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A Test of Specificity Between Emotion Regulation Repertoires And Affect: A Prospective Investigation

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A TEST OF SPECIFICITY BETWEEN EMOTION REGULATION REPERTOIRES AND AFFECT: A PROSPECTIVE INVESTIGATION

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ABSTRACT

Depression is marked by depressive affects which consists of dysphoric mood reflected by increased levels of negative affect (NA), and anhedonia which is characterized by decreased positive affect (PA). The dysregulation of affective states that characterizes depressive disorders may reflect emotion regulation deficits. Prior work has frequently linked maladaptive responses with depression, while evidence linking adaptive responses and depression has been mixed. Additionally, emerging evidence has shown a degree of specificity between emotion regulation and affect. Therefore, the present study examined whether emotion regulation responses show specificity with NA and PA across 7-day and 12-month periods within a large sample of those with various depression histories. Community dwelling and undergraduate participants (N= 241) completed self-report surveys to assess their trait emotion regulation tendencies, positive and negative affect, and a measure of their depression symptoms in-lab. They then engaged in a 7-day ecological momentary assessment protocol. Lastly, at 4-, 8-, and 12-months post-lab visit, participants completed self-report surveys to assess NA and PA over the past month. As expected, maladaptive responses significantly predicted increased NA and adaptive responses significantly predicted increased PA across all time points. Further, there was specificity as adaptive ER was more strongly linked to trait positive affect and maladaptive ER was more strongly linked to trait negative affect (p's< .001). Results

suggest specific ties across maladaptive responses and negative affect as well as adaptive responses and positive affect, though they may be different across measurement type.

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CHAPTER I

INTRODUCTION

Unipolar depressive disorders represent a common family of syndromes that are characterized by dysphoric and anhedonic mood states along with cognitive and psycho-vegetative symptoms that result in distress and functional impairment (American Psychiatric Association, 2013). Major Depressive Disorder (MDD) is one of the most investigated unipolar depressive disorders, and is hallmarked by the above mentioned mood disturbance that remains for at least two weeks and is accompanied with weight loss or weight gain, change of appetite, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or loss of energy, excessive/inappropriate guilt or feelings of worthlessness, indecisiveness or diminished ability to concentrate or think, and recurrent thoughts of death or suicidal ideation (American Psychiatric Association, 2013).

MDD is one of the most common mental health disorders in the world and the prevalence has risen sharply over recent years. Prevalence estimates suggest that over 10% of the US population suffers from MDD annually, and over 20% will experience the disorder within their lifetimes (Hasin et al., 2018). These estimates mirror those from the World Health Organization (WHO) that point to 322 million people globally suffering

from depression, an estimate that reflects an 18.4% increased prevalence since 2005 (WHO, 2017).

Risk for MDD onset is fairly low until early teens, when it begins to rise in a roughly linear fashion, with an increasingly steep slope in more recent cohorts (Kessler et al., 2003). Once present, MDD shows an episodic course with periods of inter-episode recovery (Spijker et al., 2002). For instance, Spijker and colleagues (2002) observed a 50% 3-month recovery rate that increased to 76% within 1 year, though co-occurring depressive disorders may protract the course of the disorder. However, such inter-episode recovery is often transient, with up-to 79% experiencing three or more relapses (Solomon et al., 2000).

Importantly, MDD is linked to notable personal and societal costs. At the personal level, MDD causes clinically significant distress or impairment in one or various domains of functioning and is associated with numerous adverse outcomes concerning areas such as marriage, parental functioning, employment status, and financial success (Druss et al., 2009; Kessler, 2012). It is also associated with a wide variety of chronic physical disorders including arthritis, asthma, cancer, cardiovascular disease, diabetes, hypertension, and a variety of chronic pain conditions (Kessler, 2012), as well as suicidal ideation, hospitalization, or death by suicide. Nearly 40% of those with a lifetime prevalence of MDD contemplated suicide when their depression was at its worst and 13.6% attempted suicide (Hasin et al. 2018). Suicide was ranked in the top 20 leading causes of death worldwide and was the second leading cause of death among 15-29-year-olds globally in 2015 (WHO, 2017).

At the societal level, MDD creates a large economic burden both in direct costs pertaining to medical services and prescriptions as well as indirect costs that include workplace lost productivity and suicide-related costs. In the US, the incremental economic burden of individuals with MDD increased from $173.2 billion in 2005 to $210.5 billion in 2010, a 21.5% increase. In 2010, $98.9 billion was spent on direct costs of MDD, over half of which was attributed to inpatient and outpatient treatment provision (Greenberg et al., 2015). In fact, Hasin et al. (2018) found that 10% of those diagnosed with MDD reported going to an emergency department and 11.8% reported being hospitalized overnight or longer. Further, suicide-related costs accounted for $9.4 billion in 2005, or 5% of the overall economic burden in the US which is dwarfed in comparison to the $100 billion lost through the reduced productivity and missed worked days attributed to MDD (Greenberg et al., 2015).

1.1 Depression as an Affective Disorder

Contemporary models of depression and depressive disorders are marked by a distress dimension and anhedonia. The Hierarchical Taxonomy of Psychopathology (HiTOP) model has emerged to address the limitations of traditional taxonomies which have failed to explain the dimensional nature of psychopathology, the heterogeneous nature of many disorders, and the high rates of comorbidity between disorders (Kotov et al., 2017). The HiTOP model proposes a hierarchical structure, in which, lies the internalizing spectrum that is composed of syndromes from fear, distress, eating pathology, and sexual problems subfactors. Further, this model proposes that distress underlies depressive disorders and subsumes disorders such as MDD, dysthymic disorder, and generalized anxiety disorder (GAD). Distress in the HiTOP model reflects dysphoria

and anhedonia and is clustered into symptoms including lassitude, suicidality, agitation, retardation, (low) well-being, and irritability (Kotov et al., 2017). These states of dysphoria and anhedonia have been modeled in a precursory model, the Tripartite model.

