Impact of Cognitive Behavioral Therapy on Emotional Diversity among Adults with Major Depressive Disorder and Depressive Mood Symptoms

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Recommended Citation
Yu, Stephanie, "Impact of Cognitive Behavioral Therapy on Emotional Diversity among Adults with Major Depressive Disorder and Depressive Mood Symptoms" (2018). Undergraduate Honors Theses. 1698.
https://scholar.colorado.edu/honr_theses/1698
Impact of Cognitive Behavioral Therapy on Emotional Diversity among Adults with
Major Depressive Disorder and Depressive Mood Symptoms

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Abstract

Recent research has shown that emotional diversity – or the variety and relative abundance of the emotions one experiences – can be beneficial to one’s mental and physical well-being. However, it is less clear if cognitive behavioral therapy (CBT) – an empirically-supported and well-studied psychosocial treatment for depression – may impact emodiversity among adults diagnosed with major depression or experiencing symptoms of depression. To examine the potential clinical efficacy of CBT on emodiversity, the present investigation conducted a systematic review of the literature surveying a total of 87 studies and obtained full datasets from two CBT studies on depression in adults. Specifically, Study 1 included adults with major depressive disorder who were randomly assigned to receive either CBT ($n = 70$) or SSRIs ($n = 22$) and a non-psychiatric control group ($n = 35$), all of whom completed self-reported positive and negative affect before and after a 12-week treatment period (Siegle et al., 2012). Study 2 included adults ($n = 41$) treated with CBT naturalistically in private practice for depression and anxiety who completed weekly measures of positive and negative affect over a variable treatment period (Kring, Persons, & Thomas, 2007). Results suggested that CBT was effective in decreasing negative emodiversity among adults with a history of MDD or depressive symptoms (Study 1 and Study 2) and that CBT was associated with greater increases in positive emodiversity compared to SSRI among MDD adults (Study 1). Findings provide insight into affective mechanisms underlying empirically-supported treatments for depression.

**Keywords:** Depression; Cognitive Behavioral Therapy; Emotion; Emodiversity
Impact of Cognitive Behavioral Therapy on Emotional Diversity among Adults with Major Depressive Disorder and Depressive Mood Symptoms

As humans, we experience a variety of emotions – both positive and negative – that are integral to our everyday life. Take an example: suppose you have two individuals, A and B. Individual A experiences happiness, excitement, and interest in a day. Individual B, on the other hand, experiences happiness, nervousness, and embarrassment in a day. If asked which individual was happier or had greater psychological health in general, most people would likely choose Individual A. However, recent research has shown that the choice may not be so straightforward. Indeed, recent work suggests that diversity in one’s emotions in everyday life – both positive and negative – is beneficial to one’s mental and physical well-being as well. This has been referred to as “emodiversity,” defined as the variety and relative abundance of the emotions that humans experience and research suggests that experiencing diverse and more abundant emotions, whether they are positive or negative, may confer adaptive psychological health benefits (e.g., Quoidbach, Gruber, Mikolajczak, Kogan, Kotsou, & Norton, 2014; 2018; Ong, Benson, Zautra, & Ram, 2017). Although encouraging, few studies to date have examined whether emodiversity is impacted among adults suffering from emotional disorders such as depression (e.g., Rottenberg, Gross, & Gotlib, 2005), and whether empirically-supported treatments for depression including Cognitive Behavioral Therapy (or CBT; Butler, Chapman, Forman, & Beck, 2006) may improve emodiversity. Indeed, given the potential positive impacts of emodiversity for one’s psychological health, emodiversity could possibly be used as a measure of treatment efficacy or even become a focus in the future for treatment for mood disorders such as depression.

Emodiversity: Where Does It Fit Into Understanding Emotion?
Emotions are central to everyday experiences as humans and serve important survival functions (e.g., Darwin, 1872; Cosmides & Tooby, 2000). It is critical to our survival that we have many emotions in order to adapt readily to different opportunities and challenges in the environment. For example, the functionalist theory of emotion states that emotions are fundamental for our survival and each emotion has a specific survival purpose (Cambria, Livingstone, & Hussain, 2011). For instance, the emotion of fear allows us to react in dangerous situations, such as running away from something whereas sadness can trigger motivation to change our behavior in order to deal with an obstacle. Beyond survival, emotions also allow people to communicate and interact socially with one another (e.g., Keltner & Kring, 1998; Shariff & Tracy, 2011). Given emotions are vital for our daily functioning, it is critical to better understand how emotions impact our health and in what ways it can do so.

Although researchers converge on agreement on the importance of emotions to our functioning and well-being, to date little empirical work has specifically examined the importance of experiencing a variety of emotions. Yet recent work has demonstrated that experiencing a variety and relative abundance of the emotions – referred to as “emodiversity” – is also associated with one’s physical and mental health (e.g., Quoidbach et al., 2014; 2018). The concept of emodiversity leverages from work on biodiversity which focuses on the variety and abundance of life in an ecosystem. Specifically, biodiversity is advantageous to physical ecosystems given that having a wide variety of flora and fauna allows for the ecosystem to adapt and survive as it flexibly accommodates to changes in the physical ecosystem (Hooper et al., 2005). Genetic diversity also prevents diseases from ravaging all of the organisms in an ecosystem, preventing extinction (Hooper et al., 2005). This concept of biodiversity can also be conceptually applied to the human’s internal emotional and psychological ecosystem. One way
to measure emodiversity includes utilizing self-report or questionnaire methods that include emotion rating scales, such as the PANAS (Positive and Negative Affect Scale; Watson, Clark, & Tellegen, 1988) which sample a variety of individual positive and negative emotions on a Likert Scale (e.g., from “1” being very slightly or not at all to “5” being extremely). The PANAS scale is a widely and commonly used measure to evaluate one’s emotions that has been shown to be reliable and valid (Crawford & Henry, 2004). However, considerations for future work include sampling multiple time points or using an experience sampling approach sampling emotions multiple times in a day to better capture the variety of emotions experienced daily.

The operationalization of emodiversity is adapted from the Shannon biodiversity index, which “quantifies the number of species and the evenness of species in a biological ecosystem” (Quoidbach et al., 2014). Applied to the emotional and psychological ecosystem, this quantifies emodiversity, or “the richness (how many specific emotions are experienced) and evenness (the extent to which specific emotions are experienced in the same proportion) in the human emotional ecosystem” (Quoidbach et al., 2014). “Emodiversity can be calculated using the following formula derived from Shannon’s entropy:

\[ \text{Emodiversity} = \sum_{i=1}^{s} (p_i \times \ln p_i) \]

Where \( s \) equals the total number of emotion experienced (richness) and \( p_i \) equals the proportion of \( s \) made up of the \( i \)th emotions. Specifically, in order to compute emodiversity:

1. Divide the number of times an individual experiences emotion #1 by the total number of times she experiences all types of emotions, which gives \( p_1 \),

2. Multiply this proportion by its natural log \( (p_1 \times \ln p_1) \),

3. Repeat this for each specific emotion assessed, and
4. Sum all the \((p_i \times \ln p_i)\) products and multiply the total by -1.” (Quoidbach et al., 2014).

