



Fall 2022

Individual Differences and Neural Correlates of Emotion Reactivity and Regulation: Potential Intervention Targets in Depression

Ian James Kahrilas

Follow this and additional works at: https://ecommons.luc.edu/luc_diss



Part of the [Clinical Psychology Commons](#)

Recommended Citation

Kahrilas, Ian James, "Individual Differences and Neural Correlates of Emotion Reactivity and Regulation: Potential Intervention Targets in Depression" (2022). *Dissertations*. 3970.

https://ecommons.luc.edu/luc_diss/3970

This Dissertation is brought to you for free and open access by the Theses and Dissertations at Loyola eCommons. It has been accepted for inclusion in Dissertations by an authorized administrator of Loyola eCommons. For more information, please contact ecommons@luc.edu.



This work is licensed under a [Creative Commons Attribution-Noncommercial-No Derivative Works 3.0 License](#).
Copyright © 2021 Ian James Kahrilas

LOYOLA UNIVERSITY CHICAGO

INDIVIDUAL DIFFERENCES AND NEURAL CORRELATES OF EMOTION REACTIVITY
AND REGULATION: POTENTIAL INTERVENTION TARGETS IN DEPRESSION

A DISSERTATION SUBMITTED TO
THE FACULTY OF THE GRADUATE SCHOOL
IN CANDIDACY FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

PROGRAM IN CLINICAL PSYCHOLOGY

BY

IAN J. KAHRILAS

CHICAGO, IL

DECEMBER 2021

Copyright by Ian J. Kahrilas, 2021
All rights reserved.

ACKNOWLEDGMENTS

I express my sincerest gratitude to my mentor, Dr. Rebecca Siltan, for her time and dedication to my dissertation and training as a scientist and clinical psychologist. I also thank Dr. Colleen Conley, Dr. Fred Bryant, Dr. Robert Morrison, and Dr. Gregory A. Miller for serving on my committee and for sharing their expertise for the benefit of this project. I also thank my wife, Jacqueline, for her constant support throughout my training. Last, but certainly not least, I could not have done this without the love and support of my family and friends.

TABLE OF CONTENTS

ACKNOWLEDGMENTS	iii
LIST OF TABLES	v
LIST OF FIGURES	vi
ABSTRACT	vii
CHAPTER ONE: INTRODUCTION	1
CHAPTER TWO: SAVORING THE MOMENT: A LINK BETWEEN AFFECTIVITY AND DEPRESSION	7
Method	15
Results	18
Discussion	23
CHAPTER THREE: NEURAL CHRONOMETRY OF EMOTION REACTIVITY AND REGULATION	30
Method	37
Results	47
Discussion	56
CHAPTER FOUR: A POTENTIAL NEURAL MECHANISM SUPPORTING THE BRIGHTENING EFFECT IN DEPRESSION	63
Method	77
Results	93
Discussion	99
CHAPTER FIVE: DISCUSSION	109
REFERENCE LIST	115
VITA	142

LIST OF TABLES

Table 1. Descriptive Statistics Among Positive and Negative Affectivity and Savoring for each Depression Severity Group.	19
Table 2. Correlations among affectivity, temporal domains of savoring, depression severity, and anxious arousal measures.	19
Table 3. Unstandardized path coefficients from the structural equation model using positive and negative affectivity to predict depression with savoring subscales as mediators.	23
Table 4. Repeated measures ANOVA results with block as the independent variable for each extracted ERP component from the PCA and behavioral ratings at dependent variables.	48
Table 5. Pairwise t test results for watch conditions for each extracted ERP component as well as arousal and valence behavioral ratings.	55
Table 6. Pairwise t test results for upregulation and downregulation conditions for each extracted ERP component as well as arousal and valence behavioral ratings.	56
Table 7. Summary of polynomial contrasts exploring linear (valence: comparing positive with negative) and quadratic (arousal: comparing positive and negative with neutral) orthogonal univariate trends on the block factor for each component.	94
Table 8. Parameter estimates, standard errors, and test statistics from each structural equation model using ERP components retained from PCA.	95
Table 9. Fit measures and chi-square difference test results for each model.	96

LIST OF FIGURES

Figure 1. Standardized structural diagram of path model with three mediators.	21
Figure 2. Experimental paradigm utilized in the present study.	42
Figure 3. Grand average ERP waveform collapsed across experimental blocks at PO7/PO8 electrode sites and resulting PCA component waveforms.	46
Figure 4. Grand average ERP waveform collapsed across experimental blocks at CPz/Pz electrode sites and PO3/PO4, PO7/PO8, P1/P2, P3/P4, P5/P6, and Pz as well as resulting PCA component waveforms.	47
Figure 5. Statistical findings for pairwise <i>t</i> tests among each block for arousal and valence ratings.	50
Figure 6. Pre- and post-PCA topographical plots collapsed across all blocks and statistical findings for pairwise <i>t</i> tests among each block for each component.	54
Figure 7. Visualization of possible emotion reactivity patterns.	66
Figure 8. Experimental paradigm utilized in the present study.	82
Figure 9. Topographic and ERP plots of the negative 257 ms PCA component before and after temporal PCA.	86
Figure 10. Topographic and ERP plots of the negative 371 ms PCA component before and after temporal PCA.	87
Figure 11. Topographic and ERP plots of the negative 736 ms PCA component before and after temporal PCA.	88
Figure 12. Model diagrams.	90

ABSTRACT

In the present set of studies, we investigated personality traits and neural correlates associated with positive emotion reactivity and regulation within the context of depression. In Study One, we identified potential modifiable psychological mechanisms in ameliorating depression symptoms by exploring of individual differences pertaining to the experience of positive emotion (i.e., positive affectivity and savoring) and how they relate to depression symptomatology. We addressed fundamental questions regarding the neural temporal course of emotion reactivity and regulation and highlighted differences and similarities in the reactivity and regulation across positive and negative emotion in Study Two. This is key information in developing targeted neuroscience-informed interventions for forms of transdiagnostic psychopathology symptoms characterized by dysregulation emotion reactivity and regulation. In Study Three, we investigated relations among the neural temporal course of emotion reactivity and personality traits relevant to the experience of positive emotion in depression. This work identified potential neural mechanisms supporting dysregulated emotion reactivity in depression that may be an intervention target for those with low positive affectivity in depression. Overall, this set of studies contributes to the development of neuroscience-based interventions that bolster the experience of positive emotions for individuals with depression.

CHAPTER ONE

INTRODUCTION

There are an estimated 322 million people in the world living with Major Depressive Disorder (MDD; World Health Organization, 2017), which marks an 18.4% increase from 2005 to 2015 (World Health Organization, 2017). This is compounded by increases in mental illness due to the current global pandemic (Twenge & Joiner, 2020). Depression is associated with decrements in interpersonal relationships, poor work performance and burnout, and reductions in physical wellbeing (Druss et al., 2009; Kessler et al., 2006; Lewinsohn et al., 2003). Given its ubiquity, the economic impact of depression is profound. Depression is associated with higher levels of health care utilization and spending (Donohue & Pincus, 2007). In the United States, loss in work productivity related to depression is estimated to surpass \$36 billion (Kessler et al., 2006). The cost associated with “presenteeism” (i.e., being present at work with depression) is estimated to be \$5524 per person in the United States (Evans-Lacko & Knapp, 2016). The impacts of MDD are far reaching, and more effective treatments will improve quality of life and ease economic burden.

Current treatments for depression are frequently ineffective. Approximately 45-65% of those with depression undergoing CBT do not achieve remission (DeRubeis et al., 2005; Dimidjian et al., 2006). Treatment outcomes for depression suggest that even after completion of therapy, 29% of individuals relapse within one year following treatment and 54% do so within

two years (Vittengl et al., 2007). Depression is currently a highly intractable disorder with a high rate of recurrence and relapse. Mainstream treatments for depression largely focus on mitigating symptoms associated with negative emotions (Argyropoulos & Nutt, 2013; Beck, 1979).

However, patients undergoing treatment typically equally emphasize the importance of bolstering positive emotions as they do reducing negative emotion (Demyttenaere et al., 2015). Other research further illustrates unique relations among positive emotion and depression symptoms while adjusting for the effects of negative emotion (Dunn et al., 2019; Kahrilas et al., 2020).

Moving forward, translational research that strives to facilitate development of evidence-based strategies targeting impairments in positive emotion in depression could be critical to improving the low treatment outcome rate in depression.

In order to contribute to informing the development of neuroscience-based interventions that bolster the experience of positive emotions for individuals with depression, I conducted a trio of studies aimed to investigate personality traits and neural correlates associated with positive emotion reactivity and regulation within the context of depression. First, I investigated relations among individual differences pertaining to the experience of positive emotion (i.e., positive affectivity and savoring) and how they relate to depression symptomatology. This initial line of inquiry elucidated possible modifiable psychological mechanisms in treating depression for individuals with depression and low disposition to experience positive emotions. Next, we addressed fundamental questions regarding the neural temporal course of emotion reactivity and regulation. This highlighted differences and similarities in the reactivity and regulation across positive and negative emotion, which is key information in developing targeted neuroscience-

informed interventions for forms of transdiagnostic psychopathology symptoms characterized by dysregulation emotion reactivity and regulation. Finally, informed by findings in the previous two studies, I investigated relations among the neural temporal course of emotion reactivity and personality traits relevant to the experience of positive emotion in depression. This work identified potential neural mechanisms supporting dysregulated emotion reactivity in depression that may be an intervention target for those with low positive affectivity in depression.

Overview of Study One, Savoring the Moment: A Link Between Affectivity and Depression

The first study, “Savoring the moment: A link between affectivity and depression,” which was published in the *International Journal of Wellbeing* (Kahrilas et al., 2020), investigated the relations among affectivity (negative and positive), savoring, and depression. More specifically, it investigated the mediating role of the temporal domains of savoring (anticipating, savoring the moment, reminiscing) in the relation between factors of Clark and Watson’s (1991) tripartite model (Positive Affectivity, PA; and Negative Affectivity, NA) and depression. Participants ($N = 1,618$) completed questionnaires measuring PA, NA, savoring capacity, and depression. A path analysis was conducted with PA and NA as exogenous variables, the three temporal domains of savoring as mediators, and depression the outcome. Worry, sex, and anxious arousal were included as covariates for all paths.

Overall, the findings from this study indicated that PA and NA were associated with depression as well as all three savoring temporal domains. This suggests that all temporal domains of savoring may bolster PA and mitigate NA. Notably, momentary savoring distinctly mediated the relationship between both PA and NA and depression, suggesting that momentary

savoring may reduce depression symptoms in individuals with low PA and high NA. This study underscored the importance of momentary awareness and upregulation of positive emotions in managing depression. Increased attention to positive stimuli is likely one mechanism through which savoring influences depression symptoms (Carl et al., 2013). While there are distinctions between savoring and mindfulness (Bryant & Veroff, 2007), the importance of moment-to-moment awareness in treating depression is corroborated by the efficacy of mindfulness-based interventions (Gu et al., 2015). Study One supports the importance of enhancing momentary awareness in treating depression in those with low PA, but its use of cross-sectional data precluded causal inference. Further, it did not disentangle the neural time course of positive emotion reactivity and regulation and how these concepts related to individual differences (i.e., PA and savoring capacity), which is crucial to devise neuroscience-informed interventions for those with anhedonic depression. Studies Two and Three were conducted to address these gaps.

Overview of Study Two, Neural Chronometry of Emotion Reactivity and Regulation

To extend findings from Study One, Study Two addressed fundamental questions regarding the neural time course of emotion reactivity and regulation. This study implemented a temporal principal components approach (PCA) to investigate the temporal course of enhancement and attenuation of neural response to positive and negative valenced affective images in a non-selected sample ($N = 54$). Certain ERP components are well established in emotion regulation literature (i.e., LPP), while other earlier visual components (i.e., N170 & EPN) are not. I examined these earlier ERP components with a PCA approach in order to provide a more holistic view on potentially critical windows of neural processing involved in emotion reactivity and regulation,

with the ultimate goal of eventually identifying modifiable neural mechanisms in order to ameliorate depression symptoms. The questions of how emotion reactivity relates to depression symptoms remains unanswered, however. This was the focus of Study Three.

Overview of Study Three, A Potential Neural Mechanism Supporting the Brightening Effect in Depression

Building on findings from Study One and Study Two, Study Three aimed to investigate the relations among low PA in depression and the neural time course of emotion reactivity. To this end, participants ($n = 80$) endorsing clinically significant levels of depression were recruited and completed batteries of questionnaires (including measures of depression and PA). Additionally, EEG data were collected using the same experimental paradigm validated in Study Two. Research suggests that the relation between neural correlates of emotion reactivity and depression varies as a function of depression severity (e.g., Demenescu et al., 2011; Weinberg et al., 2017). Thus, the samples from Study Two (unselected sample) and Study Three (recruited on the basis of depression symptoms) were harmonized, resulting in a sample with greater variability in depression symptoms. This study sought to compare different models of emotion reactivity in depression by using structural equation modeling. Relations among depression symptoms and emotion reactivity were analyzed to determine how neural correlates of emotion reactivity vary depending on depression severity. I hypothesized that positive and negative emotion reactivity across ERP components derived from a temporal PCA would be associated with PA. More specifically, reduced levels of PA would be associated with blunted ERP activity in a manner congruent with the ECI view. Further, we tested whether this model would outperform other competing models of emotion

reactivity as informed by a previous study (Hill et al., 2019). This study aims to advance knowledge regarding impairments in emotion reactivity for individuals with depression as well as inform potential intervention targets. This research will serve as a foundational step for future studies focused on investigating positive emotion regulation and the modification of positive emotion regulation in individuals with depression.

CHAPTER TWO

SAVORING THE MOMENT: A LINK BETWEEN AFFECTIVITY AND DEPRESSION

Affective theories of mood and anxiety disorders have posited that low positive affectivity is a specific risk factor for depression (Clark & Watson, 1991; Lewinsohn & Graf, 1973; Watson, Weber, et al., 1995; Watson, Clark, et al., 1995), whereas high negative affectivity may be a more general indicator of distress that is observed across depression and anxiety disorders, as well as other psychopathology types (Watson & Clark, 1984). Positive affectivity reflects a tendency to experience intense and frequent episodes of pleasant moods (Watson, 2009), while negative affectivity refers to a tendency to experience negative moods (Watson & Clark, 1984).

Positive and negative affectivity are theorized to represent stable individual differences indicative of an individual's disposition to experience positive and negative affect (i.e., transient emotional experiences) respectively. To this extent, positive affectivity and negative affectivity are closely associated with highly stable personality traits such as extraversion and neuroticism, correspondingly (Costa & McCrae, 1980; Warr et al., 1983). While certain major life events (e.g., unemployment, disability) are associated with long-term changes in subjective well-being, others (e.g., marriage, widowhood, divorce) only have short-term effects with a tendency to revert to baseline levels of positive affectivity following these major events (Lucas, 2007; Suh et al., 1996; Watson, 2009). With regard to depression, the frequency of positive life events is not fully explanatory of depression symptoms (Needles & Abramson, 1990). Rather, the relationship

between positive events and depression symptomatology is likely modulated by individual differences ranging from cognitive style (Needles & Abramson, 1990) to neurobiological factors (Watson, 2009) that influence the interpretation and/or experience of positive events. Related, research has indicated that emotional inertia (i.e., degree to which one's affective state is predicted by a previous affective state) may be a risk factor for future development of mood disorders (Koval et al., 2012; Kuppens et al., 2010). Research has also concurrently indicated that affective instability is related to depression symptoms (Thompson et al., 2012). Seeking to reconcile these findings, research utilizing experience sampling and controlled laboratory methods found that more resistant negative emotion, specifically, was related to depression while positive emotion continues to fluctuate (Koval et al., 2013). While research has explored the role of negative emotion regulation strategies (e.g., reappraisal, acceptance, problem solving; Aldao et al., 2010) in the context of depression, the same cannot be said about positive emotion regulation (Silton et al., 2020). Characterizing specific positive emotion regulation strategies that mediate the relationship between affectivity and depression could inform treatment and intervention approaches to depression.

Emotion Regulation and Depression

The connection between negative emotion regulation and depression is well established (Gross, 1998; Gross & Muñoz, 1995; Joormann & Gotlib, 2010; Joormann & Vanderlind, 2014; Nolen-Hoeksema et al., 1993). Individuals with depression are more likely to engage in maladaptive regulation strategies such as rumination, expressive suppression, and catastrophizing and less likely to utilize adaptive strategies like reappraisal and self-disclosure compared to

individuals without depression (Garnefski & Kraaij, 2007; Gross & John, 2003; Joormann & Gotlib, 2010). More recently, research has begun to examine the influence of positive emotion regulation on depression symptoms (Feldman et al., 2008; Nelis et al., 2015; Raes et al., 2012; Werner-Seidler et al., 2013). Beck (1979) theorized that dampening positive emotions intensifies and perpetuates depression. Accordingly, greater dampening of positive affect is prospectively associated with increased depression symptoms three and five months later (Raes et al., 2012). Other research has shown that those with depression tend to be apprehensive about experiencing positive affect and thus engage in maladaptive emotion regulation strategies, such as dampening positive affect (Werner-Seidler et al., 2013). Furthermore, Werner-Seidler et al. (2013) showed that depression symptoms are inversely related to strategies implemented to amplify positive affect and depression. In sum, although research is beginning to establish that those with depression tend to engage in maladaptive positive emotion regulation strategies that reduce positive emotion (Carl et al., 2013; Garnefski & Kraaij, 2007; Gross, 2013; Siltan et al., 2020), there has been less focus on identifying strategies that may successfully enhance positive emotion in individuals who are at risk for depression. As such, the present study evaluates the hypothesis that savoring may ameliorate depression symptoms.

Savoring Responses and Savoring Beliefs

Savoring refers to an awareness of positive experiences and the use of positive emotion regulation strategies to enhance and extend positive feelings that are derived from those experiences (Bryant, 1989; Bryant et al., 2011; Bryant & Veroff, 2007; Smith & Bryant, 2017). People initiate savoring responses in reaction to a positive event or affect as a way to maintain,

intensify, or prolong positive experience (Bryant & Veroff, 2007). The original conceptual formulation of savoring (Bryant & Veroff, 2007) is predicated on the theory that people typically engage in savoring responses in reaction to positive events or affect, which people regulate through cognitive or behavioral strategies. Chronically low levels of positive affectivity would be expected to reduce savoring responses, which over time would lower self-evaluations of savoring ability. While savoring, one may anticipate the enjoyments of future positive experiences, focus on ongoing positive experiences as they occur, or reminisce about past positive experiences. Regardless of the temporal focus, savoring processes regulate positive emotions in the present moment.

In contrast to savoring responses, savoring beliefs are self-perceptions of one's capacity to savor (Bryant, 2003). Although related to ways in which people regulate positive feelings in response to positive events, savoring beliefs are dispositional tendencies that are distinct from specific savoring strategies in which people engage. Stronger savoring beliefs are associated with lower levels of depression symptoms (Bryant, 2003; Eisner et al., 2009; Hou et al., 2016; Ramsey & Gentzler, 2014; Smith & Hollinger-Smith, 2015). Examining the correlations between savoring beliefs and depression in two separate samples, Bryant (2003) found a significant negative correlation between savoring the moment and depression in both samples, a significant negative correlation between positive reminiscence and depression in one of the samples, and no correlation between positive anticipation and depression in either sample. In another study, savoring the moment, but not anticipation, was also identified as a unique predictor of lifetime depression symptoms (Carver & Johnson, 2009). These results suggest that the capacity to savor

ongoing positive experiences as they occur may have the strongest relationship with level of depression symptoms. Furthermore, those endorsing higher levels of savoring beliefs report similar experiences as those endorsing high levels of positive affectivity—namely, intense and frequent episodes of positive affect (Bryant & Veroff, 2007; Watson, 2009).

Savoring and Positive Affect

Savoring strategies that amplify positive emotions are associated with greater frequency of positive affect (Gentzler et al., 2013; Quoidbach et al., 2010; Smith et al., 2014). As an example, college students who reminisced for one week using either memorabilia or cognitive imagery reported greater increases in frequency of happy feelings compared to participants in a control condition (Bryant et al., 2005). In addition, a present-focused savoring strategy, mindfully photographing beautiful or meaningful subjects, led to more positive moods compared to photographing neutral subjects (Kurtz, 2015). Research has also found that greater savoring beliefs are associated with higher intensity of positive affect and less negative affect (Bryant, 2003; Smith & Bryant, 2013). Across two separate samples, people who reported stronger savoring beliefs also tended to report higher levels of personality traits associated with increased positive affect, such as intensity of happy feelings, self-esteem, frequent happy feelings, and less frequent unhappy feelings (Bryant, 2003). Using experience sampling methodology, (2012) found that momentary savoring mediated the relationship between daily positive events and momentary happiness, and this effect was stronger for people with higher trait levels of amplifying (i.e., broad types of savoring strategies) and weaker for people with higher trait levels of dampening. Research has also shown that a combination of low capacity to savor the moment and

experiencing less positive events is most strongly associated with lower positive affect and less life satisfaction (Hurley & Kwon, 2013; Jose et al., 2012). Collectively, these studies illustrate that higher savoring capacity results in greater frequency and intensity of positive affect, which is representative of elevated positive affectivity. However, while this research has investigated the relationship between savoring beliefs and trait-like attributes such as intensity and frequency of affect and increased self-esteem, previous research has not explicitly examined the relationship between trait affectivity and savoring beliefs within the context of depression. The present study evaluates savoring as a positive emotion regulation strategy that may modify low positive affectivity and thus reduce depression symptoms.

The Focus of the Present Study

A variety of interventions have been developed that focus on each of the three temporal domains of savoring (i.e., reminiscing, savoring the moment, and anticipating) to boost happiness (Smith et al., 2014). For example, cultivating the ability to imagine future positive events can enhance anticipating (Quoidbach et al., 2009), taking mindful photographs aids in momentary savoring (Kurtz, 2015), and increasing awareness of recent positive events serves to bolster reminiscing (Seligman et al., 2005). Thus, if savoring mediates the relationship between affectivity and depression, it may be an effective and modifiable target for bolstering positive affectivity and reducing depression symptoms.

Common treatments for depression, including cognitive behavioral therapy (CBT) and antidepressant medication (Price & Drevets, 2010), leave considerable room for improvement that may be fulfilled by focusing on enhancing PA. These treatments predominantly focus on

ameliorating distorted thought patterns and neurotransmitter systems pertaining to negative emotions (Argyropoulos & Nutt, 2013; Beck, 1979). Approximately 45-65% of those with depression undergoing CBT do not achieve remission (DeRubeis et al., 2005; Dimidjian et al., 2006). The importance of enhancing PA is corroborated by the fact that patient definitions of depression recovery equally emphasize repair of PA and NA (Demyttenaere et al., 2015). Further, Dunn and colleagues (2019) conducted two separate studies investigating the efficacy of therapeutic and pharmacological approaches to treating depression and mitigating high NA and PA. Results indicated that NA and PA were both uniquely related to depression and that current treatments (CBT, selective serotonin reuptake inhibitors, selective norepinephrine reuptake inhibitors, tricyclic antidepressants, and monoamine oxidase inhibitors) improve NA, but do little to bolster PA (Dunn et al., 2019). This is despite the fact that reductions in PA were more marked than elevated NA in the samples tested, indicating that PA may be a more pressing intervention target (Dunn et al., 2019). Novel treatments have theorized that increased use of dampening appraisals may be a psychological mechanism of reduced PA (Dunn et al., 2018). This implicates positive emotion regulation and savoring, which is underscored by literature illustrating that greater savoring beliefs are correlated with lower levels of depressive symptoms (Bryant, 2003; Eisner et al., 2009; Hou et al., 2016; Ramsey & Gentzler, 2014; Smith & Hollinger-Smith, 2015). A more thorough understanding of the mechanisms of PA and savoring may pave the way for improving depression treatments.

The present study characterizes the relationship between affectivity, savoring beliefs, and depression symptomatology. We evaluated the following hypotheses: 1) replicating past research,

positive affectivity will be negatively associated with depression symptoms and negative affectivity will be positively associated with depression symptoms, 2) positive affectivity will be positively associated with all three temporal domains of savoring and negative affectivity will be negatively associated with all three temporal domains of savoring, 3) savoring the moment will be distinctly associated with depression, and 4) savoring the moment will distinctly mediate the relationship between affectivity and depression. Despite the present study's focus on PA, mitigating NA remains an important factor to address in depression, with research demonstrating that both are independently related to depression symptoms (Dunn et al., 2019). Additionally, given the negative associations that exist between stable negative traits and savoring (e.g., neuroticism; Bryant, 2003), NA was included as an exogenous variable in the exploratory path analysis. Further, including NA facilitates the analysis of PA and NA's unique relationship with depression via the temporal domains of savoring by statistically controlling for the effects of the other. Because anxiety and depression symptoms frequently co-occur (American Psychiatric Association, 2013; Clark, 1989; Mineka et al., 1998), we accounted for the effects of anxiety by including measures of anxious arousal (somatic) and anxious apprehension (worry) symptoms (Sharp et al., 2015) in our analyses. Additionally, research suggests that worry spans temporal domains and is associated with rumination about past events and hopelessness regarding the future (Andrews & Borkovec, 1988; MacLeod & Byrne, 1996) as well as procrastination (Stöber & Joormann, 2001), each of which may overlap with the temporal domains of savoring. Further, since research indicates that females tend to report greater savoring capacity than do males (Bryant & Veroff, 2007), gender was included as a covariate.

The present study utilized a parallel mediation model with cross-sectional data. Some researchers have advocated against this practice since correlational data do not afford causal interpretation (Maxwell et al., 2011). Alternatively, Hayes (2018) advocated for a more relaxed stance: “We should not let the limitations of our data collection efforts constrain the tools we bring to the task of trying to understand what our data might be telling us about the processes we are studying” (p. 18). Many researchers share this sentiment as indicated by their use of mediation analyses with cross-sectional data (Blashill & Vander Wal, 2010; Gaunt & Scott, 2014; Goodin et al., 2009; Kung et al., 2016; Lee et al., 2014; Li et al., 2011; Osborne et al., 2015; Pollack et al., 2012; Rees & Freeman, 2009; Smith et al., 2016; Thai et al., 2016; Thomas & Bowker, 2015; Torres & Taknint, 2015; Webb et al., 2016). Thus, the present study utilized cross-sectional mediation analyses while recognizing its preclusion of causal inference.

Method

Participants

Participants ($N = 2,482$) were recruited from introductory psychology courses. Participants received course credit for completion of an online survey. Case wise deletion was used to omit participants ($n = 864$) who had missing responses to any questionnaires used in the present study. The final sample of 1,618 participants ($n = 375$ males, $n = 1,243$ females) ranged in age from 17 - 40 ($M = 18.99$ years, $SD = 1.33$) and was 70.0% Caucasian, 19.1% Asian, 4.5% Black or African American, 0.9% Native Hawaiian or other Pacific Islander, 0.7% American Indian or Alaskan Native, and 4.9% Biracial; 11.4% reported that they were Hispanic/Latinx and 88.6% were not Hispanic/Latinx. The study was approved by the University’s Institutional

Review Board, and informed consent was provided to all participants prior to beginning the survey.

Questionnaire Measures

Savoring Capacity

We administered the Savoring Beliefs Inventory (Bryant, 2003) to assess participants' savoring capacity. The SBI measures the perception of one's ability to feel pleasure through anticipating positive experiences, savoring positive moments as they occur, and reminiscing about past positive events (Bryant & Veroff, 2007). The SBI was analyzed as three eight-item subscales pertaining to temporal forms of savoring: anticipating (e.g., "Before a good thing happens, I look forward to it in ways that give me pleasure in the present."), savoring the moment (e.g., "I know how to make the most of a good time."), and reminiscing (e.g., "I enjoy looking back on happy times from my past."). Items are rated on a seven-point Likert scale from "1" (strongly disagree) to "7" (strongly agree). The three temporal subscales of the SBI were originally conceived as separate, intercorrelated measures of individuals' characteristic capacities to savor. Test-retest reliability assessments provide cross-cultural evidence for the stability of the SBI subscales (Bryant, 2003; Kawakubo et al., 2019). Prospective research testing predictive validity found that participants' SBI scores predicted ability to anticipate, momentarily savor, or reminisce upon winter break three months later (Bryant, 2003). This empirical evidence collectively supports the conclusion that the three SBI subscales reflect stable traits that manifest themselves in predictable forms of behavioral and emotion experience over time. Bryant (2003) found moderate to high internal consistency across all three subscales. The present study

replicated these findings (Anticipating, $\omega = .87$; Savoring the Moment, $\omega = .85$; and Reminiscing, $\omega = .86$).

Depression Severity

To evaluate depression severity, the nine-item Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) was administered. PHQ-9 items are scored from “0” (not at all) to “3” (nearly every day) and are based on the depression criteria from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Internal consistency of the PHQ-9 in the present study ($\omega = .87$) was consistent with past research (Kroenke et al., 2001).

Affectivity and Anxiety Symptoms

To assess for positive and negative affectivity as well as anxiety, the 39-item Mood and Anxiety Symptoms Questionnaire (Watson, Weber, et al., 1995; Watson, Clark, et al., 1995) was administered. The MASQ was analyzed as three separate subscales: the eight-item negative affectivity (NA) Scale (MASQ-NA8; e.g., “Felt withdrawn from other people”), the 14-item positive affectivity (PA) Scale (MASQ-PA14; e.g., “Felt cheerful”), and the 17-item Anxious Arousal Scale (MASQ-AA; e.g., “Startled easily”). Previous literature has supported these oblique factors in two independent samples of individuals at-risk for depression and anxiety (Kendall et al., 2015; Nitschke et al., 2001). Participants rated their affectivity and anxiety symptoms using a five-point Likert scale from “1” (not at all) to “5” (extremely). Consistent with previous research (Bredemeier et al., 2010), the MASQ scales demonstrated good to excellent internal consistency (MASQ-NA8, $\omega = .80$; MASQ-PA14, $\omega = .94$; and MASQ-AA, $\omega = .88$).

Worry

The 16-item Penn State Worry Questionnaire (Meyer et al., 1990) was used to assess for the trait of worrying. PSWQ items are rated on a five-point Likert scale from “1” (not at all typical) to “5” (very typical). Meyer and colleagues (1990) found a high degree of internal consistency in the PSWQ, and the present study found good consistency ($\omega = .87$).

Results

Depression Severity

To provide clinically relevant descriptive information regarding depression severity in the sample, participants were categorized into five depression severity groups based on established PHQ-9 cutoff scores: minimal (1-4), mild (5-9), moderate (10-14), moderately severe (15-19), and severe (20-27; Kroenke et al., 2001). Group means, standard deviations, and reliability coefficients of primary variables of interest (PA, NA, and savoring the moment) were calculated for each of the depression severity groups (see Table 1). Pairwise t-test comparisons with Holm correction were conducted to assess mean differences between each depression group for PA, NA, and savoring the moment. Analyses indicated significant differences between all groups for all variables except for the difference in mean PA between moderately severe and severe depression groups.

Table 1. Descriptive Statistics Among Positive and Negative Affectivity and Savoring the Moment for each Depression Severity Group.

Depression Group	n	%	Positive Affectivity			Negative Affectivity			Savoring the Moment		
			Mean	SD	ω	Mean	SD	ω	Mean	SD	ω
Minimal	613	37.9	47.9	10.8	.93	14.8	4.2	.70	43.9	7.8	.81
Mild	561	34.7	43.2	10.2	.92	18.4	4.5	.65	40.3	8.3	.83
Moderate	269	16.6	38.9	10.3	.91	23.0	4.2	.50	35.9	8.0	.78
Moderately Severe	125	7.7	34.5	11.3	.93	26.3	4.3	.49	31.5	8.7	.82
Severe	50	3.1	33.0	14.6	.96	29.3	4.8	.55	27.9	9.4	.83

Note. Holm corrected pairwise t-tests indicated significant differences between each depression group for all variables except between moderately severe and severe groups for PA. SD = standard deviation, ω = McDonald's Omega.

Correlations Among Study Variables

Pearson correlations were computed among the primary study variables (see Table 2). As expected, PA was positively associated with all temporal domains of savoring. NA, depression, and anxious arousal were negatively associated with all temporal savoring domains. All correlations were statistically significant.

Table 2. Correlations among affectivity, temporal domains of savoring, depression severity, and anxious arousal measures ($N = 1,618$).

	1.	2.	3.	4.	5.	6.	7.	8.
1. Anticipating								
2. Savoring the Moment	.68							
3. Reminiscing	.75	.72						
4. Positive Affectivity	.45	.59	.46					
5. Negative Affectivity	-.36	-.55	-.39	-.40				
6. Anxious Arousal	-.28	-.31	-.28	-.07	.58			
7. Depression	-.34	-.51	-.36	-.42	.74	.54		
8. Worry	-.12	-.32	-.16	-.20	.46	.35	.46	

Positive Affectivity, Negative Affectivity, and Anxious Arousal as Predictors of Depression

The tripartite model posits that depression and anxiety have specific relations with three affective dimensions: PA, NA, and anxious arousal (Clark et al., 1995; Watson, Clark, et al., 1995). Given the present study's focus on PA and depression, we wanted to confirm the unique relation between each of these dimensions and depression symptoms in the present sample. Per the tripartite model, we anticipated that low levels of PA and high levels of NA would be positively associated with depression symptoms, and that anxious arousal would be unrelated to depression symptoms. Multiple regression analyses were conducted with PA, NA, and anxious arousal as predictors and depressions with no covariates. Diverging from the specific association between PA and depression proposed by Clark and Watson (1991, 1995a, 1995b), results from the test sample indicated that each of the predictors were uniquely associated with depression. NA ($b = .52$, $\beta = .55$, $t(1614) = 25.47$, $p < .001$) was the strongest predictor of depression, followed by anxious arousal ($b = .11$, $\beta = .21$, $t(1614) = 10.38$, $p < .001$), and lastly PA ($b = -.09$, $\beta = -.19$, $t(1614) = -10.55$, $p < .001$).

Mediation Analyses

Path analyses were run in R (R Core Team, 2018) using the lavaan package (Rosseel, 2011) to assess the indirect effects of PA and NA on depression via three temporal domains of savoring capacity while accounting for the variance associated with gender, worry, and anxious arousal (see Figure 1). Exogenous variables (PA and NA) were allowed to correlate with one another, as were the residual variances of each of the temporal domains of savoring.