The Tripartite model was proposed to characterize the traits underlying the heterogeneity in emotional symptoms. Depression is marked by depressive affects which consists of dysphoric mood reflected by increased levels of negative affect (NA), and anhedonia which is characterized by decreased positive affect (PA) (Watson & Clark, 1991). Specifically, NA was put forth as a general factor that explains covariation among depression and anxiety symptoms, and reflects upset, angry, guilty, afraid, sad, scornful, disgusted, and worried emotions. Conversely, PA represents the extent to which a person feels a zest for life and is reflected in expressions of energy, pleasurable engagement, and affective states of delight, interest, enthusiasm, and pride (Clark & Watson, 1991; Watson et al., 1995). In the context of the Tripartite model, reduced PA, and increased physiological hyperarousal (PH) were viewed as specific components of depression and anxiety respectively (Watson & Clark, 1991). This observation represents the heart of the Tripartite model, which provides a framework for understanding co-occurrence of depressive and anxiety disorders in terms of their specific and shared components. The Tripartite model poses a three-factor structure to explain anxiety and depression symptom co-occurrence that is evident in self-report measures across clinical and community samples and across ages (Costa & McCrae, 1992; Gotlib, 1984; Watson et al., 1995; Wolfe et al., 1987).

The Tripartite models of NA and PA have garnered nearly 3 decades of empirical support and has been well-applied in explaining the association between anxiety and

depression cross-sectionally in community youth (Joiner & Lonigan, 2000), undergraduate students (Joiner, 1996; Watson et al., 1995), and older adult samples (Teachman et al., 2007), as well as among those in substance abuse treatment (Watson et al., 1995). Specifically, Watson and colleagues (1995) sought to explore the factor structure of 90 self-report anxiety and depressive symptoms and found support for the three symptom factors: general distress (negative affect), anhedonia (positive affect), and somatic tension and arousal (physiological hyperarousal), in each of their five samples (three student samples, one community-dwelling adult sample, and one psychiatric patient sample). Further, Joiner (1996) utilizing confirmatory factor analysis found that a three-factor structure was supported, and a two-factor hierarchical structure in which the first-order factors of depression and anxiety loaded onto a higher-order general distress factor, was not supported. Additionally, Teachman et al. (2007) examined the structural invariance of the Tripartite model in young, middle-aged, and older adults and found that the 3-factor model best fit each age group, compared to 1- and 2-factor models tested.

Empirical works have shown that NA is strongly related to anxiety and depressive disorder symptoms and syndromes. Both Dyck et al. (1994) and Jolly et al. (1994) examined self-report symptom data from adult psychiatric patients and found that NA was significantly correlated with a broad range of anxiety and depressive symptoms but was not useful for the differentiation of anxiety from depression (Dyck et al., 1994; Jolly et al., 1994; Watson et al., 1988). PA has also been tied to depression as empirical findings indicate that low PA and anhedonia are specific markers of depression (Leventhal et al., 2006). Additionally, PA was shown to consistently and negatively correlate with depression but not anxiety (Dyck et al., 1994; Jolly et al., 1994).

In addition to the cross-sectional data, numerous studies have tied decreased PA to heightened levels of depression longitudinally (Kasch et al., 2002; Khazanov & Ruscio, 2016; Naragon-Gainey et al., 2013). Further support comes from studies showing that initial levels of anhedonia predict increased odds of depression two years later (Wardenaar et al., 2012). Similarly, literature has frequently tied NA to depression longitudinally in both youth and adults (Clark et al., 2003; Joiner & Lonigan, 2000; Lonigan et al., 2003). For example, Naragon-Gainey et al. (2013) administered self-report questionnaires at a baseline, 6-month, and 12-month time frames and found that both NA and PA were predictive of elevated levels of depression at all three time points.

Such associations are mirrored in the daily lives of those with depression, as evidenced by adults with MDD reporting less PA and more NA than healthy controls (aan het Rot et al., 2012; Bylsma & Rottenberg, 2011; Peeters et al., 2003). Barge-Schaapveld et al. (1999) used experience sampling methodology self-reports during daily activities 10 times per day for 6 days and found that depressed individuals had lower PA and elevated NA compared to controls. Additionally, Myin-Germeys et al. (2003) found that individuals in a current episode of depression, reported significantly higher NA and lower PA than the bipolar, non-affective psychosis, and control groups. Similarly, Bylsma and colleagues (2011) utilizing experience-sampling methods evidenced elevated NA for major and minor depressive groups as compared to a healthy control group.

1.2 Emotion Regulation

The dysregulation of affective states that characterize MDD and other depressive disorders may reflect emotion regulation deficits. Emotion regulation (ER) is the process through which individuals modulate their emotions consciously or unconsciously to

appropriately respond to environmental demands, and is linked to outcomes such as mental health, physical health, relationship satisfaction, and work performance (Gross, 1998; Koole, 2009; Rottenberg & Gross, 2003). Many models have been put forward to explain the mechanisms and development of emotion regulation, of which Gross's (1998) process model has received particular attention. The model focuses on the time course of an emotional response unfolding, and takes an information-processing approach to ER processes that are distinguished by the point in the emotion-generative process they modulate: situation selection, situation modification, attentional deployment, cognitive change, and response modulation (Gross, 1998).

Gross (1998) proposes that ER can occur at any stage of the emotion generative process, and there are different types of ER strategies that target each stage. However, there are also higher-order commonalities as the first four emotion regulatory families may be considered antecedent-focused, in that they occur before the response tendency occurs and an ER strategy may be used at this stage to preemptively up- or down-regulate an emotion. This antecedent-focus can be contrasted with response-focused emotion regulation, in which response tendencies occur after the emotion is generated (Gross & Thompson, 2007). Though Gross's model allows for ER to take place before emotion is experienced, most empirical works examine response-focused strategies in relation to depression (e.g., Aldao et al., 2010; Ehring et al., 2008; Joormann & Gotlib, 2010; Joormann & Stanton, 2016; Nolen-Hoeksema et al., 2008; Visted et al., 2018). Antecedent-focused strategies, as previously mentioned, occur prior to the onset of an emotional reaction and as such, measurement may be difficult and could include questions regarding to avoidance. Contrastingly, response-focused strategies attempt to

influence the ongoing emotional expression or intensity regulation after the emotional reaction has been instigated and are more easily measured (Joormann & Stanton, 2016; Visted et al., 2018).