Higher values represent more diverse experiences of emotions. Experiencing only one emotion would get an emodiversity score of 0, and the maximal emodiversity score can vary depending on what emotion scale is used. Scores in the present study range from 0 to 2.96 in Study 1 and 0 to 2.88 in Study 2.

Emodiversity can also be separated into global, positive, and negative emodiversity. Global emodiversity refers to diversity across both positive and negative emotions. Positive emodiversity refers to diversity only across positive emotions, such as the extent to which one feels the emotions excitement, interested, and proud. Negative emodiversity refers to diversity only across negative emotions, such as the extent to which one feels the emotions ashamed, nervous, and guilty. Analyzing positive and negative emotional diversity in addition to global emotional diversity allows further insight to see where changes in emodiversity are deriving from or whether there are different patterns for positive and negative emodiversity. For example, a high positive and high negative emodiversity would give a high global emodiversity score. However, a low positive emodiversity and high negative emodiversity would effectively cancel out to give a middle-range global emodiversity score.

Although a recent construct, several studies converge to suggest the importance of emodiversity for psychologically relevant outcomes. For example, greater emodiversity, both positive and negative, has been associated with lower depression symptoms and measures of better physical health, such as fewer mean number of visits to the doctor per year, in a large community sample of adults (Quoidbach et al., 2014). Specifically, Quoidbach and colleagues found that across 35,844 participants, greater positive emodiversity was associated with lower
depression independent of mean positive emotion. Similarly, they also found that greater global and negative emodiversity was also associated with lower depression symptom scores in this same study. In a second dataset included in the same study, Quoidbach and colleagues reported that across 10,000 participants, greater positive, negative, and global emodiversity was associated with better physical health, measured by number of visits to the doctor, days spent at the hospital, doctor-related costs to Belgian Social Security, hospital-related costs to Social Security, and mean defined daily dose (DDD) of medication. Additional findings by Quoidbach et al. (2018) further supported that the relationship between emodiversity and health is robust, replicable, and theoretically-grounded. In this follow-up, they show that the PANAS emotion scale is a reliable and practical measure for richness of emotions, and that future studies could potentially switch to a 21-point response for the PANAS instead of a 5-point Likert scale to better measure the evenness of the distribution of emotions. They emphasize that across such large participant samples, their initial findings are valid and robust.

Another study by Ong et al. (2017) on emodiversity had 175 healthy adult participants provide reports of their daily emotions using the PANAS and collected blood samples to assess levels of IL-6 (Interleukin 6), CRP (C-Reactive Protein), and fibrinogen, which are all markers of inflammation, which is linked to physical health disease and poorer health in general. Results suggested that global and negative emodiversity were not significantly related to inflammation, but that self-reported positive emodiversity across the 175 adults was associated with lower levels of systemic inflammation (e.g., lower levels of CRP and fibrinogen). Results held even after controlling for mean levels of positive and negative emotion, age, gender, anti-inflammatory medications, body mass index, comorbid medical conditions, and self-reported personality (Ong et al., 2017).
In addition to these studies on emodiversity, a handful of related studies provide indirect conceptual support for the importance of experiencing diverse emotions. For example, work by Demiralp et al. (2012) on emotion differentiation – defined as having very specific emotional experiences such as excitement and happiness as opposed to a general feeling of pleasure – found that people with a clinical history of major depressive disorder (MDD) had less differentiated negative emotions when compared to a healthy control group. This suggests that people who have more specific emotions rather than general feelings may be less prone to MDD. Other studies have looked at related constructs such as psychological flexibility (Kashdan & Rottenberg, 2010) – defined as the ability to recognize and adapt to various situational demands and shift mindsets or behavioral repertoires when these strategies compromise personal or social functioning – and mixed emotions (Adler & Hershfield, 2012) – defined as the concurrent experience of positive and negative emotions – and their impact on psychological health. Research on psychological flexibility has found that people who demonstrate greater coping flexibility report fewer depressive and anxiety symptoms (Kashdan & Rottenberg, 2010). Results for mixed emotional experiences demonstrate a positive relationship with psychological well-being, but this study did not specifically examine its role with depressive symptoms (Adler & Hershfield, 2012). Taken together, these studies converge with the literature on emodiversity suggesting the importance of understanding the diverse array of emotions experienced in daily life and their links with clinically relevant psychological health outcomes, such as depression.

**Empirical Gaps in Emodiversity: Clinical Applications**

These studies demonstrate the importance of emodiversity in human health, yet further research is warranted in order to determine whether or not an individual’s emodiversity can be improved as a function of empirically-supported psychological treatments. To date, however, we
are not aware of any studies systematically examining emodiversity among treatment-seeking or clinically diagnosed adults, including those diagnosed with major depressive disorder – a common, severe, and chronic psychiatric disorder – or whether well-studied empirically-supported treatments for depression (such as CBT) positively impact emodiversity levels. This is an important empirical gap to address for several reasons. First, this could potentially help depressed individuals improve their quality of daily emotions and serve as a focus of future targeted treatments. Second, given the prevalence and functional impairment associated with depression, it is important to isolate potential processes that may contribute to improved well-being and quality of life for adults suffering from mood disorders. One potentially fruitful avenue is to study the clinical application of emodiversity; i.e., whether one can detect changes over time in emodiversity among adults with depression as a function of well-known and widely-used psychological and pharmacological clinical interventions for depression.

**Emotion in Major Depressive Disorder (MDD).** Depression is a chronic and recurrent psychiatric disease marked by significant impairments in emotion related disturbances (American Psychiatric Association, 2013). The link between emotion and depression has been recently advanced. For example, in one study by Rottenberg (2005) studying the relationship between mood and emotion in major depression, important conclusions were drawn about depression and how it affects people’s emotions. Participants were shown a neutral film and a sad film. The depressed group reported greater sadness than the healthy controls for the neutral film. However, contrary to their predictions and to common intuition, when responses to the neutral film were used as the reference point, depressed participants actually reported smaller increases in sadness than the healthy controls. Follow-up experiences demonstrated this was due to depressed patients’ sadness already being at an upper limit and therefore not able to increase
as much. What they concluded from these results was that depression flattens the emotional landscape, not only for positive emotions but negative ones as well (e.g., Rottenberg, Gross, & Gotlib, 2005). Other studies have also supported this notion of flattened emotions in depressed people; i.e., MDD patients tend to have a harder time generating and sustaining positive emotions over time (e.g., Garber, Braadfladt, & Weiss, 1995; McMakin, Santiago, & Shirk, 2009) and controlling their emotions, both positive and negative (Kang & Gruber, 2013). Taken together, this suggests that adults diagnosed with MDD or experiencing depressive mood symptoms may experience attenuated (i.e., and potentially less diverse) emotional experiences. Yet to date we are not aware of studies specifically examining the possibility of interventions that may lead to changes in emotional diversity among adults with MDD or depressive symptoms. One promising avenue may be to examine changes in emotional diversity as a function of participation in well-studied empirically-supported treatments for depression.

