the baseline and lab visit, and the 22 participants who completed the baseline measures only. Results showed no significant differences in demographics and all baseline measures (p >.10).

Table 1
Participant Demographics

		N	%
Biological Sex			
	Male	23	16.8
	Female	114	83.2
Gender			
	Male	25	18.2
	Female	112	81.8
Age			
	18	59	43.1
	19	42	30.7
	20	17	12.4
	21	8	5.8
	22	3	2.2
	23	1	.7
	24	1	.7
	27	2	1.5
	29	1	.7
	Missing	3	2.2
Ethnicity			

Hispanic/Latino	19	13.9
Not Hispanic/Latino	116	84.6
Did not report	2	1.5
Race		
Caucasian	102	74.5
African American	5	3.6
Asian American	19	13.9
Native American	1	.7
Pacific Islander	3	2.2
Other	2	1.5
Mixed	5	3.6
Year In College		
Freshman	89	65.0
Sophomore	21	15.3
Junior	17	12.4
Senior	6	4.4
Other	4	2.9

Recruitment. The SPU Institutional Review Board approved all procedures of recruitment. Participants were recruited from Introductory Psychology courses on campus. Students interested in participating in the current study signed up online. Students were presented a brief description of the study and potential risks and benefits.

Procedure

The current study involved a baseline questionnaire, a lab visit, and 6 daily follow-up questionnaires administered across a 14-day follow-up period. All questionnaires were completed using Qualtrics, an electronically administered online survey tool. Time 1 (T1) included self-report on demographics and measures of baseline depressive symptoms, trait shame-proneness, trait guilt-proneness, trait brooding, trait avoidance, and trait suppression (see Figure 3). T2 involved a lab visit in which participants completed self-report of state shame, a brief shame induction, followed by self-report of state shame, brooding, avoidance, and

suppression, a few days to a few weeks following T1. In the shame-induction participants were given five minutes to write about a shameful experience from their life with the follow prompt: "Shame has been described as the feeling experienced when you notice a shortcoming in yourself. Describe a personal experience in which you felt very ashamed. Continue writing until I ask you to stop."

T3 included daily questionnaires participants completed that assess state shame, brooding, avoidance and suppression in response to actual daily stressors, and depressive symptoms beginning a few days to a week following the lab visit. All T3 daily questionnaires were identical to each other. All data was transferred from the online survey tool to a secure online database by members of the research team to be accessible to the faculty sponsor and myself. The data was accessed by members of the research team on a secure password-protected computer.

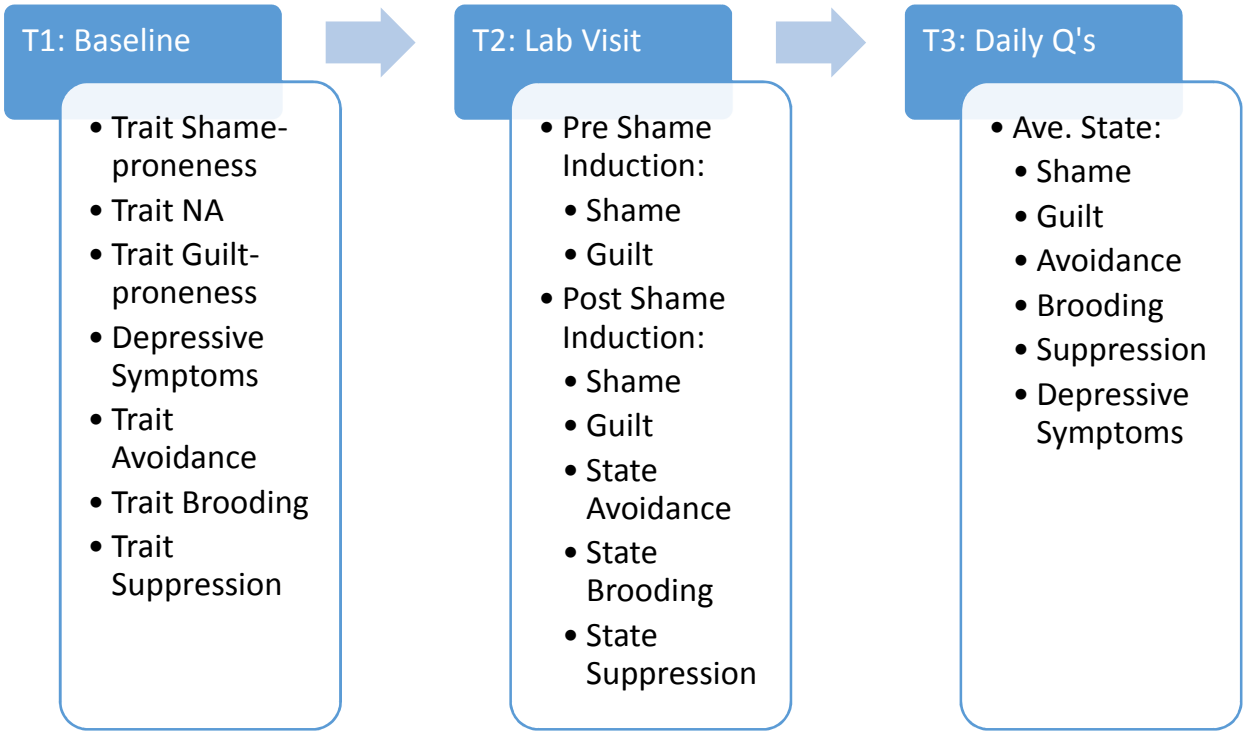


Figure 3. Variables assessed across 3-part study design

Measures

Shame.

Shame-proneness. In line with Lewis's theory of shame (1971) distinguishing the focus of the experience (i.e., the self or the behavior) we measured and differentiated between shame and guilt. At baseline, participants completed a self-report measure of shame-proneness called the Test of Self-Conscious Affect-3 (TOSCA-3; Tangney, Dearing, Wagner, & Gramzow, 2000). This measure is a scenario-based assessment (10 negative and 5 positive scenarios) that includes a set of responses that represent different affective tendencies (i.e., shame-proneness and guilt-proneness). There are fifteen scenarios such as "You are driving down the road and you hit a small animal…" with responses that you rate on a 5-point Likert scale from 1 (*not likely*) to 5 (*very likely*) including responses such as, "You'd feel bad you hadn't been more alert driving down the road." (Guilt response) or "You would think, 'I'm terrible.' (Shame response)." Scale scores are the sum of item ratings and range from 0 to 15, with higher scores representing greater proneness to the scaled emotion (i.e., shame, guilt or pride). The original TOSCA measure (Tangney, 1990; Tangney et al., 1989) has been examined and used often in research. Less research has been done with TOSCA-3 but this 3rd version includes 14 of the original items. Therefore, I included psychometric details of the TOSCA as it is likely similar to the TOSCA-3. Internal consistency reliability alpha coefficients for the shame-proneness scale in the TOSCA have been reported ranging from .74 to .77 in general population samples (Tangney & Dearing 2002; Tangney et al., 1996). For the guilt-proneness scale in the TOSCA, the reported internal consistency reliability alpha coefficients range from .69 to .78 in the general population (Tangney & Dearing, 2002; Tangney et al., 1996). The subscales of shame-proneness and guilt-proneness showed significant correlation (r = .45 - .56; Averill, Diefenbach, Stanley,

Breckenridge, & Lusby, 2002; Tangney et al., 1992). When shared variance was partialled out, studies found that shame-proneness was associated with depression, maladaptive anger responses, bulimia, anxiety, paranoia, phobia, psychosis, hostility, obsessions, and somatization (Averill, Diefenbach, Stanley, Breckenridge, & Lusby, 2002; Sanftner, Barlow, Marschall, & Tangney, 1995; Tangney, Burggraf, & Wagner, 1995; Tangney et al., 1996). Studies that did not partial out shared variance with the guilt-proneness subscale found the subscale shame-proneness correlated with low self-esteem, self-consciousness, depression, and proneness to anger (Tangney, Wagner, & Gramzow, 1992; Tangney et al., 1995). In addition, the shame-proneness subscale showed convergent validity with another shame measure, the Personal Feelings Questionnaire-2 (PFQ-2), $r = .27$. In our study, the alpha coefficient for the shame-proneness scale was .81 and guilt-proneness scale was .72.

State shame. Participants reported on their current emotions of shame and guilt at three separate parts of the study: 1) prior to the shame induction in the lab visit (T2), 2) following the shame induction in the lab visit (T2), and 3) each daily questionnaire in reference to their worst event of the day (T3). Developed as a manipulation check for a shame induction, the State Shame and Guilt Scale (SSGS; Marschall, Sanftner, & Tangney, 1994) was used to measure state shame for each of these three parts. The SSGS is originally a 15-item self-report that includes three scales: shame, guilt, and pride and for this study, participants will report on shame (4 items) and guilt (4 items). At the lab visit, on a 5-point Likert scale from 1 (*not feeling this way at all*) to 5 (*feeling this way very strongly*), participants were asked to report their experience of items such as, "I want to sink into the floor and disappear." Scale scores are the sum of item ratings and range from 4 to 20, with higher scores representing greater presence of the scale emotion (i.e., shame, guilt or pride). In the daily questionnaires, participants used a 4-point

Likert scale from 1 (*I didn't do this at all*) to 4 (*I did this a lot*), with scale scores summed and ranging from 4 to 16. Internal consistency reliability alpha coefficients for the SSGS have been reported ranging from .80 - .89 for the shame subscale and .82 for the guilt subscale (Marschall, Sanftner, & Tangney, 1994; Tilghman-Osborne, Cole, Felton, & Ciesla, 2008). Test-retest reliability tested at 5 months ranged from .53 (guilt) to .57 (shame; Tilghman-Osbourne, Cole, Felton, & Ciesla, 2008). The SSGS showed convergent validity with other measures shame and guilt (Personal Feelings Questionnaire-2, r = .52- .59; and Body and Appearance-related Self-Conscious Emotions Scale, r = .43- .50. In our study, the alpha coefficient for the shame scale was .59 pre-induction and .84 post-induction at lab visit (T2) and on average .84 across the daily questionnaires. The alpha coefficient for the guilt scale was .79 pre-induction and .86 post-induction at lab visit (T2).