Specific emotion regulation strategies have been argued to be either adaptive or maladaptive based on their long-term effects on affect, behavior, and cognition, as well as on their relationships to psychopathology (Aldao & Nolen-Hoeksema, 2012; Gross, 1998). Responses that effectively attenuate sadness and dysphoria in the short- and long-term have historically been viewed as adaptive, while those that fail to do so or exacerbate distress are maladaptive (Aldao et al., 2010; Aldao & Nolen-Hoeksema, 2012; Gross, 1998; Kovacs et al., 2009). A large empirical body of work links the frequent use of maladaptive responses with elevation in depression levels (Aldao et al., 2010), histories of depression (Joormann & Gotlib, 2010; Joormann & Stanton, 2016; Visted et al., 2018) and risk for new incidents of the disorder (Kovacs et al., 2016). For example, putatively maladaptive responses have been found to produce detrimental outcomes in experimental studies, including rebounds in negative affect following exposure to emotion-eliciting stimuli (Aldao & Nolen-Hoeksema, 2012), and elevation in depression and anxiety levels across community and clinical samples that NA underlies (Aldao et al., 2010). Likewise, those recovered from depression have been shown to deploy maladaptive ER responses at greater frequencies than healthy controls (Ehring et al., 2008; Visted et al., 2018). In turn, the abundant use of maladaptive responses has been shown to prognosticate increased depression severity and risk for new incidents of depressive disorders in longitudinal samples (Kovacs et al., 2009, 2016; Nolen-Hoeksema, 2000). This association between NA and depression is mirrored in daily life

as depressed individuals more frequently employ maladaptive responses that result in subsequent rises in NA (Havermans et al., 2007; Myin-Germeys et al., 2003).

In contrast, evidence connecting adaptive ER responses with depression outcomes has been mixed with some reporting lower use of such responses among depressed and those with depression histories (remitted) relative to healthy controls (Ehring et al., 2008), others failing to show such differences between remitted and control group (Liu & Thompson, 2017; Visted et al., 2018), and others finding no differences between adaptive ER use between depressed, remitted, and healthy controls (Siemer et al., 2007). For example, Liu and Thompson (2017) observed that while depressed participants reported lesser tendencies to engage in cognitive reappraisal, an adaptive ER response, than remitted and healthy controls, those with depression histories were comparable in their tendency to use the response as their healthy peers. In a similar vein, Ehring et al., (2010) showed that remitted individuals were equally effective as healthy controls in using reappraisal. Conversely, Joormann and colleague (2007) found no difference between those actively depressed, remitted, and control participants who refocused their attention following a negative mood induction task.

Such mixed findings have led some to pose that depressed people can implement adaptive ER strategies successfully if instructed in a laboratory setting, but fail to do so spontaneously (e.g., Joormann & Stanton, 2016). Others have proposed that the association between adaptive ER use and depression may in part be methodologically confounded (Yaroslavsky et al., 2020). Specifically, Yaroslavsky and colleagues (2020) posed that depression symptom measures and those used to diagnose depressive disorders obscure the unique ties between adaptive ER deficits and PA, for which emerging

evidence shows a degree of specificity (Brans et al., 2013; Brockman et al., 2017; Yaroslavsky et al., 2020). For instance, adaptive ER use predicted increased PA within adult samples (Brans et al., 2013; Brockman et al., 2017) and those with borderline personality disorder (BPD) (Chaudhury et al., 2017), but was to a lesser extent or unrelated with NA attenuation (Brockman et al., 2017; Chaudhury et al., 2017). As depressed mood, an index of NA, is more prevalent in depression than anhedonia, a marker of low PA, (Baji et al., 2009; Smith et al., 2008), it may be that depression symptom measures are more reflective of NA than PA.

CHAPTER II

THE CURRENT STUDY

While a literature ties maladaptive ER with depression, the findings on the role of adaptive ER in depression risk is mixed, due perhaps in part to differential links between the two ER repertoires and depression symptoms domains. Examining adaptive emotion regulation's ties with depressive affects (positive and negative affect) may therefore shed light on the mixed findings, as anhedonia, which manifests in low positive affect levels, is less prevalent in depression than sadness, which hallmarks negative affect. This study tested whether ER responses show specificity with negative affect and positive affect concurrently within a laboratory setting, and across 7-day and 12-month periods within a large sample of those with various depression histories.

2.1 Validity Check

Based on the tripartite model, elevated negative and low positive affect are expected to reflect depressive affects in this study. Therefore, a preliminary step before hypothesis testing involved assessing the validity of both affects to characterize depression. This step involved examining the relationship between depression symptoms ascertained in-lab and positive and negative affect was examined prior to examining the constructs of interest. Depression symptom severity, acquired from a common depressive measure, was

expected to predict elevated negative affect, and decreased positive affect across its three measurement periods (contemporaneously, across 7 days, and across 12 months).

2.2 Hypotheses

Hypothesis 1. Trait maladaptive ER will predict elevated negative affect and decreased positive affect. Further, trait maladaptive ER will predict significantly greater elevation in trait negative affect relative to trait positive affect. Contrastingly, trait adaptive ER will predict elevated positive affect and decreased negative affect, and further, trait adaptive ER will predict significantly greater elevation in trait positive affect relative to trait negative affect (see Figure 1).

Hypothesis 2. Maladaptive ER will predict elevated negative affect and decreased positive affect across the EMA measurement period. Further, maladaptive ER use will predict significantly greater elevation in negative relative to positive affect across the EMA measurement period. Conversely, adaptive ER will predict the opposite pattern, elevations in positive affect and reductions in negative affect across the EMA measurement period. Further, adaptive ER use will predict significantly elevated positive affect relative to negative affect across the EMA measurement period (see Figure 1).

Hypothesis 3. Maladaptive ER will predict elevated negative affect and decreased positive affect across the long-term follow-up period. Further, maladaptive ER use will predict significantly greater elevation in negative relative to positive affect across the long-term follow-up period. A reverse pattern will be observed for adaptive ER, as it will predict significantly elevated positive affect and decreased negative affect across the long-term follow-up period. Further, adaptive ER will predict significantly greater positive relative to negative affect across the long-term follow-up period (see Figure 1).