**Cognitive Behavioral Therapy.** Cognitive Behavioral Therapy (CBT) is a well-known and empirically-supported psychological intervention for depression (Beck, 1970). CBT pioneered originally by Aaron Beck (Beck, 1970), also sometimes called Cognitive Therapy (CT), is used to treat a variety of disorders, among which are bipolar and unipolar mood disorders (Butler et al., 2006). From behavior therapy, cognitive therapy gradually arose as several psychologists discovered just how important cognitive processes are in determining our behavior and feelings (Beck, 1970). The focus of CBT is to help patients first recognize and then modify their maladaptive thoughts by challenging these misconceptions. The theory behind this is that encouraging patients to recognize these distortions in their thoughts and to challenge them on their own will in turn lead to changes in their potentially destructive patterns of behavior. The technique behind CBT is outlined by Beck (1970). He breaks down CBT into three steps:
recognizing idiosyncratic cognitions, distancing, and correcting cognitive distortions and deficiencies. The first step is to train the patient to recognize their automatic thoughts, the previously mentioned maladaptive thoughts patients may not be aware of. Most of the time these thoughts are involuntary, and the person may not even realize they have those thoughts until they really pay attention to exactly what they are feeling and thinking in situations that make them anxious. The next step is having the patient distance themselves from their cognitions, meaning that they try to examine their thoughts from an objective standpoint. This may be hard for some people, even after they begin to recognize their cognitions, as they often have a distorted view that makes their thoughts seem very reasonable. The last step is correcting these maladaptive thoughts. Maladaptive thoughts can be sorted into some general categories, and cognitive behavioral therapists may use these to help a patient correct their distorted thinking (Beck, 1970).

**CBT for MDD.** There exists a robust literature on CBT in the alleviation of depressive symptoms. For example, over 75 clinical trials of CBT on depression have been conducted, and there is large consensus that CBT is an effective treatment for depression (e.g., DeRubeis et al., 2005; Gloaguen, Cottraux, Cucherat, & Blackburn, 1998). Because antidepressant medications (ADMs) have been a well-established treatment for depression, CBT is often compared to ADMs to test its efficacy. For example, one meta-analysis by Gloaguen et al. (1998) reported that CBT was even superior to antidepressant medication. However, subsequent studies showed that the studies included in that meta-analysis used methodology that may have favored CBT (Butler et al., 2006). Other recent meta-analyses conclude that antidepressant medications and CBT are equally effective in treating symptoms of depression. DeRubeis, Siegle, & Hollon (2008) concluded in a meta-analysis that CBT and antidepressants are comparable in the treatment of symptoms of depression. However, they also found that CBT seems to be more effective than
antidepressant medications at preventing relapse (DeRubeis et al., 2005). Although there is robust support for the efficacy of CBT on depressive symptoms, one gap in our understanding of CBT is whether it is effective in shifting emotional patterns experienced by those with depressive symptoms and whether such changes are enduring beyond changes in symptom severity.

**The Present Investigation**

This present investigation examines a theory-based mechanism of emotional health and emodiversity, looking at it from a clinical translational perspective to see if it can effectively track change over the course of CBT for depression. Specifically, we conducted a systematic review of the literature surveying over a total of 87 studies on CBT for MDD and obtained two full datasets from the original study authors. Specifically, Study 1 included adults with major depressive disorder who were randomly assigned to receive either CBT ($n = 70$) or SSRIs ($n = 22$) and a non-psychiatric control group ($n = 35$) who completed self-reported positive and negative affect before and after a 12-week treatment period (Siegle et al., 2012). Study 2 included adults ($n = 41$) treated with CBT naturalistically in private practice for depression and anxiety who completed weekly measures of positive and negative affect over a variable treatment period (Kring et al., 2007). This enabled us to compute novel measures of emodiversity using existing syntax (e.g., Quoidbach et al., 2014) and examine changes in emodiversity as a function of CBT treatment. This design enabled us to test the following two main study aims:

**Aim 1: Impact of CBT on Emodiversity in Depression.** The first aim examined whether cognitive-behavioral therapy (CBT) for depression is associated with changes in self-reported global, positive, and negative emodiversity over the course of treatment in adults with a diagnosis of major depressive disorder (Study 1) and experiencing symptoms of depression
We hypothesize that CBT will increase global (Hypothesis 1a), positive (Hypothesis 1b), and negative (Hypothesis 1c) emodiversity among adults with a history of MDD comparing pre-CBT treatment to post-CBT treatment time points. This hypothesis is based on literature suggesting that emodiversity is cross-sectionally associated with reductions in depressive symptom severity in a large community sample (Quoidbach et al., 2014) as well as literature suggesting reductions in depressive symptoms as a function of CBT intervention (Gloaguen et al., 1998).

Aim 2: Impact of CBT on Emodiversity in Depression Independent of Symptom Improvement. The second aim examined whether any changes in emodiversity as a function of CBT are robust after controlling for potential depressive symptom improvement. We predict that any changes in emodiversity as a function of CBT will hold even when controlling for depressive symptom severity across Study 1 and Study 2, (Hypothesis 2) suggesting emodiversity may be a unique and important marker of effective treatment change in adults experiencing symptoms of depression. This is based on literature suggesting CBT is more effective than antidepressants at preventing against relapse of depression (e.g., DeRubeis et al., 2005). This would further validate the efficacy of CBT for depression, as well as illuminate the possibility that CBT treats beyond symptoms and actually changes emodiversity, at least partially.

Methods

Data Acquisition and Literature Review.

To acquire the datasets used to compute emodiversity in the present investigation, we used a three-pronged approach to systematically review studies for CBT treatments for depressive symptoms in mood disorders literature (See Figure 1). The first approach included a
comprehensive literature search for the top five (i.e., most highly cited according to Google Scholar) relevant meta-analyses using key terms “CBT,” “depression,” “bipolar disorder,” and “medications.” All individual studies from these top cited meta-analyses were reviewed in detail. Corresponding authors for each article were then emailed for solicitation of their data and when the contact information for the primary corresponding author of an article could not be found, another author form the same article was contacted. In the case that none of the authors’ information could be found, the article was excluded for the study and they were not contacted for data. Authors were emailed a standardized emailed template (See Figure 2 for sample solicitation email). A follow-up email was sent approximately one week later to authors who didn’t respond to the first email. Out of the 47 individual authors contacted for data, 1 responded providing partial data for the present investigation (DeRubeis et al., 2005), 20 responded saying they didn’t collect measures of emotion or didn’t have access to the data, and 26 did not respond. Of these, 0 were eligible for data analysis for the present investigation.