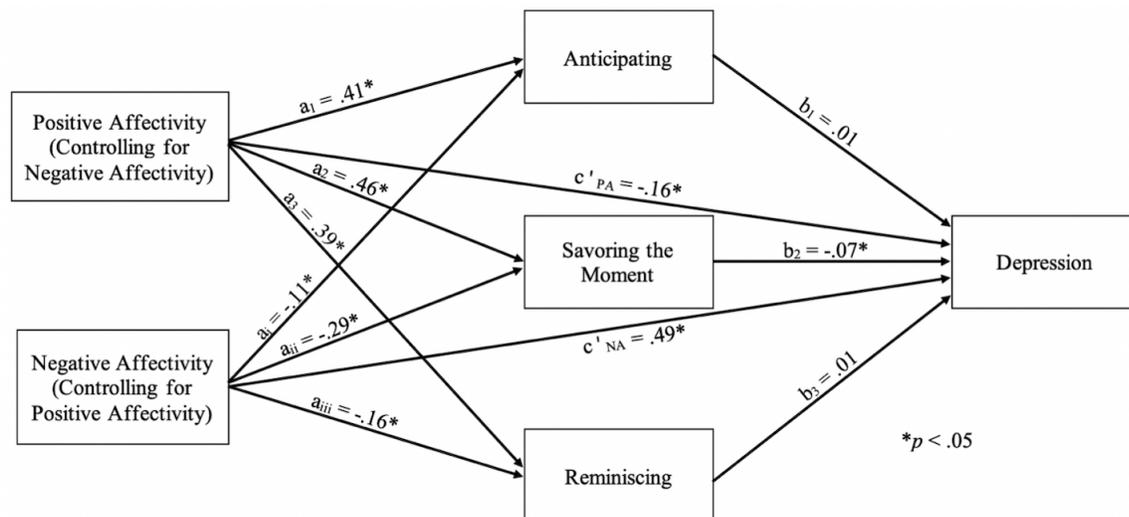


Figure 1. Standardized structural diagram of path model with three mediators: anticipating, savoring the moment, and reminiscing (N = 1,618). The diagram shows A) the effect of positive and negative affectivity on depression severity and B) the direct and indirect pathways associating affectivity with temporal domains of savoring and depression. Sex, worry, and anxious arousal were included as covariates in the model, but were omitted from the figure to streamline presentation. Positive and negative affectivity were allowed to correlate with each other and with the three covariates, which were also allowed to intercorrelate. These intercorrelations were omitted from the path diagram to streamline presentation. The residual variances in the mediators and in depression were also estimated parameters in the structural model. These residual variances were also omitted from the path diagram to streamline presentation.

Maximum likelihood estimation was used to estimate path coefficients, and bias-corrected bootstrap confidence intervals based on 10,000 bootstrap samples were used to estimate indirect effects of temporal domains of savoring between affectivity and depression. The fully saturated model contained 45 free parameters with zero degrees of freedom, resulting in perfect goodness of fit indices. Results (see Figure 1 and Table 3) showed that PA was associated with anticipating (standardized: $a_1 = .41$, unstandardized: $a_1 = .30$, $p < .001$), savoring the moment (standardized: $a_2 = .46$, unstandardized: $a_2 = .36$, $p < .001$), and reminiscing (standardized: $a_3 = .39$, unstandardized: $a_3 = .28$, $p < .001$). There were significant associations between NA

and anticipating (standardized: $a_i = -.11$, unstandardized: $a_i = -.16$, $p < .001$), savoring the moment (standardized: $a_{ii} = -.29$, unstandardized: $a_{ii} = -.44$, $p < .001$), and reminiscing (standardized: $a_{iii} = -.16$, unstandardized: $a_{iii} = -.23$, $p < .001$). Further, savoring the moment was related to lower depression severity (standardized: $b_2 = -.07$, unstandardized: $b_2 = -.04$, $p = .024$), while anticipating and reminiscing were not. Bias-corrected bootstrap confidence intervals (based on 10,000 bootstrap samples; see Table 3) for the indirect effects of temporal domains of savoring between PA and depression did not include zero for savoring the moment (95% CI [-.028, -.002], $a_2b_2 = -.01$) but did include zero for anticipating (95% CI [-.009, .012], $a_1b_1 = .00$) and reminiscing (95% CI [-.009, .013], $a_3b_3 = .00$). Similarly, bias-corrected bootstrap confidence intervals for the indirect effects of temporal domains of savoring between NA and depression did not include zero for savoring the moment (95% CI [.003, .035], $a_{ii}b_2 = .02$) but did include zero for anticipating (95% CI [-.007, .005], $a_i b_1 = .00$) and reminiscing (95% CI [-.011, .007], $a_{iii}b_3 = .00$). Notably, there is a negative indirect effect between PA and depression via savoring the moment with a positive relationship between PA and savoring the moment. This signifies that the positive association between PA and momentary savoring results in reduced depression scores that accounts for a portion of the significant negative relationship between PA and depression (standardized: $c'_{PA} = -.16$, unstandardized: $c'_{PA} = -.08$, $p < .001$). The direct effect of NA on depression was also significant (standardized: $c'_{NA} = .49$, unstandardized: $c'_{NA} = .02$, $p < .001$).

Table 3. Unstandardized path coefficients from the structural equation model using positive and negative affectivity to predict depression with savoring subscales as mediators ($N = 1,618$).

Antecedent	$M_1(\text{ANT})$			$M_2(\text{MOM})$			$M_3(\text{REM})$			Y(DEP)			Indirect Effects			
	Coef.	SE	<i>p</i>	Coef.	SE	<i>p</i>	Coef.	SE	<i>p</i>	Coef.	SE	<i>p</i>	Coef.	Lower	Upper	
PA	a_1	0.30	0.02	<.001	a_2	0.36	0.02	<.001	a_3	0.28	0.02	<.001	c'_{PA}	-0.08	0.01	<.001
$M_1(\text{ANT})$	—	—	—	—	—	—	—	—	—	—	—	—	b_1	0.00	0.02	0.796
$M_2(\text{MOM})$	—	—	—	—	—	—	—	—	—	—	—	—	b_2	-0.04	0.02	0.024
$M_3(\text{REM})$	—	—	—	—	—	—	—	—	—	—	—	—	b_3	0.01	0.02	0.697
NA	a_i	-0.16	0.05	<.001	a_{ii}	-0.44	0.04	<.001	a_{iii}	-0.23	0.05	<.001	c'_{NA}	0.46	0.02	<.001
$M_1(\text{ANT})$	—	—	—	—	—	—	—	—	—	—	—	—	b_1	0.00	0.02	0.796
$M_2(\text{MOM})$	—	—	—	—	—	—	—	—	—	—	—	—	b_2	-0.04	0.02	0.024
$M_3(\text{REM})$	—	—	—	—	—	—	—	—	—	—	—	—	b_3	0.01	0.02	0.697
Covariates:																
SEX	2.89	0.41	<.001	1.63	0.37	<.001	2.61	0.40	<.001	0.53	0.21	0.012	—	—	—	—
WOR	0.06	0.02	<.006	-0.07	0.02	<.001	0.04	0.02	<.088	0.07	0.01	<.001	—	—	—	—
ANX	-0.18	0.02	<.001	-0.08	0.02	<.001	-0.14	0.02	<.001	0.10	0.01	<.001	—	—	—	—
R^2		0.29			0.48			0.30			0.61					
F		132.29			296.23			138.36			315.26					
p		<.001			<.001			<.001			<.001					

Note. SE = Standard Error, PA = Positive Affectivity, NA = Negative Affectivity, ANT = Anticipating, MOM = Savoring the Moment, REM = Reminiscing, DEP = Depression, WOR = Worry, ANX = Anxious Arousal. The symbols appearing to the left of the direct effects (e.g., a_1) refer to the labeled paths in Figure 1. The symbols to the left of each indirect effect coefficient represent the products of each of the constituent direct effects that compose each indirect effect. For example, symbol a_1b_1 represents the product of the direct effect of PA on ANT (i.e., a_1) and the direct effect of ANT on DEP (i.e., b_1), or $(a_1)(b_1)$. The structural model included three covariates (sex, worry, and anxious arousal) as exogenous independent variables, each of which had direct effects on the three mediators and depression.

Discussion

The present study examined the relationship between affectivity (PA and NA) and depression symptoms via the future-, present-, and past-focused temporal domains of savoring beliefs (i.e., self-reported capacity to savor by anticipating, savor the moment, and savor by reminiscing, respectively). As predicted, results showed that PA and NA were associated with all three temporal domains of savoring. Savoring the moment distinctly mediated the relationship between affectivity and depression, whereas the other temporal domains did not mediate this relationship. Replicating past research, we also found a relationship between affectivity and depression symptoms (Chorpita, 2002; Clark & Watson, 1991; Dunn et al., 2019; Khazanov &

Ruscio, 2016; Watson, Weber, et al., 1995; Watson, Clark, et al., 1995). Of note, we found that the effect size for the bivariate relationship between NA and depression (i.e., 55% shared variance) was roughly three times larger than the effect size for the relationship between PA and depression (i.e., 18% shared variance); but given that NA is theorized to be a common factor shared among depression and anxiety (Watson, Clark, et al., 1995; Watson, Weber, et al., 1995), higher NA may also be increased due to co-occurring anxiety symptoms. These findings are similar to results from a meta-analysis suggesting that low positive emotionality creates an inherent vulnerability to depression, but perhaps to a lesser degree than previously surmised (Khazanov & Ruscio, 2016).

While findings from the present study imply that structured interventions aimed at enhancing any of the three temporal domains of savoring may serve to attenuate levels of NA and bolster levels of PA and well-being, only savoring the moment is likely to reduce depression symptoms. This latter finding is consistent with previous research showing that momentary savoring capacity has a stronger negative association with depressive symptoms than do reminiscing and anticipating (Bryant, 2003; Carver & Johnson, 2009). Together, these findings indicate that developing interventions to promote savoring the moment may be effective in reducing depression.

When partitioned by pre-established depression severity cutoff-scores (Kroenke et al., 2001) the majority of the sample fell into minimal and mild depression groups, with significantly fewer participants in the moderate to severe groups. This result is consistent with a previous study conducted by Kroenke and colleagues (2001) that categorized a sample of 580 adult participants

into groups based on PHQ-9 depression cut-off scores. It is also critical to note that 444 participants (27.5%) of the total present sample likely meet clinical criteria for a depression diagnosis as indicated by scoring 10 or higher on the PHQ-9 (Kroenke et al., 2001). Additionally, pairwise comparisons indicate that as participants increase in depression severity, levels of PA and capacity to savor the moment decrease while NA increases. This supports a dimensional conceptualization of the relationship between depression symptoms and individual difference factors, the present study indicates that a gradual decrease in PA/savoring the moment and increase in NA accompany a rise in depression severity.

The present study expands on previous research that has investigated the relationship between positive emotion regulation and depression by assessing beliefs about positive emotion regulation, specifically savoring, across past, present, and future temporal domains. As such, these findings suggest that savoring the moment, as opposed to reminiscing or anticipating, may be instrumental in bolstering PA and reducing NA to ameliorate depression symptoms. This conclusion is further supported by positive psychological interventions that include training in momentary savoring strategies to reduce depression (Hurley & Kwon, 2013; McMakin et al., 2011; Smith & Hanni, 2019). In particular, a savoring-based intervention that instructed female college students with symptoms of dysphoria to make positive self-attributions for recent positive events was effective in reducing depression symptoms and sustaining positive affect following positive events (McMakin et al., 2011). In another study, participants who received training in the use of strategies for savoring the moment, such as memory building and expressing positive emotions, displayed lower depression and lower negative affect after two weeks compared to a

control group (Hurley & Kwon, 2013). While interventions targeting reminiscing and anticipating may serve to increase PA and decrease NA, patients presenting with depression may require more specific treatment focused on savoring the present moment compared to a non-clinical sample. Supporting this notion, increased attention to positive stimuli is likely one mechanism through which savoring influences depression symptoms (Carl et al., 2013). Although it should be noted that savoring and mindfulness are not analogous (Bryant & Veroff, 2007), the importance of moment-to-moment awareness in treating depression is corroborated by mindfulness-based interventions (Gu et al., 2015).

Limitations

The present study has several limitations. First, the present results are based solely on self-report measures. Although self-report data are often inordinately criticized (Chan, 2009), such measures are not without problems. Self-report methods are prone to exaggeration or underreporting, both of which may be caused by social desirability bias, or a tendency to present oneself favorably (Fisher, 1993). While the PHQ-9 is frequently used to screen for depression and track treatment outcomes in research studies and clinical settings, the present study did not implement clinical interviews to validate the self-report data. Future research in this area might consider using a clinical interview to confirm depression diagnoses. However, the PHQ-9 allows for a dimensional conceptualization of depression symptoms, which is likely to be more consistent with the range of affective and cognitive experiences that co-vary with depression symptoms (Levin et al., 2007).

Second, the present study is limited by its cross-sectional design, which precludes

unequivocal conclusions about cause and effect. To overcome this limitation, future research should aim to extend the present findings using a longitudinal, randomized control trial (RCT) design to evaluate the impact of various savoring interventions designed to specifically target each of the three temporal domains in a sample of participants with low levels of PA and depression. In addition, more research is needed to understand the extent to which mindfulness-based interventions that focus on developing moment-to-moment awareness also indirectly enhance positive emotion regulation and savoring the moment, or whether interventions specifically designed to enhance savoring of positive events in the moment effectively reduce depression. Additionally, all subscales (anticipating, savoring the moment, reminiscing) measure one's capacity to generate positive feelings in the moment while focusing on either past, present, or future positive experiences (Bryant & Veroff, 2007). Studies utilizing experience sampling methods (e.g., Koval et al., 2013) offer stronger conclusions regarding temporal fluctuations of emotion and depression symptoms.

The presence of multicollinearity, or tautological logic, can be argued due to the high correlation between NA and depression in the present sample and the fact that depressed mood (NA) forms a central construct of depression. Other researchers analyzing the association between affectivity (measured with the MASQ) and depression concluded that tautological logic did not compromise their analyses (Dunn et al., 2019). Additionally, the constructs of NA and depression are theoretically distinct despite being related. NA is conceptualized as a general indicator of distress observed across depression, anxiety, and other psychopathology types (Watson & Clark, 1984). It is conceptually disparate from positive emotions; one with high NA

does not necessarily also lack joy, excitement, or enthusiasm, which are hallmarks of depression (Watson & Clark, 1984). Nitschke and colleagues (2001) corroborated this conceptualization via factor analysis, illustrating that the MASQ-NA subscale overlaps with anxiety and depression factors. Other research indicates that the MASQ-PA scale is a superior means of screening for depression compared to the MASQ-NA scale, further suggesting that NA is conceptually distinct from depression (Bredemeier et al., 2010). The PHQ-9, on the other hand, was designed to map onto criteria for major depressive disorder diagnosis in the Diagnostic and Statistical Manual of Mental Disorders (Kroenke & Spitzer, 2002). As such, the PHQ-9 was formulated for use in clinical settings with robust diagnostic sensitivity (Kroenke & Spitzer, 2001, 2002). Nonetheless, further clarification on the overlap between affectivity and depression are grounds for future research.

Finally, a post-hoc power analysis—using the *bmem* package (Zhang, 2014), specifying 1,000 Monte Carlo simulations, each with 1,000 bootstrap samples—indicated that the present sample provided less than optimal statistical power. In particular, our present sample ($N = 1,618$) yielded 60.5% power to detect a significant indirect effect of savoring the moment between PA/NA and depression via bootstrapping. Further analyses indicated that a sample size of 2,700 is required in order to reach 80% power for these indirect effects. This latter finding underscores the fact that researchers need to recruit large sample sizes to detect significant effects in complex mediation models.

Conclusion

This is one of the first studies to examine the degree to which the three temporal domains

of savoring mediate the relationship between affectivity and depression symptoms. Interventions that focus on bolstering any of the temporal domains of savoring may benefit those with low PA or high NA. However, enhancing momentary savoring may be the critical temporal domain to consider adapting as an intervention to ameliorate depression. Overall, our results suggest that interventions targeting positive emotion regulation may have considerable benefit for clinical and non-clinical populations. In advancing this research, RCT methodology should be implemented to confirm causality and benefit among individuals with depression disorders.

CHAPTER THREE

NEURAL CHRONOMETRY OF EMOTION REACTIVITY AND REGULATION

Healthy emotion regulation abilities are integral to wellbeing (Gross, 1998; Gross & Muñoz, 1995; Koole, 2009; Sapolsky, 2007). Emotion regulation refers to psychological processes that influence an initial emotional response (Lewis et al., 2010), including modulation of positive and negative affective states (Koole, 2009). This also includes attenuating upsetting, or enhancing positive aspects, of a situation (Gross, 1998b). Emotion regulation and reactivity are interrelated constructs. An individual's ability to regulate emotions is predicated upon emotion reactivity, or how an individual experiences an initial baseline affective response to a stimulus, which spans feelings, behavioral manifestations, and bodily reactions (Gross & Thompson, 2007). Emotion regulation implies that a change has occurred from baseline reactivity to an affective stimulus. Poor emotion regulation strategies are associated with a myriad of risks for poor health and wellbeing, including worsened social functioning (e.g., avoidance of close relationships, few positive relationships, and disinclination to share emotions with others; Gross, 1998a; Gross, 2002; Koole, 2009), as well as lower self-esteem and life satisfaction (Gross, 1998a; Koole, 2009; Ochsner et al., 2002). Poor emotion regulation is also associated with bullying and victimization among youth (Walcott & Landau, 2004), physical ailments such as hypertension and coronary heart disease (Hinton et al., 2009; Jorgensen et al., 1996), exacerbated cortisol reactivity to stressors (Wirtz et al., 2006), and impaired cognitive functioning (Keenan,

2000; Ochsner et al., 2002). Further, many types of psychopathology such as depression (Bylsma et al., 2008; Joormann & Stanton, 2016), bipolar disorder (Townsend & Altshuler, 2012), and schizophrenia (Strauss et al., 2013) involve dysfunctions in emotion regulation and/or reactivity.

Disentangling emotion reactivity and regulation is complex (Joormann & Stanton, 2016), and these two constructs share considerable temporal overlap in terms of affective chronometry (Davidson, 2018). Electroencephalography (EEG) is a psychophysiological method that can be used to evaluate the chronometry of reactivity and regulation processes. In particular, event-related potentials (ERPs) facilitate assessment of neural responses to affective stimuli with millisecond temporal resolution (Olofsson et al., 2008). The present study used EEG methods to investigate the temporal course of reactivity and regulation of visual affective stimuli, with the broader translational aim of informing innovative development of psychological treatments and interventions designed to support positive health and wellbeing outcomes.

Previous psychophysiological research has typically focused on either the neural correlates of reactivity *or* regulation, within positive *or* negative valence task conditions, which has precluded researchers from identifying a comprehensive picture of the temporal course of reactivity and regulation in the context of both positive and negative valence. Incorporating negative and positive stimuli in emotion reactivity and regulation paradigms facilitates comparison of reactivity and regulation conditions across valenced stimuli. Thus, in the present study, we implemented an experimental paradigm where participants were shown blocks of images of varying valence levels (positive, negative, and neutral) with different regulation instructions (increase/decrease emotional intensity in response to images, or passively watching).

As explained in more detail in the method section, we implemented a principal components analysis (PCA) approach to identifying ERP components (Dien, 2012; Donchin & Heffley, 1978), and previous research on the neural correlates of emotion reactivity and regulation contributed to informing our selection of components. The present study is inherently exploratory given the novel application of PCA of PCA to identify ERP components in the context of our study paradigm. Below, we provide a brief overview of relevant ERP components (ranging from early to late) that have been implicated in emotion reactivity and regulation. We also outline expected patterns of results regarding these components based on extant literature.

Early Visual ERP Components Associated with Affective Processing

The N170 and early posterior negativity (EPN) are early visual EEG components that are modulated by affective stimuli (Schupp et al., 2012). The N170 is a negative peak observed at bilateral occipito-temporal electrode sites, occurring approximately 170 ms following stimulus presentation (Blau et al., 2007). A network of posterior neural structures of the extrastriate visual cortex appear to contribute to the N170 (Haxby et al., 2000). These structures include the occipital/fusiform face areas and the superior temporal sulcus in the lateral temporal cortex (Haxby et al., 2000). However, precise identification of brain structures associated with N170 remains unclear, and it is likely that N170 is the product of concomitant activation across various brain regions that are dynamically recruited contingent upon the task at hand (Calder & Young, 2005; Hoffman & Haxby, 2000).

The N170 has commonly been observed in response to facial stimuli and is thought to reflect rapid structural encoding of faces, with increased N170 amplitude associated with threat-

related and positive facial expressions compared to neutral facial expressions (Blau et al., 2007; daSilva et al., 2016; Stockdale et al., 2020). Some research indicates comparable N170 amplitude for positive and negative facial expressions, while other studies show enhanced N170 amplitude for negative compared to positive facial expressions (for a review, see Hinojosa et al., 2015; Stockdale et al., 2020). Enhanced (i.e., more negative) N170 amplitude has also been observed in response to negatively-valenced complex affective images (i.e., IAPS; Bekhtereva et al., 2015), albeit with a slightly later peak latency compared to face stimuli (160 - 200 ms for face stimuli and 200 - 240 ms for IAPS). Limited, if any research, has studied patterns of N170 activity evoked by positive affective images (beyond facial expressions), and identifying the temporal course of early neural response to both positive and negative affective stimuli is important for advancing treatment and intervention for psychological disorders hallmarked by transdiagnostic dimensions of dysregulated emotion reactivity and regulation. In the present study, we expected enhanced N170-like activity in response to both positive and negative stimuli.

While studies have generally shown that N170 is sensitive to valence rather than arousal, the EPN has been observed in response to emotionally arousing compared to neutral stimuli in the 200 - 300 ms window over bilateral occipital-parietal sites (Olofsson et al., 2008; Schupp et al., 2000; Schupp, Junghöfer, et al., 2003). Potential neural generators of the EPN are located in the occipital cortex and may also include contributions from parietal regions (Wiens et al., 2011). Yoon et al. (2016) found that EPN amplitude in response to happy and fearful faces was augmented (i.e., enhanced negativity) compared to neutral faces in healthy control participants. Individual differences, including psychopathology symptoms, also modulate the EPN. For

example, individuals with anxiety disorders exhibited greater EPN amplitude in response to fearful compared to happy and neutral faces, suggesting that anxiety may specifically enhance response to fearful faces (Yoon et al., 2016).

Other research has observed augmented EPN amplitude in response to pleasant compared to unpleasant complex emotional stimuli, suggesting a “pleasure bias” (Frank & Sabatinelli, 2019). EPN has been observed across a variety of different tasks, inter-stimulus intervals (ISI), and stimulus presentation durations (Olofsson et al., 2008), leading researchers to surmise that EPN reflects relatively automatic processes observed even when processing resources are limited due to rapid presentation rates (Junghöfer et al., 2001; Schupp, Markus, et al., 2003; Schupp, Junghöfer, et al., 2003). This sensitivity to affective stimuli may index rapid affective amygdala processing of emotionally salient information (LeDoux, 1995; Morris et al., 1998) and is also theorized to index selective attention where evaluation of image features is contingent upon affectively arousing perceptual characteristics for additional processing (Dolcos & Cabeza, 2002; Schupp et al., 2004). Thus, highly arousing positive and negative images may elicit augmented EPN amplitude. Given the limited research on explicit emotion regulation and EPN/N170, analyses regarding regulation blocks were conducted in an exploratory manner.

Slow Wave Components Associated with Affective Processing

Research using EEG has implicated the LPP, a sustained positive slow wave observed starting as early as 300 ms following stimuli onset, as an established index of evaluative congruency and arousal (Olofsson et al., 2008). Research has consistently shown that LPP over the centroparietal cortex is augmented in response to unpleasant and pleasant compared to neutral

images (Cuthbert et al., 2000; Dillon et al., 2006; Foti & Hajcak, 2008; Hajcak, Moser, et al., 2006; Hajcak, Dunning, Foti, et al., 2007; Hajcak & Nieuwenhuis, 2006; Keil et al., 2002; Moser et al., 2006; Olofsson et al., 2008; Schupp, Markus, et al., 2003). LPP is most pronounced for images that are rated as highly arousing (Bradley & Lang, 2007). LPP has been theorized to reflect activation of a brain network engaged in processing affective visual stimuli, including subcortical structures, particularly the amygdala (Bradley & Lang, 2007; Cuthbert et al., 2000; Keil et al., 2002; Olofsson et al., 2008; Schupp, Markus, et al., 2003), which is corroborated by considerable input and output connections between the visual cortex and amygdaloid nuclei in primates (Amaral et al., 1992). Increased levels of self-reported arousal and valence in response to stimuli is associated with augmented LPP amplitude (Cuthbert et al., 2000; Hajcak & Nieuwenhuis, 2006; Schupp et al., 2000; Thiruchselvam et al., 2011; Tritt et al., 2016). In sum, the LPP is sensitive to the arousal and valence of emotional stimuli. We accordingly expected augmented LPP in response to both positive and negative images.

Enhanced LPP has been observed when participants described affective images in more negative (compared to neutral) terms (Foti & Hajcak, 2008) and when participants were instructed to evaluate images in affective (compared to non-affective) conditions (Hajcak, Moser, et al., 2006). Other research has shown reduced LPP when participants suppressed emotional intensity in response to viewing highly arousing affective images (e.g., Moser et al., 2006). However, research has not reliably demonstrated that LPP is enhanced when participants are instructed to enhance their emotional intensity in response to negative (Moser et al., 2006) and positive (Kropf et al., 2008) affective stimuli. However, increases in centroparietal and

frontal LPP were observed when participants were asked to savor (i.e., upregulate) positive images compared to passively viewing images (Wilson & MacNamara, 2021). Thus, we expected LPP modulations congruent with the regulatory goals.

Disentangling Overlapping Neural Processes Implicated in Reactivity and Regulation: A Principal Components Approach

Nearly all of the ERP research reviewed above involved submitting windowed averages, or area measures to statistical analyses, which is a common practice in ERP research. However, this method may be inadequate in dealing with the thorny issue of components that overlap temporally (Donchin & Heffley, 1978; Foti et al., 2009), which is of primary consideration in this present study since a primary aim of this study is to investigate the *temporal* courses associated with emotion reactivity and regulation. Indeed, there is speculation that N170 modulation due to emotional expressions may be due to superimposed EPN activity (Rellecke et al., 2013). Similarly, LPP may overlap with P3, a parietally maximal component peaking in the 300-400 ms window that is sensitive to task relevant and motivationally significant stimuli (Polich & Kok, 1995), and even later portions of EPN (Schupp et al., 2004). To address this issue, we used an exploratory temporal principal components analysis (PCA) to isolate distinct ERP components across time. PCA is a dimension reduction technique that identifies a new set of variables that are linear combinations of the original variables. In the context of ERPs, these new variables represent latent components that may not be apparent by visually inspecting ERP averages. For example, if there were two temporally overlapping ERP components with equal magnitude but opposite directionality (positive and negative), the two components would negate each other and

not be observable via inspection of grand average ERPs. Temporal PCA, on the other hand, would extract these components provided that the experimental paradigm reliably produces them.

The Present Study

The present study was designed to evaluate the temporal course of emotion reactivity and regulation to positive and negative stimuli, using EEG methods. Our experimental paradigm included 120 different standardized complex affective stimuli (40 positive, 40 neutral, and 40 positive) from the Open Affective Standardized Image Set (OASIS; Kurdi et al., 2017). Participants were given directions to increase or decrease (i.e., upregulate or downregulate) emotional intensity in response to positive and negative images, as well as to passively view them. Participants also passively viewed the block of 40 neutral images for a total of seven regulatory blocks. Using an exploratory PCA approach, we examined the evoked neural response to each regulatory block. We expected that highly arousing positive and negative compared to neutral stimuli would be associated with enhanced ERP components retained from the PCA (e.g., approximating N170, EPN, LPP). Related, indicative of an arousal effect, we expected reduced LPP in decrease blocks relative to passive watch conditions, and increased LPP in increase blocks relative to passive watch conditions. Analyses of regulatory effects on ERN/N170 were conducted in an exploratory manner. All data and R code are available on the Open Science Framework (<https://osf.io/p5ba9/>).

Method

Participants

54 participants were recruited from a college campus for the study ($n = 29$ women, $n = 2$

non-binary). Participants ranged in age from 18-29 ($M = 19.84$ years, $SD = 1.95$). The sample was 70.6% white, 17.6% Asian, 2.0% Black or African American, and 7.8% multiracial (2.0% declined to answer); 9.8% reported that they were Hispanic/Latinx and 90.2% were not Hispanic/Latinx. Two participants were not included in subsequent analyses due to missing EEG or questionnaire data. The study was approved by the Institutional Review Board and informed consent was provided to all participants prior to beginning the experiment.

Materials and Procedure

Emotion Task Paradigm

The study used 120 different OASIS images (Kurdi et al., 2017). The OASIS is a collection of 900 high-quality images collected from open-access online sources depicting a range of categories (people, animals, objects, & scenes). The OASIS was normed on arousal ratings collected from a diverse sample recruited through Amazon's Mechanical Turk (Kurdi et al., 2017). However, the International Affective Picture System (IAPS; Lang et al., 2008) was used in a majority of the aforementioned research. The IAPS was developed to provide a standardized stimulus set of complex affective images to facilitate the comparison of results across different studies and enhance replication across research groups (Bradley & Lang, 2007). IAPS images are rated with respect to valence (negative to positive) and arousal (low to high) on a nine-point visual analogue scale. The copyright agreement that accompanies the IAPS mandates that users cannot place any of the images on computer-accessible websites. This poses considerable restraint on increasing reliance on online samples in behavioral research that make data collection from large and diverse samples faster, less costly, and more efficient (Berinsky et

al., 2012; Buhrmester et al., 2011; Kraut et al., 2004; Mason & Suri, 2012; Paolacci et al., 2010). There are also concerns about lack of diversity and inclusion with regard to the initial normative development of the IAPS (Villanueva et al., 2021). We selected the OASIS for the present study given that the updated norming process included a more diverse sample of participants, and the use of the OASIS facilitates comparison of results with research that goes beyond the traditional walls of a research laboratory.

During the emotion task, participants viewed seven blocks of 40 images (280 total trials). Forty of the images were positive, 40 were neutral, and 40 were negative. The mean normative arousal ratings (on a scale of one to seven) from the OASIS data set were 5.71, 4.09, and 2.18 for positive, neutral, and negative pictures, respectively. The mean normative arousal ratings (on a scale of one to seven) were 4.46, 1.99, and 4.52, for positive, neutral, and negative pictures, respectively. Each block represented an experimental regulation condition: increase (positive/negative), decrease (positive/negative), and watch (positive/negative/neutral). During the increase condition, participants were instructed to appraise the stimulus image in a way that will intensify the emotion that they are experiencing. During the decrease condition, participants were asked to reduce the intensity of the emotion that they are experiencing. When prompted to watch the stimuli, participants were asked to simply view the picture as they would naturally without consciously attempting to modify their affective response to it. The passive “watch” blocks served as baseline conditions for LPP component analyses (Hajcak & Nieuwenhuis, 2006). A key reason that a block design was selected for this study was because arousing positive and negative images outnumber the neutral images two-to-one, and the lower-arousing neutral

stimuli may produce a P3 if intermixed with higher-arousing (e.g., positive/negative) stimuli in the same block (Schupp et al., 2000). Further, a block design paradigm may better emulate the natural environment, as stimuli of congruent arousal are typically encountered in clusters rather than isolation (Schupp et al., 2012); however, research comparing intermixed and block designs for LPP and EPN have shown comparable findings between the two approaches (Pastor et al., 2008; Schupp et al., 2012).

At the beginning of the task, participants completed three practice trials that consisted of 10 images that are not used elsewhere during the task (Hajcak & Nieuwenhuis, 2006). Participants were instructed to increase or decrease emotional arousal in response to a mixture of positive and negative images, respectively. Participants were also asked to watch a third practice trial comprised of positive, negative, and neutral images. Stimuli were presented in random order within each block. Prior to each practice trial, participants were provided with instructions on how to regulate (increase/decrease) their affective response to the stimuli, or to simply view the stimuli (Moser et al., 2006). Participants were instructed which regulation strategy to engage in (increase, decrease, or watch) at the start of each experimental block. Once the participant was ready, an OASIS stimulus appeared for 2000 ms (Foti et al., 2009; Hajcak & Olvet, 2008). Following stimulus offset, a blank screen appeared for 500 ms followed by a fixation cross for 1500 ms (\pm 250 ms) for a total ISI of 1750 - 2250 ms (see Figure 2 for a visual depiction of the experimental paradigm used in the present study). This interval is identical to other studies that used the IAPS and investigated LPP (Dunning & Hajcak, 2009; Hajcak et al., 2009). Stimuli within each block were presented in random order. Each block was separated by a break and

presented in pseudo-random order, such that no valence (i.e., positive or negative) nor regulation (i.e., increase, decrease, or watch) conditions were presented consecutively. After each experimental block, valence and arousal ratings were collected using a seven-point likert scale and the same instructions used in Kurdi, Lozano, and Banaji's (2017) norming study of the OASIS stimuli set. Furthermore, task difficulty ratings on a seven-point likert scale were also obtained. Affective visual stimuli elicit increased arousal and valence self-report ratings (Bradley & Lang, 2007; Kurdi et al., 2017; Olofsson et al., 2008) and are sensitive to reappraisal (e.g., Thiruchselvam et al., 2011). Thus, we conducted exploratory analyses with self-report ratings as the dependent variable and block type as the independent variable.

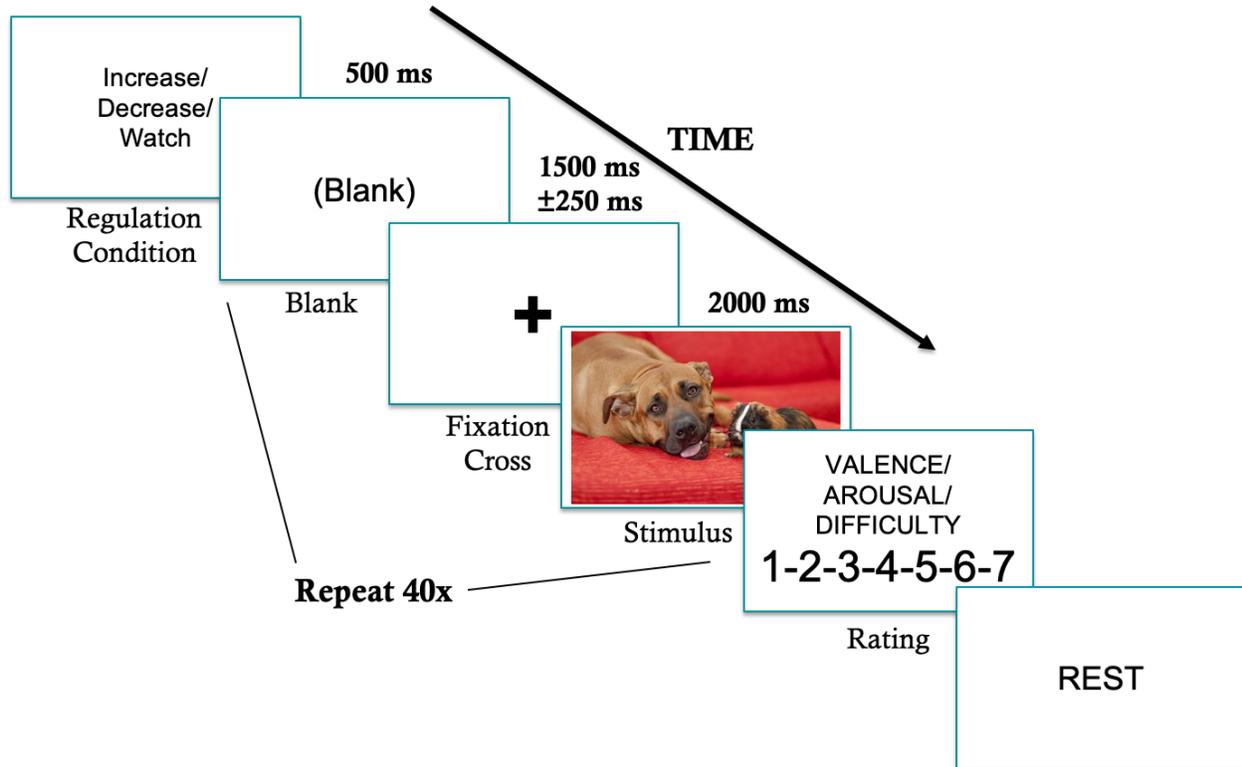


Figure 2. Experimental paradigm utilized in the present study.