CHAPTER III

METHODS

3.1 Participants

Participants were 87 community dwelling and 154 undergraduate participants between the ages of 17–63 (70% female, M_{age}= 24.15, *SD*= 9.89), of whom, a majority were Caucasian (66.7%). Participants were recruited by posting flyers in the community, ads on online bulletins, and utilizing the Undergraduate Psychology Research Pool at Cleveland State University. This sample size was based on power analyses described below. Community dwelling participants were paid to compensate them for their time, while undergraduate students received course credit for their participation. Participants also provided their contact information to be eligible as they were contacted for EMA surveys the 7-days following lab protocol as well as long-term follow up 4-, 8-, and 12-months post lab visit.

3.2 Measures

3.2.1 General measures.

Demographics. Demographic information included participants' age, biological sex, and race among other sociodemographic variables of interest.

Trait Emotion Regulation Tendencies. The Feelings and Me (FAM) is a 54-item questionnaire that measures the use of adaptive and maladaptive cognitive, behavioral, and social regulation strategies that individuals typically use in response to dysphoria. Individuals rate along a 3-point scale ranging from 0 (*not true of me*) to 2 (*many times true of me*) the degree that items describe them. Items that represent adaptive responses (i.e., "when I am sad, I look for a friend or other adult to talk to") and maladaptive responses (i.e., "when I am sad, I take pills, or drugs, or drink alcohol") are summed into two subscales. Adaptive ER is scored by summing 30 items with a potential score ranging from 0-60 and maladaptive ER is scored by summing 24 items ranging from 0-48. These adaptive and maladaptive subscales display good internal consistency (α = .80–.91) across clinical and non-clinical populations and have been shown to be related to current depression status (Kovacs et al., 2009). Both adaptive and maladaptive indices showed acceptable internal consistency in this study (adaptive α =.89, maladaptive α =.89).

Trait Positive and Negative Affect. The Positive and Negative Affect Schedule (PANAS) is a 20-item questionnaire that measures an individual's positive and negative affect. Individuals rate along a 5-point scale the extent to which they had experienced each mood state in general. The points of the scale were labeled *very slightly or not at all*, *a little, moderately*, *quite a bit*, and *very much*, respectively. The negative affect index was created by summing the negative affect items, and the positive affect index was similarly created by summing the positive affect items. PANAS's validity has been demonstrated in both clinical and non-clinical samples (Crawford & Henry, 2004; Watson et al., 1988). PANAS evidences alpha reliabilities that are all acceptably high,

ranging from .86 to .90 for PA and from .84 to .87 for NA (Watson et al., 1988). This study showed similar levels (positive affect α= .86; negative affect α= .88).

Center of Epidemiological Studies Depression Scale (CES-D). The CES-D is a 20-item self-report scale that is used to assess depression symptoms in community dwelling adults (Radloff, 1977). The CES-D measures aspects of depression including depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, psychomotor disturbance, appetite disturbances, and sleep disturbance via responses such as "My sleep was restless" and "I felt sad" as well as reverse coded items such as "I was happy" and "I felt hopeful about the future" (Radloff, 1977). Items are rated via a 4-point Likert scale indicating how often they experienced said item in the past week, from 0 (*Rarely or None of the Time - Less than 1 day*) to 3 (*Most or All of the Time 5-7 days*) (Radloff, 1977). CES-D is scored by summing the 20 items with a score ranging from 0-60. The CES-D has been shown to be a reliable measure of depression and evidences good internal consistency ($\alpha > .85$) across studies involving adults (Hann et al., 1999; Radloff, 1977). A similar level of internal consistency was reflected within this sample as well (α =.93). Concurrent validity by clinical and self-report criteria, as well as substantial evidence of construct validity have been demonstrated (Radloff, 1977).

3.2.2 Ecological momentary assessment measures.

State Positive and Negative Affect. Positive and Negative affect will be measured via items drawn from the Positive and Negative Affect Schedule. Positive affect is made up of the items: alert, strong, excited, and happy. Negative affect is made up of the items: sad, nervous, upset, distressed, angry, and frustrated (Watson et al., 1988). For feasibility

concerns only ten of the PANAS items were selected for inclusion of EMA measures. The PANAS scales have been employed shortened and elongated versions and has shown stability in moment ratings (Watson et al., 1988). The items in this study were selected as they had high factor loadings ranging from .60-.79 (Crawford & Henry, 2004; Watson & Clark, 1994). The outcomes reflect aggregates of negative affect and positive affect separately. Participants will be asked to rate these feelings on a 5-point Likert scale referring to how they feel at the time of the EMA prompt. PANAS evidences alpha reliabilities that are all acceptably high, ranging from .86 to .90 for PA and from .84 to .87 for NA (Watson et al., 1988). An acceptable level of internal consistency was reflected within this sample (positive affect α: .86; negative affect α: .88).

3.3 Procedure

3.3.1 Experimental protocol (in-lab).

Data used for this study was drawn from a larger study of mood, emotion regulation, and psychophysiology. This larger study was carried out across three phases, in-lab, the 7-day EMA measurement period, and the long-term follow up across 4-, 8-, and 12-months. When participants came into lab they were consented for the in-lab and post-lab protocols and privately completed self-report surveys to assess their trait emotion regulation tendencies (FAM), their trait positive and negative affect (PANAS), and a measure of their depression symptoms (CES-D). Next, participants completed a psychosocial interview, semi-structured clinical interview, and a psychophysiological protocol that was not used in the present study.

3.3.2 Ecological momentary assessment protocol.

After completion of the in-lab portion of the study, participants engaged in a 7-day EMA protocol. Participants received 5 prompts a day for 7 days on their cell phone to follow a link to a survey generally between 9:00 a.m. and 9:00 p.m. on SurveyHub. This schedule was created with participant input and modified as needed if/when the participant was not able to use their phone. Participants were sent a reminder prompt 15 minutes after receiving the original text message within a scheduled sampling period and allowed 30 minutes to answer the survey before the link expires. The EMA prompt led participants to SurveyHub where they reported affect ratings on a 5-point Likert-type scale ("*very slightly/not at all*", "*a little*", "*moderately*", "*quite a bit*" & "*extremely*"). These reports of affectivity reflect the emotions participants were feeling and the strength of these emotions at the time they received the EMA prompt. Positive and negative affect indices at each EMA prompt were used as dependent variables in a two-level mixed models (see Statistical Analysis 3.4 hypothesis 2/3).