Our second approach included individual contact with a list of the top 15 nationally recognized researchers in the field of cognitive behavioral therapy and mood disorders (See Table 2 for full list). Similar to the first approach, a follow-up email was sent approximately one week later to authors who didn’t respond to the first email. Out of the 15 authors contacted, 9 responded saying they didn’t collect emotion measures, 3 did not respond, and 3 collected emotion measures of which a total of 2 provided data available for analysis for the present investigation (i.e., datasets for Studies 1 and 2).

Our third approach included conducting a final literature search for articles published in the past 15 years using key terms “cognitive behavioral therapy” “depression” and “emotion.” From this approach, 7 additional researchers were emailed who were not already previously
contacted using one of the two aforementioned approaches. Once again, a reminder email was sent approximately one week after the initial email solicitation. Of the total 7 authors contacted, 1 responded who did not have data available for analysis and the remaining 6 did not respond.

In sum, we contacted a total of 69 authors and received 2 full datasets examining the impact of CBT on depressive symptoms among adults diagnosed with a history of depression (e.g., Study 1; Siegle et al., 2012) and a naturalistic sample of treatment-seeking adults with depressive symptoms (e.g., Study 2; Kring et al., 2007). We describe each of the two studies below in greater detail.

**Study 1**

The data for study 1 was provided by Dr. Greg Siegle from the University of Pittsburgh (Siegle et al., 2012). Study 1 included clinically depressed patients as well as healthy control participants who were originally recruited for a study examining the role of the subgenual anterior cingulate cortex as a reliable prognostic outcome marker of cognitive therapy for depression.

**Study 1 Participants**

The participants included three groups: the healthy control (CTL; \( n = 35 \)) group, MDD patients who were assigned to receive CBT treatment (MDD-CBT; \( n = 70 \)), and MDD patients who were assigned to take SSRIs (MDD-SSRI; \( n = 22 \)). The MDD groups were recruited in two cohorts from clinical trials (i.e., ClinicalTrials.gov, Identifier: NCT00183664 and ClinicalTrials.gov, Identifier: NCT00787501). The study included a total of 92 depressed patients (70 from cohort 1 and 22 from cohort 2) and 35 healthy controls recruited. However, only 69 depressed participants (54 from cohort 1 and 15 from cohort 2) and 33 healthy controls
had PANAS reports both pre- and post-treatment. Demographics of the sample are included in Table 1.

Study 1 Measures

Emotion and emotional diversity. The Positive and Negative Affect Scale (PANAS; Watson et al., 1988) was used to compute mean positive affect (PA) and negative affect (NA) as well as our measures of global, positive, and negative emotional diversity following guidelines reported by Quoidbach et al. (2014). The PANAS consists of 20 emotion items, including 10 positive and 10 negative, rated on a 1 (not at all) to 5 (very) scale, where higher scores mean greater emotion intensity for an individual item. The 10 positive emotion items include interested, excited, strong, enthusiastic, proud, alert, inspired, determined, attentive, and active, and the 10 negative emotion items include distressed, upset, guilty, scared, hostile, irritable, ashamed, nervous, jittery, and afraid. Internal consistency for PA ($\alpha = 0.94$ pre-treatment and $\alpha = 0.93$ post-treatment) and NA ($\alpha = 0.92$ pre-treatment and $\alpha = 0.89$ post-treatment) in the present study were good.

Depression symptoms. The Beck Depression Inventory-II (BDI-II; Beck, Steer, Ball, & Ranieri, 1996) is a self-report questionnaire used for measuring the severity of depression symptoms. The BDI-II contains 21 questions with each question rated on a scale value of 0 to 3 assessing cognitive, somatic, and affective symptoms of depression. The values for each question are totaled, with higher total scores indicating more severe depression. The BDI-II represents a revision of the BDI, with most of the items reworded and changes such as assessing increases in sleep and appetite as well instead of just decreases. Scores in the 0-13 range suggest minimal depression, 14-19 indicate mild depression, 20-28 indicate moderate depression, and 29-63
indicate severe depression severity. Individual items for the BDI-II were not provided by the
original study author, so internal consistency in the present study could not be computed.

**Study 1 Procedure**

Participants completed the PANAS before and after an experimental fMRI task and the
BDI-II after the task at baseline and following completion of treatment approximately 12 weeks
later. The PANAS was used to measure the extent to which the participants were experiencing
each emotion item at that given moment. Other experimental tasks and measures not relevant to
this study were also measured (see Siegle et al., 2012). Participants in the MDD-CBT group
underwent either 2 sessions of CT per week for the first 4 weeks followed by once a week for 8
weeks or 2 sessions per week for 8 weeks and then once a week for 4 weeks depending on if they
had early treatment responses or not, respectively. Within two weeks of completing either the
CBT or SSRI treatment group protocol (or at the week 16 time point for the CTL group who did
not complete any treatment), all participants completed the same PANAS and BDI-II protocols
again (Siegle et al., 2012). As noted in Siegle et al., 2012, CBT was successful at rates at least as
high as those observed in the literature with enough outcome variability to allow further analysis.
Participants were debriefed at the end of the study.

**Study 1 Results**

**Study 1 Results for Aim 1**

**Overview of Main Analyses.** Three separate 2 (Time: Pre-Treatment, Post-Treatment)
x 3 (Group: CTL, MDD-CBT, MDD-SSRI) repeated-measures analyses of (co)variance
(AN[C]OVAs) were conducted for each of the three emodiversity dependent variables
controlling for mean level of the respective positive and/or negative emotion intensity
following prior procedures (e.g., Quoidbach et al., 2014). A Greenhouse-Geisser correction was used when assumptions for sphericity were not met and adjusted $F$ and $p$ values are reported. Effect sizes for significant results are reported as partial eta squared ($\eta_p^2$). All reported $p$ values are two-tailed. Means and standard deviations are presented in Table 1.

**Global Emodiversity.** For Global-ED, there was a non-significant trend in Time, $F(1, 97) = 3.195, p = 0.077, \eta_p^2 = 0.032$. In other words, across all participants there was a slight decrease in global emodiversity (Pre-Treatment $M = 2.207, SE = 0.031$; Post-Treatment $M = 2.203, SE = 0.035$). For Group, there was no significant main effect, $F(2, 97) = 0.960, p = 0.387, \eta_p^2 = 0.019$. The three groups did not differ across both time points (CTL $M = 2.235, SE = 0.042$; MDD-CBT $M = 2.229, SE = 0.029$; MDD-SSRI $M = 2.151, SE = 0.053$). For the Group x Time interaction there was also no significant interaction, $F(2, 97) = 1.325, p = 0.270, \eta_p^2 = 0.027$ (See Figure 3).