Depressive Symptoms. Participants' depressive symptoms were reported at baseline using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), which is a 20 item measure of depressive symptoms in the general population with an emphasis on affective components. The CES-D Short Form (CES-D SF; Martens et al., 2006), which is a nine item measure, was administered during the lab visit and in the daily questionnaires following the lab visit. In each questionnaire participants were asked to rate how they felt and behaved during the past week. Items were selected from existing scales, including the Beck Depression Inventory and Zung Self-Rating Depression Scale. Rating responses range from 0 (*rarely or none of the time*) to 3 (*most or all of the time*) for items such as, "I felt sad." Scores are the sum of item ratings with the reverse score of four items and range from 0 to 60, with higher scores representing greater depressive symptoms and scores of 16 or more indicating a significant level

of depression. I calculated a total score ranging from 0 to 60 in the CES-D and 0 to 27 in the CES-D SF. Higher scores represent greater depressive symptoms.

Internal consistency reliability alpha coefficients for the CES-D have been reported ranging from .85 in general population samples to .91 in patient samples (Himmelfarb & Murrell, 1983; Radloff, 1977; Roberts, 1980). Test-retest reliability tested from two weeks to 12 months ranged from .45 to .70 (Radloff, 1977). Radloff (1977) identified four factors in the CES-D, including depressed affect, positive affect, somatic symptoms, and interpersonal problems and these four factors were identified in multiple other studies (Joseph & Lewis, 1995; Roberts, 1980; Zich, Attkisson, & Greenfield, 1990). It is not recommended to use these findings to form subscales. In order to reduce response bias and also assess positive affect, 4 items have been worded in a positive direction. In addition, the CES-D showed convergent validity with other depression measures (Lubin scale, r = .37-.70; Bradburn Negative Affect scale, r = .55-.63; and Hamilton Rating Scale for Depression, r = .49-.85) and discriminant validity with Bradburn Positive Affect scale, r = -.55 to -.21 and with Beck Anxiety Inventory, r = .68. In our study, the alpha coefficient was .89 at baseline and between .76 and .80 across daily questionnaires.

Brooding. Participants reported their pattern of brooding using the brooding 5-item subscale of the Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991). This self-report inventory asks participants to rate items on a 4-point Likert scale ranging from 1 (*almost never*) to 4 (*almost always*). An example of a brooding item is: "I think 'What am I doing to deserve this?'" Mean scored were calculated for the brooding scale range from 1 to 4 with higher scores indicating greater tendency to brood about negative mood or events. As noted

above, the brooding subscale demonstrates good internal consistency (α = .89; Mezulis, Simonson, McCauley, & Vander Stoep, 2011). In our study, the alpha coefficient was .78.

The Event-Anchored Ruminative Response Scale (EA-RRS; Nolen-Hoeksema & Morrow, 1991) includes the 5-item brooding subscale from the RRS. The EA-RRS measures perseveration on negative moods and negative events. Participants were asked to rate items according to how frequently they engaged in the brooding response following the shame-induction during the lab visit ranging from 1 (*not at all*) to 4 (*a lot*) and following the worst event of the previous few days during the follow up questionnaires, ranging from 0 (*I didn't do this at all)* to 3 (*I did this a lot*). In order to analyze the use of brooding across six daily questionnaires, I calculated the average mean score of the daily questionnaires completed. In our study, the brooding alpha coefficient for the shame-induction was .79 and for the worst event, the average brooding alpha coefficient was .81.

Avoidance. Participants reported on their pattern of experiential avoidance at baseline using the Brief Experiential Avoidance Questionnaire (BEAQ; Gamez, Chmielewski, Kotov, Ruggero, Suzuki, & Watson, 2014), which is a 15-item measure in which participants were asked to rate how much they agree or disagree with the items. The items were previously selected from an existing measure, the Multidimensional Experiential Avoidance Questionnaire (MEAQ; Gamez, Chmielewski, Kotov, Ruggero, & Watson, 2011) to capture the six dimensions of the original scale including behavioral avoidance, distress aversion, procrastination, distraction/suppression, repression/denial, distress endurance. Rating responses range from 1 (*strongly disagree*) to 6 (*strongly agree*) for items such as, "I work hard to keep out upsetting feelings." Scores are the sum of item ratings, including 1 reverse coded item. Higher scores represent greater avoidance. I calculated a total score ranging from 15 to 90 in the BEAQ.

Internal consistency reliability alpha coefficients for the BEAQ have been reported ranging from .80 - .86 in both university and general population samples (Gamez et al., 2014). The BEAQ showed convergent validity with other avoidance measures (Acceptance and Action Questionnaire, r = .73; Acceptance and Action Questionnaire, second version, .57-.65; Cognitive –Behavioral Avoidance Scale, r = .57-.59), suppression measures (White Bear Suppression Inventory, r = .56), negative emotionality measures (Positive and Negative Affect Schedule, r = .52; Big Five Inventory, r = .51) and psychopathology measures (Inventory of Depression and Anxiety Symptoms, r = .50-.51; Fear Questionnaire, r = .53). The BEAQ showed discriminant validity with Positive and Negative Affect Schedule, r = -.52 and with Scales of Psychological Well Being, r = -.61. In our study, the alpha coefficient was .83.

The Event-Anchored Brief Experiential Questionnaire (EA-BEAQ; Gamez et al., 2014) includes the five highest loading items onto the original BEAQ scale. Participants were asked to rate items according to how frequently they engaged in the avoidance response following the shame-induction during the lab visit and following the worst event of the previous few days during the follow up questionnaires. During the lab visit, participants rated each item on a 4-point Likert scale ranging from 1 (*not at all*) to 4 (*a lot*). Scores are the sum of item ratings with higher scores representing greater avoidance. I calculated a total score ranging from 5 to 20 in the lab visit. During the follow up questionnaires, participants rated each item on a 4-point Likert scale ranging from 0 (*I didn't do this at all*) to 3 (*I did this a lot*) and therefore, resulted in a total score ranging from 0 to 15 in the follow up questionnaires in response to the worst event of the past few days. To analyze the use of brooding across six daily questionnaires, I calculated the average score of the follow up questionnaires completed. In our study, the avoidance alpha

coefficient for the shame-induction was .64 and for the worst event, the average avoidance alpha coefficient was .83.

Suppression. Participants reported on their trait cognitive suppression using the White Bear Suppression Inventory (WBSI; Wegner & Zanakos, 1994). For this study, I selected the six items that loaded highest onto the original 15-item measure to capture chronic suppression of thoughts for the purposes of inhibiting negative thoughts and feelings in a shorter time frame. Participants were asked to rate the extent to which they agree with each item (for example, "I often do things to distract myself from my thoughts.") using a 5-point Likert scale ranging from 1 (*strongly disagree*) to 5 (*strong agree*). Responses were summed to yield a total score with higher scores reflecting greater suppression. The WBSI demonstrates internal consistency ranging from .87 to .89 in undergraduate populations and test-retest reliability tested from one week to three months that ranges from .69 to .92 (Muris, Merckelbach, & Horselenberg, 1996; Wegner & Zanakos, 1994). The WBSI showed convergent validity with the Beck Depression Inventory (BDI, r = .44-.54), anxiety measures (Repression-Sensitization Scale, r = .58; State-Trait Anxiety Inventory, r = .53-.57; Anxiety Sensitivity Inventory, r = .49; Student Worry Scale, r = .38) and the Maudsley Obsessive-Compulsive Inventory (MOCI, r = .35 - .40; Muris, Merckelbach, & Horselenberg, 1996; Wegner & Zanakos, 1994). In addition, the WBSI showed convergent validity with other thought control strategies including distraction (r = .21), worry (r = .15), punishment (r = .33), and re-appraisal (r = .15) as measured by the Thought Control Questionnaire (TCQ; Wells & Davies, 1994) and discriminant validity with social control, r = -.17 (Muris et al., 1996). In our study, the alpha coefficient was .84.

The Event-Anchored White Bear Suppression Inventory (EA-WBSI; Wegner & Zanakos, 1994) is adapted for use in this study and includes the six highest loading items of the original

scale. Participants were asked to rate items according to how much they agree with the items following the shame-induction during the lab visit and following the worst event of the previous few days during the follow up questionnaires. During the lab visit, participants rated each item on a 4-point Likert scale ranging from 1 (*not at all*) to 4 (*a lot*). Scores are the sum of item ratings with higher scores representing greater suppression. I calculated a total score ranging from 6 to 24 in the lab visit. During the follow up questionnaires, participants rated each item on a 4-point Likert scale ranging from 0 (*I didn't do this at all*) to 3 (*I did this a lot*) and will, therefore, result in a total score ranging from 0 to 18 in the follow up questionnaires in response to the worst event of the past few days. To analyze the use of suppression across six daily questionnaires, I calculated the average score of the daily questionnaires completed. In our study, the suppression alpha coefficient for the shame-induction was .92 and for the worst event, the average suppression alpha coefficient was .95.

Data Analytic Plan

Based on the study design, I tested my hypotheses both cross-sectionally and prospectively. Data analyses were conducted with SPSS 24.0 and Amos 24 statistical software. For my first hypothesis, I used path analysis in Amos 24 to evaluate this multi-mediation model cross-sectionally, considering the association of shame-proneness and guilt-proneness to depressive symptoms at baseline as mediated through trait brooding, avoidance and suppression. Bias-corrected bootstrapping was used to estimate the indirect effects (see Figure 4). Second, using path analysis I evaluated the multiple-mediator model prospectively considering the relationship between trait shame-proneness at baseline and depressive symptoms in the final daily questionnaire as mediated jointly by state brooding, avoidance, and suppression measured during the daily questionnaires.

Given the associations between depressive symptoms, trait NA, guilt-proneness, and shame-proneness and to account for other possible negative emotions beyond shame, I controlled for baseline depressive symptoms, trait NA, and guilt-proneness in prospective analyses (see Figure 5).