Apparatus and Physiological Recording

Scalp electroencephalography (EEG) was measured while participants completed an emotion regulation task (Figure 2). Participants were seated in a comfortable chair, approximately 24 inches from a 24-inch LCD monitor in a quiet, dimly lit room. The viewing distance was approximately 24 in. and occupied 25° of the vertical visual angle and 30° horizontally (Cuthbert et al., 2000; Hajcak & Nieuwenhuis, 2006). The experiment was computer-administered using E-Prime 2.0 software (Schneider et al., 2002) in order to manipulate the timing and presentation of stimuli. All stimuli were presented in color and occupied the entirety of the 21-inch monitor. Participants were monitored by a task administrator in a nearby room and received task

instructions by intercom. EEG data were recorded using a Biosemi Active2 EEG system. A custom-designed Falk Minow 64-channel cap with equidistantly spaced BioSemi active Ag and AgCl electrodes was used for data collection. CMS/DRL was placed near the vertex, and two electrodes were located on the mastoid bones. After placement of the electrode cap, electrode positions were digitized. An additional electrode was placed on the inferior edge of the orbit of each eye to monitor vertical eye movements; nearby electrodes in the cap (lateral to each eye) monitored horizontal eye movements. Data were recorded with a band pass of 0 – 104 Hz at a sampling rate of 512 Hz.

EEG Data Reduction: PCA Approach

The following EEG data processing steps were implemented in Brain Electrical Source Analysis software (BESA, Version 7; Scherg & Berg, 1990). Following the adaptive artifact correction method (Ille et al., 2002), ocular artifacts were corrected using a spatial PCA filter. EEG data were re-referenced to the (Hajcak, Moser, et al., 2006; Hajcak & Nieuwenhuis, 2006) average reference of all electrodes for analyses and digitally filtered with 0.01 Hz high-pass and 30 Hz low-pass filters with a cutoff attenuation of 12 dB/octave. EEG data were re-referenced to the average activity of all electrodes. While it is convention to re-reference data to average activity of the mastoids for LPP analysis, a covariance matrix was utilized in the PCA, which is inherently mean corrected (Dien, 2012). Thus, minimal effects would be expected as a function of referencing scheme for temporal PCA (though this is not the case for spatial PCA; see Dien, 2012). Muscle activity and other artifacts were corrected through implementing an algorithmic scan of continuous EEG data in BESA (fixed threshold criteria: amplitude > 120 μ V; gradient >

75 uV, low signal <.01 uV). Data were baseline-adjusted by subtracting the average activity for 200 milliseconds (Moser et al., 2006) before stimulus onset.

To discriminate among temporal sources of variance across the time course of ERPs, ERP averages were submitted to a temporal PCA to derive linear combinations of data that distinguish activity across timepoints. A temporal PCA was performed using the Psych package (Revelle, 2020) in R version 4.0.3 (R Core Team, 2020) with a covariance matrix. A covariance matrix was used to retain the original units of measurement (i.e., voltage) for the average ERPs for each timepoint (Donchin & Heffley, 1978). The temporal PCA used all timepoints (-200 ms - 2000 ms) as variables and all 55 participants, seven stimuli blocks, and 66 recording sites as observations, yielding linear combinations of timepoints and “temporal” factors (Dien, 2012). To determine the number of components to retain for rotation, a parallel analysis (Horn, 1965) was performed on the resultant Scree plot. The parallel analysis creates Scree plots of the actual and simulated fully random data to determine the number of components that explain more variance than if only chance alone were acting. Based on the parallel analysis, 22 components were retained for rotation. An Promax rotation was then used on the 22 components because it does not impose orthogonality, and simulation studies suggest that Promax is most effective for temporal PCA (Dien, 2010; Dien et al., 2007). The time course of each of the 22 components was inspected by plotting the covariance loadings along each timepoint. Given the a priori nature of the ERP components of interest, those PCA components with covariance loadings in time windows that fit descriptions of LPP and early visual components (e.g., N170, EPN) were retained for further analyses.

Spatial information is retained in PCA; the topography of each factor is encoded in the mean amplitude of factor scores at each recording site (Dien, 1998). Thus, scalp topographies of each component for each condition was inspected to determine correspondence of components and ERP components of interest. Five components were extracted for statistical analyses based on inspection of topographic plots and ERP waveforms. Two of the retained components represented LPP-like components that have positive peaks at 381 ms and 740 ms with the former demonstrating activity at PO7 and PO8 electrode sites and the latter at PO7, PO8, PO3, PO4, P1, P2, P5/P3, P6/P4 and Pz electrode sites. Three components represented early visual components with positive peaks at 124 ms, 162 ms, and 259 ms with activity at PO7 and PO8 electrodes sites. While no a priori hypotheses were made regarding ERP components occurring as early as 124 ms, we decided to retain the component for exploratory analyses. While the time course of the 162 ms component corresponded to N170, the positive directionality of the waveform did not. Two components with temporal loading peaks 259 ms were retained: a positive component noted earlier and a negative component at CPz and Pz electrodes sites, which represents EPN. No frontal LPP component was identified. See Figures 3 and 4 for grand average ERPs before and after PCA (collapsed across all blocks) as well as the top two rows of Figure 5 for grand average topographic plots before and after PCA.

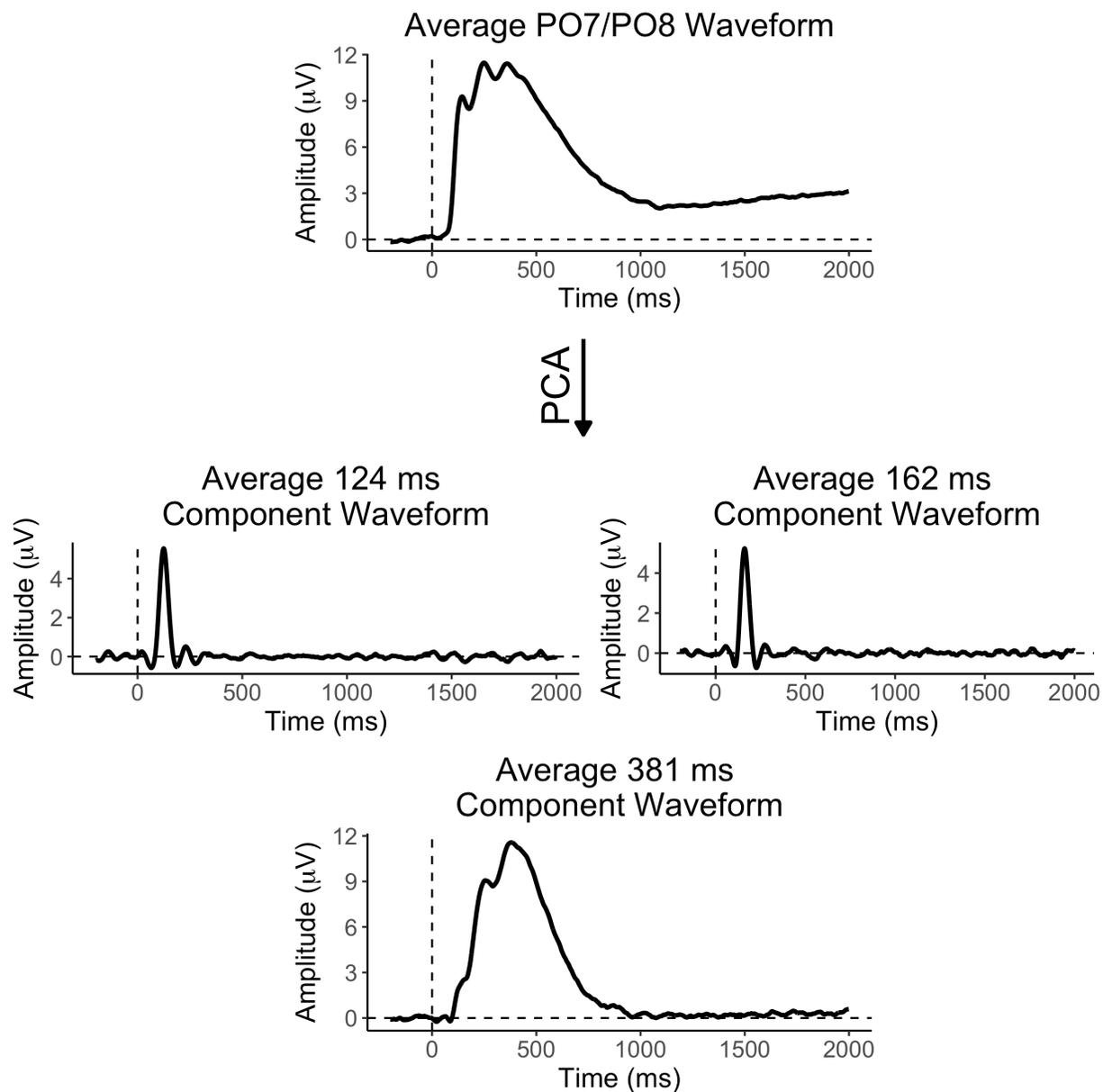


Figure 3. Grand average ERP waveform collapsed across experimental blocks at PO7/PO8 electrode sites and resulting PCA component waveforms.

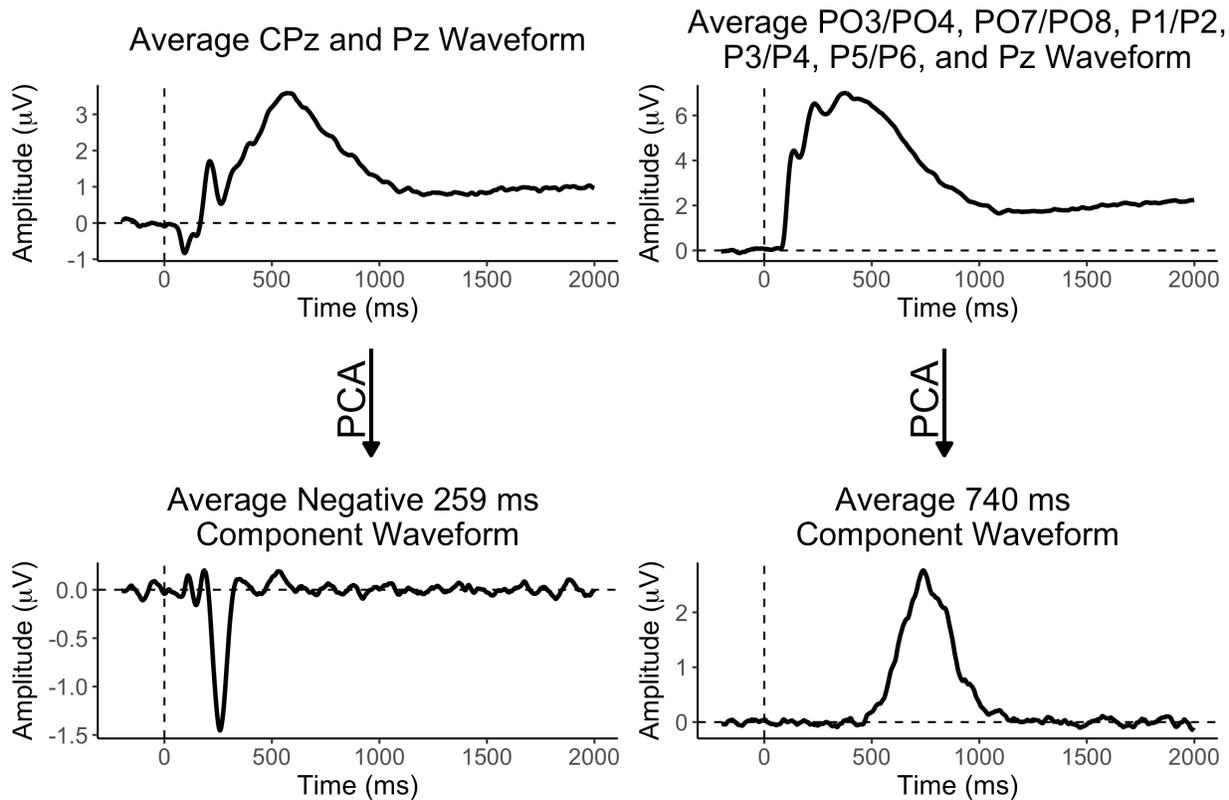


Figure 4. Grand average ERP waveform collapsed across experimental blocks at CPz/Pz electrode sites and PO3/PO4, PO7/PO8, P1/P2, P3/P4, P5/P6, and Pz as well as resulting PCA component waveforms.

Results

Analytic Strategy

Analyses were run in R version 4.0.3 (R Core Team, 2020). Multilevel modeling (MLM) analyses were conducted using the lme4 package (Bates et al., 2015). All linear mixed models were estimated using REML with participants included as random intercepts. Model assumptions were checked via visual inspection of diagnostic plots (i.e., residual, QQ, scale-location, and residuals vs. leverage) generated with the performance package (Lüdtke et al., 2019).

Repeated measures ANOVA was performed using the fitted linear mixed models to probe

for significant deviations from the average activity of each component and behavioral rating as a function of experimental block. Greenhouse-Geisser corrections were implemented to account for significant departures from sphericity as tested by Mauchly's test for sphericity. Findings are summarized in Table 4. Each analysis resulted in a significant test statistic at $\alpha = .05$, signifying that block type accounted for a significant portion of the variance in each of the retained ERP components and behavioral ratings. Follow up pairwise t tests were conducted using the emmeans package (Lenth, 2020) among passive watch and regulation conditions for PCA EEG components and behavioral ratings. Effect sizes are interpreted in accordance with Cohen's guidelines (Cohen, 1988).

Table 4. Repeated Measures ANOVA Results with Block as the Independent Variable for each Extracted ERP Component from the PCA and Behavioral Ratings as Dependent Variables ($n = 54$).

DV	F	df_1^{GG}	df_2^{GG}	MSE	p	$\hat{\eta}_G^2$
124 ms Component	2.45	4.50	229.56	0.34	.040	.008
162 ms Component	10.96	4.70	239.58	0.37	< .001	.043
259 ms Component	20.56	5.15	262.56	0.36	< .001	.120
381 ms Component	35.73	4.48	228.34	0.15	< .001	.084
740 ms Component	28.78	5.33	271.92	0.16	< .001	.165
Valence Ratings	81.55	4.26	225.68	0.86	< .001	.535
Arousal Ratings	26.27	4.89	259.16	1.45	< .001	.228
Difficulty Ratings	25.13	4.81	255.17	1.68	< .001	.222

Note. DV = dependent variable, F = F statistic with Greenhouse-Geisser corrected degrees of freedom, df_1^{GG} = Greenhouse-Geisser corrected numerator degrees of freedom, df_2^{GG} = Greenhouse-Geisser corrected denominator degrees of freedom, MSE = mean square error, p = p value, $\hat{\eta}_G^2$ = partial eta squared as measure of effect size. The above table summarized findings from repeated measures ANOVA with block as the independent variable and each of the ERP components and behavioral ratings as dependent variables (as indicated in the DV column).

Behavioral Ratings

Reactivity Conditions

Regarding behavioral ratings, we observed differences in self-reported arousal and valence ratings among passive watch conditions (positive, negative, neutral). Statistical results for behavioral ratings are summarized in Figure 6, and more detailed statistical findings for each comparison are provided in Tables 5 and 6. Participants rated negative images more negatively than neutral images and positive images and more positively than neutral images. There were significant, medium sized reactivity effects when comparing negative and neutral conditions, as well as small effects between positive and neutral conditions and negative and positive conditions. On average, participants rated negative images as more arousing than positive images and positive images as more arousing than neutral images. Medium sized differences in valence ratings were also observed between negative and neutral conditions, small sized effects for positive and neutral conditions, as well as large differences between positive and negative conditions.

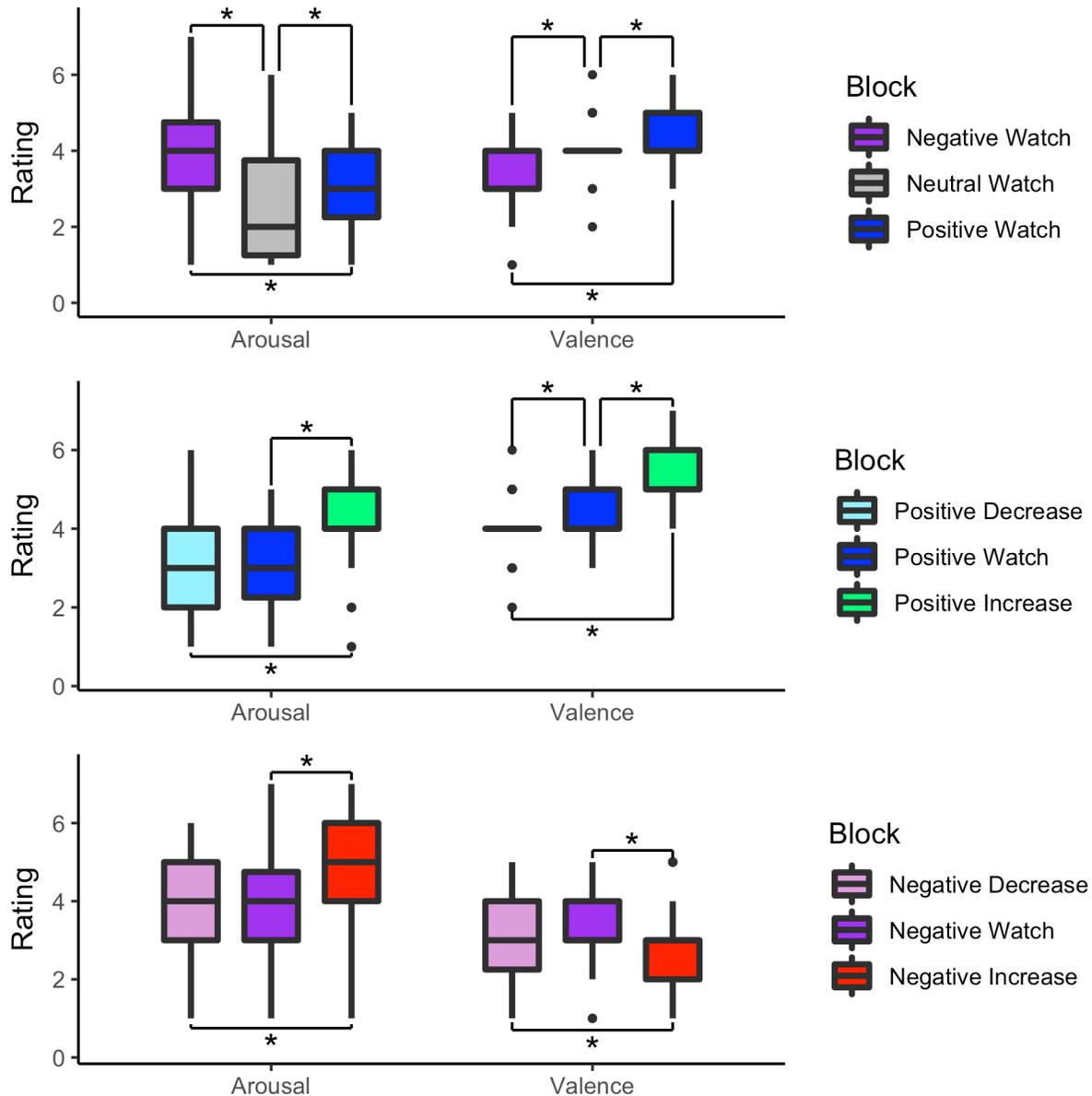


Figure 5. Statistical findings for pairwise t tests among each block for arousal and valence ratings. Differences among blocks are illustrated with box-and-whisker plots. The area between the lower and upper hinges correspond to the interquartile range (the 25th and 75th percentiles). The upper whisker extends from the hinge to the largest value no further than 1.5 times the interquartile range from the hinge. The lower whisker extends from the hinge to the smallest value at most 1.5 times the interquartile range of the hinge. Data beyond the end of the whiskers are plotted individually as dots. Significance at the $\alpha = .05$ is denoted with asterisks.

Regulation Conditions

In terms of regulation conditions, differences among passive viewing and increase (positive, negative) conditions were generally observed. For positive regulation conditions, medium-sized differences in arousal ratings were observed when comparing increase and watch conditions, as well as large differences between increase and decrease conditions. No significant difference was noted between the decrease and watch conditions. Thus, while decrease conditions did not meaningfully deviate from watch conditions for positive stimuli, they did deviate from increase conditions. Medium-sized differences in valence ratings were also noted when comparing increase and watch conditions, as well as small differences between decrease and watch conditions, and large differences between the decrease and increase conditions. In terms of negative regulation conditions, medium-sized differences in arousal ratings were observed when comparing increase and watch conditions as well as increase and decrease conditions. There was no difference between the decrease and watch conditions. Valence rating differences that were medium sized also emerged when comparing increase and watch conditions as well as decrease and increase conditions.

EEG Results

124 – 162 ms

Statistical results for EEG components are summarized in Figure 5. More detailed statistical findings for each comparison are provided in Tables 5 and 6 of the Appendix. Starting at the earliest component extracted from the PCA with a peak at 124 ms, no reactivity or positive regulatory effects were observed. However, a small difference was noted for between the

negative increase and negative watch conditions, with greater amplitudes for the negative increase relative to the negative watch condition. This illustrates a difference in time course among regulation and reactivity starting at early neural processes. Reactivity effects began unfolding at the 162 ms component, with medium and small differences in negative versus neutral and positive versus neutral contrasts, respectively. There was also a small-sized effect between negative and positive images, with negative images eliciting larger 162 ms component amplitude. No regulation effects were observed for the 162 ms component, suggesting that this component may be unique to reactivity effects.

259 ms

Reactivity and regulation effects were noted at the 259 ms component. Medium-sized significant differences were noted between negative and neutral watch conditions for the negative and positive 259 ms components. In addition, there was a small difference between positive and negative images, with negative images eliciting more negative (i.e., enhanced) 259 ms amplitude relative to positive images. Small effects were noted between increase and watch conditions for positive and negative regulatory conditions, with more negative amplitudes in the increase relative to watch conditions. Results suggest that the 259 ms is flexible to task demands (e.g., reactivity or regulation) and also marks the point at which positive emotion regulation effects begin to unfold and negative emotion regulation ends.

381 ms

There were reactivity and positive regulatory effects observed at the 381 ms component. Large- and medium-sized effects were observed for negative and positive stimuli relative to

neutral stimuli, respectively, and negative stimuli elicited greater amplitude than positive images (small-sized effect). There was a small difference between the positive watch and positive increase conditions, with greater 381 ms component amplitude in increase conditions, as well as an additional small difference between increase and decrease conditions.

740 ms

Finally, reactivity effects were observed among positive, neutral, and negative images at the 740 ms peak that ranged from large (negative vs. neutral) to medium (positive vs. neutral, negative vs. positive). No regulation effects were observed except for a small effect when comparing the positive increase and decrease conditions.

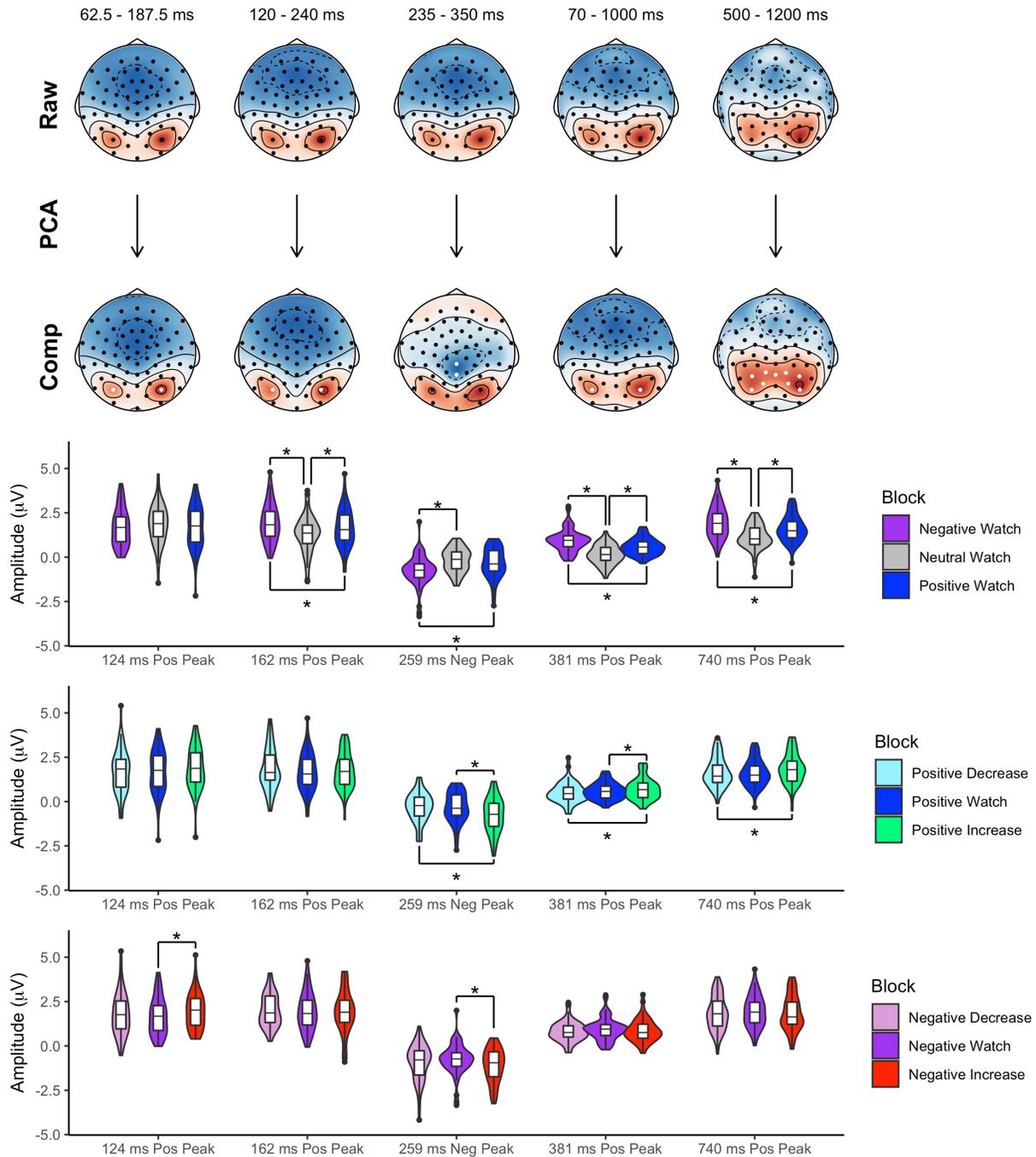


Figure 6. Pre- (top row of topographical plots) and post-PCA (second row of topographical plots) topographical plots collapsed across all blocks and statistical findings for pairwise *t* tests among each block for each component. Pre-PCA topographical plots represent average activity across the time window above each plot. Electrode sites used to measure each component are highlighted in

the post-PCA topographic plots. Differences among blocks are illustrated with box-and-whisker/violin plots below the topographic plots. The width of the violin plots corresponds to the number of cases, or density, with that level of the dependent variable. Regarding the box-and-whisker plots, the area between the lower and upper hinges correspond to the interquartile range (the 25th and 75th percentiles). The upper whisker extends from the hinge to the largest value no further than 1.5 times the interquartile range from the hinge. The lower whisker extends from the hinge to the smallest value at most 1.5 times the interquartile range of the hinge. Data beyond the end of the whiskers are plotted individually as dots. Significance at the $\alpha = .05$ is denoted with asterisks.

Table 5. Pairwise t test Results for Watch Conditions for each Extracted ERP Component as well as Arousal and Valence Behavioral Ratings.

Contrast	Estimate (95% CI)	Std. Beta (Label)	Sig.
124 ms Component			
Negative - Neutral	-0.15 (-0.38, 0.08)	0.13 (Very Small)	.292
Negative - Positive	-0.00 (-0.24, 0.23)	0.00 (Very Small)	.999
Neutral - Positive	0.14 (-0.09, 0.38)	0.13 (Very Small)	.314
162 ms Component			
Negative - Neutral	0.65 (0.40, 0.89)	0.59 (Medium)	<.001
Negative - Positive	0.31 (0.07, 0.56)	0.29 (Small)	.008
Neutral - Positive	-0.33 (-0.58, -0.09)	0.30 (Small)	.005
259 ms Component			
Negative - Neutral	-0.60 (-0.85, -0.34)	0.63 (Medium)	<.001
Negative - Positive	-0.43 (-0.69, -0.17)	0.46 (Small)	<.001
Neutral - Positive	0.16 (-0.09, 0.42)	0.17 (Very Small)	.297
381 ms Component			
Negative - Neutral	0.81 (0.66, 0.97)	0.91 (Large)	<.001
Negative - Positive	0.34 (0.19, 0.50)	0.38 (Small)	<.001
Neutral - Positive	-0.47 (-0.63, -0.32)	0.53 (Medium)	<.001
740 ms Component			
Negative - Neutral	0.82 (0.64, 0.99)	1.29 (Large)	<.001
Negative - Positive	0.37 (0.19, 0.54)	0.58 (Medium)	<.001
Neutral - Positive	-0.45 (-0.62, -0.28)	0.71 (Medium)	<.001
Arousal Ratings			
Negative - Neutral	1.24 (0.75, 1.73)	0.84 (Large)	<.001
Negative - Positive	0.61 (0.12, 1.10)	0.41 (Small)	.011
Neutral - Positive	-0.63 (-1.12, -0.14)	0.43 (Small)	.008
Valence Ratings			
Negative - Neutral	-0.91 (0.75, 1.73)	0.75 (Medium)	<.001
Negative - Positive	-1.37 (0.12, 1.10)	1.13 (Large)	<.001
Neutral - Positive	-0.46 (-1.12, -0.14)	0.38 (Small)	.006

Note. Std. Beta (Label) = Absolute value of standardized beta coefficient as measure of effect size derived by fitting model to standardized dataset with effect size label as per Cohen's (1988) recommendations, Sig. = p value. P values and confidence intervals adjusted using the Tukey method for comparing a family of three estimates.

Table 6. Pairwise t test Results for Upregulation and Downregulation Conditions for each Extracted ERP Component as well as Arousal and Valence Behavioral Ratings.

Contrast	<i>Positive Images</i>			<i>Negative Images</i>		
	Estimate (95% CI)	Std. Beta (Label)	Sig.	Estimate (95% CI)	Std. Beta (Label)	Sig.
124 ms Component						
Decrease - Increase	-0.21 (-0.44, 0.02)	0.18 (Very Small)	.085	-0.16 (-0.40, 0.07)	0.14 (Very Small)	.224
Decrease - Watch	-0.02 (-0.25, 0.21)	0.02 (Very Small)	.974	0.10 (-0.13, 0.34)	0.09 (Very Small)	.550
Increase - Watch	0.19 (-0.04, 0.42)	0.17 (Very Small)	.135	0.27 (0.03, 0.50)	0.23 (Small)	.020
162 ms Component						
Decrease - Increase	0.09 (-0.16, 0.34)	0.08 (Very Small)	.674	0.10 (-0.15, 0.35)	0.09 (Very Small)	.615
Decrease - Watch	0.19 (-0.06, 0.44)	0.17 (Very Small)	.162	0.07 (-0.17, 0.32)	0.07 (Very Small)	.757
Increase - Watch	0.10 (-0.14, 0.35)	0.09 (Very Small)	.589	-0.02 (-0.27, 0.22)	0.02 (Very Small)	.971
259 ms Component						
Decrease - Increase	0.45 (0.19, 0.71)	0.48 (Small)	<.001	0.18 (-0.08, 0.43)	0.19 (Very Small)	.245
Decrease - Watch	0.03 (-0.23, 0.28)	0.03 (Very Small)	.971	-0.16 (-0.42, 0.10)	0.17 (Very Small)	.327
Increase - Watch	-0.42 (-0.68, -0.17)	0.45 (Small)	<.001	-0.33 (-0.59, -0.07)	0.35 (Small)	.007
381 ms Component						
Decrease - Increase	-0.21 (-0.37, -0.06)	0.24 (Small)	.004	-0.01 (-0.16, 0.15)	0.01 (Very Small)	.989
Decrease - Watch	0.04 (-0.12, 0.19)	0.04 (Very Small)	.844	-0.10 (-0.26, 0.05)	0.11 (Very Small)	.273
Increase - Watch	0.25 (0.09, 0.40)	0.28 (Small)	.001	-0.09 (-0.25, 0.06)	0.10 (Very Small)	.343
740 ms Component						
Decrease - Increase	-0.22 (-0.39, -0.05)	0.35 (Small)	.008	-0.05 (-0.22, 0.13)	0.07 (Very Small)	.809
Decrease - Watch	-0.09 (-0.26, 0.08)	0.14 (Very Small)	.435	-0.13 (-0.31, 0.04)	0.21 (Small)	.162
Increase - Watch	0.13 (-0.04, 0.30)	0.21 (Small)	.178	-0.09 (-0.26, 0.08)	0.14 (Very Small)	.449
Arousal Ratings						
Decrease - Increase	-1.28 (-1.77, -0.78)	0.87 (Large)	<.001	-0.85 (-1.34, -0.36)	0.58 (Medium)	<.001
Decrease - Watch	-0.28 (-0.77, 0.22)	0.19 (Very Small)	.382	0.07 (-0.42, 0.57)	0.05 (Very Small)	.933
Increase - Watch	1.00 (0.51, 1.49)	0.68 (Medium)	<.001	0.93 (0.43, 1.42)	0.63 (Medium)	<.001
Valence Ratings						
Decrease - Increase	-1.41 (-1.76, -1.05)	1.16 (Large)	<.001	0.41 (0.05, 0.76)	0.58 (Medium)	.019
Decrease - Watch	-0.56 (-0.91, -0.20)	0.46 (Small)	.001	-0.13 (-0.48, 0.22)	0.05 (Very Small)	.664
Increase - Watch	0.85 (0.50, 1.21)	0.70 (Medium)	<.001	-0.54 (-0.89, -0.18)	0.63 (Medium)	.001

Note. Std. Beta = Absolute value of standardized beta coefficient as measure of effect size derived by fitting model to standardized dataset with effect size label as per Cohen's (1988) recommendations, Sig. = *p* value. *P* values and confidence intervals adjusted using the Tukey method for comparing a family of three estimates.