**Positive Emodiversity.** For Positive-ED, there was a significant trend for the main effect of Time, $F(1, 98) = 16.020, p < 0.001, \eta_p^2 = 0.140$. In other words, across all participants there was a significant increase in positive emodiversity (Pre-Treatment $M = 1.594, SE = 0.059$; Post-Treatment $M = 1.911, SE = 0.037$). For Group, there was no significant main effect, $F(2, 98) = 1.766, p = 0.176, \eta_p^2 = 0.035$. In other words, the three groups did not differ across both time points, although the CTL group still scored higher in positive emodiversity compared to the other two groups, (CTL $M = 1.834, SE = 0.052$; MDD-CBT $M = 1.762, SE = 0.038$; MDD-SSRI $M = 1.662, SE = 0.071$). For the Group x Time interaction there was a non-significant trend, $F(2, 98) = 2.511, p = 0.086, \eta_p^2 = 0.049$. To identify the source of the Group x Time interaction, two separate one-way ANOVAs were run for each group to compare Pre-Treatment and Post-Treatment separately. Results suggest the three groups did not differ in
positive emodiversity Pre-Treatment, $F(2, 124) = 0.055, p = 0.946, \eta^2_p = 0.001$ (CTL $M = 1.623, SE = 0.086$; MDD-CBT $M = 1.590, SE = 0.055$; MDD-SSRI $M = 1.613, SE = 0.099$). However, there was a non-significant trend for the groups differing in positive emodiversity Post-Treatment, $F(2, 101) = 2.943, p = 0.057, \eta^2_p = 0.057$ (CTL $M = 1.954, SE = 0.046$; MDD-CBT $M = 1.987, SE = 0.035$; MDD-SSRI $M = 1.806, SE = 0.066$). In other words, the mean scores for positive emodiversity for the MDD-CBT group were higher than those of the CTL group, which were higher than those for the MDD-SSRI group (MDD-CBT > CTL > MDD-SSRI), with a significant difference between the MDD-CTL group and the MDD-SSRI group (Mean difference = 0.181, $p = 0.017$) (See Figure 4).

To test the significance of differences within groups for positive emodiversity Pre- and Post-Treatment, a repeated measures ANCOVA was conducted separately for each group controlling for mean positive emotion. There was no significant effect within the CTL group at Pre- and Post-Treatment $F(1, 31) = 0.434, p = 0.515, \eta^2_p = 0.014$, (Pre-Treatment $M = 2.054, SE = 0.040$; Post-Treatment $M = 2.074, SE = 0.036$). However, there was a significant increase in positive emodiversity within the MDD-CBT group from Pre- to Post-Treatment, $F(1, 52) = 21.548, p < 0.001, \eta^2_p = 0.293$, (Pre-Treatment $M = 1.375, SE = 0.081$; Post-Treatment $M = 1.936, SE = 0.049$). There was no significant effect within the MDD-SSRI group at Pre- and Post-Treatment, although there was still a trend of increasing positive emodiversity, $F(1, 13) = 2.618, p = 0.130, \eta^2_p = 0.168$, (Pre-Treatment $M = 1.352, SE = 0.128$; Post-Treatment $M = 1.725, SE = 0.096$) (See Figure 4).

**Negative Emodiversity.** For Negative-ED, there was a significant trend for the main effect of Time, $F(1, 98) = 9.908, p = 0.002, \eta^2_p = 0.092$. In other words, across all participants there was a significant decrease in negative emodiversity (Pre-Treatment $M = 1.136, SE =
0.053; Post-Treatment \(M = 0.726, SE = 0.069\). For Group, there was a significant main effect as well, \(F(2, 98) = 3.748, \ p = 0.027, \ \eta_p^2 = 0.071\). In other words, the three groups did differ across both time points (CTL \(M = 0.780, SE = 0.070\); MDD-CBT \(M = 1.028, SE = 0.051\); MDD-SSRI \(M = 0.984, SE = 0.095\)). For the Group x Time interaction there was a significant trend, \(F(2, 98) = 6.719, \ p = 0.002, \ \eta_p^2 = 0.121\). To identify the source of the Group x Time interaction, two separate one-way ANOVAs were run for each group to compare Pre-Treatment and Post-Treatment separately. Results suggest the CTL group differed significantly from the MDD groups in negative emodiversity Pre-Treatment, \(F(2, 123) = 14.679, \ p < 0.001, \ \eta_p^2 = 0.197\) (CTL \(M = 0.830, SE = 0.080\); MDD-CBT \(M = 1.367, SE = 0.052\); MDD-SSRI \(M = 1.333, SE = 0.093\)). However, there was a non-significant trend for the groups differing in negative emodiversity Post-Treatment, \(F(2, 101) = 1.063, \ p = 0.349, \ \eta_p^2 = 0.021\) (CTL \(M = 0.670, SE = 0.077\); MDD-CBT \(M = 0.810, SE = 0.059\); MDD-SSRI \(M = 0.723, SE = 0.112\)). In other words, the mean scores for negative emodiversity for the MDD-CBT group were higher than those of the MDD-SSRI group, which were higher than those for the CTL group (MDD-CBT > MDD-SSRI > CTL) (See Figure 5).

To test the significance of differences within groups for negative emodiversity Pre- and Post-Treatment, a repeated measures ANCOVA was conducted separately for each group controlling for mean negative emotion. There was no significant effect within the CTL group at Pre- and Post-Treatment \(F(1, 31) = 0.167, \ p = 0.686, \ \eta_p^2 = 0.005\), (Pre-Treatment \(M = 0.339, SE = 0.065\); Post-Treatment \(M = 0.435, SE = 0.061\)). However, there was a significant decrease in negative emodiversity within the MDD-CBT group from Pre- to Post-Treatment, \(F(1, 52) = 8.314, \ p = 0.006, \ \eta_p^2 = 0.138\), (Pre-Treatment \(M = 1.478, SE = 0.069\); Post-Treatment \(M = 0.914, SE = 0.084\)). There was no significant effect within the MDD-SSRI group at Pre- and
Post-Treatment, although there was still a trend of increasing positive emodiversity, $F(1, 13) = 2.071, p = 0.174, \eta^2_p = 0.137$ (Pre-Treatment $M = 1.624, SE = 0.117$; Post-Treatment $M = 0.863, SE = 0.198$) (See Figure 5).