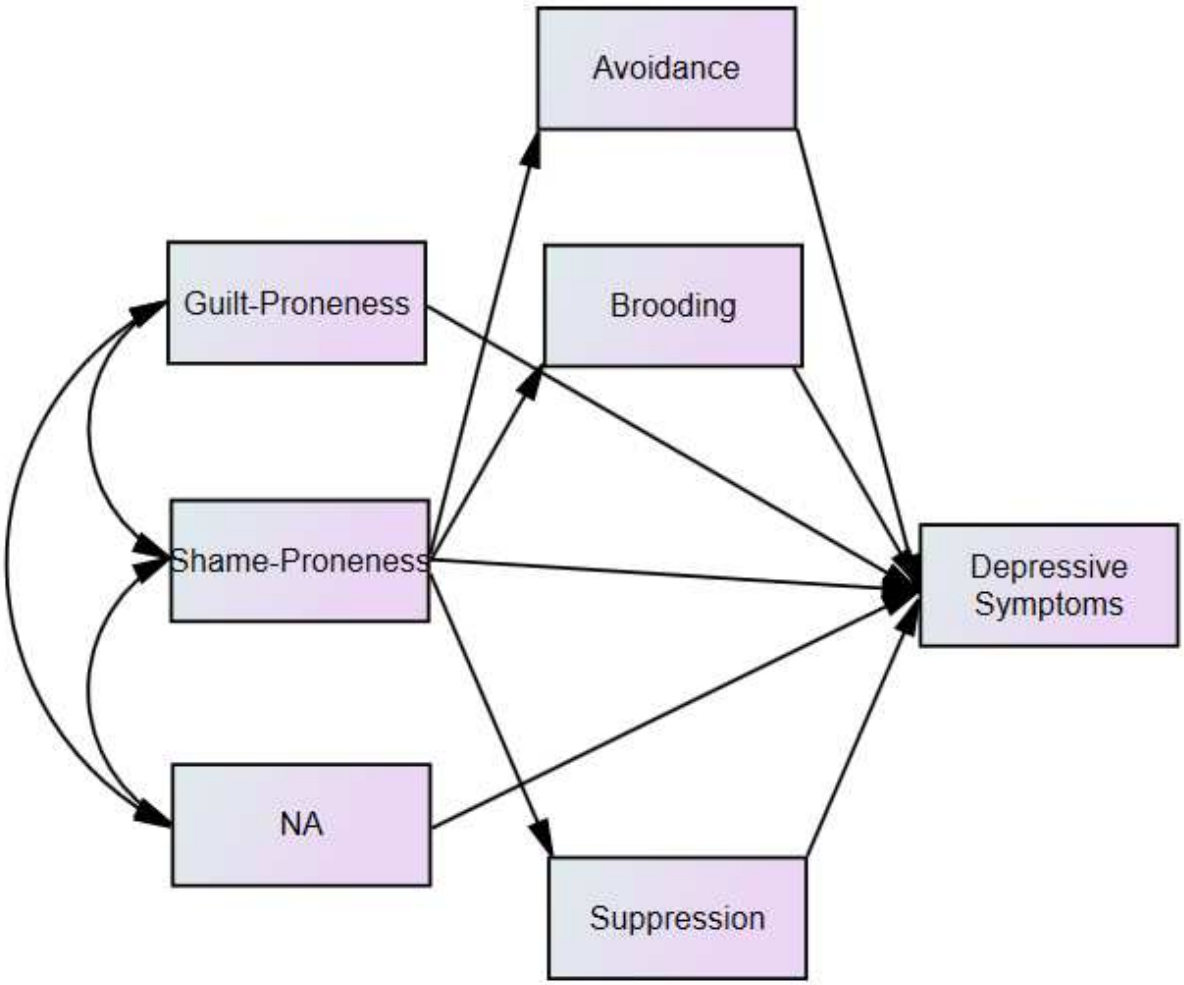


Figure 4. Hypothesis 1 Multimediation Model

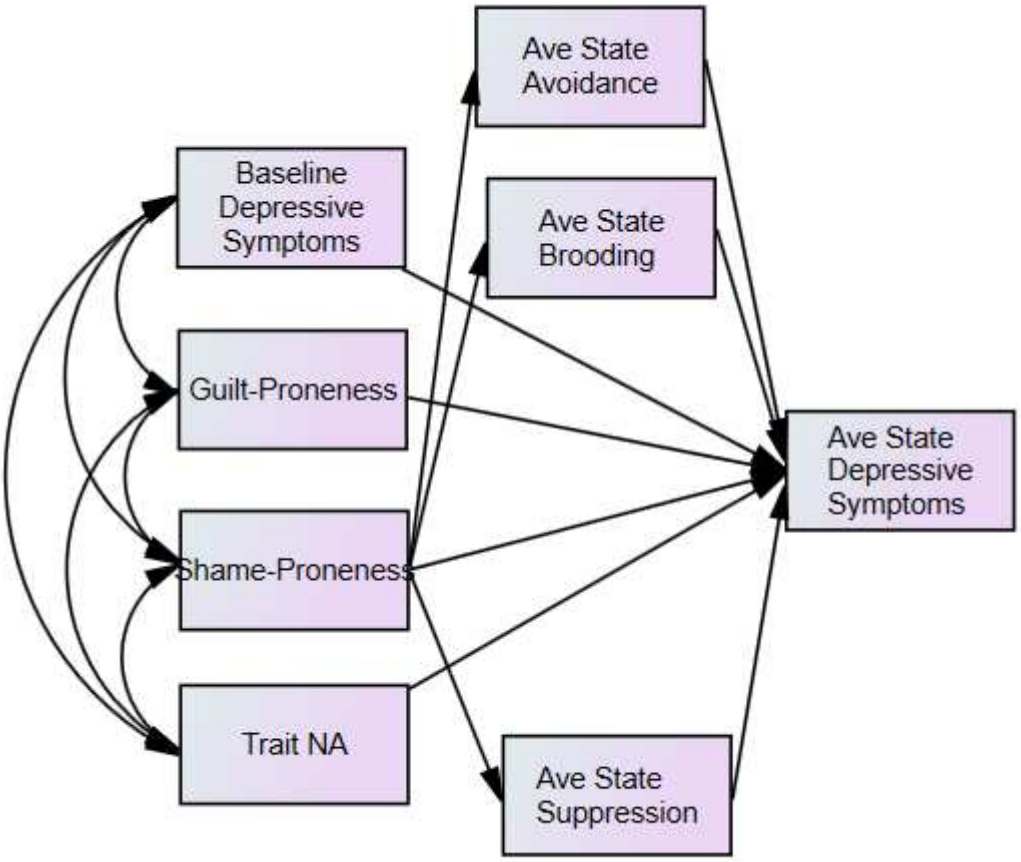


Figure 5. Hypothesis 2 Multimediation Model

To assess my final hypothesis, I first conducted a shame induction manipulation check using a paired-sample t-test to examine whether self-reported state shame measured prior to the shame induction differs significantly from state shame reported immediately following the shame induction. Second, I used hierarchical linear regression to examine the main effect of trait shame-proneness on the prediction of state response of brooding, avoidance and suppression and the indirect effects of trait shame-proneness on the prediction of state brooding, avoidance, and suppression through the state shame reactivity. Baseline depressive symptoms, guilt-proneness, and sex were entered in Step 1 to isolate the unique predictive effects of trait shame-proneness on the use of brooding, avoidance and suppression in the moment. Trait shame-

proneness was entered in Step 2, followed by state shame following induction in step 3. To determine significant mediation, I used Hayes PROCESS macro for testing indirect effects and bootstrapping 95% confidence intervals provided in the output (Hayes & Scharkow, 2013).

CHAPTER III

Results

Preliminary Analysis

Prior to testing hypotheses, I inspected the data for missing data, normality of distribution, outliers, and descriptive statistics described below.

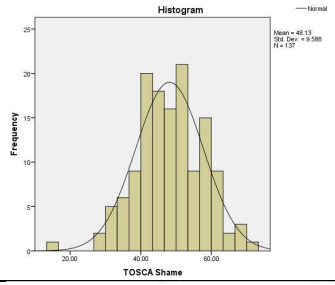
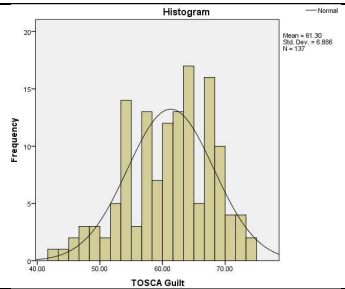
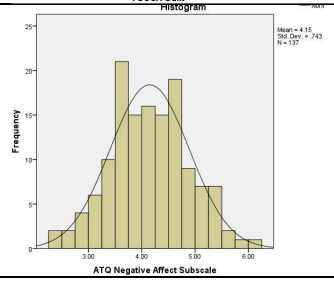
Data Preparation. No data imputation was used at the raw data level. Instead, scale scores were calculated when individuals completed 80% of the items in the measure. Using these guidelines, 137 participants had complete data on all baseline variables. In addition, 115 of 137 participants had complete data in the lab visit and 113 of 137 participants had complete data on daily questionnaire variables for prospective analyses.

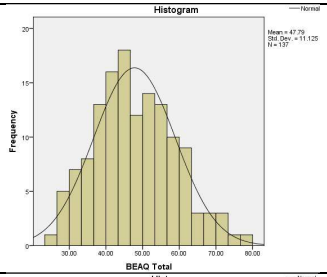
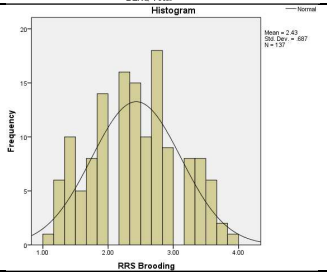
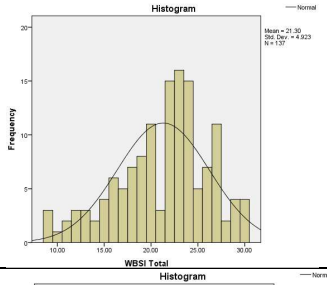
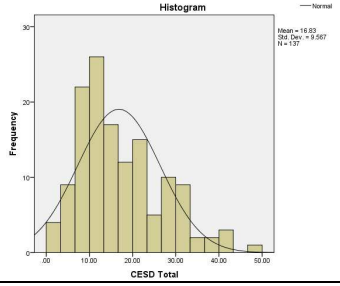
Normality and outlier analysis. Normality was assessed graphically through histograms (see Table 2), normal curves, P-P plots, Q-Q plots and box plots. It was assessed numerically through skewness and kurtosis output and computed standardized z-scores. Normality was also assessed with significance testing, specifically, the Shapiro-Wilk test (S-W test; Field 2005). Scores on measures of shame-proneness, trait negative affect, trait avoidance, and trait brooding did not significantly differ from the normal distribution. The distribution of all other scores used for analyses were significantly different than normal (see Table 2). I examined variables that showed skewness (Table 2). Positive skewness values indicate scores lean to the left of the distribution and negative skewness values indicate the scores lean to the right. Baseline and daily average variables with significant skewness were transformed using square root transformation and lab visit variables were transformed using log10 transformation. While most variables were adequately transformed, a few of the lab visit variables were still skewed, including pre-shame induction state shame and state guilt, post-shame induction state shame,