Discussion

We explored the neural time course of emotion reactivity and regulation with an exploratory temporal PCA approach in the present study. We found that emotion reactivity unfolded starting at 162 ms and generally continued through the first 1000 ms following stimulus presentation, with negative stimuli consistently eliciting enhanced amplitude relative to positive stimuli. Regarding emotion regulation, different patterns emerged that were contingent upon

whether the stimuli were positive or negative, and negative regulatory effects unfolded early in the time course at 124 ms while positive regulatory effects were observed slightly later, starting at 259 ms. Only upregulation effects were observed for positive- and negative-valenced conditions relative to passive watch conditions, and no downregulation effects were found. Overall, this suggests that emotion reactivity unfolds more consistently throughout the neural time course, with enhanced activity for negative stimuli compared to positive stimuli. Regulatory effects are smaller, and their time course is contingent upon valence.

The earliest component retained from our PCA, a positive 124 ms peak, did not display any reactivity or positive regulatory effects. However, it is the point at which we first observed a regulatory effect for increasing affect in response to negative images (relative to passive viewing). This finding challenges the notion of emotion regulation implying change from *initial* reactivity (Lewis et al., 2010) and begs the question: does emotion reactivity necessarily precede regulation? More research is needed to adequately address this question, though it appears that our findings support Joormann's and Stanton's assertion that disentangling the two processes is complex, and they may share overlap in terms of chronometry (Joormann & Stanton, 2016). This finding also indicates that negative emotion regulatory processes may be implicated earlier in the neural time course when compared to positive emotion regulation or emotion reactivity. Interventions that aim to modify neural mechanisms of negative emotion regulatory processes should likely focus on these earlier mechanisms.

For the next extracted component at 162 ms, passively viewing positive and negative stimuli elicited amplitude relative to neutral stimuli. Within the present study, this marks the

timepoint at which reactivity processes begin to unfold throughout the rest of the neural time course of extracted PCA components (i.e., the first 1000 ms following stimulus presentation). We also found that negative images elicited augmented amplitude relative to positive images (and for all subsequent components). This pattern of findings largely fits with research investigating later slow wave components (i.e., enhanced amplitude for emotional stimuli, with greater effects for negative stimuli, e.g., Foti et al., 2009; Frank & Sabatinelli, 2019). No regulatory effects were found for this 162 ms component, implying that activity within this time window is specific to emotion reactivity. This also illustrates that negative emotion regulation is not necessarily a continuous process. Rather, it is implicated at certain points in the time course. In sum, interventions targeted at emotion reactivity may consider neural processes starting very early in the time course and through the time course.

The next timepoint, 259 ms, is the sole timepoint in the present study where reactivity, positive emotion regulation, and negative emotion regulation effects were observed concurrently. This implies that the 259 ms component is flexible to task demands (e.g., reactivity or regulation) and also marks the point at which positive emotion regulation effects begin to unfold and negative emotion regulation ends. With regard to reactivity, no effect was found when comparing neutral and positive stimuli. Rather, negative images elicited augmented amplitude relative to neutral and positive stimuli. This contradicts a “pleasure bias” observed in another negative peak component (the EPN) often observed in this time window (Frank & Sabatinelli, 2019). One possible explanation for this discrepancy is our use of PCA, which addresses the issue of overlapping waveforms - a problem that windowed averages are vulnerable to (Donchin &

Heffley, 1978). Concerning regulatory effects, significant differences were observed for both positive and negative stimuli, ultimately implying that early visual ERP processes may play a larger role in emotion regulation than previously surmised. Of note, only upregulation effects were observed. It may be that this component is specific to increasing emotional intensity, or that our paradigm was unsuccessful in eliciting downregulation effects, as indicated by null findings in other ERP components and behavioral arousal ratings.

There was a continuation of reactivity effects in the two extracted positive slow wave components at 381 ms and 740 ms. Highly arousing positive and negative images eliciting enhanced slow wave ERPs (i.e., LPP) is a common finding in research investigating the neural time course of affective processing with the IAPS (Cuthbert et al., 2000; Hajcak, Dunning, & Foti, 2007; Hajcak et al., 2009; Krompinger et al., 2008; Moser et al., 2006; Schupp et al., 2000; Tritt et al., 2016). It is also largely consistent with past research examining emotion reactivity with PCA (e.g., Foti et al., 2009). Smaller effects were found for positive images compared to negative images in the 381 ms and 740 ms components, which is also consistent with previous literature (Foti et al., 2009; Frank & Sabatinelli, 2019). Of note, these findings are somewhat at odds with a 2012 study by Liu and colleagues (2012) that found comparable effect sizes between positive and negative stimuli for centroparietal LPP. Differences between the present study and Liu et al. (2012) may be attributed to different high-pass filters (.01 Hz versus .1 Hz), experimental paradigms (block vs. randomized design), and use of PCA vs. averaging of time windows. Future researchers should be mindful of these factors as to facilitate replicability of results.

Regulation effects were found in the 381 ms component for the positive increase condition relative to the neutral and decrease conditions. This contrasts with previous literature reporting null findings in positive increase conditions for positive slow wave components (Krompinger et al., 2008; Moser et al., 2009), which was attributed to a theorized ceiling effect that results from automatic capture of attentional resources by motivationally relevant stimuli. However, our results did align with Wilson and MacNamara (2021), who adopted a novel paradigm that instructed participants to savor positive IAPS stimuli, with language consistent with Bryant's (Bryant, 1989; Bryant & Veroff, 2007) conceptualization of savoring. Notably, our instructions prompted participants to reappraise the images in a manner that elicited greater emotional intensity, while Wilson and MacNamara intentionally did not use such language on the basis of the notion that the cognitive demands of reappraisal may actually contradict the goal of increasing emotional intensity. This may explain why our 740 ms component was not modulated by regulatory condition, while Wilson and MacNamara found such an effect at a parieto-occipital LPP site occurring in the 1,000 - 6,000 ms time window using windowed averages. Future researchers would benefit from incorporating elements of Wilson and MacNamara's study as well as ours: inspection of earlier EEG components, use of PCA rather than windowed averages, and employing paradigm instructions designed to more directly tap into the psychological constructs of interest. The lack of negative regulatory effects in our slow wave components is surprising given findings showing a reduction in LPP for when decreasing emotional intensity in response to negative and positive images (Krompinger et al., 2008; Moser et al., 2009, 2014, 2006). Indeed, it is possible that our block-design paradigm with seven different regulatory conditions failed to

capture such an effect, as other researchers have utilized randomized designs. The presence of type II error also cannot be ruled out, and future replication is the only way to ascertain the presence of a true effect.

There are several limitations to the present study. The psychophysiological data collected are cross-sectional and causation cannot be inferred. The present study's exclusionary criteria (only recruited participants who were right-handed, not color-blind, and learned English as a first language) and reliance on a convenience sample mostly comprised of white college students limits ability to generalize findings to a more diverse population. While much research on this topic (the present study included) relies on convenience samples (e.g., Hajcak & Nieuwenhuis, 2006; Kim & Hamann, 2007; Moser et al., 2006), future research should investigate the chronometry of emotion reactivity and regulation across the lifespan and study diverse samples.

Conclusion

In conclusion, the present study illustrated a cascade of early and slow wave ERP components that were modified in reactivity conditions. There were consistent emotion reactivity effects observed for positive and negative stimuli starting at 162 ms and generally throughout the first 1000 ms of the neural temporal course. Different temporal windows of regulatory effects emerged depending on stimulus valence, with negative regulatory effects unfolding from 124 ms to 159 ms, and positive regulatory effects unfolding from 259 ms to 381 ms. These are important considerations in devising neuroscience-informed treatments for individuals who experience dysregulated emotion reactivity and/or regulation. Additionally, this is one of the first studies to examine the neural time course of affective processing using the OASIS stimuli set, which

includes high quality images, spans a number of semantic categories, and is open access (Kurdi et al., 2017). The present study provides a more holistic perspective of the time course of emotion reactivity/regulation in response to positive and negative stimuli. Identifying neural implementations of emotion reactivity and regulation will be crucial for developing therapeutic and pharmacological interventions for those suffering from psychological and physiological ailments. It may also bring us a step closer to harnessing the mechanisms of wellbeing.

CHAPTER FOUR

A POTENTIAL NEURAL MECHANISM SUPPORTING THE BRIGHTENING EFFECT IN DEPRESSION

Impaired emotion reactivity has been observed in individuals with major depressive disorder (MDD; e.g., Joormann & Stanton, 2016; Rottenberg, 2005) which is associated with decrements in interpersonal relationships, poor work performance and burnout, and reductions in physical wellbeing (Druss et al., 2009; Kessler et al., 2006; Lewinsohn et al., 2003). Emotion reactivity refers to an individual's initial baseline affective response to a stimulus, and it is marked by alterations in a number of response systems, such as perception, expressive behavior, feeling, and physiology (Ekman, 1992; Keltner & Gross, 1999). Consistent with Russell's (1980) affective circumplex framework, reactivity can vary with regard to arousal (not arousing to very arousing) and valence (negative to positive; Gross & Jazaieri, 2014). There are three predominant views of emotion reactivity in depression based on laboratory-based research: negative potentiation, positive attenuation, and emotion context insensitivity (ECI). The negative potentiation view states that negative mood (i.e., diffuse and slow-moving feeling states with weaker ties to specific events and stimuli in one's environment; Watson, 2000) in MDD is due to potentiated emotional reactivity in response to negative emotional stimuli. Positive attenuation holds that those with MDD will have reduced emotional reactivity in response to positive stimuli (Rottenberg, 2005). The negative potentiation and positive attenuation views are not

mutually exclusive; those with MDD can theoretically display both attenuated positive and potentiated negative emotion reactivity. The ECI theory maintains that MDD is marked by a pattern of reduced reactivity to both positive and negative stimuli (Rottenberg, 2005). While the ECI theory is compatible with the positive attenuation theory, it hypothesizes attenuated reactivity in response to negative stimuli as well, which is at odds with the negative potentiation theory.

Bylsma and colleagues (Bylsma et al., 2008) conducted a meta-analysis examining the evidence for each of these three theories of emotional reactivity in MDD. Studies used in the meta-analysis included participants with a diagnosis of current MDD using Diagnostic and Statistical Manual of Mental Disorders (DSM) criteria, a reference group, and measures of emotional reactivity that included self-report, expressive behavioral, or peripheral physiological measures/indicators (Bylsma et al., 2008). Results illustrated that MDD was consistently associated with consistent reductions in positive emotion reactivity and negative emotion reactivity (i.e., providing support for the ECI theory), and that effect sizes were greater for reductions in positive emotional reactivity than negative emotional reactivity (Bylsma et al., 2008). Additional research is needed to reconcile these findings with research that has found support for the negative potentiation theory (Chan et al., 2007; Ellis et al., 2010), though results support that positive attenuation is a robust feature of depression. Despite some inconsistent findings regarding negative emotion reactivity in depression, research using self-report and physiological methods to measure emotion reactivity supports the ECI view (Bylsma et al., 2008), or that there is attenuated emotion reactivity in response to positive as well as negative

stimuli.

Research grounded in daily life experiences using ecological momentary assessment methods have found results in the opposite direction than those derived in laboratory assessments, or that individuals with MDD exhibit a “brightening effect” with greater reactivity to positive events (Khazanov et al., 2019a). One potential explanation for these findings is that those with depression harbor more negative expectations for future events (Khazanov et al., 2019a), and these lower expectations amplify responses to positive stimuli (McNamara et al., 2013). It is unclear why laboratory settings have failed to replicate a brightening effect, though it is possible that standardized stimuli are less salient than real day-to-day experiences (Khazanov et al., 2019a).

While four possible frameworks (negative potentiation, positive attenuation, ECI, brightening effect) have been previously discussed in the literature, these frameworks do not fully embrace all eight possible patterns of emotion reactivity that might be observed in individuals with depression (see Figure 7). The present study will evaluate all possible patterns of emotion reactivity that might covary with depression symptoms, beyond the four existing frameworks that are included in the literature. Given the laboratory context of the present study, our hypotheses were ultimately informed from other laboratory-based studies, which appear to provide the most support for the ECI view. Therefore, consistent with the ECI theory, we hypothesized that attenuated reactivity to positive and negative stimuli, as measured via electroencephalography (EEG), would be associated with increased depression symptoms.

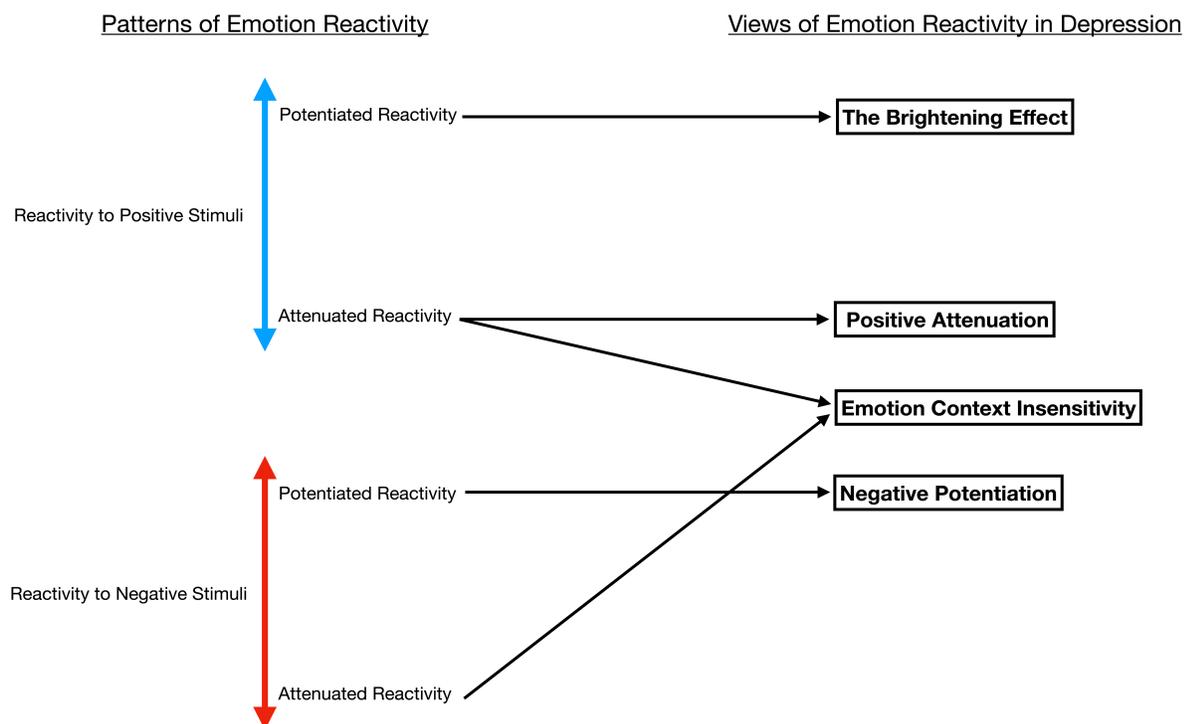


Figure 7. Visualization of possible emotion reactivity patterns. The left column represents dimensions of emotion reactivity in terms of reactivity to positive and negative stimuli. The right column illustrates combinations of these possible patterns based on four existing frameworks of emotion reactivity in depression; however, other combinations may also exist, such as concurrent positive attenuation and negative potentiation and concurrent positive and negative potentiation.

EEG offers millisecond temporal resolution (Olofsson et al., 2008) that contributes to further refining our understanding of the temporal course of neural correlates implicated in emotion reactivity dysfunction in depression in order to contribute to the development of neuroscience-informed treatments for depression. Previous research investigating emotion reactivity in depression using EEG has predominately focused on later positive slow wave components that are measured with windowed averages (Hill et al., 2019; Weinberg et al., 2016, 2017). A relatively narrow focus on slow wave components might overlook important neural processes occurring earlier in the time course of emotion reactivity and might be susceptible to

measuring overlapping components (Donchin & Heffley, 1978). Thus, in the present study we implemented a principal components analysis (PCA) approach to measuring event-related potentials (ERPs) in order to identify distinct temporal components, and we also investigated earlier indices of neural activity.

Low Positive Affectivity and Emotion Reactivity in Depression

Anhedonia is a hallmark symptom of MDD (American Psychiatric Association 2013; Treadway & Zald, 2011). Anhedonia negatively impacts daily function, predicts poor treatment response, indicates risk for future depressive episodes, and shows specificity with regard to depression diagnosis (Khazanov et al., 2019a; Watson & Naragon-Gainey, 2010). While high negative affectivity (NA) may be a global indicator of distress in depression and anxiety disorders (Watson & Clark, 1984), low positive affectivity (PA) is a trait that is closely related to anhedonia (De Fruyt et al., 2020). Low PA is a catalyst for exacerbated depressive symptomatology (Clark & Watson, 1991; Davidson, 1998; Kahrilas et al., 2020; Watson et al., 2015). PA reflects one's disposition to experience intense and frequent episodes of pleasant moods (Watson, 2009), such as happiness, interest, energy, and self-assurance (Mineka et al., 1998; Watson & Naragon-Gainey, 2010) as well as level of pleasurable engagement with one's environment and ability to respond to positive events (Watson et al., 1988; Watson, 2009; Watson & Naragon-Gainey, 2010). Further, PA influences reactivity to positive emotions and experiences, as well as disposition to recognize, attend to, and ultimately experience positive emotions (e.g., happiness, interest, energy, and self-assurance; Watson, 2009). Moving forward, developing evidence-based strategies to target low positive affectivity and related anhedonic

depressive symptoms could be critical to increasing the effectiveness of successful treatment for depression (Forbes & Dahl, 2005; Kahrilas et al., 2020; Siltan et al., 2020; Treadway & Zald, 2011).

Common treatments for depression, such as cognitive behavioral therapy (CBT) and antidepressant medication (e.g., selective serotonin reuptake inhibitors, selective norepinephrine reuptake inhibitors, tricyclic antidepressants, and monoamine oxidase inhibitors; Price & Drevets, 2010) have primarily focused on ameliorating distorted thought patterns and neurotransmitter systems pertaining to negative affect (Argyropoulos & Nutt, 2013; Beck, 1979; Dunn et al., 2019). Dunn and colleagues (2019) investigated the efficacy of therapeutic and pharmacological approaches to treating depression. Findings showed that high negative affect (i.e. up-regulation of a negative valence system that promotes withdrawal from negative stimuli) and low positive affect (down-regulation of a positive valence system that promotes approach to rewarding stimuli) were both important psychological targets in treating depression, and that treatments aimed at enhancing the experience of positive emotions (i.e., enhancing reactivity to positive events/stimuli) in depression may improve efficacy. Previous depression research that focused on emotion reactivity has generally illustrated attenuated reactivity in response to positive stimuli (i.e., the positive attenuation view and ECI theory); thus, attenuated reactivity to positive stimuli may represent a critical mechanism related to low positive affectivity and anhedonic symptoms in depression. Although disordered emotion reactivity to positive stimuli may be influenced by a myriad of intersecting contextual factors and individual differences, emotion reactivity occurs “online,” and in the moment, and likely influences a cascade of other affective events that unfold

following initial reactivity. Thus, baseline emotion reactivity to positive stimuli may be a critical target for future depression treatments. As such, in the present study, we aimed to examine relations among PA and emotion reactivity to positive stimuli.

Regional Brain Activity Implicated in Emotion Reactivity in Non-Clinical and Clinical Samples

Non-Clinical Samples

In functional magnetic resonance imaging (fMRI) research conducted in non-clinical samples, brain network activity during emotion reactivity to visual stimuli involves a visual-subcortical network including right occipital, visual cortical, and amygdala activation in response to viewing affective stimuli that is modulated by arousal, with some variability in valence effects (Bradley et al., 2003; Lang et al., 1998; Sabatinelli et al., 2005). Right occipital cortex (i.e., fusiform cortex, lateral occipital cortex, and medial parietal cortex; Bradley et al., 2003), as well as visual cortex and amygdala (Sabatinelli et al., 2005) are implicated in an arousal effect in response to viewing pleasant and unpleasant affective images compared to neutral images. The largest changes in brain activity are associated with viewing highly arousing stimuli, regardless of valence (Bradley & Lang, 2007). However, positive emotional stimuli eliciting regional brain activity has been observed (e.g., Frank & Sabatinelli, 2019), as has greater activity for negative emotional stimuli (Kim & Hamann, 2007).

Clinical Samples

In individuals with MDD, attenuated ventral striatum activity has been observed in response to positive visual stimuli (Epstein et al., 2006; Lawrence et al., 2004; Surguladze et al.,

2005). Ventral striatum activity is associated with processing of rewarding/positive stimuli (Epstein et al., 2006; Fliessbach et al., 2007; Schultz et al., 1992). Attenuated striatum activity has been related to self-reported lack of interest and pleasure in activities (Epstein et al., 2006; Treadway & Zald, 2011), which provides support for both the positive attenuation and ECI views of emotion reactivity in depression. Other studies have found enhanced activity in frontal brain structures involved in emotion regulation (e.g., middle frontal gyrus, cingulate cortex, dorsolateral PFC) during emotion reactivity to positive stimuli in those with MDD (Demenescu et al., 2011; Ochsner et al., 2009). This pattern of findings may index increased attention orientation and processing demands to mood-incongruent stimuli (Demenescu et al., 2011). Alternatively, it is possible that patterns of enhanced activity are consistent with the brightening effect (Khazanov et al., 2019b; Khazanov & Ruscio, 2016) such that individuals with depression have greater reactivity to positive events and stimuli.

Regarding negative emotion reactivity, studies have indicated increased responsiveness to, and memory for, negative stimuli as illustrated by enhanced amygdala response to negative emotional stimuli (Fales et al., 2008; Savitz & Drevets, 2009) in those with MDD compared to controls. Increased amygdala reactivity to negative stimuli has been observed in those with severe depression (Sheline et al., 2001), and antidepressant medication regimen may be linked to down-regulation of amygdala response (Fu et al., 2004; Lawrence et al., 2004; Sheline et al., 2001). In contrast, participants with mild-to-moderate depression severity (with and without medication) do not exhibit differences in amygdala activity in emotion reactivity compared to controls (Demenescu et al., 2011). Therefore, support for negative potentiation in depression appears to be

contingent upon depression severity.

Neuroimaging Results Presently Offer an Opaque Picture of Emotion Reactivity in Depression

Based on the existing neuroimaging research, there is not year a clear pattern of support for any of the four existing frameworks regarding abnormalities in emotion reactivity in depression (positive attenuation, negative potentiation, brightening effect, or ECI). In part this opaque picture may be a result of the relatively slow time course of the blood-oxygen-level-dependent (BOLD) response as measured via fMRI, which most likely fails to fully capture the temporal course of emotion reactivity, which is observed early as 100 ms in EEG research (Frank & Sabatinelli, 2019; Hinojosa et al., 2015; Kahrilas et al., in prep; Stockdale et al., 2020). Additionally, individual differences (e.g., differences in depression severity, medication status, and levels of co-occurring anxiety) across studies may be contributing to obscuring clear patterns of brain activity.

Neuroscience research investigating depression that does not account for co-occurring anxious apprehension and anxious arousal may yield mixed findings (Herrington et al., 2010; Sharp et al., 2015). Anxious apprehension refers to a tendency to engage in negative, ruminative thinking, and is analogous to enduring state worry (Burdwood et al., 2016; Ruscio et al., 2001), while anxious arousal entails hypervigilance and sympathetic nervous system hyperarousal to mild stressors (Nitschke et al., 1999). Accounting for co-occurring dimensions of anxiety can help clarify distinct patterns of emotion reactivity and neural activity associated with PA (Herrington et al., 2010; Sharp et al., 2015). Altogether, a dimensional approach to measuring

depression symptoms in the context of neural emotional reactivity would allow different patterns of neural activity to covary with degrees of illness severity and anxiety comorbidity. Therefore, in the present study we included measures of anxious arousal and anxious apprehension in our statistical models in order to more adequately capture distinct relations among low PA and neural activity, with the broad aim of improving the characterization of patterns of neural correlates of emotion reactivity in depression. Further, fMRI-based neuroimaging research does not capture the temporal course of emotion reactivity, which occurs as early as 100 ms (Frank & Sabatinelli, 2019; Hinojosa et al., 2015; Kahrilas et al., in prep; Stockdale et al., 2020); thus, it is critical to also understand the temporal course of regional brain activity implicated in emotion reactivity.

Evaluating the Temporal Course of Emotion Reactivity in Depression: Event-Related

Potentials Index Dynamic Cascade of Neural Processes

Early ERP Components

Research conducted by our lab and others has identified early visual ERP components that are sensitive to reactivity to affective stimuli (i.e., N170 & EPN; Frank & Sabatinelli, 2019; Kahrilas et al., in prep.). Studies investigating whether these ERP components are modified in depression have produced mixed findings. Research from Foti and colleagues (2010) illustrated that early ERP components are unmodified in individuals with MDD compared to individuals without MDD (as indexed by enhanced vertex parietal potential, a component theorized to reflect perceptual processing and encoding of facial stimuli, in response to fearful/angry compared to neutral faces). In contrast, Dai and Feng (2012) found smaller N170 amplitudes in response to happy faces in sub-clinical MDD cases and enhanced N170 in response to negative faces in

clinical MDD cases, which is congruent with the positive attenuation and negative potentiation views. Chen and colleagues (2014) noted reduced N170 amplitude in response to happy, neutral, and sad faces in a visual emotions oddball paradigm using face stimuli for individuals with a current first episode of major depression. The same study indicated that participants with recurrent depression exhibited enhanced N170 amplitude in response to sad faces and attenuated N170 amplitude in response to happy and neutral faces (Chen et al., 2014), demonstrating patterns of emotion reactivity that vary as a function of illness history. Research investigating N170/EPN and emotional reactivity in response to complex affective images (e.g., International Affective Pictures System, or IAPS) in depression is scant, with one study finding enhanced EPN activation in response to negative compared to neutral images for individuals with depression among binge drinkers (Connell et al., 2015). Following these findings, we hypothesized that PA would be associated with attenuated N170 and EPN amplitude in response to passively viewing positive stimuli compared to neutral stimuli. Given the equivocal findings regarding these early components in response to negative images, analyses regarding the relationship between PA and N170/EPN in response to passively viewing negative compared to neutral stimuli were conducted in an exploratory manner.

LPP

Affective disorders such as MDD and dysthymia are associated with abnormal emotional reactivity as indexed by the late positive potential (LPP) in response to affective stimuli (Grunewald et al., 2019). LPP is a sustained positive slow wave EEG component observed starting as early as 300 ms following stimuli onset and is as an established index of evaluative

congruency, valence, and arousal in response to visual stimuli (Olofsson et al., 2008). LPP has been theorized to reflect activation of a brain network engaged in processing affective visual stimuli, including subcortical structures, particularly the amygdala (Bradley & Lang, 2007; Cuthbert et al., 2000; Keil et al., 2002; Olofsson et al., 2008; Schupp et al., 2003). LPP over the centroparietal cortex is augmented in the presence of unpleasant and pleasant compared to neutral images (Cuthbert et al., 2000; Dillon et al., 2006; Foti & Hajcak, 2008; Hajcak et al., 2006, 2007; Hajcak & Nieuwenhuis, 2006; Keil et al., 2002; Moser et al., 2006; Olofsson et al., 2008; Schupp et al., 2003) and is most pronounced for images that are rated as highly arousing (Bradley & Lang, 2007).

Attenuated LPP in response to both unpleasant and pleasant affective stimuli has been observed in those with MDD (Foti et al., 2010; Kayser et al., 2000; MacNamara et al., 2016), which is consistent with the ECI view of emotion reactivity in depression (Rottenberg et al., 2005). A study conducted by Weinberg and colleagues (2016) found that, in a sample with diagnoses of MDD and anxiety disorders, a diagnosis of MDD was uniquely associated with attenuated LPP in response to rewarding visual stimuli while a diagnosis of an anxiety disorder was not. The aforementioned study did not demonstrate an association with MDD diagnosis and reduced LPP in response to threatening stimuli (Weinberg et al., 2016), thus ultimately showing support for the positive attenuation view of emotion reactivity. A follow-up study found that those with MDD and previous suicide attempts (an index for illness severity) had blunted LPP in response to threatening stimuli compared to those with MDD and without a history of suicide attempts (Weinberg et al., 2017). This effect was unique to threatening (not rewarding) images,

which is consistent with fMRI accounts of depression severity altering amygdala activity patterns in response to negative stimuli (Demenescu et al., 2011). Overall, this illustrates differences in neural reactivity depending on illness severity and further underscores the need for dimensional measures of psychopathology rather than categorical nosology.

Findings that illustrate negative associations among LPP elicited in response to positive and negative images and depression symptoms (Hill et al., 2019) provide additional support for the ECI view. Of note in the Hill and colleagues (2019) study was the use of structural equation modeling (SEM). In contrast to more ubiquitous OLS regression methods, SEM can explicitly model measurement error, assess overall model fit, and allow for statistical comparison of competing models. Hill and colleagues (2019) constructed two separate factors: a neural reactivity and psychopathology symptoms model. Results from this study indicated that the model corresponding to the ECI view provided the best fit to the data. Related, Weinberg and colleagues (2018) found blunted LPP in response to rewarding and threatening images in those endorsing lower levels of PA, a pattern of results ultimately lending support to the ECI view. Thus, we hypothesized that PA would be uniquely and negatively associated with LPP amplitude in response to positive and negative stimuli.

Temporal PCA Approach to ERPs Improve Measurement of Distinct Components Across Time

Most of the aforementioned research utilizes windowed averages, or area measures, for measuring ERP components. However, this method is inadequate in dealing with the thorny issue of overlapping components (Donchin & Heffley, 1978). For example, there is speculation that

N170 modulation due to emotional expressions may be due to superimposed EPN activity (Rellecke et al., 2013). Similarly, LPP may overlap with P3, a parietally maximal component peaking in the 300-400 ms window that is sensitive to task relevant and motivationally significant stimuli (Polich & Kok, 1995), and even later portions of EPN (Schupp et al., 2004). To address this issue, we utilized an exploratory temporal PCA to better isolate distinct ERP components across time. PCA is a dimension reduction technique that identifies a new set of variables that are linear combinations of the original variables. In the context of ERPs, these new variables represent latent components that may not be apparent by visually inspecting ERP averages. For example, if there were two temporally overlapping ERP components with equal magnitude but opposite directionality (positive and negative), the two components would negate each other and not be observable via inspection of grand average ERPs. Temporal PCA, on the other hand, would theoretically be able to retain these components provided that the experimental paradigm reliably produces them. Thus, the present study used a temporal PCA approach to evaluating neural components evoked by emotion reactivity.

Primary Objectives of the Present Study

To clarify the pattern of the temporal course of the neural correlates of emotion reactivity in individuals with depression symptoms, the present study used an SEM approach to evaluate several statistical models characterizing patterns of emotion reactivity (as indexed by N170, EPN, and LPP, derived with PCA) with PA (while accounting for co-occurring anxious arousal and anxious apprehension symptoms). With regard to earlier visual components (N170, EPN), we hypothesized that emotion reactivity in response to positive images would be negatively

associated with PA. No hypotheses were made regarding early visual component activity in response to negative images and its relation to PA, and these analyses were carried out in an exploratory manner.

Method

Overview of Study Harmonization Approach

In order to facilitate addressing our primary research question regarding the impact of depression symptoms on the neural correlates of emotion reactivity, we strategically harmonized across two college student samples in order to increase variability in the primary variables of interest (e.g., low PA). Participants in both samples completed the same self-report symptom measures and underwent the same EEG experimental paradigm (described below in detail). The two samples included the following: a) an unselected non-clinical sample from an EEG experiment ($N = 52$; Kahrilas et al., in prep) and b) pre-trial baseline (T0) assessment data from a three-month randomized controlled trial that investigated the efficacy of Headspace in mitigating depression symptoms in college students (Supported Mindful Learning Study; SMiLe; $N = 82$). Participants who had elevated depression symptoms (PHQ-8 score of 10 or higher) were recruited into the SMiLe study for baseline assessment. Only self-report and EEG data from pre-trial baseline assessment were used in the present study. Approximately 15 students per each fall and spring semester were recruited over the course of three years (Fall 2017 - Spring 2020) for a total of six semesters. After harmonization, the total sample size for the present study was $N = 134$.

Participants

Participant Recruitment and Inclusion Procedures Across Two Studies

We recruited a non-selected sample of undergraduate students ($N = 52$) for an EEG study from a psychology department participant pool (Kahrilas et al., in preparation). Eighty undergraduate participants with elevated depression symptom scores (score of 10 or higher on the PHQ-8) were invited to participate in the Supported Mindful Learning (SMiLe) study, which was a three-month randomized controlled trial that investigated effectiveness of Headspace (www.headspace.com). Participants were recruited using listserv emails, flyers, and the psychology department participant pool postings. Participants that met inclusion criteria were randomized to one of three experimental groups and completed batteries of surveys at four separate timepoints.

To determine eligibility for the SMiLe study, interested participants completed an online screening survey through Opinio. Inclusion criteria included the following: participants had to be an undergraduate at the institution from which the study was being conducted, at least 18 years old, and endorse clinically significant levels of depressive symptoms as indicated by a score equal to or greater than 10 on the Patient Health Questionnaire-8 (PHQ-8, Kroenke & Spitzer, 2002; Kroenke et al., 2009). For screening purposes, an eight-item version of the PHQ was utilized with the suicidality item omitted (per IRB request). Individuals were excluded if they had a history of neurological conditions or head trauma, regular practice of mindfulness in the past six months (which is consistent with exclusion criteria used in previous research; e.g., Van Dam et al., 2018), prior use of the Headspace app within the past six months, and unwillingness to join the peer

support group (if randomized to the Headspace with Peer Support group).

Of the 80 participants recruited for the SMiLe study, 73 underwent the EEG paradigm, and 11 participants were excluded from analyses due to large amounts of EEG artifact. This resulted in a final total of 62 from the SMiLe study. The 62 SMiLe Study participants were harmonized with the sample of 52 from Kahrilas and colleagues (in preparation) for a total n of 114. In the harmonized sample, participants were 18 to 29 years old ($M = 19.4$, $SD = 1.64$), 75% of which identified as female, 21% as male, 0.9% as transgender, 0.9% as non-binary, and 1.7% as other. Seventy-one percent identified as heterosexual, 21% as bisexual, 4% as gay, and 4% as a different sexual orientation. Sixty-nine percent of participants identified as white, 15% Asian, 8% Hispanic or Latinx, 5% Multi-Racial, 1% as Black, and 1% as Other.

Materials and Procedure

To evaluate depression severity, the nine-item Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) was administered in the laboratory setting. PHQ-9 items are scored from “0” (not at all) to “3” (nearly every day) and are based on the depression criteria from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Internal consistency of the PHQ-9 in the present study was good (George & Mallery, 2003; $\omega = .82$).