**Study 1 Results for Aim 2**

To test Aim 2, we re-ran the same repeated-measures ANCOVA described above for Aim 1 while additionally controlling for mean depressive symptom severity (i.e., averaged across Pre- and Post-Treatment time points). As before, a Greenhouse-Geisser correction was used when assumptions for sphericity were not met and adjusted $F$ and $p$ values are reported. Effect sizes for significant results are reported as partial eta squared ($\eta^2_p$).

**Global Emodiversity.** For Global-ED, the main effect of Time was not significant, $F(1, 93) = 1.532, p = 0.219, \eta^2_p = 0.016$ suggesting no differences across participants as a function of treatment (Pre-Treatment $M = 2.226, SE = 0.032$; Post-Treatment $M = 2.215, SE = 0.036$). There was no main effect for Group, $F(2, 93) = 0.510, p = 0.602, \eta^2_p = 0.011$ suggesting that the three groups did not differ across both time points (CTL $M = 2.260, SE = 0.055$; MDD-CBT $M = 2.226, SE = 0.035$; MDD-SSRI $M = 2.176, SE = 0.058$). For the Group x Time interaction, there was also no significant interaction, $F(2, 93) = 1.053, p = 0.353, \eta^2_p = 0.022$.

**Positive Emodiversity.** For Positive-ED, the main effect of Time was still significant, $F(1, 94) = 7.449, p = 0.008, \eta^2_p = 0.073$ suggesting an increase in positive emodiversity from Pre-Treatment to Post-Treatment across all participants (Pre-Treatment $M = 1.601, SE = 0.061$; Post-Treatment $M = 1.936, SE = 0.037$). There was no main effect for Group, $F(2, 94) = 0.751, p = 0.475, \eta^2_p = 0.016$ suggesting that the three groups did not differ across both time points (CTL $M = 1.839, SE = 0.075$; MDD-CBT $M = 1.772, SE = 0.048$; MDD-SSRI $M = 1.695, SE = 0.079$). For the Group x Time interaction, there was no increasing trend in positive emodiversity in the
groups $F(2, 94) = 1.083, p = 0.343, \eta_p^2 = 0.023$. This was in contrast to the results found for Aim 1, in which there was a non-significant trend with positive emodiversity increasing from Pre-Treatment to Post-Treatment in each group.

To test the significance of differences within the groups for positive emodiversity Pre- and Post-Treatment, a repeated measures ANCOVA was conducted separately for each group controlling for mean positive emotion as well as mean depressive symptom severity. There was still no significant effect within the CTL group $F(1, 30) = 0.558, p = 0.461, \eta_p^2 = 0.018$, (Pre-Treatment $M = 2.054, SE = 0.038$; Post-Treatment $M = 2.074, SE = 0.034$). Within the MDD-CBT group, the increase in positive emodiversity from Pre- to Post-Treatment was no longer significant as it was without controlling for mean depressive symptom severity, $F(1, 49) = 3.948, p = 0.053, \eta_p^2 = 0.075$, (Pre-Treatment $M = 1.382, SE = 0.082$; Post-Treatment $M = 1.937, SE = 0.051$). Within the MDD-SSRI group, the increase in positive emodiversity became significant, also in contrast to without controlling for mean depressive symptom severity $F(1, 11) = 9.181, p = 0.011, \eta_p^2 = 0.455$, (Pre-Treatment $M = 1.370, SE = 0.128$; Post-Treatment $M = 1.798, SE = 0.082$).

**Negative Emodiversity.** For Negative-ED, there was a significant main effect of Time $F(1, 94) = 8.398, p = 0.005, \eta_p^2 = 0.082$ suggesting a decrease in negative emodiversity across all participants (Pre-Treatment $M = 1.165, SE = 0.055$; Post-Treatment $M = 0.720, SE = 0.071$).

There was no significant main effect for Group, $F(2, 94) = 0.353, p = 0.704, \eta_p^2 = 0.007$ suggesting that the groups did not differ in negative emodiversity Pre- and Post-Treatment (CTL $M = 0.870, SE = 0.098$; MDD-CBT $M = 0.986, SE = 0.063$; MDD-SSRI $M = 0.971 SE = 0.104$). For the Group x Time interaction there was a significant trend, $F(2, 94) = 5.802, p = 0.004, \eta_p^2 = 0.110$. To identify the source of the Group x Time interaction, two separate one-way ANOVAs
were run for each group to compare the Pre-Treatment and Post-Treatment separately. Results suggest that although there was no significant difference between all three groups Pre-Treatment, $F(2, 117) = 2.491, p = 0.087, \eta^2 = 0.042$, (CTL $M = 0.931, SE = 0.132$; MDD-CBT $M = 1.321, SE = 0.069$; MDD-SSRI $M = 1.344, SE = 0.106$), there was a significant difference between the CTL group and the MDD groups (CTL vs. MDD-CBT: Mean difference = -0.390, $p = 0.032$, CTL vs. MDD-SSRI: Mean difference = -0.413, $p = 0.038$). The three groups did not differ Post-Treatment $F(2, 88) = 0.100, p = 0.905, \eta^2 = 0.002$, (CTL $M = 0.714, SE = 0.080$; MDD-CBT $M = 0.763, SE = 0.067$; MDD-SSRI $M = 0.741, SE = 0.130$). However, the mean scores for negative emodiversity for the MDD-CBT group were still higher than those of the MDD-SSRI group, which were higher than those for the CTL group (MDD-CBT > MDD-SSRI > CTL), as seen in the results for Aim 1.

To test the significance of differences within the groups for negative emodiversity Pre- and Post-Treatment, a repeated measures ANCOVA was conducted separately for each group controlling for mean negative emotion as well as mean depressive symptom severity. There was still no significant change within the CTL group, $F(1, 30) = 0.973, p = 0.332, \eta^2 = 0.031$, (Pre-Treatment $M = 0.339, SE = 0.064$; Post-Treatment $M = 0.435, SE = 0.060$). Within the MDD-CBT group, the decrease in negative emodiversity from Pre- to Post-Treatment was also no longer significant $F(1, 49) = 2.537, p = 0.118, \eta^2 = 0.049$, (Pre-Treatment $M = 1.484, SE = 0.072$; Post-Treatment $M = 0.919, SE = 0.087$). Within the MDD-SSRI group, the decrease in negative emodiversity was still not significant when controlling for mean depressive symptom severity $F(1, 11) = 3.358, p = 0.094, \eta^2 = 0.234$, (Pre-Treatment $M = 1.740, SE = 0.095$; Post-Treatment $M = 0.875, SE = 0.216$).

In summary, after controlling for mean depressive symptom severity, both the increase
in positive emodiversity and decrease in negative emodiversity within the MDD-CBT group were no longer significant. By contrast, the increase in positive emodiversity within the MDD-SSRI group became significant.