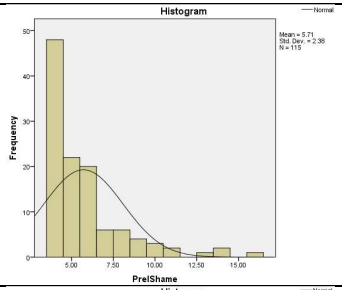
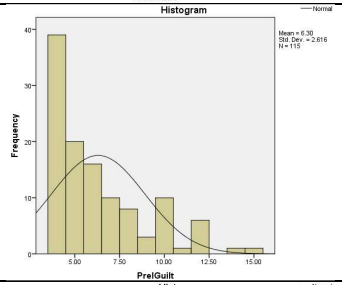
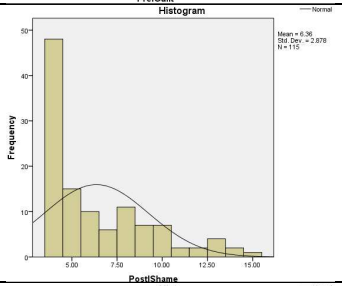
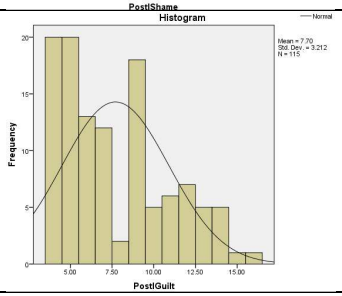
state guilt, state brooding, and state avoidance. Transformations will be reflected in the correlation tables to follow (Tables 3-5). I examined variables for problems in kurtosis. Positive kurtosis values indicate more weight in the tails while negative kurtosis values indicate less weight in the tails compared to what would be expected with normal distribution (Westfall, 2014). Variables that showed kurtotic deviation from normal distribution were inspected. There was no sufficient evidence to support deleting any problematic outliers, and visually most variables showed sufficient tails. Those variables that did not present with sufficient tails, including pre-shame induction state shame, post-shame induction state suppression, and average state avoidance, were addressed by the transformations noted above. Pre-shame induction state shame and post-shame induction state suppression remained slightly kurtotic following the transformations.

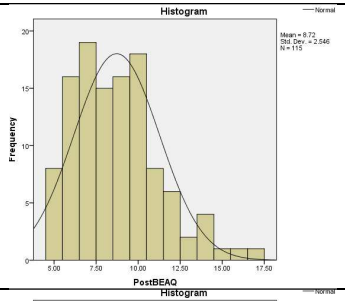
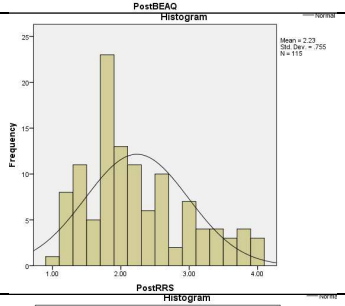
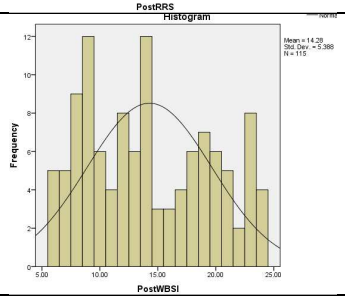
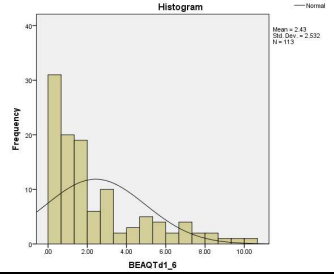
Table 2

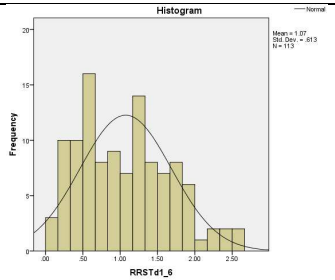
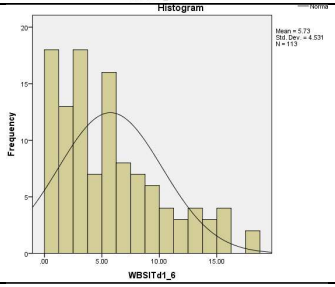
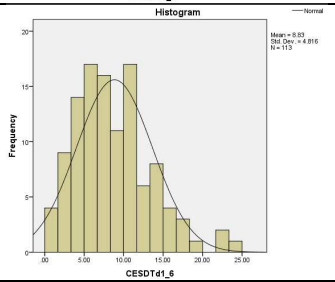
Assessing Univariate Normality of Continuous Variables

		Shapiro-Wilk Test of Normality			Kurtosis		Skewness	
Variables	Histogram	*R*	*Df*	*P*	kurtosis	*z* kurt	skewness	*z* skew
Baseline								
Shame-Proneness		.994	137	.809	.225	.547	-.170	-.821
Guilt-Proneness		.979	137	.033	-.362	-.881	-.407	-1.966*
Trait NA		.994	137	.880	-.152	-.370	.063	.304

Trait Avoidance		.988	137	.294	-.340	-.827	.261	1.261
Trait Brooding		.979	137	.031	-.691	1.681	.011	.053
Trait Suppression		.966	137	.002	-.170	-.414	-.520	-2.512*
Depressive Symptoms		.261	137	.000	.261	.635	.836	4.039***
Shame Induction		*R*	*Df*	*P*	kurtosis	*z* kurt	skewness	*z* skew

Pre Shame		.731	115	.000	4.732	10.586***	2.069	9.155***
Pre Guilt		.828	115	.000	.723	1.617	1.201	5.314***
Post Shame		.806	115	.000	.469	1.049	1.170	5.177***
Post Guilt		.906	115	.000	-.684	1.530	.630	2.788**

Post Avoidance		.941	115	.000	.521	1.166	.785	3.473***
Post Brooding		.932	115	.000	-.357	-.799	.683	3.022**
Post Suppression		.940	115	.000	-1.169	-2.615**	.241	1.066
Daily Questionnaires		*R*	*Df*	*P*	kurtosis	*z* kurt	skewness	*z* skew
Ave State Avoidance		.842	113	.000	1.028	2.279*	1.319	5.811***

Ave State Brooding		.965	113	.005	-.495	1.098	.468	2.062*
Ave State Suppression		.921	113	.000	-.048	.106	.849	3.740***
Ave State Depressive Symptoms		.956	113	.001	.778	1.725	.828	3.648***

Note. R is the Shapiro-Wilk Test Statistic. To facilitate interpretation, z values for kurtosis and skewness are calculated by dividing by their respective standard error. An absolute value greater than 1.96 is significant $p < .05$, above 2.58 is significant at $p < .01$, and above 3.29 is significance at $p < .001$. * denotes $p < .05$, ** $p < .01$, *** $p < .001$.

Descriptive. Bivariate correlations, means, and standard deviations for baseline study variables are presented in Table 3, descriptive data for the lab visit variables are presented in Table 4, and descriptive data for the average daily measures in the daily questionnaires are presented in Table 5.

Baseline. Depressive symptoms at baseline were positively correlated with trait negative affect, shame-proneness, trait avoidance, trait brooding, and trait suppression (see Table 3). In addition, shame-proneness was positively correlated with trait negative affect, guilt-proneness, trait avoidance, and trait brooding. In addition, trait avoidance, trait brooding, and trait suppression were all positively correlated. As expected, guilt-proneness was not correlated with depressive symptoms, trait avoidance, trait brooding, or trait suppression. Contrary to hypotheses, shame-proneness was not correlated with trait suppression.

Table 3

Bivariate Correlations and Descriptives among Variables at Baseline

N	*Variable*	1	2	3	4	5	6	7	*M (SD)*
137	1. Bio Sex (0 = Male, 1 = female)								.83 (.38)
137	2. Trait NA	.338**							4.15 (.74)
137	3. Trait Shame	.209*	.482**						48.13 (9.59)
137	4. Trait Guilt	.175*	.246**	.599**					3.02 (.94)
137	5. Trait Avoidance	.125	.278**	.226**	-.070				47.79 (11.12)
137	6. Trait Brooding	.073	.361**	.336**	.143	.263**			2.43 (.69)
137	7. Trait Suppression	-.051	.320**	.152	.080	.313**	.464**		1.69 (.83)
137	8. Baseline CES-D	.124	.445**	.378**	.129	.312**	.448**	.407**	3.93 (1.18)

Note. CES-D = Center for Epidemiological Studies Depression Scale; NA = Negative Affect; *$p < .05$, ** $p < .01$.

Lab Visit. The pre induction shame and post induction shame correlated positively with state brooding and suppression (see Table 4). Contrary to hypotheses, pre induction shame did not correlate significantly with state avoidance. In addition, state avoidance, brooding and suppression were positively correlated.

Table 4

Bivariate Correlations and Descriptives among Variables at Lab Visit

N	*Variable*	1	2	3	4	5	6	7	*M (SD)*
137	1. Bio Sex								.83 (.38)
115	2. Pre Induction Shame	.085							.73 (.15)
115	3. Pre Induction Guilt	.143	.489**						.77 (.16)
115	4. Post Induction Shame	.191*	.645**	.494**					.77 (.18)
115	5. Post Induction Guilt	.244**	.316**	.574**	.640**				.85 (.18)
115	6. Post Induction Avoidance	.074	.177	.274**	.247**	.284**			.92 (.12)
115	7. Post Induction Brooding	.159	.409**	.491**	.559**	.577**	.446**		.33 (.14)
115	8. Post Induction Suppression	.123	.321**	.549**	.507**	.605**	.512**	.624**	1.12 (.17)

Note. *$p < .05$, **$p < .01$.

Weekly. Biological sex correlated with average state shame, brooding, and depressive symptoms. As expected, average state shame was positively correlated with average state guilt, avoidance, brooding, suppression, and depressive symptoms. Contrary to hypotheses, average state guilt also correlated with average state avoidance, brooding, suppression, and depressive symptoms. In addition, average state avoidance, brooding and suppression were positively correlated and all three emotion regulation strategies were positively correlated with average state depressive symptoms (see Table 5).

Table 5
Bivariate Correlations and Descriptives among Average Daily Questionnaire Variables

N	*Variable*	1	2	3	4	*M (SD)*
137	1. Bio Sex					.83 (.38)
113	2. State Avoidance	.193*				1.31 (.84)
113	3. State Brooding	.212*	.751**			.99 (.31)
113	4. State Suppression	.137	.730**	.717**		2.17 (1.02)
113	5. State CES-D	.232*	.539**	.544**	.605**	2.86 (.82)

Note. CES-D = Center for Epidemiological Studies Depression Scale; *$p < .05$, ** $p < .01$.