3.3.3 Long term follow up protocol (LTFU).

At each of the respective follow ups (4-, 8-, and 12 months) participants were contacted via email and phone call to complete self-report surveys via an online link to SurveyHub to assess their affect (PANAS) over the past month (see Statistical Analysis 3.4 hypothesis 2/3).

3.4 Statistical Analyses

Descriptive statistics and bivariate associations among study constructs were examined using Mplus software v8.1. Biological sex was included as a covariate because of the differences in depression rates and emotion regulatory strategies utilized.

Specifically, the prevalence of major depression is higher in women than men (Albert, 2015) and women are more likely than men to report engaging in most types of emotion regulation including rumination, reappraisal, and distraction (Nolen-Hoeksema, 2012). Additionally, age was included as a covariate because this sample included a 46-year age range and affectivity and depression have been shown to change across development. For example, Charles et al., (2001) found that negative affect decreased with age, while positive affect was stable across young and middle-aged adults but the older group evidenced a slight decrease. Additionally, major depressive disorder risk was significantly greater for groups <65-years old, with the strongest risk among those 45 to 64-years old (Hasin et al., 2005). The Mplus software v8.1 was used to test the three study hypotheses as well as the validity check utilizing CES-D. The CES-D provides cutoff scores (e.g., 16 or greater) that aid in identifying individuals at risk for clinical depression. In this study, the range of scores was 0-53, and 137 participants had a score of <16 while 102 participants had a score >16 (M= 16.23, SD= 12.0) with one participant missing data.

Validity Check: To test the validity of CES-D as a predictor of depressive affects (elevated negative affect and reduced positive affect), both positive and negative affect were entered as dependent variables, where residuals were allowed to covary, and regressed onto CES-D for the cross-sectional, EMA, and long term follow up data.

Hypothesis 1. To test specificity between ER repertoires and both trait affects, negative affect and positive affect were entered as dependent variables in a multivariate regression model where residuals were allowed to covary; both affects were regressed on the two ER indices and covariates (i.e., age and sex). It is expected that maladaptive ER will

positively predict negative affect and negatively predict positive affect, while adaptive ER will positively predict positive affect and negatively predict negative affect. If specificity is present, then the relationship between a given ER index will be expected to be stronger for its affiliated affect. That is, adaptive ER's relationship with positive affect would be expected to be stronger than its association with negative affect and conversely, maladaptive ER's relationship with negative affect would be expected to be stronger than its association with positive affect. Specificity will be evident by a significant difference between regression weights between a given index and its associations with both affects (see Figure 1).

Hypothesis 2-3. The approach to testing these hypotheses mirrors that reported above, with the exception that multilevel modeling was employed to accommodate the nesting of respondent's data within themselves. I employed the TWO-LEVEL procedure in Mplus to disentangle variability tied to a given EMA or Follow-up measurement observation (Level 1) from its more stable, person-level component (Level 2). For hypothesis 1, level 1 data reflected all within-subject EMA observations, while level 1 hypothesis 3 data included all available 4-, 8-, and 12-month data. As with hypothesis 1, negative affect and positive affect were allowed to correlate at each measurement level and were regressed on the predictors and potential covariates. It should be noted that the predictors and potential covariates were grand-mean centered. Specificity will be evident by a significant difference between regression weights between a given index and its associations with both affects (see Figure 1).

Power Analysis. Power calculation was based on effect sizes drawn from a pilot sample wherein effects of maladaptive ER on negative affect and adaptive ER on positive affect

were large in magnitude across self-report, EMA, and the long-term follow-up period (Cohen's d = .79-1.51), while the association between maladaptive ER and positive affect adaptive ER and negative affect were small in magnitude (Cohen's d < .34), with exception for maladaptive ER and positive affect, which were of medium-to-large effect sizes (Cohen's d = .67-.76). Sample size requirements were determined via Monte Carlo simulations in Mplus and reflect the minimum sample size requirements to detect ER specificity. Based on these simulations, it was determined that N = 36 was sufficient to test the first hypothesis, N=280 for the second, and N=365 to test the third hypothesis. To accommodate feasibility concerns, the target sample size was reduced to N=241, which reduced the second and third Hypothesis' Power to .79 and .65, respectively.

CHAPTER IV

RESULTS

4.1 Validity Check

In a series of multivariate regressions, negative affect and positive affect were regressed onto CES-D scores for the cross-sectional, EMA, and longitudinal data; residuals were allowed to covary in all models. In accordance with expectation, CES-D significantly predicted depressive affects as evidenced by elevated negative affect and reduced positive affect across all time points (see Table 2).

4.2 Descriptive Statistics/Validity Check

Pearson correlations were performed to examine the relationship between all self-reported variables (see Table 1). Age was positively associated with sex and negatively with maladaptive emotion regulation. Biological sex was negatively correlated with maladaptive emotion regulation and negative affect (long term follow-up), while being positively related to positive affect (across all time points). Additionally, maladaptive emotion regulation evidenced a significant negative association with adaptive emotion regulation and positive affect across all time points, and contrastingly, having a positive correlation with negative affect across all time points. Similarly, adaptive emotion

regulation was negatively related to negative affect across all time points, and positively related to positive affect across all time points (see Table 1).

4.3 Hypothesis Testing

4.3.1 Hypothesis 1:

To test specificity between emotion regulation repertoires and both trait affects, negative affect and positive affect were entered as dependent variables in a multivariate regression in which the two emotion regulation indices were predictors and demographic characteristic covariates (see Table 3).

In support of hypothesis 1, there was a robust association between both emotion regulation indices and trait affects. Adaptive emotion regulation significantly and positively predicted increased positive affect and decreased negative affect. Conversely, maladaptive emotion regulation significantly and positively predicted increased negative affect and decreased positive affect. Additionally, there was specificity as adaptive ER was more strongly linked to trait positive affect relative to negative affect (B_{PA}= .32; B_{NA} = -.14; $\Delta B_{PA\text{-}NA}$= .46, p< .001), and maladaptive ER was more strongly linked to trait negative affect (B_{NA}= .56; B_{PA}= -.33; $\Delta B_{NA\text{-}PA}$= .89, p< .001).