**Study 1 Brief Discussion**

Study 1 found no significant differences in global emodiversity between the three groups and no significant changes within each group. For positive emodiversity, the MDD-CBT group had the most robust result, with a significant increase even after controlling for mean level of positive emotion. This was consistent with our predictions that CBT would increase positive emodiversity, as greater emodiversity seems to be beneficial to one’s mental and physical well-being. However, contrary to our hypothesis that negative emodiversity would increase as well, it actually significantly decreased in the MDD-CBT group when controlling for mean emotion. When depressive symptoms were also controlled for, some of the results lost their significance, such as the increase in positive emodiversity and decrease in negative emodiversity in the MDD-CBT group. However, some results remained significant such as the difference in negative emodiversity between the CTL group and the MDD groups Pre-Treatment, suggesting that emodiversity may track some changes above and beyond symptom improvement, which could make CBT more effective at preventing against relapse of MDD.

**Study 2**

The data from study 2 was provided by Drs. Jacqueline Persons, Ann Kring, and Cannon Thomas (e.g., as published in Kring et al., 2007). Study 2 included adults who were treated with CBT naturistically in private practice for depression and anxiety and completed weekly measures
of positive and negative affect over a variable treatment period. Three aspects of Study 2 represent a novel extension beyond Study 1. First, Study 2 employed a naturalistic study design in which people voluntarily sought out treatment for their depression (i.e., as therapy occurs in the real world), thus increasing its ecological validity. Second, Study 2 included all participants enrolled in treatment and enabled the examination of changes in emodiversity in association with continuous symptoms of depression. Third, Study 2 included a less severely depressed sample as compared with the MDD group in Study 1, thus enabling the examination of whether CBT may differentially impact potential changes in emodiversity depending on depressive symptom severity at baseline.

**Study 2 Participants**

Study 2 enrolled 44 treatment-seeking adult participants as clients in private practice for one of the original study authors (e.g., Dr. Jacqueline Persons). These participants were recruited for the study testing the tripartite model of anxiety and depression (Kring et al., 2007). Of the 44 adult participants, more than two-thirds of them were diagnosed with at least one mood disorder and at least one anxiety disorder. Because the study was addressing the relationship between depression and anxiety symptoms, three participants without a diagnosis of major depressive disorder or an anxiety disorder were excluded for the study, leaving a total sample size of 41. Demographics of the sample are included in Table 1.

**Study 2 Measures**

Participants completed weekly measures of emotion and depression symptoms, in addition to other measures not relevant to the present investigation, before their therapy session (Kring et al., 2007). We describe each below.
Depression symptoms. The Beck Depression Inventory (BDI; Beck et al., 1996) is a self-report 21-item questionnaire used to determine the severity of current depression symptoms. Each question is rated on a scale value of 0 to 3, with higher scores indicating greater depressive symptom severity. Scores in the 0-9 range suggest minimal depression, 10-18 indicate mild depression, 19-29 indicate moderate depression, and 30-63 indicate severe depression severity. Internal consistency for the BDI ($\alpha = 0.87$ pre-treatment and $\alpha = 0.92$ post-treatment) were good in the present investigation.

PANAS. The same PANAS measure was used in Study 2 as described above for Study 1. Unlike Study 1, where participants rated the extent to which they felt each emotion at the given moment before and after each task, participants in Study 2 rated the extent to which they felt each emotion in the past week, analogous to the time range for BDI symptom measures. Internal consistency for PA ($\alpha = 0.80$ pre-treatment and $\alpha = 0.84$ post-treatment) and NA ($\alpha = 0.92$ pre-treatment and $\alpha = 0.88$ post-treatment) in the present study were also good.

Study 2 Procedure

As described in Kring et al., (2007), participants were recruited by flyers that were part of new patient intake packets at the San Francisco Bay Area Center for Cognitive Therapy (SFBACCT). Sixteen of the 41 participants received treatment from the second author of the paper, Dr. Jacqueline Persons, a PhD level clinical psychologist with more than 20 years of experience. The remaining 28 participants received treatment from PhD level clinicians with between 1 and 7 years of experience at the SFBACCT. CBT sessions were held weekly, with the participants receiving an average of 18.00 (SD = 12.67) sessions. One participant received only 2 sessions, and the rest received at least 6. Seventy percent of the participants were also taking
medications. The effectiveness of CBT treatment was evidenced by a significant decrease in the BDI across the course of treatment. Patients were debriefed at the end of the study.

**Study 2 Results**

**Study 2 Results for Aim 1**

**Preliminary Analyses.** We first examined associations between baseline symptoms of depression with global, positive, or negative emodiversity. Bivariate correlation analyses indicated that greater depression symptoms were associated with increased negative emodiversity ($r = 0.36, p = 0.03$) but were not correlated with global ($r = 0.14, p = 0.42$) or positive ($r = -0.04, p = 0.83$) emodiversity.

**Overview of Main Analyses.** Three separate repeated-measures analyses of covariance (ANCOVAs) were conducted for the two time points (Pre-Treatment and Post-Treatment) for each of the three separate emodiversity variables (Global-ED, Positive-ED, Negative-ED), while controlling for mean PA and/or NA respectively. In addition, because this study was a naturalistic study and the number of sessions each participant received varied, the total number of sessions was also controlled for in all analyses. A Greenhouse-Geisser correction was used when assumptions for sphericity were not met and adjusted $F$ and $p$ values are reported. Effect sizes for significant results are reported as partial eta squared ($\eta_p^2$). All reported $p$ values are two-tailed. Means and standard deviations are presented in Table 1.

**Global Emodiversity.** For Global-ED, there was no significant main effect of Time, $F(1, 31) = 2.310, p = 0.139, \eta_p^2 = 0.069$. This suggests that there was no change in global emodiversity across all participants as a function of treatment (Pre-Treatment $M = 2.544, SE = 0.041$; Post-Treatment $M = 2.435, SE = 0.057$) (See Figure 6).
**Positive Emodiversity.** For Positive-ED, there was a significant main effect of Time, $F(1, 33) = 5.388, p = 0.027, \eta_p^2 = 0.140$. This suggests that positive emodiversity decreased across all participants as a result of treatment (Pre-Treatment $M = 1.868, SE = 0.060$; Post-Treatment $M = 1.820, SE = 0.090$) (See Figure 7).

**Negative Emodiversity.** For Negative-ED, there was also a significant main effect of Time, $F(1, 34) = 4.931, p = 0.033, \eta_p^2 = 0.127$. This suggests that negative emodiversity also decreased across all participants as a result of treatment (Pre-Treatment $M = 1.817, SE = 0.054$; Post-Treatment $M = 1.467, SE = 0.088$) (See Figure 8).

**Study 2 Results for Aim 2**

To test Aim 2 for Study 2, we re-ran the same repeated-measures ANCOVA while additionally controlling for mean depressive symptom severity (i.e., averaged across Pre- and Post-Treatment time points), in addition to the covariates noted above for Aim 1. As before, a Greenhouse-Geisser correction was used when assumptions for sphericity were not met and effect sizes for significant results are reported as partial eta squared ($\eta_p^2$).