Cross-Sectional Analyses

Model Fit. I assessed my mediation hypotheses with path analysis in Amos 24. Path analysis was used to determine the best-fitting model of the data (Kenny & Milan, 2008). I assessed the adequacy of hypothesis 1 model by evaluating the fit statistics including the chi-square (χ^2) likelihood ratio statistic, the comparative fit index (CFI), and root mean square error of approximation (RMSEA). The fit indices were: $\chi^2(6) = 26.235$, $p < .001$; CFI = .913; RMSEA = .157. According to Byrne (2013), the model fit was poor. Therefore, I proceeded to making theory-driven changes to the model (see Table 6). Following this process, the modified hypothesis 1 model was used (see Figure 6).

Table 6
Concurrent Model Fitting

Model	X^2	*df*	*p*	Model Comparison	ΔX^2	Δdf	CFI	RMSEA	Δ in Model
Hyp1 Model	26.235	6	<.001				.913	.157	
M1	10.841	3	.013	Hyp1 vs M1	15.394	3	.956	.139	Deleted NA
M2	11.895	4	.018	M1 vs M2	1.054	1	.956	.120	Deleted G→DS
M3	1.948	3	.583	M2 vs M3	9.947	1	1.000	.000	Added G→Avoidance

Note. NA = negative affect, DS = depressive symptoms, G = guilt-proneness,

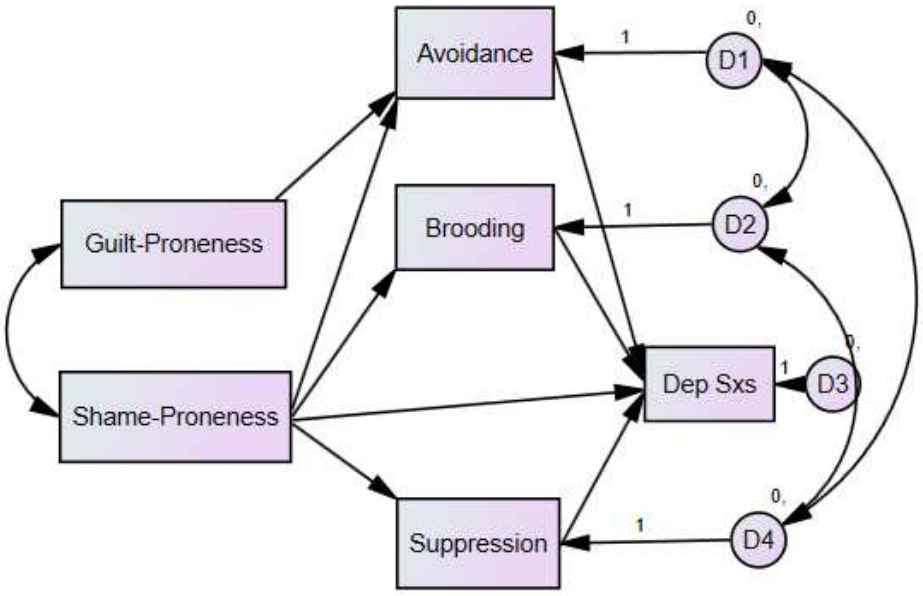


Figure 6. Modified Hypothesis 1 Model

Analysis. Once adequate model fit was established, I assessed the joint effects of multiple mediators. Phantom variables allow for calculation of the unique effect of each individual mediator. Bias-corrected bootstrapping was used to estimate the indirect effects.

Indirect effects are considered significant when the confidence intervals do not include 0; confidence intervals that include 0 are considered non-significant.

C' path: Did shame-proneness predict baseline depressive symptoms? Greater shame-proneness predicted of greater baseline depressive symptoms (B = .029, 95% CI = .010 to .048, p = .003). The specific indirect effects, total effects, and direct effects are discussed below and reported in Table 7.

A path: Did shame-proneness predict the proposed mediator?

Avoidance as a proposed mediator. Within the full model, the direct effect of greater shame-proneness on greater trait avoidance was significant (B = .477, 95% CI = .234 to .700, p = .002).

Brooding as a proposed mediator. The direct effect of greater shame-proneness on greater trait brooding was significant (B = .024, 95% CI = .013 to .037, p = .001).

Suppression as a proposed mediator. The direct effect of greater shame-proneness on greater trait suppression was non-significant (B = .013, 95% CI = -.003 to .028, p = .114).

B path: Did proposed mediator predict baseline depressive symptoms?

Avoidance as a proposed mediator. Greater trait avoidance did not predict greater baseline depressive symptoms (B = .013, 95% CI = -.002 to .030, p = .095).

Brooding as a proposed mediator. Greater trait brooding predicted greater baseline depressive symptoms (B = .396, 95% CI = .117 to .684, p = .010).

Suppression as a proposed mediator. Greater trait suppression predicted greater baseline depressive symptoms (B = .319, 95% CI = .059 to .529, p = .011).

Indirect effects.

Avoidance as a mediator. The specific indirect effect (greater shame-proneness through greater trait avoidance) was a marginally significant predictor of greater baseline depressive symptoms (B = .006, 95% CI = .000 to .017, p = .064).

Brooding as a mediator. The specific indirect effect (greater shame-proneness through greater trait brooding) predicted greater baseline depressive symptoms (B = .010, 95% CI = .003 to .019, p = .005).

Suppression as a mediator. The specific indirect effect (greater shame-proneness through greater trait suppression) did not predict greater baseline depressive symptoms (B = .004, 95% CI = .000 to .012, p = .075).

Table 7

Hypothesis 1 Bootstrap Analysis of Magnitude and Statistical Significance of Indirect and Direct Effects

IV→ Mediator (if any)			→	DV	*β* (standardized path coefficient and product)				Mean effect *B*	SE of mean	95% CI Lower	95% CI Upper	Two-tailed significance	
Indirect Effects														
SH	→	A	→	DS	.412	X	.127	=	.052	.006	.004	.000	.017	.064	
SH	→	B	→	DS	.336	X	.231	=	.078	.010	.004	.003	.019	.005	**
SH	→	S	→	DS	.152	X	.224	=	.034	.004	.003	.000	.012	.075	
G	→	A	→	DS	-.310	X	.224	=	-.086	-.050	.036	-.141	.002	.062	
Direct Effects														
G	→	A							-.310	-3.670	1.117	-5.824	-1.396	.002	**
SH	→	DS							.238	.029	.009	.010	.048	.003	**
SH	→	A							.412	.477	.117	.234	.700	.002	**
SH	→	B							.336	.024	.006	.013	.037	.001	**
SH	→	S							.152	.013	.008	-.003	.028	.114	
A	→	DS							.127	.013	.008	-.002	.030	.095	
B	→	DS							.231	.396	.146	.117	.684	.010	**
S	→	DS							.224	.319	.118	.059	.529	.011	*

Notes. A = avoidance, B = brooding, S = suppression, DS = depressive symptoms, G = guilt-proneness, SH = shame-proneness, NA = trait negative affect. *$p < .05$. ** $p < .01$.

Prospective Analyses: Hypothesis 2

Data analytic plan. In hypothesis 2, the independent variable was trait shame-proneness. The dependent variable was depressive symptoms averaged across 6 weeks of the daily questionnaires. The proposed mediators were average state brooding, avoidance, and suppression reported across 6 daily questionnaires. Trait guilt-proneness, trait NA, and depressive symptoms at baseline were controlled for in the multiple mediation model. See Table 7 for the correlations and descriptives among hypothesis 2 variables. Path analysis was used to determine the best-fitting model of the data (Kenny & Milan, 2008). I assessed the adequacy of hypothesis 2 model by evaluating the fit statistics including the chi-square (χ^2) likelihood ratio statistic, the comparative fit index (CFI), and root mean square error of approximation (RMSEA). The fit indices were: $\chi^2(9) = 28.262$, $p = .001$; CFI = .955; RMSEA = .138. According to Byrne (2013), the model fit is poor. Therefore, I proceeded to making theory-driven changes to the model. Specifically, given that trait negative affect and baseline depressive symptoms were so highly correlated with shame-proneness and depressive symptoms prospectively, I wondered if given the sample size, I did not have enough variance in the model to identify significant relationships between shame-proneness and depressive symptoms. Therefore, I removed trait NA and baseline depressive symptoms from the model. The modified hypothesis 2 model has the following fit indices: $\chi^2(3)$ 3.825, $p = .281$; CFI = .997; RMSEA = .050. According to Byrne (2013), the model fit is adequate. Therefore, the modified hypothesis 2 model was used to further assess indirect effects (see Figure 7).

Table 8

Bivariate Correlations and Descriptives among Hypothesis 2 Variables

N	*Variable*	1	2	3	4	5	6	7	*M (SD)*
137	1. Trait NA								4.15 (.74)
137	2. Trait Shame	.482**							48.13 (9.59)
137	3. Trait Guilt	.246**	.599**						3.02 (.94)
137	4. Baseline CES-D	.445**	.378**	.129					3.93 (1.18)
113	5. State Avoidance	.302**	.328**	.065	.418**				47.79 (11.12)
113	6. State Brooding	.420**	.421**	.176	.453**	.751**			2.43 (.69)
113	7. State Suppression	.377**	.380**	.133	.475**	.730**	.717**		1.69 (.83)
113	8. State CES-D	.484**	.316**	.016	.679**	.539**	.544**	.605**	2.86 (.82)

Note. CES-D = Center for Epidemiological Studies Depression Scale; NA = Negative Affect; *$p < .05$, ** $p < .01$.

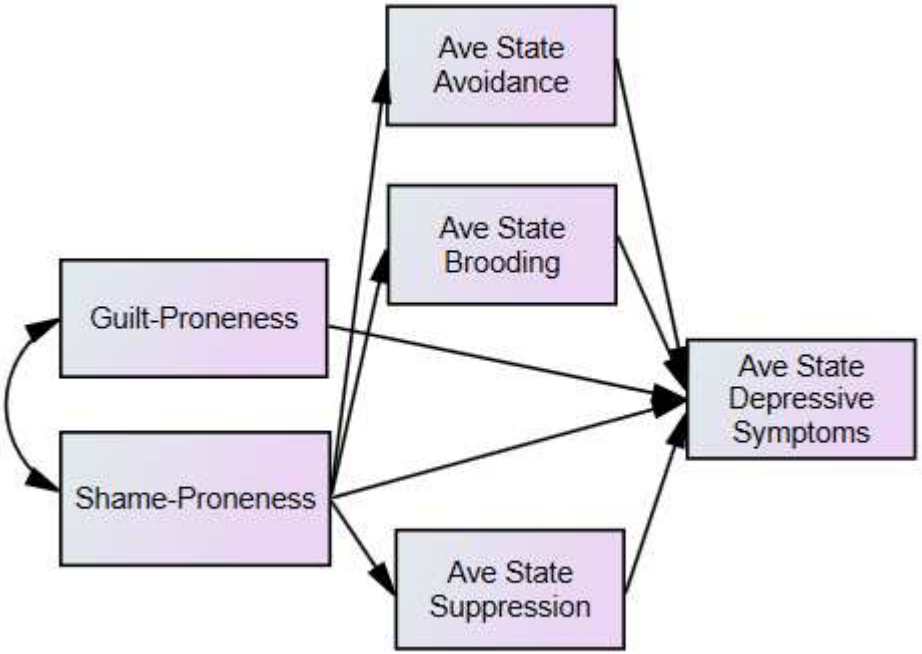


Figure 7. Modified Hypothesis 2 Model

C' path: Did shame-proneness predict baseline depressive symptoms? There was a marginally significant direct effect of greater shame-proneness on greater average daily depressive symptoms (*B* = .016, 95% CI = -.002 to .033, *p* = .074). The specific direct and indirect effects are discussed below and reported in Table 8.