4.3.2 Hypothesis 2:

Multilevel modeling was employed to test specificity in daily life as respondent's data were nested within themselves. To test specificity, negative affect and positive affect were allowed to correlate at each measurement level and EMA time point and were regressed on the aforementioned predictors and potential covariates, the same as hypothesis 1 (see Table 3).

In accordance with expectation, there was a robust association between both emotion regulation indices and their affiliated affect (e.g., adaptive ER with positive affect). Adaptive emotion regulation significantly positively predicted increased positive affect but did not significantly predict negative affect. On the other hand, maladaptive emotion regulation significantly positively predicted increased negative affect but did not significantly predict positive affect. Additionally, there was specificity as adaptive ER was more strongly linked to positive affect in daily life (B_{PA}= .11; B_{NA}= -.03; $\Delta B_{\text{PA-NA}}$= .14, p< .001), and maladaptive ER was more strongly linked to negative affect in daily life (B_{NA}= .19; B_{PA}= -.03; $\Delta B_{\text{NA-PA}}$= .22, p< .001).

4.3.3 Hypothesis 3:

The approach to test this hypothesis mirrors that reported above, negative affect and positive affect were allowed to correlate at each measurement level and were regressed on the predictors and covariates while employing multilevel modeling to accommodate for the nesting of respondent's data (see Table 3).

Consistent with expectations, there was a robust association between both emotion regulation indices and affects. Adaptive emotion regulation significantly positively predicted increased positive affect and decreased negative affect. Conversely, maladaptive emotion regulation significantly positively predicted increased negative affect and decreased positive affect. Additionally, there was specificity as adaptive ER was more strongly linked to positive affect longitudinally (B_{PA}= .33; B_{NA}= -.10; $\Delta B_{\text{NA-PA}}$= .43, p< .001), and maladaptive ER was more strongly linked to negative affect longitudinally (B_{NA}= .52; B_{PA}= -.28; $\Delta B_{\text{NA-PA}}$= .79, p< .001).

CHAPTER V

DISCUSSION

Depression has been shown to be one of the most common mental health disorders in the world and it causes clinically significant distress or impairment in one or various domains of functioning as well as being associated with numerous adverse outcomes (Druss et al., 2009; Kessler, 2012). Depression is marked by depressive affects which consists of dysphoric mood reflected by increased levels of negative affect (NA), and anhedonia which is characterized by decreased positive affect (PA) (Watson & Clark, 1991). The dysregulation of affective states that characterizes depressive disorders may reflect emotion regulation deficits. Prior work has frequently linked maladaptive responses with depression, while the evidence linking adaptive responses and depression has been mixed (Aldao et al., 2010; Ehring et al., 2008; Joormann & Gotlib, 2010; Joormann & Stanton, 2016; Kovacs et al., 2016; Liu & Thompson, 2017; Siemer et al., 2007; Visted et al., 2018). Additionally, emerging evidence has shown a degree of specificity between ER and affect (Brans et al., 2013; Brockman et al., 2017; Yaroslavsky et al., 2020). Due to the mixed findings of adaptive emotion regulation on depression risk, further examination of its ties with affective states are warranted to elucidate these ambivalent findings. Therefore, three hypotheses were examined to test

whether ER responses show specificity with NA and PA across 7-day and 12-month periods within a large sample of those with various depression histories. Specifically, it was hypothesized that there would be specificity between a specific ER index and its corresponding trait affect (hypothesis 1), an ER index and its corresponding affect in daily life (hypothesis 2), and an ER index and its corresponding affect at 4-, 8- and 12-months longitudinally (hypothesis 3).

As a validity check, the relationship between depression levels and positive/negative affect (across all time points) was examined. As expected, CES-D significantly predicted depressive affects (increased negative affect and decreased positive affect) across all time points. This supports prior findings indicating depression includes deficient positive affect (anhedonia) and/or excessive negative affect (sadness and guilt) (Peeters et al., 2003; Rottenberg & Gross, 2007). These results align well with the tripartite model indicating depressive affects, increased negative affect (general distress) and decreased positive affect (anhedonia), being tied to depression. Additionally, the results of this validity type are expected as negative affect (distress) appears to underlie depressive disorders in the distress spectrum in the Hierarchical Taxonomy of Psychopathology (HiTOP). Further, these results highlight depression's association with negative affect and positive affect across various time points as it was examined in-lab, EMA, and long term-follow-up.

Independent of demographics, maladaptive emotion regulation significantly predicted negative affect across all time points. This is consistent with literature in which maladaptive responses have been found to produce detrimental outcomes in experimental studies, including rebounds in negative affect following exposure to emotion-eliciting

stimuli (Aldao & Nolen-Hoeksema, 2012), elevation in depression and anxiety levels across community and clinical samples that negative affect underlies (Aldao et al., 2010), and elevations in negative affect in daily life (Brans et al., 2013). Additionally, the results of this study further support the pervasive role of negative affect as found in the distress spectrum of the Hierarchical Taxonomy of Psychopathology (HiTOP). Negative affect has been identified as a transdiagnostic characteristic of several psychopathology including depressive and anxiety disorders. As such, progressing our understanding maladaptive emotion regulation's tie with negative affect may elucidate a potential area of therapeutic intervention.

Adaptive emotion regulation significantly predicted positive affect across all time points which is consistent with several literature. Specifically, Erisman & Roemer, (2010) found adaptive strategies increase positive affect experimentally, and similarly, adaptive responses have evidenced increases in positive affect in daily life (Brans et al., 2013; Brockman et al., 2017). The results of this study help elucidate the role of adaptive emotion regulation on depression as it supports the tie between adaptive emotion regulation and positive affect, which is the specific factor of depression in the tripartite model. Previous studies have found that maladaptive emotion regulation is more robustly tied to psychopathology, but it may be that the role of adaptive emotion regulation has been obscured by symptom measures that focus primarily on the distress component (elevated negative affect) rather than anhedonic (low positive affect) characteristics.