To assess for positive affectivity and anxious arousal, the 39-item Mood and Anxiety Symptoms Questionnaire (MASQ; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995) was administered. The 14-item positive affectivity (PA) Scale (MASQ-PA14; e.g., “Felt cheerful”) and the 17-item Anxious Arousal Scale (MASQ-AA; e.g., “Startled easily”) were used for subsequent analyses. Previous literature has supported these oblique factors in two independent

samples of individuals at-risk for depression and anxiety (Kahrilas et al., 2020; Kendall et al., 2015; Nitschke et al., 2001). Participants rated their affectivity and anxiety symptoms using a five-point Likert scale from “1” (not at all) to “5” (extremely). Consistent with previous research (Bredemeier et al., 2010) the MASQ scales demonstrated good to excellent internal consistency (MASQ-PA14, $\omega = .95$ and MASQ-AA, $\omega = .85$).

To measure anxious apprehension, the 16-item Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) was administered. PSWQ items are rated on a five-point Likert scale from “1” (not at all typical) to “5” (very typical). Meyer and colleagues (1990) found a high degree of internal consistency in the PSWQ, and the present study found excellent consistency ($\omega = 0.94$).

Emotion Task Paradigm

64-channel high-density electroencephalography (EEG) data were recorded while participants viewed seven blocks of 40 images (280 total trials). The paradigm used 120 different Open Affective Standardized Image Set (OASIS) images (Kurdi et al., 2017). 40 of the images were positive, 40 were neutral, and 40 were negative. The mean normative valence ratings (on a scale of one to seven) were 5.71, 4.09, and 2.18 for positive, neutral, and negative pictures, respectively. The mean normative arousal ratings (on a scale of one to seven) were 4.46, 1.99, and 4.52, for positive, neutral, and negative pictures, respectively.

Each block represented an experimental regulation condition: increase (positive/negative), decrease (positive/negative), and watch (positive/negative/neutral). During the increase condition, participants were instructed to appraise the picture in a way that will intensify the emotion that is elicited by looking at it. During the decrease condition, participants were asked to reduce the

intensity of the emotion that is elicited by looking at the picture. When prompted to watch, participants were asked to view the picture as they would naturally. The passive “watch” blocks served as baseline conditions for LPP component analyses (Hajcak & Nieuwenhuis, 2006). A block design was selected for the proposed study because valenced images outnumber the neutral images two-to-one. The relatively rare neutral stimuli may produce a P3 if intermixed with valenced stimuli in the same block (Schupp et al., 2000). Additionally, LPP signal is increased for stimuli that are perceived as incongruent within a given affective context. Thus, a neutral stimulus displayed among a series of emotional stimuli would likely evoke an enhanced LPP activity, thus decreasing effect size (Schupp et al., 2000). Further, a block design paradigm may better emulate one’s natural environment, as stimuli of congruent valence are typically encountered in clusters rather than isolation (Schupp et al., 2012). Research comparing intermixed and block designs for LPP and EPN have shown comparable findings between the two approaches (Pastor et al., 2008; Schupp et al., 2012).

First, participants completed three practice trials that consisted of 10 images not used during the actual task (Hajcak & Nieuwenhuis, 2006). Participants were instructed to increase or decrease emotional arousal in response to a mixture of positive and negative images, respectively. Participants were also asked to watch a third practice trial comprised of positive, negative, and neutral images. Stimuli were presented in random order within each block. Prior to each practice trial, participants were provided with instructions on how to regulate (increase/decrease) their affective response to the stimuli, or to simply view the stimuli (Moser et al., 2006). Participants were shown which regulation strategy to engage in (increase, decrease, or watch) at the start of

each experimental block. Once the participant was ready, an OASIS stimulus appeared for 2000 ms (Foti et al., 2009; Hajcak & Olvet, 2008). Following stimulus offset, a blank screen appeared for 500 ms followed by a fixation cross for 1500 ms (± 250 ms) for a total ISI of 1750 - 2250 ms (see *Figure 8* for a visual depiction of the experimental paradigm used in the present study). This interval is identical to other studies utilizing the IAPS and investigating LPP (Dunning & Hajcak, 2009; Hajcak et al., 2009). Stimuli within each block were presented in random order. Each block was separated by a break and presented in pseudo-random order, such that no valence (i.e., positive or negative) nor regulation (i.e., increase, decrease, or watch) condition were presented consecutively. After each experimental block, valence and arousal ratings were collected using a seven-point likert scale and the same instructions used in Kurdi, Lozano, and Banaji's (2017) norming study of the OASIS stimuli set. Furthermore, difficulty ratings on a seven-point likert scale were also obtained.

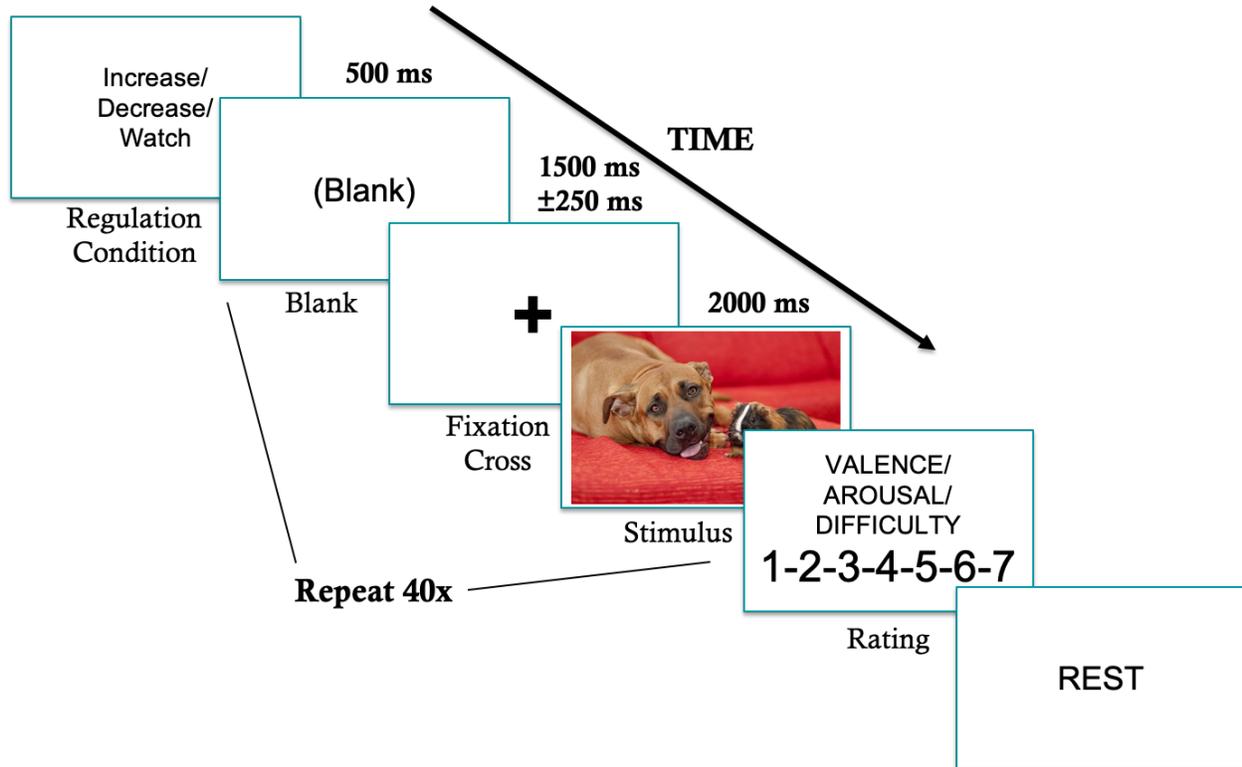


Figure 8. Experimental paradigm utilized in the present study.

Apparatus and Physiological Recording

Scalp electroencephalography (EEG) was measured while participants completed an emotion regulation task (Figure 8). Participants were seated in a comfortable chair, approximately 24 inches from a 24-inch LCD monitor in a quiet, dimly lit room. Participants were monitored by a task administrator in a nearby room and received task instructions by intercom. EEG data were recorded using a Biosemi Active2 EEG system. A custom-designed Falk Minow 64-channel cap with equidistantly spaced BioSemi active Ag and AgCl electrodes was used for data collection. CMS/DRL was placed near the vertex, and two electrodes were located on the mastoid bones. After placement of the electrode cap, electrode positions were digitized. An additional electrode

was placed on the inferior edge of the orbit of each eye to monitor vertical eye movements; nearby electrodes in the cap (lateral to each eye) monitored horizontal eye movements. Data were recorded with a band pass of 0 – 104 Hz at a sampling rate of 512 Hz.

The following EEG data processing steps were implemented in Brain Electrical Source Analysis software (BESA, Version 7; Scherg & Berg, 1990). Following the adaptive artifact correction method (Ille et al., 2002), ocular artifacts were corrected using a spatial PCA filter. EEG data were re-referenced to the average reference of all electrodes for analyses and digitally filtered with 0.01 Hz high-pass and 30 Hz low-pass filters with a cutoff attenuation of 12 dB/octave. While it is convention to re-reference data to average activity of the mastoids for LPP (e.g., Hajcak et al., 2006; Hajcak & Nieuwenhuis, 2006), a covariance matrix was utilized in the PCA, which is inherently mean corrected (Dien, 2012). Thus, minimal effects would be expected as a function of referencing scheme for temporal PCA (though this is not the case for spatial PCA; see Dien (2012)). Muscle activity and other artifacts were corrected through implementing an algorithmic scan of continuous EEG data in BESA (fixed threshold criteria: amplitude > 120 uV; gradient > 75 uV, low signal <.01 uV). Data were baseline-adjusted by subtracting the average activity for 200 milliseconds (Moser et al., 2006) before stimulus onset.

Temporal PCA

To discriminate among temporal sources of variance (i.e., ERP components) in the time course of ERPs, ERP averages were submitted to a temporal PCA to derive linear combinations of data that distinguish activity across timepoints. A temporal PCA was performed using the Psych package (Revelle, 2020) in R version 4.0.3 (Team, 2020) with a covariance matrix. A

covariance matrix was used to retain the original units of measurement (i.e., voltage) for the average ERPs for each timepoint (Donchin & Heffley, 1978). The temporal PCA used all timepoints (-200 ms - 2000 ms) as variables and all 113 participants, three blocks (passive positive, negative, and neutral viewing blocks), and 64 recording sites as observations, yielding linear combinations of timepoints and “temporal” factors (Dien, 2012).

To determine the number of components to retain for rotation, a parallel analysis (Horn, 1965) was performed on the resultant Scree plot. The parallel analysis creates Scree plots of the actual and simulated fully random data to determine the number of components that explain more variance than if only chance alone were acting. Based on the parallel analysis, 22 components were retained for rotation. A Promax rotation was then used on the 22 components because it does not impose orthogonality, and simulation studies suggest that Promax is most effective for temporal PCA (Dien, 2010; Dien et al., 2007). The time course of each of the 22 components was inspected by plotting the covariance loadings along each timepoint. To further guide our selection of components, we used a previous study conducted by our lab (Kahrilas et al., in prep) that analyzed the time course of emotion reactivity and regulation using PCA. PCA components with covariance loadings in time windows that fit descriptions of early visual and slow wave components (e.g., EPN, P300, and LPP) were retained for statistical analyses. Spatial information is retained in PCA; the topography of each factor is encoded in the mean amplitude of factor scores at each recording site (Dien, 1998). Thus, scalp topographies of each component for each condition was inspected to determine correspondence of components and ERP components of interest. Of note, portions of waveforms represented by a given PCA component can be

reproduced by multiplying covariance loadings by observations' factor scores (Dien, 1998).

Three components were retained for statistical analyses based on inspection of topographic plots and ERP waveforms. Two of the retained components represented later components that have positive peaks at 371 ms and 736 ms, with the former demonstrating activity at approximately PO7 and PO8 electrode sites and the latter at PO7, PO8, PO3, PO4, P1, P2, P5/P3, P6/P4 and Pz electrode sites. Similar to findings from Kahrilas and colleagues (in preparation), topography of a 257 ms component indicated a dipole of scalp activity, with positive going waveforms in the occipital region (PO7, PO8, O1, and O2) and negative waveforms in the centroparietal region (CPz and Pz). Based on our a priori interest in the EPN and considering that our research demonstrated EPN activity in response to emotional images (Kahrilas et al., in prep), we submitted the negative centroparietal activity to statistical analyses. No component resembling an N170 was derived from the PCA. See Figures 9, 10, and 11 for grand average ERP and topographic plots of the retained components before and after the PCA was performed. All visualizations were composed using the ggplot2 (Wickham, 2016) and eegUtils (Craddock, 2019) packages R version 4.0.3 (R Core Team, 2020).

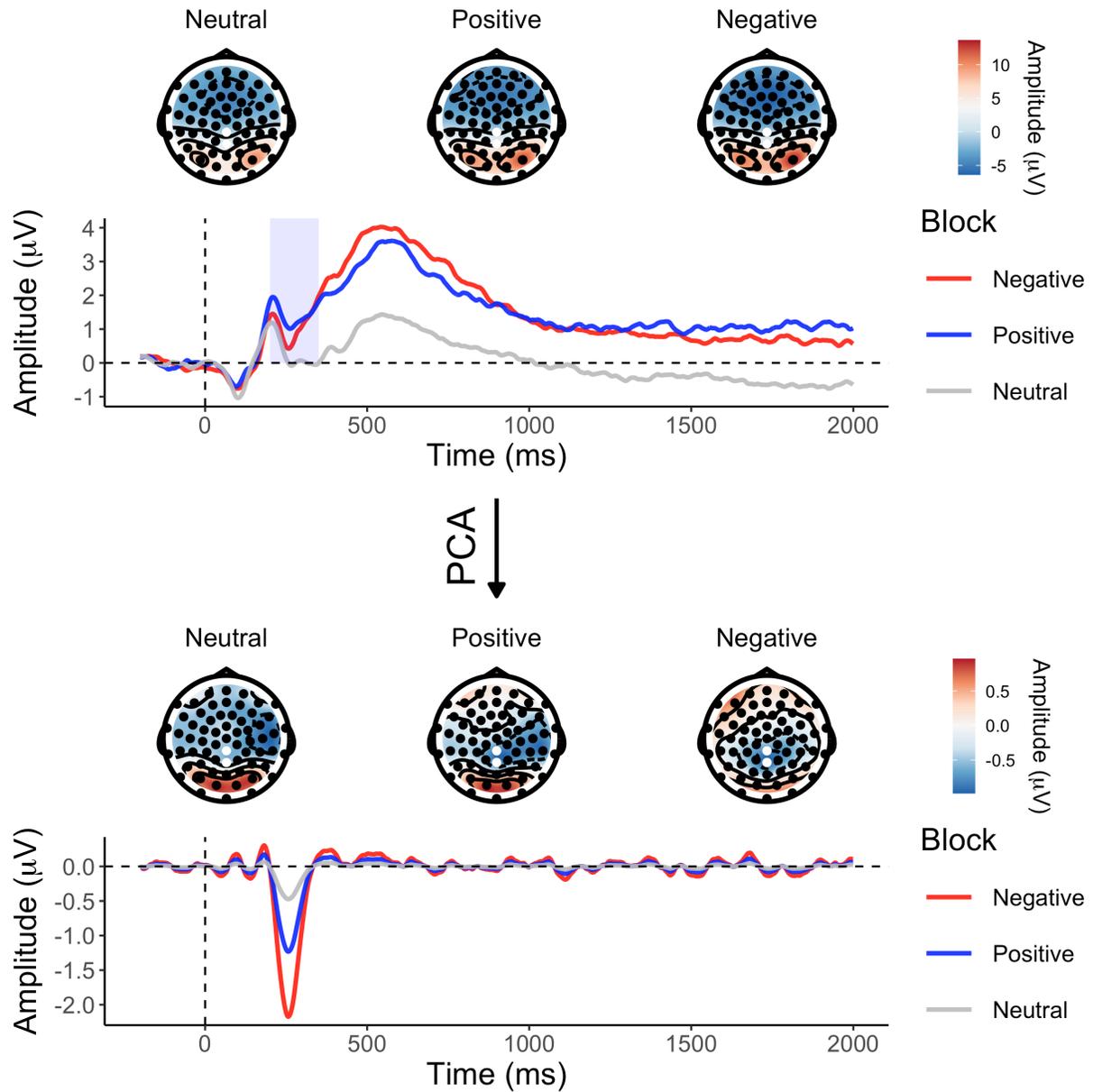


Figure 9. Topographic and ERP plots of the negative 257 ms PCA component before and after temporal PCA. Top row topographic plots represent average activity in the shaded region of the ERP plot below it (200 - 350 ms). ERP plots represent average activity in highlighted sensors topographic plots (CPz and Pz).

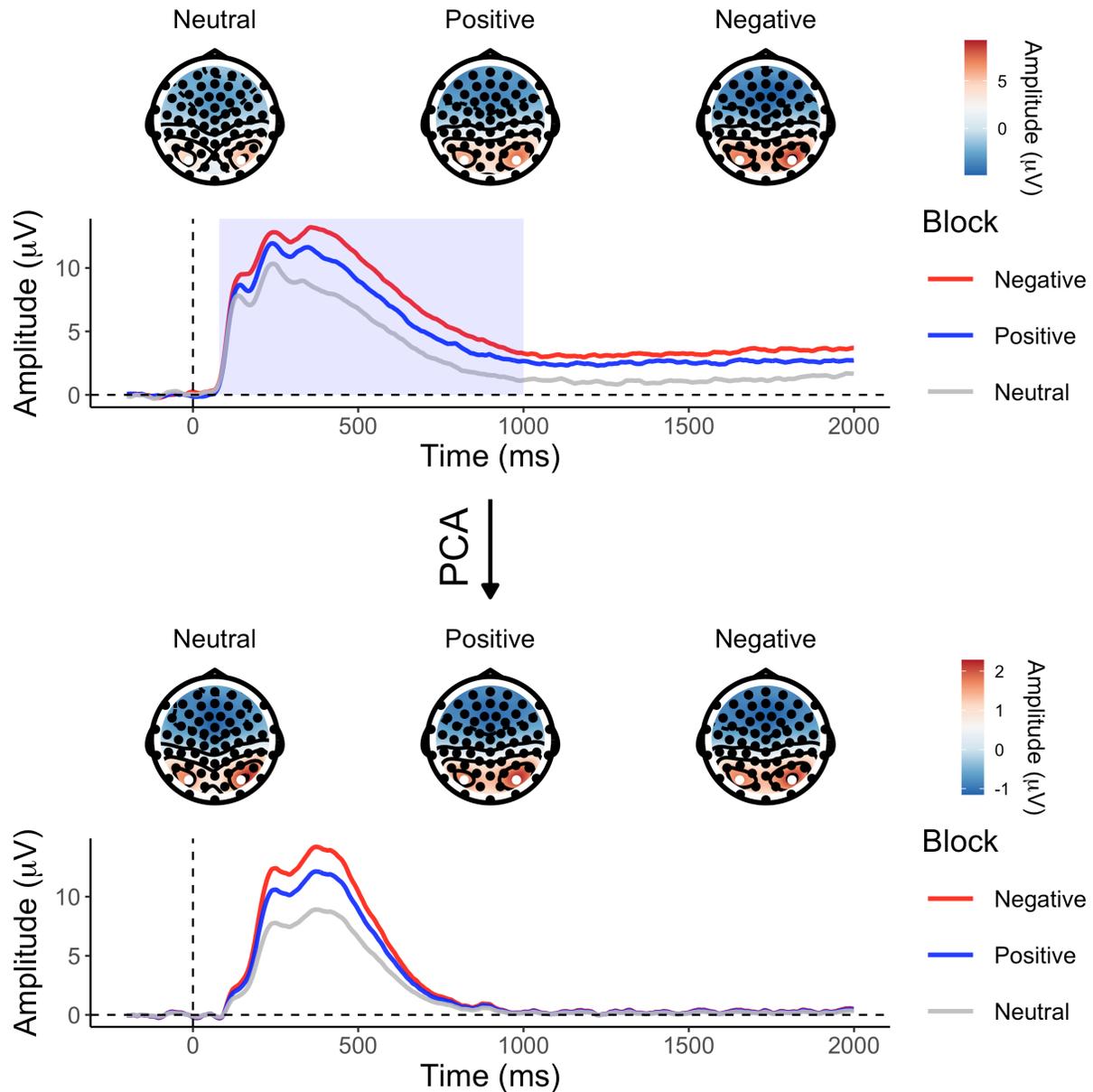


Figure 10. Topographic and ERP plots of the negative 371 ms PCA component before and after temporal PCA. Top row topographic plots represent average activity in the shaded region of the ERP plot below it (80 - 1000 ms). ERP plots represent average activity in highlighted sensors topographic plots (PO7 and PO8).

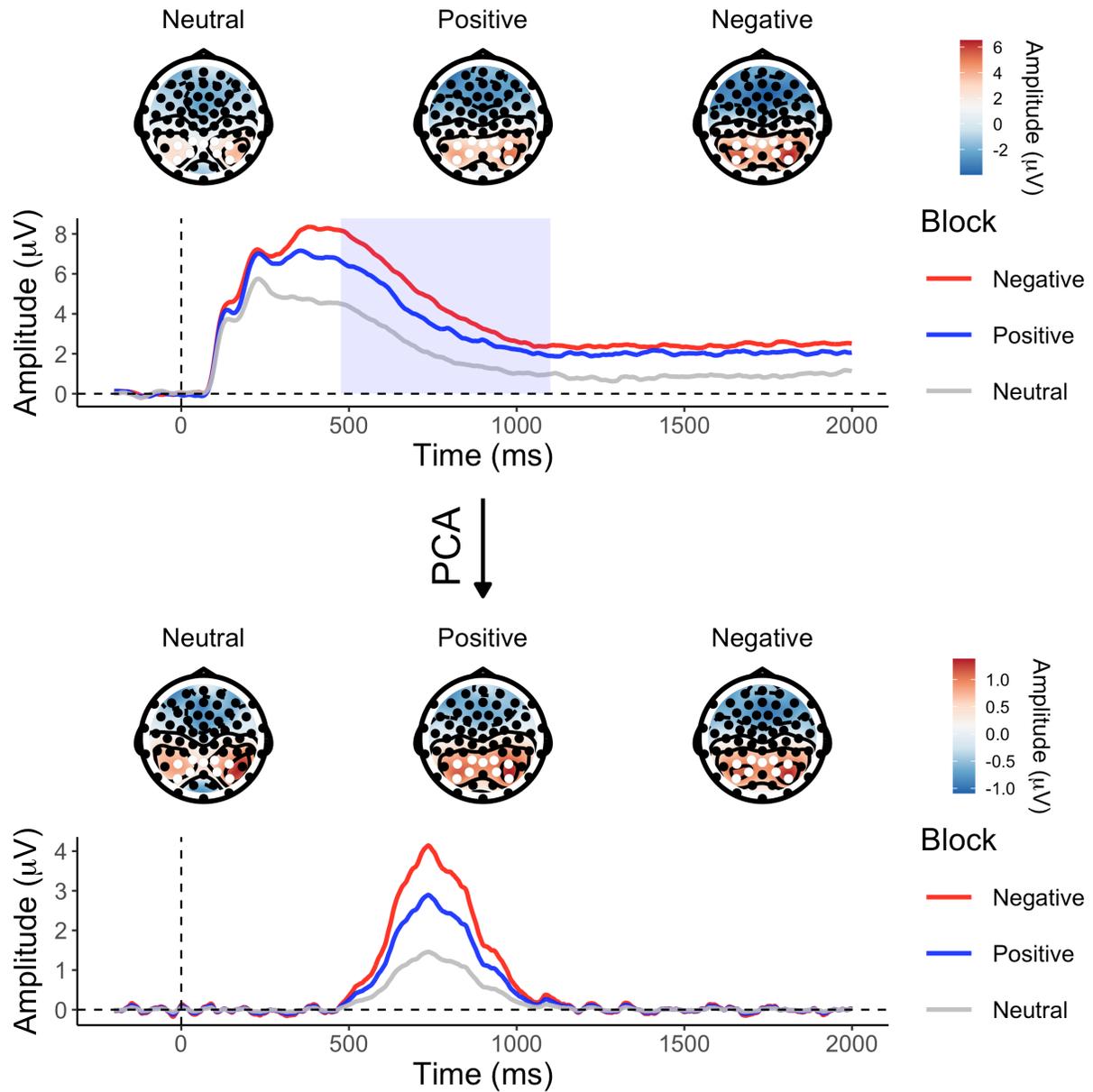


Figure 11. Topographic and ERP plots of the negative 736 ms PCA component before and after temporal PCA. Top row topographic plots represent average activity in the shaded region of the ERP plot below it (475 - 1100 ms). ERP plots represent average activity in highlighted sensors topographic plots (PO7, PO8, PO3, PO4, P1, P2, P3, P4, P5, P6, and Pz).

Analytical Approach

SEM was conducted using the lavaan package (Rosseel, 2012). Further, the first author made extensive use of the dplyr (Wickham et al., 2021) package in data cleaning and manipulation. Tables were constructed using the kableExtra package (Zhu, 2021). Our modeling process largely followed that of Hill and colleagues (2019). Our first stage of modeling was to construct a measurement model with two latent orthogonal factors (*Reactivity*, and *Psychopathology Symptoms*). Positive, negative, and neutral viewing conditions served as indicator variables of the *Reactivity* factor (see Figure 12). Measures of anxious apprehension (PSWQ), anxious arousal (MASQ-AA), and PA (MASQ-PA) served as indicator variables for the *Psychopathology Symptoms* factor. Three separate measurement models were constructed for each ERP component retained from the earlier PCA. Across these measurement models, indicators (positive, negative, and neutral viewing conditions) of the *Reactivity* factor changed to reflect each PCA component being tested, while the *Psychopathology Symptoms* factor remained identical. The variances of both latent variables were fixed to unity (i.e., a variance of one) to set the metric for factor loading and covariance estimates. All loadings were freely estimated, as were the residual variances of each indicator variable. Each of the subsequent competing models were compared to their respective measurement model with a chi-square likelihood ratio test to determine if each model offered superior fit to the data compared to the measurement model.

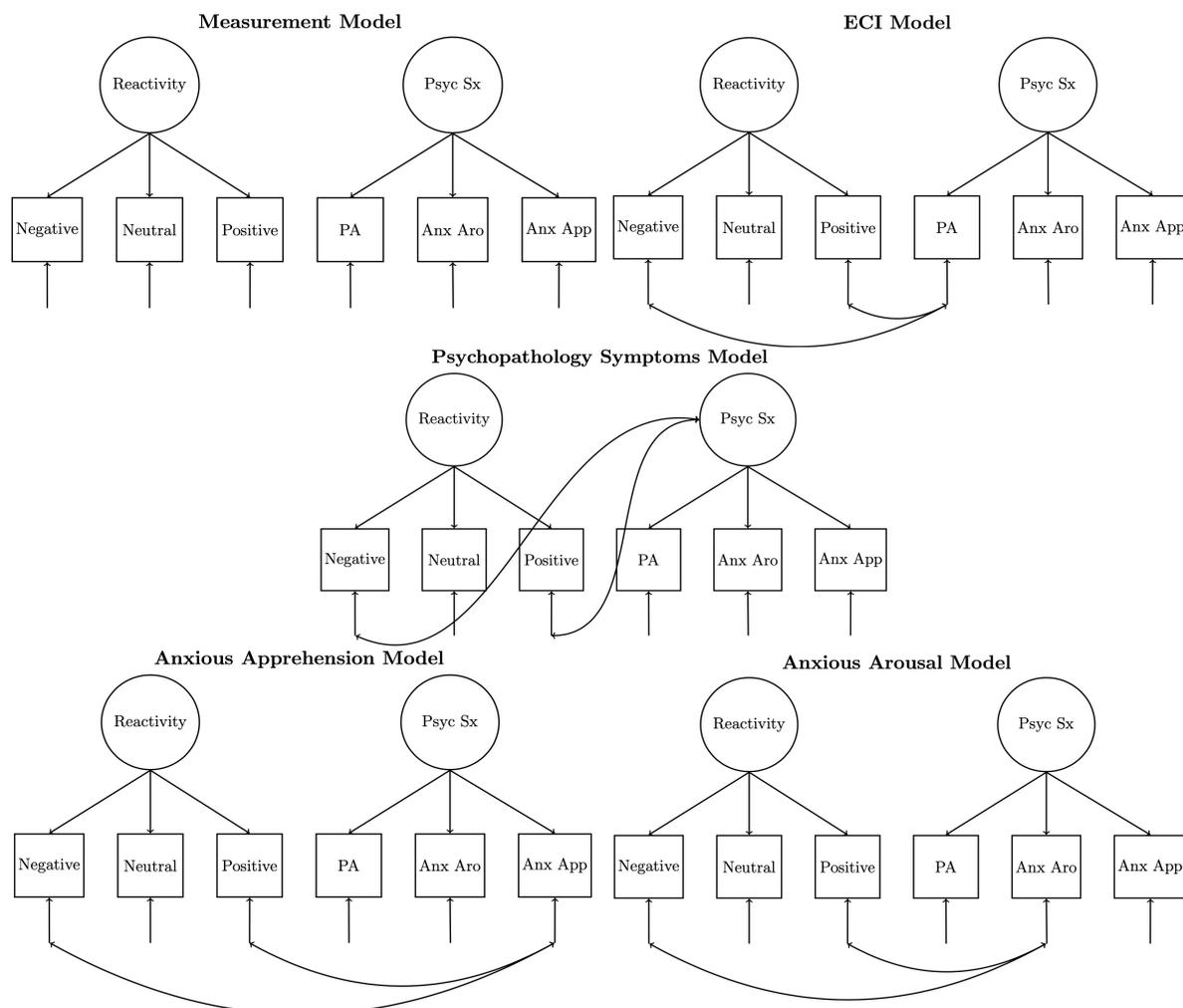


Figure 12. The model diagrams show each model that was fitted to the data for each ERP component retained from the PCA. The Measurement Model (top left) treats the latent Reactivity and Psychopathology Symptoms factors as orthogonal constructs. The ECI Model (top right) estimates covariances among the positive/negative viewing conditions for a given ERP component and positive affectivity. The Psychopathology Symptoms Model Diagram (middle) estimates covariances among positive and negative viewing conditions for a given ERP component and global psychopathology symptoms, rather than PA specifically. The Anxious Apprehension model (bottom left) estimates covariances among positive/negative viewing conditions for a given ERP component and anxious apprehension. Last, the Anxious Arousal Model (bottom right) estimates covariances among positive/negative viewing conditions for a given ERP component and anxious arousal. Psyc Sx = Psychopathology Symptoms, PA = positive affectivity, Anx Aro = anxious arousal, Anx App = Anxious apprehension, ECI = emotion context insensitivity.

We then tested the ECI model by estimating covariances among the positive/negative viewing conditions and PA. While Hill and colleagues (2019) constrained these covariances to be equal, we allowed these values to be freely estimated on the basis that the ECI view makes no hypotheses as to whether the *degree* of blunting is equal for positive and negative images, just the negative directionality. If both estimated covariances were positive (negative for negative peak waveforms) and significant, and if this model offered superior fit to the data compared to the nested comparison measurement model, then we would interpret this as support for the ECI view. Allowing these covariances to be freely estimated allowed us to determine if the positive attenuation or negative potentiation views offered superior fit, as well. If a significant positive (negative for negative peak waveforms) covariance between PA and the positive viewing condition was found with no association with the negative viewing condition, this would be evidence for the positive attenuation theory (in the presence of a significant likelihood ratio test compared to the measurement model). In contrast, if a significant negative (positive for negative peak waveforms) covariance between PA and the negative viewing condition was found in the absence of an association between the positive viewing condition and PA, this would be evidence for the positive attenuation theory (in the presence of a significant likelihood ratio test compared to the measurement model). Note that the positive attenuation and negative potentiation views are not mutually exclusive, and these patterns could be observed simultaneously. Other competing models were directly informed from Hill and colleagues' 2019 study. We then tested a psychopathology symptoms model that estimated covariances among the positive/negative viewing conditions and the latent *Psychopathology Symptoms* factor, which tested whether

emotion reactivity was associated with more general psychopathology symptoms rather than PA specifically. We also tested an anxious arousal model that estimated covariances among the positive/negative viewing conditions and the residual variance of the anxious arousal indicator. This tested the hypothesis of whether reactivity to emotional stimuli was associated with physiological symptoms associated with anxiety specifically. Lastly, we also tested an anxious apprehension model that estimated covariances among the positive/negative viewing conditions and the residual variance of the anxious apprehension indicator. This tested the hypothesis of whether reactivity to emotional stimuli was associated with apprehension symptoms associated with anxiety specifically. See Figure 12 for model diagrams of each of the models described.

All models were estimated using full information maximum likelihood (FIML) with robust (Huber-White) standard errors and a scaled test statistic. Of the 113 cases used for analyses, 10 (8.8%) were missing one questionnaire subscale used for the *Psychopathology Symptoms* latent factor. Inspection of missing cases did not reveal any indications that these data were missing not at random (Enders, 2010), and thus FIML was used to address missing data. Model fit was evaluated with measures of absolute fit, including the goodness-of-fit chi-square value with accompanying degrees of freedom, root mean squared error of approximation (RMSEA) < .08 (Hu & Bentler, 1998), and standardized root mean square residual (SRMR) < .08 (Hu & Bentler, 1998). Fit was also evaluated with measures of relative fit, including non-normed fit index (NNFI) > .90 (Marsh et al., 2004) and the Comparative Fit Index (CFI) > .90 (Marsh et al., 2004). A chi-square difference test was performed between the nested measurement model and its corresponding ECI, psychopathology symptoms, and anxious arousal, and anxious

apprehension model. The chi-square difference test evaluates the equal-fit hypothesis for two hierarchical models. If the chi-square difference value is non-significant, then the two models fit the data equally well and the more parsimonious (i.e., measurement model) is the preferred model. If the chi-square difference value is significant, then the model with fewer degrees of freedom (i.e., not the measurement model) fits the data better and is thus preferred over the measurement model. The ECI, Psychopathology Symptoms, and Anxious Arousal, and Anxious Apprehension models are not hierarchical and thus cannot be compared using a chi-square difference test. Thus, the Akaike information criterion (AIC) and Bayesian information criterion (BIC) will be used to determine best fit among these non-nested models, with lower values indicating superior fit (Markon & Krueger, 2004).

Results

Arousal and Valence Effects

A repeated measures ANOVA was conducted with block type (positive, negative, and neutral emotional stimuli) as the independent variable with Greenhouse-Geisser corrections for deviations from sphericity for each of the three ERP components. Results illustrated that block type explained a significant portion of the variance for each of the three components, 257 ms component: $F(1.95, 218.06) = 50.57, MSE = 0.29, p < .001, \hat{\eta}_G^2 = .111$, 371 ms component: $F(1.78, 199.37) = 148.96, MSE = 0.13, p < .001, \hat{\eta}_G^2 = .125$, and the 736 ms component: $F(1.90, 212.57) = 78.06, MSE = 0.17, p < .001, \hat{\eta}_G^2 = .174$. We also explored linear (valence: comparing pleasant with threat) and quadratic (arousal: comparing pleasant and threat with neutral) orthogonal univariate trends on the block factor in Table 7. Results indicated significant

valence (with greater effects for negative compared to positive emotional stimuli) and arousal effects for each of the components.