A path: Did shame-proneness predict proposed mediator?

Avoidance as a proposed mediator. Within the full model, greater shame-proneness predicted greater average state avoidance (*B* = .029, 95% CI = .012 to .044, *p* = .001).

Brooding as a proposed mediator. Greater shame-proneness predicted greater average state brooding (*B* = .014, 95% CI = .008 to .019, *p* = .001).

Suppression as a proposed mediator. Greater shame-proneness predicted greater average state suppression (*B* = .040, 95% CI = .022 to .058, *p* = .001).

B path: Did proposed mediator predict baseline depressive symptoms?

Avoidance as a proposed mediator. Greater average state avoidance did not predict greater average daily depressive symptoms (*B* = .101, 95% CI = -.143 to .335, *p* = .397).

Brooding as a proposed mediator. Greater average state brooding did not predict greater average daily depressive symptoms (*B* = .400, 95% CI = -.217 to 1.027, *p* = .173).

Suppression as a proposed mediator. Greater average state suppression predicted greater average daily depressive symptoms (*B* = .302, 95% CI = .118 to .492, *p* = .003).

Indirect effects.

Avoidance as a mediator. The specific indirect effect (greater shame-proneness through greater average state avoidance) did not predict greater average depressive symptoms (*B* = .003, 95% CI = -.004 to .012, *p* = .336).

Brooding as a mediator. The specific indirect effect (greater shame-proneness through greater average state brooding) did not predict greater average depressive symptoms (B = .005, 95% CI = -.002 to .016, p = .153).

Suppression as a mediator. The specific indirect effect (greater shame-proneness through greater average state suppression) predicted greater average depressive symptoms (B = .012, 95% CI = .004 to .024, p = .002).

Table 9

Hypothesis 2 Bootstrap Analysis of Magnitude and Statistical Significance of Indirect and Direct Effects

IV→ Mediator (if any)				DV	β (standardized path coefficient and product)					Mean effect B	SE of mean	95% CI		Two-tailed significance	
												Lower	Upper		
Indirect Effects															
SH	→	A	→	DS2	.328	X	.104	=	.034	.003	.004	-.004	.012	.336	
SH	→	B	→	DS2	.421	X	.153	=	.064	.005	.004	-.002	.016	.153	
SH	→	S	→	DS2	.380	x	.378	=	.144	.012	.005	.004	.024	.002	**
Direct Effects															
G	→	DS2							-.183	-.154	.080	-.306	.016	.070	
SH	→	DS2							.188	.016	.009	-.002	.033	.074	
SH	→	A							.328	.029	.008	.012	.044	.001	**
SH	→	B							.421	.014	.003	.008	.019	.001	**
SH	→	S							.380	.040	.009	.022	.058	.001	**
A	→	DS2							.104	.101	.120	-.143	.335	.397	
B	→	DS2							.153	.400	.318	-.217	1.027	.173	
S	→	DS2							.378	.302	.095	.118	.492	.003	**

Notes. A = average daily avoidance, B = average daily brooding, S = average daily suppression, DS2 = average daily depressive symptoms, G = guilt-proneness, SH = shame-proneness. *$p < .05$. ** $p < .01$.

Prospective Analyses: Hypothesis 3

Data analytic plan. In hypothesis 3, I conducted a shame induction manipulation check using a paired-sample t-test. The test was significant t (114) = -2.814, p = .006. Prior to the shame-induction participants endorsed lower state shame (M = .73, SD = .15) than following the shame-induction (M = .77, SD = .18), although this difference was relatively small (r = .25). For the purposes of distinguishing shame from guilt, I examined change in state guilt as well. The t-test comparing pre-induction and post-induction guilt was significant t (114) = -5.573, p < .001. Prior to the shame-induction participants endorsed lower state guilt (M = .77, SD = .16) than following the shame-induction (M = .85, SD = .18), and this difference was moderate (r = .46). I used Hayes PROCESS macro in SPSS to test indirect effects of the hierarchical linear regression. I controlled for baseline depressive symptoms, guilt-proneness, and biological sex in step 1. I entered trait shame-proneness in step 2 as the independent variable. The mediator was change in state shame from pre to post shame induction and was entered in step 3. The three dependent variables assessed separately were state avoidance, brooding and suppression measure post shame induction. The indirect effect was tested using a bootstrap estimation approach of 1000 samples and looking at the 95% confidence intervals (Hayes & Scharkow, 2013). See Table 9 for correlations and descriptive for hypothesis 3 variables.

Avoidance. The specific indirect effects, total effects, and direct effects are discussed below. Shame-proneness did predict state avoidance post shame (B = .003, SE = .002, p = .048). However, shame-proneness did not predict greater shame from pre to post shame induction (B = -.002, SE = .002, p = .386) and the change in shame did not predict state avoidance post shame induction (B = .069, SE = .081, p = .399). Therefore, state shame in the shame induction did not

mediate the relationship between shame-proneness at baseline and state avoidance following the shame induction (*B* = -.0001, *SE* = .0003, 95% CI = -.001 to .000).

Brooding. Shame-proneness was only a marginal predictor of state brooding post shame (*B* = .003, *SE* = .002, *p* = .071). However, shame-proneness did not predict greater shame from pre to post shame induction (*B* = -.002, *SE* = .002, *p* = .386). The shame induction did predict greater state brooding post shame induction (*B* = .220, *SE* = .091, *p* = .017). Taken together state shame in the shame induction did not mediate the relationship between shame-proneness at baseline and state brooding following the shame induction (*B* = -.0003, *SE* = .001, 95% CI = -.002 to .001).

Suppression. Shame-proneness did not predict state suppression post shame (*B* = .002, *SE* = .002, *p* = .368) or greater shame pre to post shame induction (*B* = -.002, *SE* = .002, *p* = .386). The shame induction did predict greater state suppression post shame induction (*B* = .309, *SE* = .110, *p* = .006). Therefore, change in shame in the shame induction did not mediate the relationship between shame-proneness at baseline and state suppression following the shame induction (*B* = -.001, *SE* = .001, 95% CI = -.003 to .001).

Table 10

Bivariate Correlations and Descriptives among Hypothesis 3 Variables

N	*Variable*	1	2	3	4	5	6	7	8	*M* (*SD*)
137	1. Biological Sex									.83 (.38)
137	2. Trait Shame	.209*								48.13 (9.59)
137	3. Trait Guilt	.175*	.599**							3.02 (.94)
137	4. Baseline CES-D	.124	.378**	.129						3.93 (1.18)
115	5. Change in Shame	.154	.077	.130	.176					.04 (.14)
115	6. Change in Guilt	.131	.161	.054	.031	.431**				.08 (.16)
115	7. Post Induction Avoidance	.074	.268**	.065	.347**	.126	.042			.92 (.12)
115	8. Post Induction Brooding	.159	.302**	.136	.393**	.278**	.152	.446**		.33 (.14)
115	9. Post Induction Suppression	.123	.195*	.057	.384**	.304**	.125	.512**	.624**	1.12 (.17)

Note. CES-D = Center for Epidemiological Studies Depression Scale; *$p < .05$, ** $p < .01$.

Post Hoc Analyses

I analyzed cross-sectional mediation models of each mediator separately in SPSS, using model 4 in the PROCESS macro. Controlling for guilt and analyzing each mediator separately, trait avoidance and brooding each mediated the relationship between shame-proneness and baseline depressive symptoms (see Table 10). Trait suppression did not mediate the relationship between shame-proneness and baseline depressive symptoms.

Table 11
Post Hoc Hypothesis 1 Individual Mediation PROCESS Results

Path/effect	*B*	*SE*	95% CI		*Z*	*P*
Avoidance			Lower	Upper	Sobel test	
c	.046	.013				.000**
a SH→A	.485	.118				.000**
b A→DS2	.023	.009				.008**
c' SH→DS2	.011	.005	.004	.023	2.200	.028*
Brooding						
c	.041	.012				.001**
a SH→B	.028	.007				.000**
b B→DS2	.608	.136				.000**
c' SH→DS2	.017	.006	.008	.029	2.879	.004**
Suppression						
c	.051	.011				.000**
a SH→S	.014	.009				.132
b S→DS2	.051	.011				.000**
c' SH→DS2	.007	.005	-.002	.019	1.418	.156

Notes. A = average daily avoidance, B = average daily brooding, S = average daily suppression, DS2 = average daily depressive symptoms, SH = shame-proneness. *$p < .05$. ** $p < .01$.

In addition, I analyzed prospective mediation models of each mediator separately in SPSS, using model 4 in the PROCESS macro. Controlling for guilt and analyzing each mediator separately, average state avoidance, brooding and suppression each mediated the relationship between shame-proneness and average state depressive symptoms (see Table 10).

Table 12
Post Hoc Hypothesis 2 Individual Mediation PROCESS Results

Path/effect	*B*	*SE*	95% CI		*Z*	*p*
Avoidance			Lower	Upper	Sobel test	
c	.024	.009				.012*
a SH→A	.040	.010				.000**
b A→DS2	.449	.082				.000**
c' SH→DS2	.018	.006	.008	.032	3.251	.001**
Brooding						
c	.021	.009				.023**
a SH→B	.016	.004				.000**
b B→DS2	1.263	.229				.000**
c' SH→DS2	.020	.006	.011	.033	3.501	.001**
Suppression						
c	.020	.009				.027*
a SH→S	.050	.012				.000**
b S→DS2	.439	.066				.000**
c' SH→DS2	.022	.006	.013	.035	3.602	.000**

Notes. A = average daily avoidance, B = average daily brooding, S = average daily suppression, DS2 = average daily depressive symptoms, SH = shame-proneness. *$p < .05$. ** $p < .01$.