Notably, maladaptive emotion regulation significantly predicted positive affect but only for the in-lab and longitudinal time points, not EMA. Similarly, adaptive emotion regulation significantly predicted negative affect across the in-lab and

longitudinal time points but not EMA. These results highlight the discrepancy between retrospective self-report and EMA collection methods, as both adaptive and maladaptive responses were more robustly tied to affect for the retrospective self-reports, and to a lesser extent EMA. This finding adds to prior work showing moderate agreement between retrospective self-report and EMA (Anestis et al., 2010; Solhan et al., 2009), and may reflect biases that are overcome through the use of EMA to examine experiences over time and across settings (Bylsma & Rottenberg, 2011).

In accordance with expectations, results suggest specific ties across maladaptive responses and negative affect as well as adaptive responses and positive affect, though they may be different across measurement type. Across all time points, there was specificity as adaptive emotion regulation was more strongly linked to positive affect than negative affect, and conversely, maladaptive emotion regulation was more strongly linked to negative affect than positive affect. This result is consistent with prior work elucidating specificity in which deployment of adaptive strategies in response to distress predicted increased PA in the daily lives of healthy adults (Brans et al., 2013; Brockman et al., 2017) and patients with borderline personality disorder (Chaudhury et al., 2017), but was less or unrelated with NA downregulation (Brockman et al., 2017; Chaudhury et al., 2017). These findings may help elucidate the mixed findings of adaptive emotion regulation's association with depression, as mentioned earlier, may have been obscured by depression measurements that have focused more on the distress (elevated negative affect) component of depression and less so on the anhedonic (low positive affect) component. These results suggest that influence of a particular affect may be acquired by focusing on its affiliated regulatory strategy. For example, when treating an individual

that is very distressed, it may be more beneficial to focus on the maladaptive regulatory strategies they are using rather than the adaptive strategies they are not. On the other hand, as low positive affect is a specific factor of depression according to the tripartite model, focusing on adaptive regulatory strategies may be useful in elevating the anhedonic state they are in.

5.1 Limitations and Recommendations for Future Research

Results of the present study should be interpreted while considering several limitations. First, the current studies relied on self-reports during the in-lab and long-term follow-up periods to assess the experience and regulation of emotions. Self-reported data are prone to distortion by a number of factors including memory biases and social desirability motivations (Lance & Vandenberg, 2009; Scollon et al., 2003). Second, participants were not from a clinical sample and as such, many did not have a diagnosed depressive disorder. Future research may benefit by recruiting higher concentrations of individuals with clinical levels of depression to ascertain whether it strengthens these relationships in a clinical sample. Lastly, emotion regulation strategies were considered as a sum rather than discretely. Future research may benefit by examining discrete strategies and their relationship with affect. For example, future work may benefit by separating a strategy such as problem solving from a strategy such as acceptance, as they appear to have differences in their ties to psychopathology (Aldao et al., 2010). Aldao and colleagues (2010) noted that problem solving may be more closely associated with psychopathology. Although they are both adaptive strategies, examining them separately rather than in a summed adaptive emotion regulation index may elucidate potential differences in positive and negative affect and how they interact with depression.

Additionally, future research on these associations may benefit from use of physiological measures to provide a more complete picture of how emotion regulation relates to affective states. For example, respiratory sinus arrythmia, an index of the parasympathetic nervous system, has been shown to be a reliable peripheral biomarker of emotion regulation (Beauchaine, 2015).

5.2 Strengths and Clinical Applications

The present study exhibits several strengths. This is the first study as far as we could find, that examined specificity between emotion regulation indices and negative/positive affect. Second, the methodology allowed us to examine affect across in-lab, daily life, and longitudinal reports. Previous research on emotion regulation and affect has relied heavily on cross-sectional designs, but with the inclusion of daily life and longitudinal data we are able to increase the generalizability of these findings and also offer several points of comparison for future research. These findings are clinically applicable as they further support the tripartite model's framework that relates depression and depressive affects (elevated negative affect and reduced positive affect). The tripartite model posits that low positive affect (anhedonia) is a specific factor of depression, and in conjunction with elevated negative affect (distress) is predictive of depression. Additionally, this study ties maladaptive emotion regulation tendencies more strongly to negative affect, and adaptive emotion regulation to positive affect. These results support prior findings which have frequently tied maladaptive responses to psychopathology, some of which fall under the distress spectrum that is marked by elevated negative affect (distress), but also elucidates the efficacy of adaptive emotion

regulation strategies on positive affect which is important as adaptive strategies play a central role in a wide variety of psychotherapeutic approaches.

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APPENDIX A: TABLES

Table 1. *Descriptive Statistics and Correlations Among Demographic, Emotion Regulation, and Affect Measures.*

Variable	*n*	*M (SD)*	1.	2.	3.	4.	5.	6.	7.	8.
1. Age	221	24.09 (9.82)	---	.14*	-.23***	.05	-.01/-.04	.04/.01	.11	.02
2. Sex	221	.30 (.46)		---	-.24***	-.12	-.10/-.17*	.16/.20**	-.04	.14*
3. MER	221	42.18 (9.03)			---	-.18**	.60/.66***	-.46/-.45***	.47***	-.20**
4. AER	221	55.38 (10.48)				---	-.27/-.25***	.47/.52***	-.20**	.37***
5. cNA/lNA	221/616	22.58/22.57 (8.47/8.33)					---	-.46/-.47***	---	---
6. cPA/lPA	221/615	32.14/31.61 (7.96/8.79)						---	---	---
7. eNA	3772	9.70 (4.56)							---	-.21**
8. ePA	3779	9.15 (4.85)								---

Note. Sex (0= female, 1=male); MER= Feelings and Me Summed Maladaptive Emotion Regulation Scales; AER= Feelings and Me Summed Adaptive Emotion Regulation Scales; cNA= Cross-Sectional PANAS Negative Affect Total; cPA= Cross-Sectional PANAS Positive Affect Total; eNA= EMA PANAS Negative Affect; ePA= EMA PANAS Negative Affect; lNA= Long-Term Follow-Up PANAS Negative Affect; lPA= Long Term Follow-Up PANAS Positive Affect. EMA, and Long-Term Follow-Up affects were taken from different data sets, so they were not correlated with each other.
***$p \leq .001$, **$p \leq .01$, * $p \leq .05$