Table 7. Summary of Polynomial Contrasts Exploring Linear (Valence: Comparing Positive with Negative) and Quadratic (Arousal: Comparing Positive and Negative with Neutral) Orthogonal Univariate Trends on the Block Factor for each Component.

Contrast	Estimate	SE	df	t	p
<i>257 ms Component</i>					
Linear	0.41	0.07	224	5.91	< .001
Quadratic	-0.99	0.12	224	-8.14	< .001
<i>371 ms Component</i>					
Linear	-0.32	0.04	224	-7.08	< .001
Quadratic	1.22	0.08	224	15.74	< .001
<i>736 ms Component</i>					
Linear	-0.32	0.05	224	-5.99	< .001
Quadratic	1.00	0.09	224	10.96	< .001

Note. Contrast = linear (valence) and quadratic (arousal) contrasts for each ERP component, *SE* = standard error; *df* = degrees of freedom; *t* = *t* ratio; *p* = *p* value.

All parameter estimates are tabulated in Table 8. Model fit indices and chi-square difference test results are contained in Table 9. The anxious arousal, PA, and anxious apprehension variables each loaded significantly onto the *Psychopathology Symptoms* factor, with correlations ranging from moderate to large (Cohen, 1988; .35, -.40, and .89, respectively). While each sequence of models contained a different ERP component, they each contained the same set of indicator variables for the *Psychopathology Symptoms* factor. Thus, the factor loadings for this factor remained the same for each model.

Table 8. Parameter estimates, standard errors, and test statistics from each structural equation model using ERP components retained from PCA ($n = 113$). Each model contains two factors: Reactivity and Psychopathology Symptoms. The Reactivity factor represents the shared variance for each of the passive viewing conditions (positive, negative, and neutral) for each PCA ERP component, and the residual variance of each indicator variable represents that variable's unique variance. The Psychopathology Symptoms factor represents the shared variance of the PA, anxious arousal, and anxious apprehension measures, and the residual variance of each indicator variable represents that variable's unique variance.

Path	257 ms Comp			371 ms Comp			736 ms Comp		
	Est/Std	SE	<i>p</i>	Est/Std	SE	<i>p</i>	Est/Std	SE	<i>p</i>
Measurement Model									
PsySx → Anx Aro	3.10/0.35	1.03	.003	3.10/0.35	1.03	.003	3.10/0.35	1.03	.003
PsySx → PA	-4.23/-0.40	1.63	.010	-4.23/-0.40	1.63	.010	-4.23/-0.40	1.63	.010
PsySx → Anx App	11.89/0.89	3.55	<.001	11.89/0.89	3.55	<.001	11.89/0.89	3.55	<.001
Reactivity → Neutral	0.53/0.71	0.07	<.001	0.67/0.89	0.06	<.001	0.41/0.68	0.06	<.001
Reactivity → Positive	0.77/0.89	0.08	<.001	0.79/0.97	0.06	<.001	0.40/0.81	0.04	<.001
Reactivity → Negative	0.59/0.70	0.08	<.001	0.83/0.90	0.06	<.001	0.49/0.75	0.05	<.001
ECI Model									
Positive ↔ PA	2.40/0.62	0.61	<.001	-0.55/-0.29	0.32	.080	0.20/0.07	0.33	.541
Negative ↔ PA	1.28/0.22	0.85	.130	-0.29/-0.08	0.55	.593	0.36/0.09	0.61	.556
Psychopathology Symptoms Model									
PsySx ↔ Positive	-0.17/-0.43	0.33	.610	-0.02/-0.12	0.04	.587	0.01/0.04	0.04	.781
PsySx ↔ Negative	-0.09/-0.15	0.19	.641	0.01/0.03	0.07	.872	0.01/0.02	0.07	.888
Anxious Arousal Model									
Positive ↔ Anx Aro	-0.36/-0.11	0.67	.591	-0.03/-0.02	0.36	.932	-0.03/-0.02	0.36	.932
Negative ↔ Anx Aro	-0.96/-0.19	0.61	.118	0.30/0.09	0.51	.565	0.30/0.09	0.51	.565
Anxious Apprehension Model									
Positive ↔ Anx App	1.30/0.79	0.75	.083	-0.49/-0.40	0.42	.246	0.15/0.08	0.41	.714
Negative ↔ Anx App	1.02/0.40	1.13	.366	-0.16/-0.07	0.79	.837	0.02/0.01	0.83	.980

Note. Path labels correspond to parameter estimates in each model. Group headings in the 'path' column denote the specific model that the following parameter estimates are unique to. PsySx = Psychological Symptoms latent factor, ECI = emotion context insensitivity, SE = standard error, Est/Std = unstandardized and standardized parameter estimate, → = latent factor loading, ↔ = covariance.

Table 9. Fit measures and chi-square difference test results for each model. Chi-square difference test results represent the comparison of each model with its respective nested measurement model (i.e., initial model with completely orthogonal *Reactivity* and *Psychopathology Symptoms* factors and indicator variables).

Model	χ^2	<i>df</i>	RMSEA	SRMR	CFI	NNFI	AIC	BIC	$\Delta\chi^2$
257 ms Component									
Measurement	18.93	9	0.10	0.08	0.92	0.87	3212.98	3262.08	Nested Measurement Model
Emotion Context Insensitivity	4.72	7	0.00	0.04	1.00	1.00	3201.12	3255.67	$\Delta\chi^2(2) = 13.52, p = .001$
Psychopathology Symptoms	20.64	7	0.13	0.07	0.89	0.77	3210.95	3265.49	$\Delta\chi^2(2) = 2.46, p = .292$
Anxious Arousal	17.02	7	0.11	0.08	0.92	0.83	3214.32	3268.87	$\Delta\chi^2(2) = 2.18, p = .336$
Anxious Apprehension	17.94	7	0.12	0.08	0.92	0.82	3214.08	3268.63	$\Delta\chi^2(2) = 1.08, p = .583$
371 ms Component									
Measurement	5.03	9	0.00	0.04	1.00	1.00	3017.62	3066.71	Nested Measurement Model
Emotion Context Insensitivity	2.84	7	0.00	0.03	1.00	1.00	3019.44	3073.99	$\Delta\chi^2(2) = 2.20, p = .332$
Psychopathology Symptoms	4.57	7	0.00	0.04	1.00	1.00	3020.86	3075.40	$\Delta\chi^2(2) = 0.62, p = .733$
Anxious Arousal	4.46	7	0.00	0.04	1.00	1.00	3020.81	3075.36	$\Delta\chi^2(2) = 0.68, p = .711$
Anxious Apprehension	3.96	7	0.00	0.03	1.00	1.00	3020.32	3074.87	$\Delta\chi^2(2) = 1.08, p = .583$
736 ms Component									
Measurement	5.20	9	0.00	0.04	1.00	1.00	2998.22	3047.31	Nested Measurement Model
Emotion Context Insensitivity	4.91	7	0.00	0.04	1.00	1.00	3001.70	3056.25	$\Delta\chi^2(2) = 0.43, p = .806$
Psychopathology Symptoms	5.32	7	0.00	0.04	1.00	1.00	3002.15	3056.70	$\Delta\chi^2(2) = 0.05, p = .974$
Anxious Arousal	3.99	7	0.00	0.04	1.00	1.00	3000.95	3055.50	$\Delta\chi^2(2) = 1.21, p = .545$
Anxious Apprehension	5.58	7	0.00	0.04	1.00	1.00	3002.10	3056.65	$\Delta\chi^2(2) = 0.09, p = .958$

Note. χ^2 = chi-square, *df* = degrees of freedom, RMSEA = root mean square error of approximation, SRMR = standardized root mean square residual, CFI = comparative fit index, NNFI = non-normed fit index, AIC = Akaike Information Criteria, BIC = Bayesian Information Criteria, $\Delta\chi^2$ = Satorra-Bentler scaled difference chi-square test comparing each model with its respective nested measurement model.

257 ms Component

The two-factor confirmatory factor analysis measurement model with the 257 ms component did not meet standards for acceptable model fit. While the CFI > .90, the SRMR, NNFI, and RMSEA indicated poor fit (Table 9). The positive, negative, and neutral viewing conditions each loaded strongly and significantly on the *Reactivity* factor. When the positive and negative indicators of the *Reactivity* factor were allowed to covary with the PA indicator of the *Psychopathology Symptoms* factor for the ECI model, model fit drastically improved, with all fit measures satisfying criteria for acceptable model fit (Table 9). Further, a Satorra-Bentler scaled difference chi-square test that compared the ECI model with the measurement model indicated superior fit to the data ($\Delta\chi^2(2) = 13.52, p = .001$; Table 9). In other words, the ECI model was

a superior characterization of the covariance structure compared to a fully orthogonal solution. Additionally, there was a large, positive correlation between the positive viewing condition and PA ($r = .61$, $p < .001$; Table 8). Of note, since the 257 ms component has a negative peak, this directionality is in the opposite direction hypothesized in the ECI view of emotion reactivity in depression. More specifically, as self-reported PA increases, the 257 ms attenuates for positive stimuli. No significant relation was found between the 257 ms component negative viewing condition and PA.

Considering that the directionality of the parameter estimates was in the opposite direction hypothesized for the ECI view of emotion reactivity and given the non-significant covariance of the negative viewing condition indicator and PA, we fit another model to the data in a post-hoc exploratory fashion. In this model, we dropped the covariance estimate between the negative viewing condition and PA (i.e., constrained the value to zero), which is in line with the brightening effect described in EMA research (e.g., Khazanov et al., 2019b). This model provided excellent fit to the data ($\chi^2(8) = 7.98$, RMSEA = 0.00, SRMR = 0.05, CFI = 1.00, NNFI = 1.00) that was superior to the nested measurement model ($\Delta\chi^2(1) = 9.43$, $p = .002$). Additionally, this brightening effect model did not result in an appreciable decrement in model fit compared to the ECI model, which has an additional parameter estimate and one fewer degree of freedom ($\Delta\chi^2(1) = 3.38$, $p = .07$). In line with the brightening effect, we found a large positive effect between the positive condition and PA (Est/Std = 1.942/.48, $SE = 0.57$, $z = 3.43$, $p = .001$), such that decreases in PA (i.e., greater depression) would result in enhanced 257 ms component activity in response to positive stimuli. The Psychopathology Symptoms, Anxious Arousal, nor

the Anxious Apprehension model provided acceptable fit to the data (Table 9), and no significant associations were found by freeing parameters in these models (Table 8).

371 ms Component

With the 371 ms component *Reactivity* factor, we found acceptable model fit with the measurement model (Table 9). Each of the positive, negative, and neutral indicator variables loaded strongly onto the *Reactivity* factor ($r = .97, .90, .89$, respectively; Table 8). While each of the subsequent competing models afforded good model fit, none fit the data significantly better than the initial nested measurement model (Table 9). None of the freed parameters unique to each competing model were significant. Of the competing models, the ECI model offered superior fit as indicated by lower AIC and BIC values (Table 9). Overall, we found that an orthogonal solution that separated the *Reactivity* factor and *Psychopathology Symptoms* factor and all indicator variables offered superior fit to the data.

736 ms Component

The 736 ms component measurement model afforded acceptable model fit to the data (Table 9), and positive, negative, and neutral indicator variables loaded significantly and strongly onto the latent *Reactivity* factor ($r = .81, .75, .68$, respectively, Table 8). Similar to the 371 ms component, we found that the initial measurement model had the best fit to the data. While competing models had model fit measures indicating acceptable fit, none constituted significantly improved fit to the data compared to the measurement model (Table 9). Additionally, we found no significant covariance estimates as a result of freeing parameters in each of the competing models.

Discussion

In the present study, we sought to evaluate the temporal course of the neural correlates of emotion reactivity in depression by applying PCA and SEM methods. We examined three separate ERP components retained from a PCA analysis, one that resembled an EPN and two others resembling later components (i.e., P300 and LPP). Our findings illustrated a unique relation between the positive viewing condition for the 257 ms component and PA, such that decreases in PA were associated with enhanced 257 ms amplitude. These findings are consistent with the brightening effect (Khazanov et al., 2019), and suggests that early visual components may be neural indicator of PA. This may be crucial information in devising neuroscience informed interventions for those with low PA and depression.

We hypothesized that EPN activity in response to positive stimuli would be negatively associated with depression symptoms (i.e., lower self-reported PA). We did not make an a priori hypothesis for negative images. However, our primary hypothesis was not supported. Rather, we found large, significant effects in the opposite direction. More specifically, lower levels of PA (i.e., more depression) were associated with enhanced EPN activity. No relation was observed among negative emotion reactivity and PA for EPN, and when this relation was constrained to zero, we obtained a model that provided the best fit to the data (albeit marginally). This pattern of observations does not match any laboratory-based views of emotion reactivity (i.e., positive attenuation, negative potentiation, ECI), which are predicated upon blunted emotion reactivity in response to positive images as a function of depressive symptomatology. However, it does fit with the brightening effect that has been observed in EMA research, or that those with depression

report greater changes in affect in response to positive events (e.g., Khazanov et al., 2019b) compared to healthy controls. Moreover, this effect is specific to MDD compared to anxiety disorders and is specific to positive emotion (Bylsma et al., 2011; Khazanov et al., 2019b; Peeters et al., 2003; Thompson et al., 2012), which is consistent with the present study's unique relation among EPN activity in response to positive stimuli and PA when accounting for anxious arousal and anxious apprehension symptoms. The EPN may be capturing neural activity that has greater ecological validity. A large body of research has found robust relations among EPN and evolutionarily salient stimuli, such as snakes and curvilinear objects (e.g., Langeslag & van Strien, 2018; Strien et al., 2016). This literature has explored relations among EPN and negative stimuli that facilitate survival. It may be that in depression, this pattern of enhanced EPN in response to naturally threatening stimuli is extended to positive stimuli as well. As such, it may be a viable indicator of dysregulated real-world emotion reactivity.

While our hypotheses for later components were in line with previous laboratory research (e.g., Hill et al., 2019), our findings in the present study did not support the ECI view of emotion reactivity (i.e., a negative relation between positive and negative emotion reactivity and PA). With regards to the 371 ms and 736 ms components, no other models outperformed the initial measurement model. In other words, our results supported that these components are distinct from psychopathology symptoms. This finding is inconsistent with a plethora of research that has examined relations between depression symptoms and LPP (Foti et al., 2010; Hill et al., 2019; Proudfit et al., 2015; Weinberg et al., 2017; Weinberg & Sandre, 2018). There are a number of possible factors contributing to the inconsistent findings. We used a block design paradigm in the

present study in contrast to a more ubiquitous randomized design that may interfere with picture processing and produce an oddball effect (Weinberg et al., 2016). Further, our relatively narrow focus on low PA as a symptom of depression may contribute to these differences, as others have analyzed relations among slow wave components and more heterogeneous depression symptomatology (e.g., Foti et al., 2010; Hill et al., 2019; Weinberg et al., 2017). However, Weinberg and colleagues (2016) also used a block design paradigm and observed a blunted LPP effect in response to rewarding images in a community sample. Further, Weinberg and Sandre (2018) investigated more granular features of psychopathology symptoms (i.e., low PA, panic) and observed small partial correlations among P300/LPP and positive images with a sample size of 211. Both of the aforementioned studies utilized windowed averages in their analyses rather than PCA components as the present study has. Research that uses windowed averages in analyses may run the risk of including superimposed ERP signals in independent/dependent variables, which may conflate the constructs of interest. Additionally, SEM allowed us to explicitly model measurement error in constructing our latent *Reactivity* and *Psychopathology Symptoms* variables, which represented variance unique to psychophysiological and psychopathology symptoms of interest (Hill et al., 2019). In conjunction, PCA and SEM allotted greater precision in our constructs, and thus allowed us to observe more specific and refined associations. However, our discrepant findings could also simply be a product of type II error that can only be clarified with additional research. Indeed, the difference in results could be attributed to an amalgamation of differences in samples and analytic choices. Further research should clarify the role of these factors in the neural correlates of depressive symptomatology.

With regards to clinical intervention, targeting early affective processes with specificity to the baseline perception of positive stimuli and events may be effective in treating individuals with low PA. This is supported by research suggesting that depression treatments focusing on enhancing positive emotions may improve efficacy as opposed to solely focusing on mitigating negative emotions (Dunn et al., 2012, 2017). Mindfulness-based meditation practices may represent an important component in interventions that aim to boost positive emotions and target low PA symptoms in individuals with depression. Mindfulness-based interventions (MBIs) emphasize the importance of cultivating non-judgmental attention and awareness in the present moment (Gu et al., 2015), and appear to enhance the capacity for positive emotions (Garland, Farb, et al., 2015; Wielgosz et al., 2019). MBIs may modify positive valence systems through multiple psychological mechanisms, including modulations in emotional reactivity, enhanced emotion awareness, increased use of cognitive reappraisal, and alterations in reward processes (Wielgosz et al., 2019).

A systematic review and meta-analysis conducted by Gu and colleagues (2015) indicated that emotional reactivity and self-reported mindfulness are primary psychological mechanisms driving the efficacy of MBIs for mood disorders. Research has also indicated relations between facets of mindfulness and factors in Clark and Watson's (1991) tripartite model (i.e., PA, NA, and anxious arousal). Studies using cross-sectional data have illustrated negative associations between specific facets of mindfulness (non-reactivity and non-judgment) and both anhedonic depression (low PA) and general distress (high NA) in participants diagnosed with depression and anxiety (Desrosiers et al., 2013). Another study using cross-sectional and

longitudinal methods in a non-clinical sample found inverse relations between the mindfulness facets of acting with awareness and non-judging with PA and NA, which suggests that adopting a non-evaluative disposition may serve to mitigate the experience of negative mood and bolster the experience of positive emotion (Raphiphatthana et al., 2016). The same study showed that acting with awareness predicted lower levels of anhedonia (i.e., higher PA) one month later, indicating that awareness to present activities may facilitate pleasant experience derived from said activities (Raphiphatthana et al., 2016). Other research also supports the importance of awareness in wellbeing. States of distraction are associated with lower levels of wellbeing, impaired executive functioning, and adverse psychological outcomes (Dahl et al., 2020). Future work in our lab will seek to investigate changes in EPN as a function of MBIs to further clarify this component as a modifiable neural correlate of low PA in depression.

Other work by our lab (Kahrilas et al., in prep) has illustrated the blurred lines between the temporal courses of the neural correlates of emotion reactivity and regulation. Thus, incorporating emotion regulation strategies that bolster positive emotions may also serve to repair early reactivity processes associated with low PA. This is corroborated by the notion that the brightening effect may stem from more negative expectations for future events in depression (Strunk et al., 2006). Research has illustrated that low expectation for positive events results in *amplified* reactions to positive stimuli (McNamara et al., 2013). Savoring-based regulatory strategies are also used to sustain and upregulate positive emotions (Heiy & Cheavens, 2014; Liu & Thompson, 2017). While savoring, one may eagerly anticipate future positive experiences, focus on ongoing positive experiences as they occur in the present moment, or reminisce about

past positive experiences. With specific relevance to depression, studies aimed at enhancing savoring capacity show that enriching any of the three temporal domains of savoring (reminiscing, savoring the moment, or anticipating) is associated with increased frequency and intensity of positive affect, and decreased negative affect (Bryant & Veroff, 1984, 2007; Kahrilas et al., 2020). Training on momentary positive emotion regulation (i.e., memory building, expressing positive emotions) resulted in decreased self-reported depression symptoms when compared to a control group after two weeks (Hurley & Kwon, 2012). Intervention studies with older adults have shown that savoring was positively related to decreased depression, increased activity engagement, and increased wellbeing (Smith et al., 2020; Smith & Hanni, 2017). Savoring may improve the capacity to recognize and enjoy positive moments, even during difficult times (Smith & Hanni, 2017). Thus, anticipatory-based interventions aimed at constructing more positive expectations for future events may scaffold more frequent experiences of positive emotions. Another important consideration is whether the brightening effect stems from a low frequency of positive events. In support of this notion, individuals with mood disorders tend to report fewer pleasant events compared to individuals without mood disorders (Bylsma et al., 2011). Enhanced reactivity to positive stimuli, then, may be adaptive as to reconcile the fewer opportunities to erode negative mood. While other researchers have suggested that psychological correlates of low PA emerge only in later attentional stages (Weinberg & Sandre, 2018), our work underscores the importance of considering the role that earlier affective processes play in depression as well.

While previous research has not observed relations between EPN and PA (Weinberg &

Sandre, 2018), it seems reasonable to attribute this to our use of PCA to isolate these components. Visual inspection of waveforms indicated that the 371 ms component (which was not found to be related to PA in the present study) starting at around 70 ms, overlapped considerably with the 271 ms peak EPN. Relying on windowed averages likely would not have successfully captured the EPN component, which may explain these previous null findings. While there is no one best way to measure one's ERP data, we do encourage other ERP researchers to add multivariate techniques such as PCA to their analysis toolbox.

There are several limitations to the present study. First, these data are cross sectional and causation cannot be inferred among neural activity and self-report measures. Future longitudinal research should focus on studying the relations between low PA and associated neurophysiological factors to further clarify the direction of these relations. Further, while our sample contained dimensional variability on constructs of interest (e.g., depression, PA), it is still primarily a white undergraduate sample, which affords limited generalizability. Future research studies should incorporate community samples and participants throughout the lifespan. Related, research has suggested that older individuals may have higher savoring capacity (Smith & Hollinger-Smith, 2015), which may have implications for EPN and PA and inform intervention strategies for older and younger individuals with depression alike. Future work should also employ other EEG methods, such as source analysis, to investigate the time course of specific neural structures and their relation with self-report data. This research will elucidate brain-behavior relations within the context of psychopathology and inform clinical and pharmacological interventions. Last, despite the methodological rigor of our analytical plan, our

results are still merely associations. Future researchers should strive to test more intricate theories of how psychophysiological constructs relate to questionnaire-based data (e.g., mediation and moderation). Such information regarding mechanisms of change among neurophysiological factors and psychopathology will be crucial to developing more refined and targeted clinical interventions. While sample size can be a limitation, SEM is one such analytical framework that is catered to more testing such theories and models. For those researchers with smaller sample sizes that wish to test more complex models, partial least squares structural equation modeling allows for such analyses with reduced demands on sample size (Sarstedt et al., 2020).

Conclusion

The present study examined relations among three ERP components derived from a temporal PCA using SEM. This framework facilitated a more accurate and refined measurement of ERP components. Our findings indicated that an early ERP component with a negative peak at 271 ms had a strong inverse relation with PA, consistent with the brightening effect observed in EMA-based depression research. This finding suggests that interventions aimed at modulating early affective processes associated with emotion reactivity may bolster PA and reduce depressive symptomatology. Mindfulness- and savoring-based interventions may be particularly effective to this end. Future work should extend our findings in a diverse, community-based sample and utilize other EEG analysis methods, such as time-frequency and source analysis. Last, we encourage other researchers to test more complex and refined theories, perhaps within an SEM framework, to provide more information regarding mechanisms of change among neurobiological factors and self-report data. Overall, incorporating these methods may bring us a

step closer to bridging the gap between patterns of self-report symptomatology and their neural correlates, in order to provide a more holistic perspective on developing treatments for those with depression and other forms of psychopathology.

CHAPTER FIVE

DISCUSSION

Broadly, the present set of studies investigated individual differences and neural correlates associated with emotion reactivity and regulation. Study One, “Savoring the moment: A link between affectivity and depression” advanced our understanding of the relations among affectivity (negative and positive), savoring, and depression. More specifically, it investigated the mediating role of the temporal domains of savoring (anticipating, savoring the moment, reminiscing) in the relation between factors of positive affectivity (PA), negative affectivity (NA), and depression. Findings from this study illustrated that PA and NA were associated with depression as well as all three savoring temporal domains. In line with previous research, this suggests that Low PA and high NA are risk factors for depression and that each of the temporal domains of savoring may bolster PA and mitigate NA. Notably, momentary savoring was the only temporal domain of savoring that was associated with depression and distinctly mediated the relationship between both PA and NA and depression. Altogether, this indicates momentary savoring may reduce depression symptoms in individuals with low PA and high NA, more so than future- or past-oriented domains of savoring. This underscores the importance of momentary awareness and upregulation of positive emotions in managing depression as well as the notion that increased attention to positive stimuli is likely one mechanism through which savoring attenuates depression symptoms (Carl et al., 2013).

Study One supports the importance of momentary savoring in managing depression for those with low PA and high NA. However, it did not disentangle the affective chronometry of emotion reactivity and regulation and how these concepts related to PA, which is crucial to devise neuroscience-informed interventions for those with depression. Studies Two and Three were conducted to address these gaps. Study Two, “Neural chronometry of positive and negative emotion reactivity and regulation,” addressed fundamental questions regarding the neural time course associated with reacting to, and regulating, positively- and negatively-valenced emotional stimuli. I created and administered a novel experimental emotion reactivity/regulation paradigm utilizing a block design presentation and a new, open-source set of standardized images (the Open Affective Standardized Image Set, or OASIS; Kurdi et al., 2017).

To extract ERP components throughout the time course of emotion reactivity/regulation, I used an exploratory temporal PCA. The temporal PCA retained five ERP components with peaks ranging from 124 ms to 740 ms. With regards to emotion reactivity (i.e., changes among passively viewing different valence categories of images), differences were noted as early as 162 ms through the entire time course. Negative and positive emotional stimuli elicited enhanced component amplitudes at 162 ms, 381 ms, and 740 ms. Negative emotional stimuli elicited augmented amplitude relative to neutral stimuli at 259 ms, though positive emotion stimuli did not. Valence effects (i.e., differences between arousal-matched negative and positive images) were noted, such that negative images consistently elicited enhanced amplitudes of all retained components throughout.

Results illustrated a temporal cascade of ERP components sensitive to emotion reactivity

in the first 1000 ms of neural activity, which may serve as modifiable intervention targets in psychopathology. Differences in chronometry emerged in emotion regulation contingent upon valence. Negative regulatory processes unfolded in the 124 ms to 259 ms, while positive regulatory processes occurred later from 259 ms to 740 ms. This may imply that those with impairment in positive emotion regulation and negative emotion regulation might benefit from interventions that focus on different processes occurring within the neural time course. Only increase conditions (i.e., differences between increasing emotional intensity in response to stimuli and passively watching them) were observed; decreasing emotional intensity in response to stimuli did not lead to reductions in component amplitudes. This may be attributed to our block design paradigm. In sum, Study Two underscored the importance of attending to the entire temporal course of emotion reactivity and regulation, which provides a more holistic view on the temporal cascade of emotional neural processing that may highlight modifiable mechanisms with relevance to depression. Further, it brought attention to the topic of measurement within ERPs with a temporal PCA approach that mitigates conflation of overlapping ERP components.

The third study in the series, “Early neural indicators of the brightening effect” built upon findings from Study Two and investigated associations among neural chronometry of emotion reactivity and PA. To obtain dimensional measures of our variables, we harmonized the sample from Study Two with a sample recruited from the SMiLe study, an eight-week mindfulness-based meditation intervention study using the Headspace smart phone app. The SMiLe study recruited participants endorsing significant depressive symptomatology (i.e., PHQ-9 > 10) from a college student population. We utilized that same experimental paradigm and temporal PCA method from

Study Two to guide our selection of ERP components.

We tested competing views of emotion reactivity in depression with SEM as informed by a previous study conducted by Hill and Foti (2019) in three separate ERP components extracted from PCA. We found that an early visual component with a negative peak at 257 ms at centroparietal electrode sites was associated with positive affectivity, such that increases in positive affectivity were associated with attenuated 257 ms component amplitudes. Further, we found that this model offered superior fit to the data compared to competing models. Later components were not associated with positive affectivity, and an orthogonal model that fixed associations between later components and internalizing symptoms to zero fit the data better than other competing models. While we hypothesized that a pattern of results consistent with the emotion context insensitivity view would emerge, we found that lower levels of positive affectivity (i.e., greater depressive symptomatology) was associated with enhanced 257 ms component activity. While inconsistent with previous laboratory findings, this does fit with the brightening effect observed in ecological momentary assessment research that observes enhanced reactivity in response to positive events for those with depression (e.g., Khazanov et al., 2019). It may be that these early neural processes are capturing activity that is more representative of day-to-day lived experiences, which is corroborated by research suggesting that EPN reflects relatively automatic processes observed even when processing resources are limited due to rapid presentation rates (Junghöfer et al., 2001; Schupp, Markus, et al., 2003; Schupp, Junghöfer, et al., 2003). Targeting early reactive processes with specificity to positive events, then, may be effective in treating those with low PA and depression.

Future Directions

Targeting early reactive processes with specificity to positive events may be effective in treating those with low PA and depression. One potentially fruitful venture would be to explore the role of mindfulness-based interventions in modifying positive affectivity and early neural activity. Mindfulness meditation is theorized to enhance the experience of positive emotions (Garland et al., 2015; Wielgosz et al., 2019). It is also theorized to promote wellbeing (Dahl et al., 2015) and modify positive valence systems through enhanced emotion awareness, modulations in emotional reactivity, increased use of cognitive reappraisal, and alterations in reward processes (Wielgosz et al., 2019). Other studies have illustrated associations between specific facets of mindfulness (e.g., directing attention to present activity, non-reactivity, and non-judgment) and PA in participants with depression and anxiety (Desrosiers et al., 2013; Moore & Malinowski, 2009, Raphiphatthana et al., 2016). Therefore, mindfulness may bolster PA and modify associated early neural processes.

The PCA approach in Study Three suggests the existence of separable temporal courses for neural generators; yet EEG scalp analyses provide minimal information regarding the spatial resolution of these generators. Therefore, future work should also employ other EEG analytic methods, such as source analysis, to investigate the time course of specific neural structures and their relation with variables pertaining to positive emotion in depression. For example, potential neural generators of the EPN lie in lateral occipital cortex and the frontoparietal network (Frank & Sabatinelli, 2019; Junghöfer et al., 2001). These specific brain regions may be modifiable intervention targets that serve to bolster wellbeing in depression.

Future research should also continue using advanced statistical methods such as PCA and SEM. To improve upon the analytic strategy from Study Three, which estimates covariances among residual variances of indicator variables that may contain measurement error, experimental paradigm blocks with similar regulatory goals (e.g., to maximize positive emotion/mitigate negative emotion and maximize negative emotion/mitigate positive emotion) can be specified as indicators of latent variables that are devoid of measurement error. The positive emotion latent factor can then be regressed on the negative emotion latent factor to derive the residual variance of the former. For self-report data, indicators could be specified to construct a latent positive emotion construct (e.g., positive affectivity, savoring capacity) while another latent factor for anxiety symptoms could be constructed. Then, a second order internalizing factor could be specified that explains the shared variance of the positive emotion and anxiety latent factors. The residual variance of the positive emotion factor, then, could be regressed on the positive neural reactivity factor. This association will be free of measurement error and more specific with regards to the constructs of interest. Building upon this approach, future work should investigate possible modifiable mechanisms that link positive emotion and early neural processes. For example, since the brightening effect may stem from negative expectations for future positive events for those with depression (Khazanov et al., 2019; Strunk et al., 2006), the ability to anticipate future positive events may constitute a modifiable psychological mechanism. Interventions targeted at bolstering anticipatory processes, then, may influence the relation between early neural and positive affectivity.