CHAPTER IV

Discussion

Depression represents one of the most prevalent health issues in the world. Specifically, Major Depressive Disorder is the leading cause of disability in the United States across ages 15-44 (World Health Organization, 2004) and worldwide across ages five and older (World Health Organization, 2007). Depression is detrimental to multiple areas of life and therefore, a thorough understanding of this mental health condition and the mechanisms causing and maintaining it is crucial. Using the well supported cognitive affective models of depression, we can better identify and examine factors that contribute to depression and ultimately develop interventions.

The purpose of the current study was to further examine the relationship between shame-proneness and depressive symptoms, and specifically, the three cognitive emotion regulation strategies, avoidance, brooding, and suppression, as potential mechanisms driving the relationship between shame-proneness and increased depressive symptoms. Using a sample of young adults, three hypotheses were examined. First, I examined cross-sectionally the relationship between shame-proneness and depressive symptoms to test the overall model that the tendency to experience shame may be associated with greater depressive symptoms and also that trait patterns of avoidance, brooding, and suppression may facilitate this relationship jointly.

Second, I examined this model prospectively to answer the question of the causal impacts of shame affect and cognitive strategies on the development and maintenance of depressive symptoms. The current study was a novel contribution to the literature as it attempted to examine jointly and prospectively three potential cognitive emotion regulation strategies as mechanisms driving the relationship between the tendency to experience shame and greater depressive symptoms.

In addition to the first two hypotheses, I also wanted to explore the relationship between trait and state shame, and state cognitive reactions to shame. To do this, I attempted to induce the shame emotion in participants and then evaluate emotion regulation strategies to that emotion using self-report immediately following the induction.

Cross-Sectional Analyses. I will first review results of hypothesis 1, which were intended to reinforce previous research findings. With a modified model, cross-sectional results found that shame-proneness, the trait tendency to experience shame in negative events, was a significant predictor of baseline depressive symptoms. In addition, with all variables in the model shame-proneness also significantly predicted greater trait avoidance and trait brooding. Contrary to my hypotheses and previous research, shame-proneness did not significantly predict greater trait suppression. Cognitive emotion regulation strategies of trait brooding and suppression did each significantly predict greater depressive symptoms within the joint model. Trait avoidance was a marginally significant predictor of depressive symptoms. Given these relationships, the indirect effects of shame-proneness on depressive symptoms was not significant through trait suppression. The indirect effects through trait brooding was significant. Trait avoidance was a marginally significant mediator. Of note, the model included guilt-proneness as covariate. Guilt-proneness, as previous studies suggests, did not significantly predict greater depressive symptoms (Bennet, Sullivan, & Lewis, 2010; Tangney et al., 1992) but did predict less trait avoidance. This finding is interesting and aligns with previous research that shows that guilt tends to elicit active problem-solving and amends-making behavior, while shame elicits avoidance and hiding (Covert, Tangney, Maddux, & Heleno, 2003; Lopez, Gover, Leskela, Sauer, Schirmer, & Wyssmann, 1997).

Prospective Analyses. In analyses of hypothesis 2, I looked at this model prospectively. Increased shame-proneness marginally predicted greater average daily depressive symptoms. Results indicated that greater shame-proneness predicted greater average daily use of avoidance, brooding, and suppression. However, only average daily suppression predicted greater average daily depressive symptoms when all emotion regulation cognitive strategies were in the model. The indirect effects through average daily suppression was significant. The model included guilt-proneness as a covariate. Contrary to cross-sectional analyses, guilt-proneness did not predict state avoidance.

I conducted post hoc analyses that assessed mediators separately and found concurrently that greater trait avoidance and brooding mediated the relationship between greater shame-proneness and greater baseline depressive symptoms. Prospectively, greater trait avoidance, brooding, and suppression mediated the relationship between greater shame-proneness and greater average depressive symptoms. Statistically, this suggests that trait NA and baseline depressive symptoms are very strong predictors of depressive symptoms and when removed from the model allowed us to examine the more subtle relationship between shame-proneness and baseline and daily depressive symptoms. Secondly, analyzing mediators jointly in the model did not support the significance of all three mediators and this may be due in part to the high correlations among the mediators. It seems that these cognitive strategies may represent significant factors predicting depressive symptoms beyond high trait NA and previous depressive symptoms and these mechanisms are similar maladaptive emotion regulation strategies.

State Shame Did Not Mediate the Relationship Between Shame-proneness and Emotion Regulation Strategies. Finally, in analyses of hypothesis 3, we found that the shame induction did elicit a significant change in state shame pre and post induction but the effect was

small. Therefore, the indirect effects of shame-proneness on each state emotion regulation cognitive strategy (avoidance, brooding, and suppression) was not significantly mediated by state shame. However, shame-proneness did significantly predict state avoidance and marginally predicted brooding suggesting that the tendency to experience shame leads individuals to use avoidance and brooding in the moment when faced with negative events.

Was Shame-proneness Associated with Greater Depressive Symptoms?

Overall, results support previous research findings that shame-proneness is associated with and predicts greater depressive symptoms. Multiple theories on the development of shame-proneness assert that the tendency to experience shame emerges from relationship factors beginning in early childhood (Gilbert, 2003; Nathanson, 1992; Tomkins, 1963). Research suggests that individuals are showing high shame-proneness by adolescence (Orth, Robins, Soto, 2010).

Similarly, Young and colleagues (2016) examined general motivational causality orientations that may develop overtime given environmental contexts. They examined a control orientation, described as an orientation that develops in environments of limited control and choice, coercive, contingent, and extrinsically designed motivations. They also examined impersonal orientation, described as an orientation that develops in environments when individuals cannot see connection between their effort and outcomes, fail to experience competence, relatedness or autonomy regardless of their performance. Young and colleagues found these two orientations were associated with greater shame-proneness and greater depressive symptoms and suggested that shame-proneness may mediate the relationship between control orientation and impersonal orientation and depressive symptoms (Young, Neighbors, Dibello, Traylor, & Tomkins, 2016). Future research should continue to examine prospectively

the relationship and environmental factors that contribute to the development of shame-proneness compared to guilt-proneness. I don't wish to discount shame as a natural and important social, moral emotion. I do wish to suggest the development of guilt-proneness tendencies may be preferable for general psychological well-being and may facilitate more adaptive prosocial responses in moral situations. In understanding the development of shame-proneness, we may find preventative interventions and reduce subsequently this vulnerability to depression.

Did Shame-proneness Predict Greater Depressive Symptoms Through Greater Avoidance, Brooding, and Suppression?

Avoidance. Results supported my hypothesis that shame-proneness would predict greater avoidance in negative events. Cross-sectional analyses suggested shame-proneness is associated with greater trait avoidance (H1). Prospectively, shame-proneness did predict greater average daily avoidance and greater use of avoidance in negative events (H2 and H3). This is consistent with previous studies and also the description of responses to the emotion of shame (Tangney & Wagner, 1992; Tangney et al., 1996). Cross-sectionally, avoidance was marginally significant mediator in the relationship between shame-proneness and depressive symptoms in the full multi-mediational model. Prospectively, state avoidance was not a significant mediator. However, in post hoc analyses, when mediators were analyzed separately, avoidance was a significant mediator concurrently and prospectively. Analyses that did not support this may have been impacted the degree of shared variance across mediators (avoidance, suppression and brooding) and reduced variance caused by the collapse of multiple data points (daily questionnaires) into an average single data point (time 3). While results were mixed, there was some support for previous research that highlights avoidance as a relevant emotion regulation

cognitive strategy in the relationship between shame-proneness and depressive symptoms (De Rubeis & Hollenstein, 2009).

Brooding. Results largely supported my hypothesis that shame-proneness would predict greater brooding in negative events. Shame-proneness was found to be associated with greater trait brooding cross-sectionally (H1). In addition, shame-proneness predicted greater average daily brooding (H2) and greater state brooding in response to the shame-induction (H3), consistent with previous studies (Cheung et al., 2004). In cross-sectional analyses, I found that trait brooding significantly mediated the relationship between greater shame-proneness and greater depressive symptoms. However, average state brooding did not significantly mediate the relationship between shame-proneness and average daily depressive symptoms with all mediators in the model. In post hoc analyses, greater brooding significantly mediated the relationship between greater shame-proneness and greater depressive symptoms cross-sectionally and prospectively, consistent with previous research (Orth et al., 2006). Previous research has strongly supported the notion that brooding is a strong cognitive predictor of the onset and maintenance of depressive symptoms, these results also provided some support to the notion that it has important implications in context of shame-proneness and its impact on depressive symptoms.

Suppression. Results largely supported my hypothesis that shame-proneness would predict greater suppression. Shame-proneness was not found to be associated with greater trait suppression cross-sectionally (H1) but prospectively, shame-proneness predicted greater average daily suppression (H2). However, shame-proneness did not predict greater suppression in response to the shame induction (H3). Trait suppression did not significantly mediate the relationship between shame-proneness and depressive symptoms cross-sectionally. Prospective

analyses supported greater average daily suppression as a mediator between greater shame-proneness and greater average daily depressive symptoms. Overall, these mixed findings regarding suppression as an emotion regulation cognitive strategy warrant future research.

Do Avoidance, Brooding, and Suppression Jointly Mediate the Relationship Between Shame-proneness and Depressive Symptoms?

One goal of this study was to examine the joint effects of avoidance, brooding and suppression in the relationship between shame-proneness and depressive symptoms. This question of joint effects was examined cross-sectionally and prospectively using path analysis. Overall, analyses did not fully support all three mediators in the model. Despite these findings, future research is warranted. This study was limited by the brevity of the prospective study period that is likely not long enough to observe a meaningful change in depressive symptoms. Future studies should continue to examine these relationships prospectively with longer time periods that might more accurately capture significant changes in depressive symptoms. Another limitation of my analyses was the collapse of variables across daily data points for an average daily score. Future studies might consider a similar design and elect to utilize analyses (e.g. multilevel modeling) that can enhance the power of the multiple data points. Overall, future research should continue to consider these emotion regulation cognitive strategies jointly and examine the relationship between them. It may also be helpful to consider additional emotion regulation cognitive strategies that have not been studied here but are theoretically relevant.

Does State Shame Mediate the Relationship Between Shame-proneness and Avoidance, Brooding, and Suppression?