Table 2. *Validity Check of Negative Affect and Positive Affect Regressed onto CES-D*

	DV: Cross-Sectional NA and PA					
		cNA			cPA	
Variables	B	SE	β	B	SE	β
CES-D	.55***	.03	.78	-.40***	.03	-.59
	DV: EMA NA and PA					
		eNA			ePA	
Variables	B	SE	β	B	SE	β
CES-D	.15***	.02	.59	-.08***	.02	-.34
	DV: LTFU NA and PA					
		lNA			lPA	
Variables	B	SE	β	B	SE	β
CES-D	.48***	.03	.79	-.37	.04	-.63

Note. CES-D= Center of Epidemiological Studies Depression Scale; cNA= Cross-Sectional PANAS Negative Affect Total; cPA= Cross-Sectional PANAS Positive Affect Total; eNA= EMA PANAS Negative Affect; ePA= EMA PANAS Negative Affect; lNA= Long-Term Follow-Up PANAS Negative Affect; lPA= Long-Term Follow-Up PANAS Positive Affect.
***$p \leq .001$, **$p \leq .01$, * $p \leq .05$

Table 3. *Cross-Sectional, EMA, and LTFU Negative and Positive Affect Regressed onto Maladaptive and Adaptive Emotion Regulation.*

	DV: Cross-Sectional NA and PA					
	cNA			cPA		
Variables	B	SE	β	B	SE	β
1. Age	.12*	.05	.14	-.07	.04	-.09
2. Sex	-.02	.92	-.001	2.39**	.82	.14
3. MER	.56***	.05	.60	-.33***	.05	-.37
4. AER	-.14***	.04	-.17	.32***	.04	.43
	Residual Correlations					
	r	*SE*				
cNA←→cPA	-.19	.07				
	DV: EMA NA and PA					
	eNA			ePA		
Variables	B	SE	β	B	SE	β
1. Age	.08**	.03	.21	.001	.02	.003
2. Sex	.20	.47	.03	1.12*	.47	.17
3. MER	.19***	.03	.51	-.03	.03	-.10
4. AER	-.03	.02	-.10	.11***	.02	.38
	Residual Correlations					
	r	*SE*				
eNA←→ePA$_{L2}$	-.11	.07				
eNA←→ePA$_{L1}$	-.31	.03				
	DV: LTFU NA and PA					
	lNA			lPA		
Variables	B	SE	β	B	SE	β
1. Age	.09	.05	.13	-.09	.05	-.12
2. Sex	-.78	.87	-.05	3.01**	.87	.20
3. MER	.52***	.05	.65	-.28***	.05	-.35
4. AER	-.10**	.04	-.15	.33***	.04	.49
	Residual Correlations					
	r	*SE*				
lNA←→lPA$_{L2}$	-.17	.10				
lNA←→lPA$_{L1}$	-.29	.06				

Note. Sex (0= female, 1=male); EMA= ecological momentary assessment; LTFU= Long-Term Follow-Up; MER= Feelings and Me Summed Maladaptive Emotion Regulation Scales; AER= Feelings and Me Summed Adaptive Emotion Regulation Scales; cNA= Cross-Sectional PANAS Negative Affect Total; cPA= Cross-Sectional PANAS Positive Affect Total; eNA= EMA PANAS Negative Affect; ePA= EMA PANAS Negative Affect; lNA= Long-Term Follow-Up PANAS Negative Affect; lPA= Long-Term Follow-Up PANAS Positive Affect.

***$p \leq .001$, **$p \leq .01$, * $p \leq .05$

APPENDIX B: FIGURES

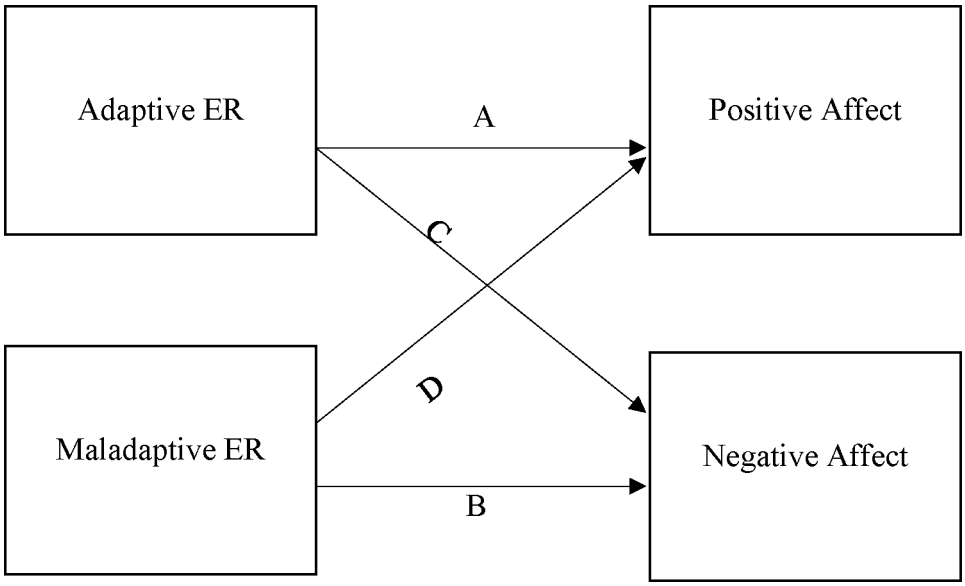

Figure 1. Adaptive ER= Feelings and Me Summed Adaptive Emotion Regulation Scales; Maladaptive ER= Feelings and Me Summed Maladaptive Emotion Regulation Scales; Negative Affect= In-Lab, EMA, or Long-Term Follow-Up PANAS Negative Affect Total; Positive Affect= In-Lab, EMA, or Long-Term Follow-Up PANAS Positive Affect Total. Specificity of Maladaptive ER and negative affect will be evident by a significant difference between B and D. Specificity of Adaptive ER and positive affect will be evident by a significant difference between A and C.