REFERENCE LIST

- Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*(2), 217–237. <https://doi.org/10.1016/j.cpr.2009.11.004>
- Amaral, D. G., Price, J. L., Pitkanen, A., & Carmichel, S. T. (1992). Anatomical organization of the primate amygdaloid complex. In *The amygdala: Neurobiological aspects of emotion, attention, and mental dysfunction* (pp. 1–66). Wiley.
- Andrews, V. H., & Borkovec, T. D. (1988). The differential effects of inductions of worry, somatic anxiety, and depression on emotional experience. *Journal of Behavior Therapy and Experimental Psychiatry, 19*(1), 21–26. [https://doi.org/10.1016/0005-7916\(88\)90006-7](https://doi.org/10.1016/0005-7916(88)90006-7)
- Argyropoulos, S. V., & Nutt, D. J. (2013). Anhedonia revisited: Is there a role for dopamine-targeting drugs for depression? *Journal of Psychopharmacology, 27*(10), 869–877. <https://doi.org/10.1177/0269881113494104>
- American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*.
- Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software, 67*(1), 1–48. <https://doi.org/10.18637/jss.v067.i01>
- Beck, A. T. (1979). *Cognitive therapy and the emotional disorders*. Penguin.
- Bekhtereva, V., Craddock, M., & Müller, M. M. (2015). Attentional bias to affective faces and complex IAPS images in early visual cortex follows emotional cue extraction. *NeuroImage, 112*, 254–266. <https://doi.org/10.1016/j.neuroimage.2015.03.052>
- Berinsky, A. J., Huber, G. A., & Lenz, G. S. (2012). Evaluating online labor markets for experimental research: Amazon. Com’s Mechanical Turk. *Political Analysis, 20*(3), 351–368. <https://doi.org/http://dx.doi.org/10.1093/pan/mpr057>
- Blashill, A. J., & Vander Wal, J. S. (2010). The role of body image dissatisfaction and depression on HAART adherence in HIV positive men: Tests of mediation models. *AIDS and*

Behavior, 14(2), 280–288. <https://doi.org/10.1007/s10461-009-9630-2>

- Blau, V. C., Maurer, U., Tottenham, N., & McCandliss, B. D. (2007). The face-specific N170 component is modulated by emotional facial expression. *Behavioral and Brain Functions*, 3(1), 7. <https://doi.org/10.1186/1744-9081-3-7>
- Bradley, M. M., & Lang, P. J. (2007). The International Affective Picture System (IAPS) in the Study of Emotion and Attention. In *Handbook of Emotion Elicitation and Assessment* (pp. 29–46). Oxford University Press.
- Bradley, M. M., Sabatinelli, D., Lang, P. J., Fitzsimmons, J. R., King, W., & Desai, P. (2003). Activation of the visual cortex in motivated attention. *Behavioral Neuroscience*, 117(2), 369–380. <https://doi.org/10.1037/0735-7044.117.2.369>
- Bredemeier, K., Spielberg, J. M., Silton, R. L., Berenbaum, H., Heller, W., & Miller, G. A. (2010). Screening for depressive disorders using the Mood and Anxiety Symptoms Questionnaire Anhedonic Depression Scale: A receiver-operating characteristic analysis. *Psychological Assessment*, 22(3), 702–710. <https://doi.org/10.1037/a0019915>
- Bryant, F. B. (1989). A four-factor model of perceived control: Avoiding, coping, obtaining, and savoring. *Journal of Personality*, 57(4), 773–797. <https://doi.org/http://dx.doi.org/10.1111/j.1467-6494.1989.tb00494.x>
- Bryant, F. B. (2003). Savoring Beliefs Inventory (SBI): A scale for measuring beliefs about savouring. *Journal of Mental Health*, 12(2), 175–196. <https://doi.org/10.1080/0963823031000103489>
- Bryant, F. B., Chadwick, E. D., & Kluwe, K. (2011). Understanding the processes that regulate positive emotional experience: Unsolved problems and future directions for theory and research on savoring. *International Journal of Wellbeing*, 1(1). <https://doi.org/10.5502/ijw.v1i1.18>
- Bryant, F. B., Smart, C. M., & King, S. P. (2005). Using the past to enhance the present: Boosting happiness through positive reminiscence. *Journal of Happiness Studies*, 6(3), 227–260. <https://doi.org/10.1007/s10902-005-3889-4>
- Bryant, F. B., & Veroff, J. (2007). *Savoring: A new model of positive experience*. Lawrence Erlbaum Associates Publishers.
- Buhrmester, M., Kwang, T., & Gosling, S. D. (2011). Amazon's mechanical turk: A new source of inexpensive, yet high-quality, data? *Perspectives on Psychological Science*, 6(1), 3–5. <https://doi.org/10.1177/1745691610393980>

- Burdwood, E. N., Infantolino, Z. P., Crocker, L. D., Spielberg, J. M., Banich, M. T., Miller, G. A., & Heller, W. (2016). Resting-state functional connectivity differentiates anxious apprehension and anxious arousal. *Psychophysiology*, *53*(10), 1451–1459. <https://doi.org/10.1111/psyp.12696>
- Bylsma, L. M., Morris, B. H., & Rottenberg, J. (2008). A meta-analysis of emotional reactivity in major depressive disorder. *Clinical Psychology Review*, *28*(4), 676–691. <https://doi.org/10.1016/j.cpr.2007.10.001>
- Bylsma, L. M., Taylor-Clift, A., & Rottenberg, J. (2011). Emotional reactivity to daily events in major and minor depression. *Journal of Abnormal Psychology*, *120*(1), 155–167. <https://doi.org/10.1037/a0021662>
- Calder, A. J., & Young, A. W. (2005). Understanding the recognition of facial identity and facial expression. *Nature Reviews Neuroscience*, *6*(8), 641–651. <https://doi.org/10.1038/nrn1724>
- Carl, J. R., Soskin, D. P., Kerns, C., & Barlow, D. H. (2013). Positive emotion regulation in emotional disorders: A theoretical review. *Clinical Psychology Review*, *33*(3), 343–360. <https://doi.org/10.1016/j.cpr.2013.01.003>
- Carver, C. S., & Johnson, S. L. (2009). Tendencies toward mania and tendencies toward depression have distinct motivational, affective, and cognitive correlates. *Cognitive Therapy and Research*, *33*, 552. <https://doi.org/10.1007/s10608-008-9213-y>
- Chan, D. (2009). *Statistical and methodological myths and urban legends: Doctrine, verity and fable in the organizational and social sciences*. Taylor & Francis.
- Chan, S. W. Y., Goodwin, G. M., & Harmer, C. J. (2007). Highly neurotic never-depressed students have negative biases in information processing. *Psychological Medicine*, *37*(9), 1281–1291. <https://doi.org/10.1017/S0033291707000669>
- Chen, J., Ma, W., Zhang, Y., Wu, X., Wei, D., Liu, G., Deng, Z., Yang, L., & Zhang, Z. (2014). Distinct facial processing related negative cognitive bias in first-episode and recurrent major depression: Evidence from the N170 ERP component. *PLoS ONE*, *9*(10), e109176. <https://doi.org/10.1371/journal.pone.0109176>
- Chorpita, B. F. (2002). The tripartite model and dimensions of anxiety and depression: An examination of structure in a large school sample. *Journal of Abnormal Child Psychology*, *30*(2), 177–190. <https://doi.org/10.1023/A:1014709417132>
- Clark, L. A. (1989). The anxiety and depressive disorders: Descriptive psychopathology and differential diagnosis. In *Personality, psychopathology, and psychotherapy. Anxiety and depression: Distinctive and overlapping features* (pp. 83–129). Academic Press.

- Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *American Psychological Association, 100*(3), 316–336. <https://doi.org/http://dx.doi.org/10.1037/0021-843X.100.3.316>
- Clark, L. A., Watson, D., & Reynolds, S. (1995). Diagnosis and classification of psychopathology: Challenges to the current system and future directions. *Annual Review of Psychology, 46*(1), 121–153.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed). L. Erlbaum Associates.
- Connell, A. M., Patton, E., & McKillop, H. (2015). Binge drinking, depression, and electrocortical responses to emotional images. *Psychology of Addictive Behaviors, 29*(3), 673–682. <https://doi.org/10.1037/adb0000071>
- Costa, P. T., & McCrae, R. R. (1980). Influence of extraversion and neuroticism on subjective well-being: Happy and unhappy people. *Journal of Personality and Social Psychology, 38*(4), 668–678. <https://doi.org/10.1037/0022-3514.38.4.668>
- Cronbach, L. J., & Furby, L. (1970). How we should measure "change": Or should we? *Psychological Bulletin, 74*(1), 68–80. <https://doi.org/10.1037/h0029382>
- Cuthbert, B. N., Schupp, H. T., Bradley, M. M., Birbaumer, N., & Lang, P. J. (2000). Brain potentials in affective picture processing: Covariation with autonomic arousal and affective report. *Biological Psychology, 52*(2), 95–111. [https://doi.org/http://dx.doi.org/10.1016/S0301-0511\(99\)00044-7](https://doi.org/http://dx.doi.org/10.1016/S0301-0511(99)00044-7)
- Dahl, C. J., Lutz, A., & Davidson, R. J. (2015). Reconstructing and deconstructing the self: Cognitive mechanisms in meditation practice. *Trends in Cognitive Sciences, 19*(9), 515–523. <https://doi.org/10.1016/j.tics.2015.07.001>
- Dahl, C. J., Wilson-Mendenhall, C. D., & Davidson, R. J. (2020). The plasticity of well-being: A training-based framework for the cultivation of human flourishing. *Proceedings of the National Academy of Sciences, 117*(51), 32197–32206. <https://doi.org/10.1073/pnas.2014859117>
- Dai, Q., & Feng, Z. (2012). More excited for negative facial expressions in depression: Evidence from an event-related potential study. *Clinical Neurophysiology, 123*(11), 2172–2179. <https://doi.org/10.1016/j.clinph.2012.04.018>
- daSilva, E. B., Crager, K., & Puce, A. (2016). On dissociating the neural time course of the processing of positive emotions. *Neuropsychologia, 83*, 123–137. <https://doi.org/10.1016/j.neuropsychologia.2015.12.001>

- Davidson, R. J. (2018). Reactivity, recovery, regulation: The three R's of emotional responding. In A. S. Fox, R. C. Lapate, A. J. Shackman, & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (Second, pp. 71–73). Oxford University Press.
- Davidson, R. J. (1998). Affective style and affective disorders: Perspectives from affective neuroscience. *Cognition & Emotion*, *12*(3), 307–330.
<https://doi.org/10.1080/026999398379628>
- De Fruyt, J., Sabbe, B., & Demyttenaere, K. (2020). Anhedonia in depressive disorder: A narrative review. *Psychopathology*, *53*(5-6), 274–281. <https://doi.org/10.1159/000508773>
- Demenescu, L. R., Renken, R., Kortekaas, R., van Tol, M.-J., Marsman, J. B. C., van Buchem, M. A., van der Wee, N. J. A., Veltman, D. J., den Boer, J. A., & Aleman, A. (2011). Neural correlates of perception of emotional facial expressions in out-patients with mild-to-moderate depression and anxiety. A multicenter fMRI study. *Psychological Medicine*, *41*(11), 2253–2264. <https://doi.org/10.1017/S0033291711000596>
- Demyttenaere, K., Donneau, A.-F., Albert, A., Anseau, M., Constant, E., & van Heeringen, K. (2015). What is important in being cured from depression? Discordance between physician and patients (1). *Journal of Affective Disorders*, *174*, 390–396.
<https://doi.org/10.1016/j.jad.2014.12.004>
- Dien, J. (1998). Addressing misallocation of variance in principal components analysis of event-related potentials. *Brain Topography*, *11*(1), 13.
<https://doi.org/10.1023/A:1022218503558>
- Dien, J. (2010). The ERP PCA Toolkit: An open source program for advanced statistical analysis of event-related potential data. *Journal of Neuroscience Methods*, *187*(1), 138–145.
<https://doi.org/10.1016/j.jneumeth.2009.12.009>
- Dien, J. (2012). Applying principal components analysis to event-related potentials: A tutorial. *Developmental Neuropsychology*, *37*(6), 497–517.
<https://doi.org/10.1080/87565641.2012.697503>
- Dien, J., Khoe, W., & Mangun, G. R. (2007). Evaluation of PCA and ICA of simulated ERPs: Promax vs. infomax rotations. *Human Brain Mapping*, *28*(8), 742–763.
<https://doi.org/10.1002/hbm.20304>
- Dillon, D. G., Cooper, J. J., Grent-'t-Jong, T., Woldorff, M. G., & LaBar, K. S. (2006). Dissociation of event-related potentials indexing arousal and semantic cohesion during emotional word encoding. *Brain and Cognition*, *62*(1), 43–57.
<https://doi.org/10.1016/j.bandc.2006.03.008>

- Dolcos, F., & Cabeza, R. (2002). Event-related potentials of emotional memory: Encoding pleasant, unpleasant, and neutral pictures. *Cognitive, Affective, & Behavioral Neuroscience*, 2(3), 252–263.
- Donchin, E., & Heffley, E. F. (1978). Multivariate analysis of event-related potential data: A tutorial review. In D. Otto (Ed.), *Multidisciplinary perspectives in event-related brain potentials research* (pp. 555–572). Multidisciplinary perspectives in event-related brain potentials research.
- DimidjDr. Kahrilas, S., Hollon, S. D., Dobson, K. S., Schmalin, K. B., Kohlenberg, R. J., Addis, M. E., Gallop, R., McGlinchey, J. B., Markley, D. K., Gollan, J. K., Atkins, D. C., Dunner, D. L., & Jacobson, N. S. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology*, 74(4), 658–670. <https://doi.org/10.1037/0022-006X.74.4.658>
- DeRubeis, R. J., Hollon, S. D., Amsterdam, J. D., Shelton, R. C., Young, P. R., Salomon, R. M., O'Reardon, J. P., Lovett, M. L., Gladis, M. M., Brown, L. L., & Gallop, R. (2005). Cognitive therapy vs medications in the treatment of moderate to severe depression. *Archives of General Psychiatry*, 62, 8.
- Desrosiers, A., Klemanski, D. H., & Nolen-Hoeksema, S. (2013). Mapping mindfulness facets onto dimensions of anxiety and depression. *Behavior Therapy*, 44(3), 373–384. <https://doi.org/10.1016/j.beth.2013.02.001>
- DimidjDr. Kahrilas, S., Hollon, S. D., Dobson, K. S., Schmalin, K. B., Kohlenberg, R. J., Addis, M. E., Gallop, R., McGlinchey, J. B., Markley, D. K., Gollan, J. K., Atkins, D. C., Dunner, D. L., & Jacobson, N. S. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology*, 74(4), 658–670. <https://doi.org/10.1037/0022-006X.74.4.658>
- Donohue, J. M., & Pincus, H. A. (2007). Reducing the societal burden of depression: A review of economic costs, quality of care and effects of treatment. *Pharmacoeconomics*, 25(1), 7–24. <https://doi.org/10.2165/00019053-200725010-00003>
- Druss, B. G., Hwang, I., Petukhova, M., Sampson, N. A., Wang, P. S., & Kessler, R. C. (2009). Impairment in role functioning in mental and chronic medical disorders in the United States: Results from the National Comorbidity Survey Replication. *Molecular Psychiatry*, 14(7), 728–737. <https://doi.org/10.1038/mp.2008.13>
- Dunn, B. D., Burr, L. A., Smith, H. B., Hunt, A., Dadgostar, D., Dalglis, L., Smith, S., Attree, E., Jell, G., Martyn, J., Bos, N., & Werner-Seidler, A. (2018). Turning gold into lead:

Dampening appraisals reduce happiness and pleasantness and increase sadness during anticipation and recall of pleasant activities in the laboratory. *Behaviour Research and Therapy*, *107*, 19–33. <https://doi.org/10.1016/j.brat.2018.05.003>

Dunn, B. D., German, R. E., Khazanov, G., Xu, C., Hollon, S. D., & DeRubeis, R. J. (2019). Changes in positive and negative affect during pharmacological treatment and cognitive therapy for major depressive disorder: A secondary analysis of two randomized controlled trials. *Clinical Psychological Science*, *216770261986342*. <https://doi.org/10.1177/2167702619863427>

Dunning, J. P., & Hajcak, G. (2009). See no evil: Directing visual attention within unpleasant images modulates the electrocortical response. *Psychophysiology*, *46*(1), 28–33. <https://doi.org/http://dx.doi.org/10.1111/j.1469-8986.2008.00723.x>

Eisner, L. R., Johnson, S. L., & Carver, C. S. (2009). Positive affect regulation in anxiety disorders. *Journal of Anxiety Disorders*, *23*(5), 645–649. <https://doi.org/10.1016/j.janxdis.2009.02.001>

Ekman, P. (1992). An argument for basic emotions. *Cognition and Emotion*, *6*, 654–660.

Ellis, A. J., Fischer, K. M., & Beevers, C. G. (2010). Is dysphoria about being *red* and *blue*? Potentiation of anger and reduced distress tolerance among dysphoric individuals. *Cognition & Emotion*, *24*(4), 596–608. <https://doi.org/10.1080/13803390902851176>

Enders, C. K. (2010). An introduction to missing data. In *Applied missing data analysis* (pp. 1–36). Guilford Press.

Epstein, J., Pan, H., Kocsis, J. H., Yang, Y., Butler, T., Chusid, J., Hochberg, H., Murrough, J., Strohmayr, E., Stern, E., & Silbersweig, D. A. (2006). Lack of ventral striatal response to positive stimuli in depressed versus normal subjects. *American Journal of Psychiatry*, *7*.

Evans-Lacko, S., & Knapp, M. (2016). Global patterns of workplace productivity for people with depression: Absenteeism and presenteeism costs across eight diverse countries. *Social Psychiatry and Psychiatric Epidemiology*, *51*(11), 1525–1537. <https://doi.org/10.1007/s00127-016-1278-4>

Fales, C. L., Barch, D. M., Rundle, M. M., Mintun, M. A., Snyder, A. Z., Cohen, J. D., Mathews, J., & Sheline, Y. I. (2008). Altered emotional interference processing in affective and cognitive-control brain circuitry in major depression. *Biological Psychiatry*, *63*(4), 377–384. <https://doi.org/10.1016/j.biopsych.2007.06.012>

Feldman, G. C., Joormann, J., & Johnson, S. L. (2008). Responses to positive affect: A self-report measure of rumination and dampening. *Cognitive Therapy and Research*, *32*(4),

507. <https://doi.org/http://dx.doi.org/10.1007/s10608-006-9083-0>

- Fisher, R. J. (1993). Social desirability bias and the validity of indirect questioning. *Journal of Consumer Research*, 20(2), 303–315. <https://doi.org/https://doi.org/10.1086/209351>
- Fliessbach, K., Weber, B., Trautner, P., Dohmen, T., Sunde, U., Elger, C. E., & Falk, A. (2007). Social comparison affects reward-related brain activity in the human ventral striatum. *Science*, 318(5854), 1305–1308.
- Foti, D., & Hajcak, G. (2008). Deconstructing reappraisal: Descriptions preceding arousing pictures modulate the subsequent neural response. *Journal of Cognitive Neuroscience*, 20(6), 977–988. <https://doi.org/http://dx.doi.org/10.1162/jocn.2008.20066>
- Forbes, E. E., & Dahl, R. E. (2005). Neural systems of positive affect: Relevance to understanding child and adolescent depression? *Development and Psychopathology*, 17(3), 827–850. <https://doi.org/10.1017/S095457940505039X>
- Foti, D., & Hajcak, G. (2008). Deconstructing reappraisal: Descriptions preceding arousing pictures modulate the subsequent neural response. *Journal of Cognitive Neuroscience*, 20(6), 977–988. <https://doi.org/http://dx.doi.org/10.1162/jocn.2008.20066>
- Foti, D., Hajcak, G., & Dien, J. (2009). Differentiating neural responses to emotional pictures: Evidence from temporal-spatial PCA. *Psychophysiology*, 46(3), 521–530. <https://doi.org/http://dx.doi.org/10.1111/j.1469-8986.2009.00796.x>
- Foti, D., Olvet, D. M., Klein, D. N., & Hajcak, G. (2010). Reduced electrocortical response to threatening faces in major depressive disorder. *Depression and Anxiety*, 27(9), 813–820. <https://doi.org/10.1002/da.20712>
- Frank, D. W., & Sabatinelli, D. (2019). Hemodynamic and electrocortical reactivity to specific scene contents in emotional perception. *Psychophysiology*, 56(6), e13340. <https://doi.org/10.1111/psyp.13340>
- Fu, C. H. Y., Williams, S. C. R., Cleare, A. J., Brammer, M. J., Walsh, N. D., Kim, J., Andrew, C. M., Pich, E. M., Williams, P. M., Reed, L. J., Mitterschiffthaler, M. T., Suckling, J., & Bullmore, E. T. (2004). Attenuation of the neural response to sad faces in major depression by antidepressant treatment: A prospective, event-related functional magnetic resonance imaging study. *Archives of General Psychiatry*, 61(9), 877. <https://doi.org/10.1001/archpsyc.61.9.877>
- Garland, E. L., Farb, N. A., Goldin, P. R., & Fredrickson, B. L. (2015). Mindfulness broadens awareness and builds eudaimonic meaning: A process model of mindful positive emotion regulation. *Psychological Inquiry*, 26(4), 293–314.

<https://doi.org/10.1080/1047840X.2015.1064294>

- Garnefski, N., & Kraaij, V. (2007). The cognitive emotion regulation questionnaire. *European Journal of Psychological Assessment, 23*(3), 141–149.
<https://doi.org/10.1027/1015-5759.23.3.141>
- Gaunt, R., & Scott, J. (2014). Parents' involvement in childcare: Do parental and work identities matter? *Psychology of Women Quarterly, 38*(4), 475–489.
<https://doi.org/10.1177/0361684314533484>
- Gentzler, A. L., Morey, J. N., Palmer, C. A., & Yi, C. Y. (2013). Young adolescents' responses to positive events: Associations with positive affect and adjustment. *The Journal of Early Adolescence, 33*(5), 663–683. <https://doi.org/10.1177/0272431612462629>
- George, D., & Mallery, P. (2003). *SPSS for Windows step by step: A simple guide and reference* (4th ed.). Allyn & Bacon.
- Goodin, B. R., McGuire, L. M., Stapleton, L. M., Quinn, N. B., FabDr. Kahrilas, L. A., Haythornthwaite, J. A., & Edwards, R. R. (2009). Pain catastrophizing mediates the relation between self-reported strenuous exercise involvement and pain ratings: The moderating role of anxiety sensitivity. *Psychosomatic Medicine, 71*(9), 1018–1025
- Gotlib, I. H. (1998). EEG alpha asymmetry, depression, and cognitive functioning. *Cognition & Emotion, 12*(3), 449–478. <https://doi.org/10.1080/026999398379673>
- Gross, J. J. (1998a). Antecedent-and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*(1), 224–237.
<https://doi.org/http://dx.doi.org/10.1037/0022-3514.74.1.224>
- Gross, J. J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*(3), 271.
<https://doi.org/http://dx.doi.org/10.1037/1089-2680.2.3.271>
- Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*(3), 281–291.
<https://doi.org/http://dx.doi.org/10.1017/S0048577201393198>
- Gross, J. J. (2013). Emotion regulation: Taking stock and moving forward. *Emotion, 13*(3), 359–365. <https://doi.org/10.1037/a0032135>
- Gross, J. J., & Jazaieri, H. (2014). Emotion, emotion regulation, and psychopathology: An affective science perspective. *Clinical Psychological Science, 2*(4), 387–401.
<https://doi.org/10.1177/2167702614536164>

- Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*(2), 348–362. <https://doi.org/10.1037/0022-3514.85.2.348>
- Gross, J. J., & Muñoz, R. F. (1995). Emotion regulation and mental health. *Clinical Psychology: Science and Practice, 2*(2), 151–164. <https://doi.org/http://dx.doi.org/10.1111/j.1468-2850.1995.tb00036.x>
- Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). Guilford Press.
- Grunewald, M., Döhnert, M., Brandeis, D., Klein, A. M., von Klitzing, K., Matuschek, T., & Stadelmann, S. (2019). Attenuated LPP to emotional face stimuli associated with parent- and self-reported depression in children and adolescents. *Journal of Abnormal Child Psychology, 47*(1), 109–118. <https://doi.org/10.1007/s10802-018-0429-3>
- Gu, J., Strauss, C., Bond, R., & Cavanagh, K. (2015). How do mindfulness-based cognitive therapy and mindfulness-based stress reduction improve mental health and wellbeing? A systematic review and meta-analysis of mediation studies. *Clinical Psychology Review, 37*, 1–12. <https://doi.org/10.1016/j.cpr.2015.01.006>
- Hajcak, G., Dunning, J. P., & Foti, D. (2007). Neural response to emotional pictures is unaffected by concurrent task difficulty: An event-related potential study. *Behavioral Neuroscience, 121*(6), 1156. <https://doi.org/http://dx.doi.org/10.1037/0735-7044.121.6.1156>
- Hajcak, G., Dunning, J. P., & Foti, D. (2009). Motivated and controlled attention to emotion: Time-course of the late positive potential. *Clinical Neurophysiology, 120*(3), 505–510. <https://doi.org/10.1016/j.clinph.2008.11.028>
- Hajcak, G., Dunning, J. P., Foti, D., & Weinberg, A. (2007). Temporal dynamics of emotion regulation. In *Handbook of Emotion Regulation* (Second, pp. 43–57). Guilford Press.
- Hajcak, G., Moser, J. S., & Simons, R. F. (2006). Attending to affect: Appraisal strategies modulate the electrocortical response to arousing pictures. *Emotion, 6*(3), 517. <https://doi.org/http://dx.doi.org/10.1037/1528-3542.6.3.517>
- Hajcak, G., & Nieuwenhuis, S. (2006). Reappraisal modulates the electrocortical response to unpleasant pictures. *Cognitive, Affective, & Behavioral Neuroscience, 6*(4), 291–297. <https://doi.org/http://dx.doi.org/10.3758/CABN.6.4.291>
- Hajcak, G., & Olvet, D. M. (2008). The persistence of attention to emotion: Brain potentials during and after picture presentation. *Emotion, 8*(2), 250. <https://doi.org/http://dx.doi.org/10.1037/1528-3542.8.2.250>

- Haxby, J. V., Hoffman, E. A., & Gobbini, M. (2000). The distributed human neural system for face perception. *Trends in Cognitive Sciences*, 4(6), 223–233. [https://doi.org/10.1016/S1364-6613\(00\)01482-0](https://doi.org/10.1016/S1364-6613(00)01482-0)
- Hayes, A. F. (2018). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. (2nd ed.). Guilford Press.
- Heiy, J. E., & Cheavens, J. S. (2014). Back to basics: A naturalistic assessment of the experience and regulation of emotion. *Emotion*, 14(5), 878–891. <https://doi.org/10.1037/a0037231>
- Herrington, J. D., Heller, W., Mohanty, A., Engels, A. S., Banich, M. T., Webb, A. G., & Miller, G. A. (2010). Localization of asymmetric brain function in emotion and depression. *Psychophysiology*, 47(3), 442–454. <https://doi.org/10.1111/j.1469-8986.2009.00958.x>
- Hill, K. E., South, S. C., Egan, R. P., & Foti, D. (2019). Abnormal emotional reactivity in depression: Contrasting theoretical models using neurophysiological data. *Biological Psychology*, 141, 35–43. <https://doi.org/10.1016/j.biopsycho.2018.12.011>
- Hinojosa, J. A., Mercado, F., & Carreti'e, L. (2015). N170 sensitivity to facial expression: A meta-analysis. *Neuroscience & Biobehavioral Reviews*, 55, 498–509. <https://doi.org/10.1016/j.neubiorev.2015.06.002>
- Hinton, D. E., Hofmann, S. G., Pollack, M. H., & Otto, M. W. (2009). Mechanisms of efficacy of CBT for cambodDr. Kahrilas refugees with PTSD: Improvement in emotion regulation and orthostatic blood pressure response. *CNS Neuroscience & Therapeutics*, 15(3), 255–263. <https://doi.org/10.1111/j.1755-5949.2009.00100.x>
- Hoffman, E. A., & Haxby, J. V. (2000). Distinct representations of eye gaze and identity in the distributed human neural system for face perception. *Nature Neuroscience*, 3(1), 80–84. <https://doi.org/10.1038/71152>
- Horn, J. L. (1965). A rationale and test for the number of factors in factor analysis. *Psychometrika*, 30(2), 179–185. <https://doi.org/10.1007/BF02289447>
- Hou, W. K., Lau, K. M., Ng, S. M., Cheng, A. C. K., Shum, T. C. Y., Cheng, S. T., & Cheung, H. Y. S. (2016). Savoring moderates the association between cancer-specific physical symptoms and depressive symptoms. *Psycho-Oncology*, 26(2), 231–238. <https://doi.org/10.1002/pon.4114>
- Hu, L., & Bentler, P. M. (1998). Fit indices in covarDr. Kahrilasce structure modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods*, 3(4), 424–453. <https://doi.org/10.1037/1082-989X.3.4.424>

- Hurley, D. B., & Kwon, P. (2012). Results of a study to increase savoring the moment: Differential impact on positive and negative outcomes. *Journal of Happiness Studies*, *13*(4), 579–588.
- Hurley, D. B., & Kwon, P. (2013). Savoring helps most when you have little: Interaction between savoring the moment and uplifts on positive affect and satisfaction with life. *Journal of Happiness Studies*, *14*(4), 1261–1271.
<https://doi.org/http://dx.doi.org/10.1007/s10902-012-9377-8>
- Ille, N., Berg, P., & Scherg, M. (2002). Artifact correction of the ongoing EEG using spatial filters based on artifact and brain signal topographies: *Journal of Clinical Neurophysiology*, *19*(2), 113–124. <https://doi.org/10.1097/00004691-200203000-00002>
- Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion*, *24*(2), 281–298.
<https://doi.org/http://dx.doi.org/10.1080/02699930903407948>
- Joormann, J., & Stanton, C. H. (2016). Examining emotion regulation in depression: A review and future directions. *Behaviour Research and Therapy*, *86*, 35–49.
<https://doi.org/10.1016/j.brat.2016.07.007>
- Joormann, J., & Vanderlind, W. M. (2014). Emotion regulation in depression: The role of biased cognition and reduced cognitive control. *Clinical Psychological Science*, *2*(4), 402–421.
<https://doi.org/10.1177/2167702614536163>
- Jorgensen, R. S., Johnson, B. T., Kolodziej, M. E., & Schreer, G. E. (1996). Elevated blood pressure and personality: A meta-analytic review. *Psychological Bulletin*, *120*(2), 293–320. <https://doi.org/10.1037//0033-2909.120.2.293>
- Jose, P. E., Lim, B. T., & Bryant, F. B. (2012). Does savoring increase happiness? A daily diary study. *The Journal of Positive Psychology*, *7*(3), 176–187.
<https://doi.org/10.1080/17439760.2012.671345>
- Junghöfer, M., Bradley, M. M., Elbert, T. R., & Lang, P. J. (2001). Fleeting images: A new look at early emotion discrimination. *Psychophysiology*, *38*(02), 175–178.
- Kahrilas, I. J., Silton, R. L., Bryant, F. B., & Miller, G. A. (n.d.). *The neurobiological correlates of emotion reactivity and regulation*.
- Kahrilas, I. J., Smith, J. L., Silton, R. L., & Bryant, F. B. (2020). Savoring the moment: A link between affectivity and depression. *International Journal of Wellbeing*, *10*(2), 19–36.
<https://doi.org/10.5502/ijw.v10i2.779>

- Kawakubo, A., Bryant, F. B., Miyakawa, E., & Oguchi, T. (2019). Development and validation of the Japanese version of the Savoring Beliefs Inventory (SBI-J). *Journal of Positive Psychology and Wellbeing*, 3(2), 119–136.
- Kayser, J., Bruder, G. E., Tenke, C. E., Stewart, J. E., & Quitkin, F. M. (2000). Event-related potentials (ERPs) to hemifield presentations of emotional stimuli: Differences between depressed patients and healthy adults in P3 amplitude and asymmetry. *International Journal of Psychophysiology*, 36(3), 211–236.
- Keenan, K. (2000). Emotion dysregulation as a risk factor for child psychopathology. *Clinical Psychology: Science and Practice*, 7(4), 418–434. <https://doi.org/http://dx.doi.org/10.1093/clipsy.7.4.418>
- Keil, A., Bradley, M. M., Hauk, O., Rockstroh, B., Elbert, T., & Lang, P. J. (2002). Large-scale neural correlates of affective picture processing. *Psychophysiology*, 39(5), 641–649. <https://doi.org/http://dx.doi.org/10.1111/1469-8986.3950641>
- Keltner, D., & Gross, J. J. (1999). Functional Accounts of emotions. *Cognition & Emotion*, 13(5), 467–480. <https://doi.org/10.1080/026999399379140>
- Kendall, A. D., Zinbarg, R. E., Mineka, S., Bobova, L., Prenoveau, J. M., Revelle, W., & Craske, M. G. (2015). Prospective associations of low positive emotionality with first onsets of depressive and anxiety disorders: Results from a 10-wave latent trait-state modeling study. *Journal of Abnormal Psychology*, 124(4), 933–943. <https://doi.org/10.1037/abn0000105>
- Kessler, R. C., Akiskal, H. S., Ames, M., Birnbaum, H., Greenberg, P., Hirschfeld, R. M. A., Jin, R., Merikangas, K. R., Simon, G. E., & Wang, P. S. (2006). Prevalence and effects of mood disorders on work performance in a nationally representative sample of U.S. Workers. *The American Journal of Psychiatry*, 163(9), 1561–1568. <https://doi.org/10.1176/ajp.2006.163.9.1561>
- Keune, P. M., Bostanov, V., Hautzinger, M., & Kotchoubey, B. (2013). Approaching dysphoric mood: State-effects of mindfulness meditation on frontal brain asymmetry. *Biological Psychology*, 93(1), 105–113. <https://doi.org/10.1016/j.biopsycho.2013.01.016>
- Kim, S. H., & Hamann, S. (2007). Neural correlates of positive and negative emotion regulation. *Journal of Cognitive Neuroscience*, 19(5), 776–798. <https://doi.org/http://dx.doi.org/10.1162/jocn.2007.19.5.776>
- Khazanov, G. K., & Ruscio, A. M. (2016). Is positive emotionality a specific risk factor for depression? A meta-analysis of longitudinal studies. *Psychological Bulletin*, 142(9), 991–1015. <https://doi.org/10.1037/bul0000059>

- Khazanov, G. K., Ruscio, A. M., & Swendsen, J. (2019). The “brightening” effect: Reactions to positive events in the daily lives of individuals with major depressive disorder and generalized anxiety disorder. *Behavior Therapy, 50*(2), 270–284. <https://doi.org/10.1016/j.beth.2018.05.008>
- Kim, S. H., & Hamann, S. (2007). Neural correlates of positive and negative emotion regulation. *Journal of Cognitive Neuroscience, 19*(5), 776–798. <https://doi.org/http://dx.doi.org/10.1162/jocn.2007.19.5.776>
- Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion, 23*(1), 4–41. <https://doi.org/http://dx.doi.org/10.1080/02699930802619031>
- Koval, P., Kuppens, P., Allen, N. B., & Sheeber, L. (2012). Getting stuck in depression: The roles of rumination and emotional inertia. *Cognition & Emotion, 26*(8), 1412–1427. <https://doi.org/10.1080/02699931.2012.667392>
- Koval, P., Pe, M. L., Meers, K., & Kuppens, P. (2013). Affect dynamics in relation to depressive symptoms: Variable, unstable or inert? *Emotion, 13*(6), 1132–1141. <https://doi.org/10.1037/a0033579>
- Kraut, R., Olson, J., Banaji, M., Bruckman, A., Cohen, J., & Couper, M. (2004). Psychological research online: Report of board of scientific affairs’ advisory group on the conduct of research on the internet. *American Psychologist, 59*(2), 105–117. <https://doi.org/10.1037/0003-066X.59.2.105>
- Kroenke, K., & Spitzer, R. L. (2002). The PHQ-9: A new depression diagnostic and severity measure. *Psychiatric Annals, 32*(9), 509–515. <https://doi.org/10.3928/0048-5713-20020901-06>
- Kroenke, K., Spitzer, R. L., & Williams, J. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine, 16*(9), 606–613. <https://doi.org/10.1046/j.1525-1497.2001.016009606.x>
- Krompinger, J. W., Moser, J. S., & Simons, R. F. (2008). Modulations of the electrophysiological response to pleasant stimuli by cognitive reappraisal. *Emotion, 8*(1), 132–137. <https://doi.org/10.1037/1528-3542.8.1.132>
- Kung, F. Y. H., Eibach, R. P., & Grossmann, I. (2016). Culture, fixed-world beliefs, relationships, and perceptions of identity change. *Social Psychological and Personality Science, 7*(7), 631–639. <https://doi.org/https://doi.org/10.1177/1948550616652208>
- Kuppens, P., Allen, N. B., & Sheeber, L. B. (2010). Emotional inertia and psychological maladjustment. *Psychological Science, 21*(7), 984–991.