State shame was not successfully elicited in the shame induction task and subsequently did not mediate the relationship between shame-proneness and state avoidance, brooding, and

suppression. Continued research on emotional regulation cognitive strategies occurring at a state level is encouraged and may benefit from additional refinement of shame induction strategies for the purposes of better understanding event specific responses and how to intervene in the moment. It may also be helpful to compare this state shame measure with other state measures and see whether the SSGS is the most effective tool to measure and distinguish state shame and state guilt. Interestingly, even though state shame was not effectively elicited, shame-proneness did significantly predict greater use of avoidance and brooding following the shame induction. This finding further supports the notion that shame-proneness leads to problematic emotion regulation cognitive strategies to cope with this difficult, painful emotion.

Limitations and Future Directions

There are several limitations of the current study that are important to consider. First, the shame induction did not significantly elicit shame as intended, preventing us from assessing successfully the mediation model of hypothesis 3. Not a lot of research has attempted to induce state shame and those studies that have tried used a writing task that asked participants to reflect on a shameful memory or read about a selected shameful experience. Given the possible uses in this research area, it is recommended that methods be established to effectively induce shame and multiple measures be used to assess the induction of shame or guilt. Future research should investigate the relationship between state shame and state emotion regulation cognitive strategies to further tease apart the processes occurring following state shame contributing to depressive symptoms and other mental health concerns.

A second limitation of the study was the loss of power when I collapsed multiple daily variable data points into one average variable in prospective analyses. Future research should continue to pursue prospective design and analyses, with time frames that range from the few

weeks, as in our study, to months and years, for the purposes of exploring causal relationships between these affective and cognitive variables and depressive symptoms. Third, our measures were all self-report and therefore, there may be threats to reliability. Fourth, our sample was limited to college students and may not generalize to the larger public and clinical populations. Sixth, future research should examine cultural factors that may moderate the effect of shame on depressive symptoms and on the use of maladaptive emotion regulation cognitive strategies.

Considering the assessment of the three cognitive processes jointly, it may be interesting for future studies to assess whether they are more related than distinct. The literature distinguishes between suppression and avoidance sometimes but not others and across all three mediators there is high correlation. Considering the results in this study, rumination and suppression separately mediated the relationship between shame-proneness and depressive symptoms cross-sectionally and prospectively, respectively. Rumination represents a strategy of perseverating on negative thoughts, emotions, and events, which can include voluntary or involuntary perseveration. Suppression represents a strategy to not think about negative thoughts, emotions, and events, which research shows leads to increased negative thoughts and emotions. It may be that a person uses suppression to respond to difficulty managing rumination processes. It may be interesting to examine the sequence of how these strategies are employed.

A previous study assessed the model fit of 3 cognitive processes (i.e., worry, experiential avoidance, and thought suppression) versus one single factor combined and found that while model fit was equivalent, a one factor model accounted for more variance predicting depression and anxiety symptoms prospectively (Bird, Mansell, Dickens, & Tai, 2012). This suggests that there is significant overlap between cognitive emotion regulation strategies that may be better captured in one construct. Thus, it is recommended that cognitive emotion regulation strategies

be examined for their similar and/or distinct presentations and effects on development and maintenance depressive symptoms to determine whether these cognitive emotion regulation strategies could be better captured by a larger maladaptive emotion regulation construct.

At a broader level, future directions of research could benefit from examining the multiple areas of shame research together to distinguish the unique and similar ways that shame impacts different populations. While this study has largely focused on research on the relationship between shame proneness and depression, it has hinted at the larger area of research examining shame as an emotional state impacting non clinical populations at a state level. This study also acknowledges a large area of research examining shame-proneness as a problematic pattern leading the mental health problems in clinical populations (Leskela, Dieperink, & Thuras, 2002; Robinaugh & McNally, 2010). A third large body of research on shame also comes from trauma research. This research posits that traumatic events, which often threaten self-appraisal and self-concept, lead to acute and highly problematic shame experiences (Dyer, Dorahy, Shannon, & Corry, 2013; Srinivas, DePrince, & Chu, 2015). Even within this trauma research are bodies of research that examine effects of shame on PTSD. While shame is not a diagnostic symptom of PTSD, the presence shame has been to contribute to the development and maintenance of PTSD (Øktedalen, Hoffart, & Langkaas, 2015) and has led to a body of research on moral injury, which is painful emotional experience (i.e., shame, guilt) that results from dissonance between an experience and one's fundamental beliefs about the world and self (Litz et al., 2009. In addition, trauma research has also examined the impact of shame on complex PTSD and DID, distinguished from PTSD by the symptoms that surface above and beyond PTSD symptoms following the chronic, repeated traumatic events (Courtois & Ford, 2012; Herman, 2015; Pelcovitz et al., 1997). Recently, Dyer and colleagues (2016) attempted to bridge

the research in these areas and examine state shame across four groups: 1) individuals diagnosed with DID, 2) individuals diagnosed with complex trauma, 3) individuals with a general mental health disorder, and 4) individual without mental health diagnoses. Cross-sectional analyses found that all three clinical groups had significantly greater state shame than the non clinical group. In addition, groups with complex trauma and general mental health concerns had less state shame than the group with DID. Dyer and colleagues also examined shame coping styles and how these were similar or different across different groups. Future studies should continue from this work and examine the use of avoidance, brooding, and suppression across these different groups of people to determine if these emotion regulation cognitive strategies are being used across multiple different populations and if they are contributing to the maintenance of their symptoms.

Clinical Application

The current study and previous research demonstrated a significant detrimental relationship between shame-proneness and greater depressive symptoms (Andrews et al., 2002; Cheung et al., 2004; Kim et al., 2011; Pineles et al., 2006; Tangney et al., 1992). Similarly, the current study and previous research has identified avoidance, brooding and suppression as trait and state level cognitive responses that contribute to greater depressive symptoms (Aldao et al., 2010; Beevers & Meyer, 2004; Blalock & Joiner, 2000; Borton & Casey, 2006; Borton et al., 2005; Holahan et al., 2005; Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1991; 1993; Nolen- Hoeksema, et al., 1993; Seiffge-Krenke & Klessinger, 2000; Wenzlaff & Luxton, 2003). Therefore, attention to these pieces in treating depressive symptoms will be helpful. While shame is a natural human emotion, as mentioned before, it can become problematic when elicited frequently and as a result of rigid cognitive conclusions individuals make about themselves.

Considering shame and reactions to shame is important in treating clinical depression, as well as, other mental health disorders. Among the many increased negative emotions people with depressive symptoms face, shame represents a particularly problematic emotion associated with a number of maladaptive responses.

Shame is a self-focused, as well as other-focused, emotion and at a fundamental cognitive level is elicited when a person expects valued others to negatively evaluate them. The natural reactions to shame involve inhibiting interaction and communication with others (Keltner & Harker, 1998). While this may play out in a number of ways, it is important to acknowledge that it will often surface in therapeutic work. Despite good rapport, patients find it difficult to admit to another person something they are ashamed of, which can be a barrier to therapy and ultimately, the work of healing shame. Perceiving oneself as flawed in some way can affect motivation to make positive changes. Living through a horrific event where you had to act in a way dissonant with who you believe yourself to be and feeling shame as a result of the change in your self-concept can be a barrier to moving forward. These are a few examples of the ways in which shame may present in therapy.

Shame and vulnerability researcher Brene Brown writes in depth about overcoming shame and her work has been well received as a self-help and therapy resource. At the most basic level, she speaks of a shame-culture that struggles to embrace differences, and equates behavior to a trait, leaving little room for choice. In her work, she describes tools needed overcome the emotion of shame, tools for shame resilience, 1) Ability to recognize and understand shame triggers, 2) high levels of critical awareness, 3) ability to reach out and tell your story, and 4) ability to speak shame. Broadly speaking these tools make therapeutic sense and align with research on addressing shame that I will review briefly now (Brown, 2012).

One of her biggest recommendations is sharing and communicating about personal shame. While there are stipulations for this process (i.e., finding someone who can support you in sharing personal shame), the underlying purpose undermines a core problem with shame and responses to shame. By sharing personal shame, individuals do not avoid others, do not hide away, and it increases the opportunity for another person to challenge the individual's fears around another's judgement of them. For example, if person A shares their personal shame and person B supports them, validates the courage it took to share, and even identifies with their shame emotion, the outcome was person B has provided an alternative response to person A's expectation and reduced person A's motivation to isolate. Therapy, whether it is individual or group, is a great possible sharing space where a person can share their shame in a safe place, have a corrective experience, and likely reduce the powerful emotion of shame.

The cognitive component influencing the experience of shame comes when a person attributes a personal behavior to a trait flaw. Considering cognitive interventions may be helpful in treating shame by providing increased awareness of the thoughts that precede the emotion, space to evaluate the accuracy of those thoughts, and opportunity to reframe those thoughts and reduce the emotional response. In addition, using cognitive interventions at the point of emotion regulation to reduce brooding, avoidance and suppression could help reduce the intensity of the emotion. Mindfulness may be one intervention, as it has been shown to reduce brooding (Eisendrath et al., 2008; Michalak, Hölz, & Teismann, 2011; Ramel, Goldin, Carmona, & McQuaid, 2004). In addition, mindfulness is a core component of Acceptance and Commitment Therapy (ACT), which targets avoidance and suppression tendencies. (Hayes, Strosahl, & Wilson, 1999).

Research on moral injury in PTSD literature is examining the harmful effects of shame and other moral emotions following traumatic events. Moral injury posits that when a person is faced with situations dissonant to their fundamental beliefs about the world and the self, they may experience more shame as a result of the dissonance and try to avoid future situations that may remind or reawaken the dissonance (Litz et al., 2009). Interventions for moral injury in PTSD have emphasized moving toward self-forgiveness, described as motivational changes through which there is less motivation to avoid shame inducing stimuli and to engage in self-punishing and self-destructive behaviors, and more motivation to act compassionately with the self (Hall & Fincham, 2005). Practicing self-compassion has been found to predict lower levels of depression, and is associated with less rumination and less suppression (Neff, 2003; Neff, Hsieh, & Dejitterat, 2005). Self-compassion, self-forgiveness, and self-acceptance may allow for movement away from paralyzing shame and toward an individual's personal valued goals. Overall, current interventions for shame are focused on facing the emotion of shame with openness and acceptance, reorganization the cognitive beliefs individual's hold that elicit shame, and reducing the emotion regulation strategies that enhance shame for the purposes of reducing the emotion pain in the moment and hopefully the impact of shame on depression and other mental health concerns.

In conclusion, this study suggests that shame is a significant emotion to consider in understanding depression and specific mechanisms of avoidance, brooding, and suppression account for some of this relationship. Further exploration of the mechanisms occurring at a state shame level would be useful for implementing treatment.

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