<https://doi.org/10.1177/0956797610372634>

- Kurdi, B., Lozano, S., & Banaji, M. R. (2017). Introducing the Open Affective Standardized Image Set (OASIS). *Behavior Research Methods*, *49*(2), 457–470.
<https://doi.org/10.3758/s13428-016-0715-3>
- Kurtz, J. L. (2015). Seeing through new eyes: An experimental investigation of the benefits of photography. *Journal of Basic & Applied Sciences*, *11*, 354–358.
<https://doi.org/http://dx.doi.org/10.6000/1927-5129.2015.11.51>
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). International Affective Picture System (IAPS): Affective ratings of pictures and instruction manual. *Technical Report A-8*.
- Lang, P. J., Bradley, M. M., Fitzsimmons, J. R., Cuthbert, B. N., Scott, J. D., Moulder, B., & Nangia, V. (1998). Emotional arousal and activation of the visual cortex: An fMRI analysis. *Psychophysiology*, *35*(2), 199–210.
- Langeslag, S. J. E., & van Strien, J. W. (2018). Early visual processing of snakes and angry faces: An ERP study. *Brain Research*, *1678*, 297–303.
<https://doi.org/10.1016/j.brainres.2017.10.031>
- Lawrence, N. S., Williams, A. M., Surguladze, S., Giampietro, V., Brammer, M. J., Andrew, C., Frangou, S., Ecker, C., & Phillips, M. L. (2004). Subcortical and ventral prefrontal cortical neural responses to facial expressions distinguish patients with bipolar disorder and major depression. *Biological Psychiatry*, *55*(6), 578–587.
<https://doi.org/10.1016/j.biopsych.2003.11.017>
- LeDoux, J. E. (1995). Emotion: Clues from the brain. *Annual Review of Psychology*, *46*, 27.
<https://doi.org/http://dx.doi.org/10.1146/annurev.ps.46.020195.001233>
- Lee, J.-S., Ahn, Y.-S., Jeong, K.-S., Chae, J.-H., & Choi, K.-S. (2014). Resilience buffers the impact of traumatic events on the development of PTSD symptoms in firefighters. *Journal of Affective Disorders*, *162*, 128–133. <https://doi.org/10.1016/j.jad.2014.02.031>
- Lenth, R. (2020). *Emmeans: Estimated marginal means, aka least-squares means*.
- Levin, R. L., Heller, W., Mohanty, A., Herrington, J. D., & Miller, G. A. (2007). Cognitive deficits in depression and functional specificity of regional brain activity. *Cognitive Therapy and Research*, *31*(2), 211–233. <https://doi.org/10.1007/s10608-007-9128-z>
- Lewis, A. R., Zinbarg, R. E., & Durbin, C. E. (2010). Advances, problems, and challenges in the study of emotion regulation: A commentary. *Journal of Psychopathology and Behavioral Assessment*, *32*(1), 83–91. <https://doi.org/10.1007/s10862-009-9170-0>

- Lewinsohn, P. M., & Graf, M. (1973). Pleasant activities and depression. *Journal of Consulting and Clinical Psychology, 41*(2), 261–268.
<https://doi.org/http://dx.doi.org/10.1037/h0035142>
- Lewinsohn, P. M., Rohde, P., Seeley, J. R., Klein, D. N., & Gotlib, I. H. (2003). Psychosocial functioning of young adults who have experienced and recovered from major depressive disorder during adolescence. *Journal of Abnormal Psychology, 112*(3), 353–363.
<https://doi.org/10.1037/0021-843x.112.3.353>
- Li, N. P., Patel, L., Balliet, D., Tov, W., & Scollon, C. N. (2011). The incompatibility of materialism and the desire for children: Psychological insights into the fertility discrepancy among modern countries. *Social Indicators Research, 101*(3), 391–404.
<https://doi.org/10.1007/s11205-010-9665-9>
- Liu, Y., Huang, H., McGinnis-Deweese, M., Keil, A., & Ding, M. (2012). Neural substrate of the late positive potential in emotional processing. *Journal of Neuroscience, 32*(42), 14563–14572. <https://doi.org/10.1523/JNEUROSCI.3109-12.2012>
- Liu, D. Y., & Thompson, R. J. (2017). Selection and implementation of emotion regulation strategies in major depressive disorder: An integrative review. *Clinical Psychology Review, 57*, 183–194. <https://doi.org/10.1016/j.cpr.2017.07.004>
- Lovibond, S. H., & Lovibond, P. F. (1996). *Manual for the depression anxiety stress scales*. Psychology Foundation of Australia.
- Lucas, R. E. (2007). Adaptation and the set-point model of subjective well-being: Does happiness change after major life events? *Current Directions in Psychological Science, 16*(2), 75–79. <https://doi.org/10.1111/j.1467-8721.2007.00479.x>
- Lüdecke, D., Makowski, D., & Waggoner, P. (2019). Performance: Assessment of regression models performance. *R Package Version 0.4, 2*.
- MacLeod, A. K., & Byrne, A. (1996). Anxiety, depression, and the anticipation of future positive and negative experiences. *Journal of Abnormal Psychology, 105*(2), 286–289.
<https://doi.org/http://dx.doi.org/10.1037/0021-843X.105.2.286>
- MacNamara, A., Kotov, R., & Hajcak, G. (2016). Diagnostic and symptom-based predictors of emotional processing in generalized anxiety disorder and major depressive disorder: An event-related potential study. *Cognitive Therapy and Research, 40*(3), 275–289.
<https://doi.org/10.1007/s10608-015-9717-1>
- Markon, K. E., & Krueger, R. F. (2004). An empirical comparison of information-theoretic selection criteria for multivariate behavior genetic models. *Behavior Genetics, 34*(6), 593–

610. <https://doi.org/10.1007/s10519-004-5587-0>

- Marsh, H. W., Hau, K.-T., & Wen, Z. (2004). In search of golden rules: Comment on hypothesis-testing approaches to setting cutoff values for fit indexes and dangers in overgeneralizing Hu and Bentler's (1999) Findings. *Structural Equation Modeling: A Multidisciplinary Journal*, *11*(3), 320–341. https://doi.org/10.1207/s15328007sem1103_2
- Mason, W., & Suri, S. (2012). Conducting behavioral research on Amazon's Mechanical Turk. *Behavior Research Methods*, *44*(1), 1–23. <https://doi.org/10.3758/s13428-011-0124-6>
- Maxwell, S. E., Cole, D. A., & Mitchell, M. A. (2011). Bias in cross-sectional analyses of longitudinal mediation: Partial and complete mediation under an autoregressive model. *Multivariate Behavioral Research*, *46*(5), 816–841. <https://doi.org/10.1080/00273171.2011.606716>
- McMakin, D. L., Siegle, G. J., & Shirk, S. R. (2011). Positive affect stimulation and sustainment (PASS) module for depressed mood: A preliminary investigation of treatment-related effects. *Cognitive Therapy and Research*, *35*(3), 217–226. <https://doi.org/10.1007/s10608-010-9311-5>
- McNamara, J. M., Fawcett, T. W., & Houston, A. I. (2013). An adaptive response to uncertainty generates positive and negative contrast effects. *Science*, *340*(6136), 1084–1086. <https://doi.org/10.1126/science.1230599>
- Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, *28*(6), 487–495. [https://doi.org/http://dx.doi.org/10.1016/0005-7967\(90\)90135-6](https://doi.org/http://dx.doi.org/10.1016/0005-7967(90)90135-6)
- Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology*, *49*(1), 377–412. <https://doi.org/http://dx.doi.org/10.1146/annurev.psych.49.1.377>
- Monroe, S. M., & Anderson, S. F. (2015). Depression: The shroud of heterogeneity. *Current Directions in Psychological Science*, *24*(3), 227–231. <https://doi.org/10.1177/0963721414568342>
- Moore, A., & Malinowski, P. (2009). Meditation, mindfulness and cognitive flexibility. *Consciousness and Cognition*, *18*(1), 176–186. <https://doi.org/10.1016/j.concog.2008.12.008>
- Morris, J. S., Öhman, A., & Dolan, R. J. (1998). Conscious and unconscious emotional learning in the human amygdala. *Nature*, *393*(6684), 467–470. <https://doi.org/10.1038/30976>

- Moser, J. S., Hajcak, G., Bukay, E., & Simons, R. F. (2006). Intentional modulation of emotional responding to unpleasant pictures: An ERP study. *Psychophysiology*, *43*(3), 292–296. <https://doi.org/10.1111/j.1469-8986.2006.00402.x>
- Moser, J. S., Hartwig, R., Moran, T. P., Jendrusina, A. A., & Kross, E. (2014). Neural markers of positive reappraisal and their associations with trait reappraisal and worry. *Journal of Abnormal Psychology*, *123*(1), 91–105. <https://doi.org/10.1037/a0035817>
- Moser, J. S., Kropfing, J. W., Dietz, J., & Simons, R. F. (2009). Electrophysiological correlates of decreasing and increasing emotional responses to unpleasant pictures. *Psychophysiology*, *46*(1), 17–27. <https://doi.org/10.1111/j.1469-8986.2008.00721.x>
- Needles, D. J., & Abramson, L. Y. (1990). Positive life events, attributional style, and hopefulness: Testing a model of recovery from depression. *Journal of Abnormal Psychology*, *99*(2), 156–165. <https://doi.org/http://dx.doi.org/10.1037/0021-843X.99.2.156>
- Nelis, S., Holmes, E. A., & Raes, F. (2015). Response styles to positive affect and depression: Concurrent and prospective associations in a community sample. *Cognitive Therapy and Research*, *39*(4), 480–491. <https://doi.org/10.1007/s10608-015-9671-y>
- Nitschke, J. B., Heller, W., Imig, J. C., McDonald, R. P., & Miller, G. A. (2001). Distinguishing dimensions of anxiety and depression. *Cognitive Therapy and Research*, *25*(1), 1–22. <https://doi.org/https://doi.org/10.1023/A:1026485530405>
- Nitschke, J. B., Heller, W., Palmieri, P. A., & Miller, G. A. (1999). Contrasting patterns of brain activity in anxious apprehension and anxious arousal. *Psychophysiology*, *36*(5), 628–637. <https://doi.org/10.1111/1469-8986.3650628>
- Nolen-Hoeksema, S., Morrow, J., & Fredrickson, B. L. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology*, *102*(1), 20–28. <https://doi.org/10.1037//0021-843X.102.1.20>
- Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, *14*(8), 1215–1229. <https://doi.org/http://dx.doi.org/10.1162/089892902760807212>
- Ochsner, K. N., Ray, R. R., Hughes, B., McRae, K., Cooper, J. C., Weber, J., Gabrieli, J. D. E., & Gross, J. J. (2009). Bottom-Up and Top-Down Processes in Emotion Generation: Common and Distinct Neural Mechanisms. *Psychological Science*, *20*(11), 1322–1331. <https://doi.org/10.1111/j.1467-9280.2009.02459.x>
- Olofsson, J. K., Nordin, S., Sequeira, H., & Polich, J. (2008). Affective picture processing: An integrative review of ERP findings. *Biological Psychology*, *77*(3), 247–265.

<https://doi.org/10.1016/j.biopsycho.2007.11.006>

- Osborne, D., Huo, Y. J., & Smith, H. J. (2015). Organizational respect dampens the impact of group-based relative deprivation on willingness to protest pay cuts. *British Journal of Social Psychology, 54*(1), 159–175. <https://doi.org/10.1111/bjso.12069>
- Paolacci, G., Chandler, J., & Ipeirotis, P. G. (2010). Running experiments on amazon mechanical turk. *Judgment and Decision Making, 5*, 411–419.
- Pastor, M. C., Bradley, M. M., Löw, A., Versace, F., Molt'o, J., & Lang, P. J. (2008). Affective picture perception: Emotion, context, and the late positive potential. *Brain Research, 1189*, 145–151. <https://doi.org/10.1016/j.brainres.2007.10.072>
- Peeters, F., Nicolson, N. A., Berkhof, J., & Devries, M. (2003). Effects of daily events on mood states in major depressive disorder. *Journal of Abnormal Psychology, 211*.
- Polich, J., & Kok, A. (1995). Cognitive and biological determinants of P300: An integrative review. *Biological Psychology, 41*(2), 103–146. [https://doi.org/10.1016/0301-0511\(95\)05130-9](https://doi.org/10.1016/0301-0511(95)05130-9)
- Pollack, J. M., Vanepps, E. M., & Hayes, A. F. (2012). The moderating role of social ties on entrepreneurs' depressed affect and withdrawal intentions in response to economic stress. *Journal of Organizational Behavior, 33*(6), 789–810. <https://doi.org/https://doi.org/10.1002/job.1794>
- Price, J. L., & Drevets, W. C. (2010). Neurocircuitry of mood disorders. *Neuropsychopharmacology, 35*(1), 192–216. <https://doi.org/https://doi.org/10.1038/npp.2009.104>
- Proudfit, G. H., Bress, J. N., Foti, D., Kujawa, A., & Klein, D. N. (2015). Depression and event-related potentials: Emotional disengagement and reward insensitivity. *Current Opinion in Psychology, 4*, 110–113. <https://doi.org/10.1016/j.copsyc.2014.12.018>
- Quoidbach, J., Berry, E. V., Hansenne, M., & Mikolajczak, M. (2010). Positive emotion regulation and well-being: Comparing the impact of eight savoring and dampening strategies. *Personality and Individual Differences, 49*(5), 368–373. <https://doi.org/10.1016/j.paid.2010.03.048>
- Quoidbach, J., Wood, A., & Hansenne, M. (2009). Back to the future: The effect of daily practice of mental time travel into the future on happiness and anxiety. *Journal of Positive Psychology, 4*, 349–355. <https://doi.org/http://dx.doi.org/10.1080/17439760902992365>
- R Core Team (2018). *R: A language and environment for statistical computing*. R Foundation for

Statistical Computing.

- Raes, F., Smets, J., Nelis, S., & Schoofs, H. (2012). Dampening of positive affect prospectively predicts depressive symptoms in non-clinical samples. *Cognition & Emotion*, *26*(1), 75–82. <https://doi.org/10.1080/02699931.2011.555474>
- Ramsey, M. A., & Gentzler, A. L. (2014). Age differences in subjective well-being across adulthood: The roles of savoring and future time perspective. *The International Journal of Aging and Human Development*, *78*(1), 3–22. <https://doi.org/10.2190/AG.78.1.b>
- Raphiphatthana, B., Jose, P. E., & Kielpikowski, M. (2016). How do the facets of mindfulness predict the constructs of depression and anxiety as seen through the lens of the tripartite theory? *Personality and Individual Differences*, *93*, 104–111. <https://doi.org/10.1016/j.paid.2015.08.005>
- Rees, T., & Freeman, P. (2009). Social support moderates the relationship between stressors and task performance through self-efficacy. *Journal of Social and Clinical Psychology*, *28*(2), 244–263. <https://doi.org/10.1521/jscp.2009.28.2.244>
- Rellecke, J., Sommer, W., & Schacht, A. (2013). Emotion effects on the N170: A question of reference? *Brain Topography*, *26*(1), 62–71. <https://doi.org/10.1007/s10548-012-0261-y>
- Revelle, W. (2020). *Psych: Procedures for psychological, psychometric, and personality research*. Northwestern University.
- Rosseel, Y. (2011). *Lavaan: An R package for structural equation modeling and more Version 0.4-9 (BETA)*.
- Rottenberg, J. (2005). Mood and emotion in major depression. *Current Directions in Psychological Science*, *14*(3), 167–170. <https://doi.org/10.1111/j.0963-7214.2005.00354.x>
- Rottenberg, J., Gross, J. J., & Gotlib, I. H. (2005). Emotion context insensitivity in major depressive disorder. *Journal of Abnormal Psychology*, 627–639.
- Ruscio, A. M., Borkovec, T. D., & Ruscio, J. (2001). A taxometric investigation of the latent structure of worry. *Journal of Abnormal Psychology*, 412–422.
- Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology*, *39*(6), 1161–1178. <https://doi.org/http://dx.doi.org.flagship.luc.edu/10.1037/h0077714>
- Sabatinelli, D., Bradley, M. M., Fitzsimmons, J. R., & Lang, P. J. (2005). Parallel amygdala and inferotemporal activation reflect emotional intensity and fear relevance. *NeuroImage*, *24*(4), 1265–1270. <https://doi.org/10.1016/j.neuroimage.2004.12.015>

- Sapolsky, R. M. (2007). Stress, stress-related disease, and emotional regulation. In *Handbook of emotion regulation*. Guilford Press.
- Sarstedt, M., Hair, J. F., Nitzl, C., Ringle, C. M., & Howard, M. C. (2020). Beyond a tandem analysis of SEM and PROCESS: Use of PLS-SEM for mediation analyses! *International Journal of Market Research*, *62*(3), 288–299. <https://doi.org/10.1177/1470785320915686>
- Savitz, J. B., & Drevets, W. C. (2009). Imaging phenotypes of major depressive disorder: Genetic correlates. *Neuroscience*, *164*(1), 300–330. <https://doi.org/10.1016/j.neuroscience.2009.03.082>
- Scherg, M., & Berg, P. (1990). BESA Brain electric source analysis handbook. *Munich: Max-Planck Institute for Psychiatry*.
- Schneider, W., Eschman, A., & Zuccolotto, A. (2002). *E-Prime User's Guide*. Psychology Software Tools Incorporated.
- Schultz, W., Apicella, P., Scarnati, E., & Ljungberg, T. (1992). Neuronal activity in monkey ventral striatum related to the expectation of reward. *Journal of Neuroscience*, *12*(12), 4595–4610.
- Schupp, H. T., Cuthbert, B. N., Bradley, M. M., Cacioppo, J. T., Ito, T., & Lang, P. J. (2000). Affective picture processing: The late positive potential is modulated by motivational relevance. *Psychophysiology*, *37*(2), 257–261. <https://doi.org/http://dx.doi.org/10.1111/1469-8986.3720257>
- Schupp, H. T., Junghöfer, M., Weike, A. I., & Hamm, A. O. (2003). Attention and emotion: An ERP analysis of facilitated emotional stimulus processing: *NeuroReport*, *14*(8), 1107–1110. <https://doi.org/10.1097/00001756-200306110-00002>
- Schupp, H. T., Junghöfer, M., Weike, A. I., & Hamm, A. O. (2004). The selective processing of briefly presented affective pictures: An ERP analysis: Selective emotional processing. *Psychophysiology*, *41*(3), 441–449. <https://doi.org/10.1111/j.1469-8986.2004.00174.x>
- Schupp, H. T., Markus, J., Weike, A. I., & Hamm, A. O. (2003). Emotional facilitation of sensory processing in the visual cortex. *Psychological Science*, *14*(1), 7–13. <https://doi.org/http://dx.doi.org/10.1111/1467-9280.01411>
- Schupp, H. T., Schmälzle, R., Flaisch, T., Weike, A. I., & Hamm, A. O. (2012). Affective picture processing as a function of preceding picture valence: An ERP analysis. *Biological Psychology*, *91*(1), 81–87.
- Seligman, M. E. P., Steen, T. A., Park, N., & Peterson, C. (2005). Positive psychology progress:

- Empirical validation of interventions. *American Psychologist*, *60*(5), 410–421.
<https://doi.org/10.1037/0003-066X.60.5.410>
- Sharp, P. B., Miller, G. A., & Heller, W. (2015). Transdiagnostic dimensions of anxiety: Neural mechanisms, executive functions, and new directions. *International Journal of Psychophysiology*, *98*(2), 365–377. <https://doi.org/10.1016/j.ijpsycho.2015.07.001>
- Sheline, Y. I., Barch, D. M., Donnelly, J. M., Ollinger, J. M., Snyder, A. Z., & Mintun, M. A. (2001). Increased amygdala response to masked emotional faces in depressed subjects resolves with antidepressant treatment: An fMRI study. *Biological Psychiatry*, *50*(9), 651–658. [https://doi.org/10.1016/S0006-3223\(01\)01263-X](https://doi.org/10.1016/S0006-3223(01)01263-X)
- Silton, R. L., Kahrilas, I. J., Skymba, H. V., Smith, J., Bryant, F. B., & Heller, W. (2020). Regulating positive emotions: Implications for promoting well-being in individuals with depression. *Emotion*, *20*(1), 93–97. <https://doi.org/10.1037/emo0000675>
- Smith, J. L., Bihary, J. G., O'Connor, D., Basic, A., & O'Brien, C. J. (2020). Impact of savoring ability on the relationship between older adults' activity engagement and well-being. *Journal of Applied Gerontology*, *39*(3), 323–331.
<https://doi.org/10.1177/0733464819871876>
- Smith, J. L., & Bryant, F. B. (2017). Savoring and well-being: Mapping the cognitive-emotional terrain of the happy mind. In M. D. Robinson & M. Eid (Eds.), *The Happy Mind: Cognitive Contributions to Well-Being* (Eds, pp. 139–156). Springer.
- Smith, J. L., & Bryant, F. B. (2013). Are we having fun yet? Savoring, type A behavior, and vacation enjoyment. *International Journal of Wellbeing*, *3*(1), 1–19.
<https://doi.org/10.5502/ijw.v3i1.1>
- Smith, J. L., & Hanni, A. A. (2019). Effects of a savoring intervention on resilience and well-being of older adults. *Journal of Applied Gerontology*, *38*(1), 137–152.
<https://doi.org/10.1177/0733464817693375>
- Smith, J. L., Harrison, P. R., Kurtz, J. L., & Bryant, F. B. (2014). Nurturing the capacity to savor: Interventions to enhance the enjoyment of positive experiences. In *The Wiley Blackwell Handbook of Positive Psychological Interventions* (pp. 42–65). John Wiley & Sons.
- Smith, J. L., & Hollinger-Smith, L. (2015). Savoring, resilience, and psychological well-being in older adults. *Aging & Mental Health*, *19*(3), 192–200.
<https://doi.org/http://dx.doi.org/10.1080/13607863.2014.986647>
- Smith, N. A., Martinez, L. R., & Sabat, I. E. (2016). Weight and gender in service jobs: The importance of warmth in predicting customer satisfaction. *Cornell Hospitality Quarterly*,

57(3), 314–328. <https://doi.org/10.1177/1938965515622564>

- Stewart, J. L., Coan, J. A., Towers, D. N., & Allen, J. J. B. (2011). Frontal EEG asymmetry during emotional challenge differentiates individuals with and without lifetime major depressive disorder. *Journal of Affective Disorders, 129*(1-3), 167–174. <https://doi.org/10.1016/j.jad.2010.08.029>
- Stöber, J., & Joormann, J. (2001). Worry, procrastination, and perfectionism: Differentiating amount of worry, pathological worry, anxiety, and depression. *Cognitive Therapy and Research, 25*(1), 49–60. <https://doi.org/http://dx.doi.org/10.1023/A:1026474715384>
- Stockdale, L. A., Morrison, R. G., Palumbo, R., Garbarino, J., & Silton, R. L. (2017). Cool, callous and in control: Superior inhibitory control in frequent players of video games with violent content. *Social Cognitive and Affective Neuroscience, 12*(12), 1869–1880. <https://doi.org/10.1093/scan/nsx115>
- Stockdale, L. A., Morrison, R. G., & Silton, R. L. (2020). The influence of stimulus valence on perceptual processing of facial expressions and subsequent response inhibition. *Psychophysiology, 57*(2), e13467. <https://doi.org/10.1111/psyp.13467>
- Strien, J. W. V., Christiaans, G., Franken, I. H. A., & Huijding, J. (2016). Curvilinear shapes and the snake detection hypothesis: An ERP study. *Psychophysiology, 53*(2), 252–257. <https://doi.org/10.1111/psyp.12564>
- Strunk, D. R., Lopez, H., & DeRubeis, R. J. (2006). Depressive symptoms are associated with unrealistic negative predictions of future life events. *Behaviour Research and Therapy, 44*(6), 861–882. <https://doi.org/10.1016/j.brat.2005.07.001>
- Strauss, G. P., Kappenman, E. S., Culbreth, A. J., Catalano, L. T., Lee, B. G., & Gold, J. M. (2013). Emotion regulation abnormalities in schizophrenia: Cognitive change strategies fail to decrease the neural response to unpleasant stimuli. *Schizophrenia Bulletin, 39*(4), 872–883. <https://doi.org/10.1093/schbul/sbs186>
- Suh, E., Diener, E., & Fujita, F. (1996). Events and subjective well-being: Only recent events matter. *Journal of Personality and Social Psychology, 70*(5), 1091. <https://doi.org/http://dx.doi.org/10.1037/0022-3514.70.5.1091>
- Surguladze, S., Brammer, M. J., Keedwell, P., Giampietro, V., Young, A. W., Travis, M. J., Williams, S. C. R., & Phillips, M. L. (2005). A differential pattern of neural response toward sad versus happy facial expressions in major depressive disorder. *Biological Psychiatry, 57*(3), 201–209. <https://doi.org/10.1016/j.biopsych.2004.10.028>
- Thai, N., Taber-Thomas, B. C., & P'erez-Edgar, K. E. (2016). Neural correlates of attention

- biases, behavioral inhibition, and social anxiety in children: An ERP study. *Developmental Cognitive Neuroscience*, *19*, 200–210. <https://doi.org/10.1016/j.dcn.2016.03.008>
- Thibodeau, R., Jorgensen, R. S., & Kim, S. (2006). Depression, anxiety, and resting frontal EEG asymmetry: A meta-analytic review. *Journal of Abnormal Psychology*, *115*(4), 715–729. <https://doi.org/10.1037/0021-843X.115.4.715>
- Thiruchselvam, R., Blechert, J., Sheppes, G., Rydstrom, A., & Gross, J. J. (2011). The temporal dynamics of emotion regulation: An EEG study of distraction and reappraisal. *Biological Psychology*, *87*(1), 84–92. <https://doi.org/10.1016/j.biopsycho.2011.02.009>
- Thomas, K. K., & Bowker, J. C. (2015). Rejection sensitivity and adjustment during adolescence: Do friendship self-silencing and parent support matter? *Journal of Child and Family Studies*, *24*(3), 608–616. <https://doi.org/10.1007/s10826-013-9871-6>
- Thompson, R. J., Mata, J., Jaeggi, S. M., Buschkuhl, M., Jonides, J., & Gotlib, I. H. (2012). The everyday emotional experience of adults with major depressive disorder: Examining emotional instability, inertia, and reactivity. *Journal of Abnormal Psychology*, *121*(4), 819–829. <https://doi.org/10.1037/a0027978>
- Torres, L., & Taknint, J. T. (2015). Ethnic microaggressions, traumatic stress symptoms, and Latino depression: A moderated mediational model. *Journal of Counseling Psychology*, *62*(3), 393–401. <https://doi.org/10.1037/cou0000077>
- Townsend, J., & Altshuler, L. L. (2012). Emotion processing and regulation in bipolar disorder: A review. *Bipolar Disorders*, *14*(4), 326–339. <https://doi.org/10.1111/j.1399-5618.2012.01021.x>
- Treadway, M. T., & Zald, D. H. (2011). Reconsidering anhedonia in depression: Lessons from translational neuroscience. *Neuroscience and Biobehavioral Reviews*, *35*(3), 537–555. <https://doi.org/10.1016/j.neubiorev.2010.06.006>
- Tritt, S. M., Peterson, J. B., Page-Gould, E., & Inzlicht, M. (2016). Ideological reactivity: Political conservatism and brain responsivity to emotional and neutral stimuli. *Emotion*, *16*(8), 1172–1185. <https://doi.org/10.1037/emo0000150>
- Twenge, J. M., & Joiner, T. E. (2020). U.S. Census Bureau-assessed prevalence of anxiety and depressive symptoms in 2019 and during the 2020 COVID-19 pandemic. *Depression and Anxiety*, 1–3. <https://doi.org/10.1002/da.23077>
- Van Dam, N. T., van Vugt, M. K., Vago, D. R., Schmalzl, L., Saron, C. D., Olendzki, A., Meissner, T., Lazar, S. W., Kerr, C. E., Gorchov, J., Fox, K. C. R., Field, B. A., Britton,

- W. B., Brefczynski-Lewis, J. A., & Meyer, D. E. (2018). Mind the hype: A critical evaluation and prescriptive agenda for research on mindfulness and meditation. *Perspectives on Psychological Science, 13*(1), 36–61. <https://doi.org/10.1177/1745691617709589>
- Villanueva, C. M., Silton, R. L., Heller, W., Barch, D. M., & Gruber, J. (2021). Change is on the horizon: Call to action for the study of positive emotion and reward in psychopathology. *Current Opinion in Behavioral Sciences, 39*, 34–40. <https://doi.org/10.1016/j.cobeha.2020.11.008>
- Vittengl, J. R., Clark, L. A., Dunn, T. W., & Jarrett, R. B. (2007). Reducing relapse and recurrence in unipolar depression: A comparative meta-analysis of cognitive-behavioral therapy's effects. *Journal of Consulting and Clinical Psychology, 75*(3), 475–488. <https://doi.org/10.1037/0022-006X.75.3.475>
- Warr, P. B., Barter, J., & Brownbridge, G. (1983). On the independence of positive and negative affect. *Journal of Personality and Social Psychology, 44*(3), 644–651. <https://doi.org/http://dx.doi.org/10.1037/0022-3514.44.3.644>
- Walcott, C. M., & Landau, S. (2004). The relation between disinhibition and emotion regulation in boys with attention deficit hyperactivity disorder. *Journal of Clinical Child and Adolescent Psychology, 33*(4), 772–782. https://doi.org/http://dx.doi.org/10.1207/s15374424jccp3304_12
- Watson, D. (2000). *Mood and temperament*. Guilford Press.
- Watson, D. (2009). Positive affectivity. In C. R. Snyder & S. J. Lopez (Eds.), *Oxford Handbook of Positive Psychology* (2nd ed., pp. 106–119). Oxford University Press.
- Watson, D., & Clark, L. A. (1984). Negative affectivity: The disposition to experience aversive emotional states. *Psychological Bulletin, 96*, 465–490. <https://doi.org/http://dx.doi.org/10.1037/0033-2909.96.3.465>
- Watson, D., Clark, L. A., & Carey, G. (1988). Positive and negative affectivity and their relation to anxiety and depressive disorders. *Journal of Abnormal Psychology, 97*(3), 346. <https://doi.org/http://dx.doi.org/10.1037/0021-843X.97.3.346>
- Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104*(1), 15–25. <https://doi.org/http://dx.doi.org/10.1037/0021-843X.104.1.15>
- Watson, D., & Naragon-Gainey, K. (2010). On the specificity of positive emotional dysfunction

in psychopathology: Evidence from the mood and anxiety disorders and schizophrenia/schizotypy. *Clinical Psychology Review*, 30(7), 839–848.
<https://doi.org/10.1016/j.cpr.2009.11.002>

- Watson, D., Stasik, S. M., Ellickson-Larew, S., & Stanton, K. (2015). Extraversion and psychopathology: A facet-level analysis. *Journal of Abnormal Psychology*, 124(2), 432–446. <https://doi.org/10.1037/abn0000051>
- Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology*, 104(1), 15–25. <https://doi.org/http://dx.doi.org/10.1037/0021-843X.104.1.15>
- Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology*, 104(1), 3–14. <https://doi.org/http://dx.doi.org/10.1037/0021-843X.104.1.3>
- Webb, J. B., Fiery, M. F., & Jafari, N. (2016). “You better not leave me shaming!”: Conditional indirect effect analyses of anti-fat attitudes, body shame, and fat talk as a function of self-compassion in college women. *Body Image*, 18, 5–13.
<https://doi.org/10.1016/j.bodyim.2016.04.009>
- Weinberg, A., May, A. M., Klonsky, E. D., Kotov, R., & Hajcak, G. (2017). Decreased neural response to threat differentiates patients who have attempted suicide from nonattempters with current ideation. *Clinical Psychological Science*, 5(6), 952–963.
<https://doi.org/10.1177/2167702617718193>
- Weinberg, A., Perlman, G., Kotov, R., & Hajcak, G. (2016). Depression and reduced neural response to emotional images: Distinction from anxiety, and importance of symptom dimensions and age of onset. *Journal of Abnormal Psychology*, 125(1), 26–39.
<https://doi.org/10.1037/abn0000118>
- Weinberg, A., & Sandre, A. (2018). Distinct associations between low positive affect, panic, and neural responses to reward and threat during late stages of affective picture processing. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 3(1), 59–68.
<https://doi.org/10.1016/j.bpsc.2017.09.013>
- Weinberg, A., Venables, N. C., Proudfit, G. H., & Patrick, C. J. (2015). Heritability of the neural response to emotional pictures: Evidence from ERPs in an adult twin sample. *Social Cognitive and Affective Neuroscience*, 10(3), 424–434.
<https://doi.org/10.1093/scan/nsu059>

- Werner-Seidler, A., Banks, R., Dunn, B. D., & Moulds, M. L. (2013). An investigation of the relationship between positive affect regulation and depression. *Behaviour Research and Therapy, 51*(1), 46–56. <https://doi.org/10.1016/j.brat.2012.11.001>
- Wielgosz, J., Goldberg, S. B., Kral, T. R. A., Dunne, J. D., & Davidson, R. J. (2019). Mindfulness meditation and psychopathology. *Annual Review of Clinical Psychology, 15*(1), 285–316. <https://doi.org/10.1146/annurev-clinpsy-021815-093423>
- Wiens, S., Sand, A., & Olofsson, J. K. (2011). Nonemotional features suppress early and enhance late emotional electrocortical responses to negative pictures. *Biological Psychology, 86*(1), 83–89. <https://doi.org/10.1016/j.biopsycho.2010.11.001>
- Wilson, K. A., & MacNamara, A. (2021). Savor the moment: Willful increase in positive emotion and the persistence of this effect across time. *Psychophysiology, 58*(3). <https://doi.org/10.1111/psyp.13754>
- Wirtz, P. H., von Känel, R., Mohiyeddini, C., Emini, L., Ruedisueli, K., Groessbauer, S., & Ehlert, U. (2006). Low social support and poor emotional regulation are associated with increased stress hormone reactivity to mental stress in systemic hypertension. *The Journal of Clinical Endocrinology & Metabolism, 91*(10), 3857–3865. <https://doi.org/10.1210/jc.2005-2586>
- World Health Organization (2017). *Depression and other common mental disorders: Global health estimates*.
- Yoon, S., Shim, M., Kim, H. S., & Lee, S.-H. (2016). Enhanced early posterior negativity to fearful faces in patients with anxiety disorder. *Brain Topography, 29*(2), 262–272. <https://doi.org/10.1007/s10548-015-0456-0>
- Zhang, Z. (2014). Monte Carlo based statistical power analysis for mediation models: Methods and software. *Behavior Research Methods, 46*(4), 1184–1198. <https://doi.org/10.3758/s13428-013-0424-0>
- Zotev, V., Yuan, H., Misaki, M., Phillips, R., Young, K. D., Feldner, M. T., & Bodurka, J. (2016). Correlation between amygdala BOLD activity and frontal EEG asymmetry during real-time fMRI neurofeedback training in patients with depression. *NeuroImage: Clinical, 11*, 224–238. <https://doi.org/10.1016/j.nicl.2016.02.003>

VITA

Dr. Ian Kahrilas was a doctoral student at Loyola University Chicago and studied clinical psychology and neuroscience. He received his B.S. in Psychology from the University of Illinois at Urbana-Champaign in 2014. During his time as an undergraduate at the University of Illinois, he received the Emanuel Donchin award for outstanding undergraduate research assistant in cognitive neuroscience. He also conducted research under the guidance of Dr. Florin Dolcos. After graduating, Dr. Kahrilas worked as the laboratory research assistant for a health psychology lab at Northwestern University Feinberg School of Medicine, studying how psychological variables influence symptomatology associated with functional gastrointestinal disorders. He started graduate school in 2016 at Loyola University Chicago as a member of Dr. Rebecca Siltan's research lab, the Wellbeing and Emotion Lab at Loyola (WELL). Dr. Kahrilas received his M.A. in Clinical Psychology in 2018. His graduate research focused on identifying and understanding potential modifiable neural mechanisms related to the experience of positive emotion in depression. He has presented research at multiple regional and national conferences and co-authored several peer-reviewed journal articles. Dr. Kahrilas completed his predoctoral internship in Clinical Psychology at Edward Hines, Jr. VA Hospital.