**Global Emodiversity.** For Global-ED, there was still no significant main effect of Time, $F(1, 30) = 3.389, p = 0.076, \eta_p^2 = 0.101$ (Pre-Treatment $M = 2.544, SE = 0.038$; Post-Treatment $M = 2.435, SE = 0.058$). This was consistent with Aim 1 results.

**Positive Emodiversity.** For Positive-ED, there was still a significant main effect of Time, $F(1, 32) = 4.537, p = 0.041, \eta_p^2 = 0.124$ (Pre-Treatment $M = 1.868, SE = 0.061$; Post-Treatment $M = 1.820, SE = 0.090$). This was consistent with Aim 1 results.

**Negative Emodiversity.** For Negative-ED, there was also still a significant main effect of Time, $F(1, 33) = 6.746, p = 0.014, \eta_p^2 = 0.170$ (Pre-Treatment $M = 1.817, SE = 0.051$; Post-Treatment $M = 1.467, SE = 0.088$). This was consistent with Aim 1 results.
In sum, results did not change when additionally controlling for depressive symptoms for global, positive, and negative emodiversity for Study 2.

**Study 2 Brief Discussion**

Study 2 examined changes in emodiversity among a naturalistic sample of treatment-seeking adults receiving CBT in private practice. Results indicated that both positive and negative emodiversity decreased after CBT treatment across all participants, and global emodiversity did not change. These results held even after controlling for depressive symptom severity. These results diverged from Study 1.

**General Discussion**

The association between emodiversity and better mental and physical health has been shown to be robust and theoretically-grounded (e.g., Quoidbach et al., 2014; 2018). This multi-study analysis sheds some new light on emotional diversity. While previous studies have shown that emodiversity is beneficial independent of mean level of emotion across general community samples (e.g., Quoidbach et al., 2014; 2018), the present investigation was the first to examine whether empirically-supported psychological interventions for emotional disorders – focused on depression – is associated with improvements or changes in emodiversity. Given that there is substantial literature on CBT as an effective treatment for MDD in adults as well as literature on the importance of emodiversity, analyzing whether emodiversity changes as a function of CBT could have important clinical implications.

**Changes in Emodiversity as a Function of CBT Treatment.** Our first aim examined whether CBT was associated with changes (i.e., increases) in emodiversity as a function of CBT treatment. In Study 1, there were no significant results found for global emodiversity between or
within groups. For positive emodiversity, the MDD-CBT group had the most robust result, with a significant increase even after controlling for mean level of positive emotion. This was consistent with our predictions that CBT would increase emodiversity, as greater emodiversity seems to be beneficial to one’s mental and physical well-being (e.g., Quoidbach et al., 2014; 2018). This significant increase in positive emodiversity as a result of CBT treatment indicates that CBT for depression does indeed change emodiversity. However, contrary to our hypothesis that negative emodiversity would increase as well, it actually significantly decreased in the MDD-CBT group when controlling for mean emotion. This suggests that patients with MDD may have too much negative emodiversity compared to healthy people and decreasing negative emodiversity in depressed people may actually be more beneficial for them. This supports literature on the ability to control or regulate negative emotions and its associations with improved health outcomes (e.g., Gross & John, 2003; Ong, Bergeman, Bisconti, & Wallace, 2006).

We aimed to replicate these findings in Study 2 examining CBT naturalistically among a treatment-seeking adult sample. In Study 2, all participants received CBT, although with varying number of sessions. Surprisingly, and in contrast to Study 1 results and our a priori predictions, both positive and negative emodiversity actually decreased after CBT treatment, and global emodiversity did not change. These results suggest that changes in emodiversity as a function of CBT treatment for depression may be more nuanced. There are several potential explanations for these findings. First, the nature of the two studies differed in many aspects, with Study 2 being more naturalistic and therefore having variable number of sessions between patients. At the same time, when we ran a correlation analysis between the number of sessions and emodiversity, we
did not find a significant correlation\(^1\). Second, it is possible that the difference may be accounted for by the severity of depression across both studies. Indeed, Study 1 contained a more severely depressed sample on average compared with Study 2. This sample difference could possibly indicate that the severity of depression may affect their emotion diversity and response to treatment. This suggests that the impact of emodiversity as a function of CBT may be quadratic rather than linear, such that for more severely depressed participants (e.g., with greater affective flattening; Rottenberg et al., 2005) CBT may function to increase emotional diversity and broaden emotion experience whereas for less severely depressed participants who may experience more current distress it may function to constrain and hence contain overwhelming emotional experiences. Further work is needed to test this possibility.

Another possibility is worth noting. Specifically, given that Study 1 and Study 2 used the same PANAS scale with the same emotion items, we compared the global, positive, and negative emodiversity of the MDD-CBT group in Study 1 with the global, positive, and negative emodiversity of the participants in Study 2. We found that the participants in Study 2 had higher global, positive, and negative emodiversity at baseline compared to MDD-CBT participants in Study 1. In other words, Study 2 participants had higher positive emodiversity at baseline that decreased after CBT treatment, whereas Study 1 MDD-CBT participants had lower positive emodiversity at baseline that increased after CBT treatment. This could suggest that there may be an optimal range for positive emodiversity. So since Study 2 started higher, positive emodiversity decreased, whereas Study 1 started lower and therefore increased after treatment. The different trends could also simply be an effect of Study 1 participants being a severely depressed sample and Study 2 participants being mildly depressed. Perhaps higher positive

\(^1\) Global ED: \(r = 0.18, p = 0.30\); Positive ED: \(r = 0.25, p = 0.14\); Negative ED: \(r = 0.01, p = 0.94\)
emodiversity is more beneficial to more severely depressed patients, and CBT helps achieve this in a severely depressed population.

For negative emodiversity, the MDD-CBT group from Study 1 had a larger decrease in negative emodiversity compared to the participants from Study 2. This may support the possibility of having an optimal range for negative emodiversity as well. Depressed patients from both MDD groups in Study 1 had much higher levels of negative emodiversity. Negative emodiversity was also positively correlated to the BDI in Study 2, meaning that the more severe the depressive symptoms, the higher the negative emodiversity. This indicates that depressed patients may have higher levels of negative emodiversity in general and may benefit from CBT to lower these levels. Again, this could support that the relationship between level of diversity and better psychological well-being could be quadratic rather than linear. Greater emodiversity in general might not always be beneficial, and maybe too much emodiversity can be harmful as well, especially for negative emodiversity, which could explain the findings from these two studies included in this analysis.

**Examining Robustness of Results when Controlling for Depressive Symptom Severity.** We then examined whether there were significant changes in the results after controlling for mean depressive symptom severity to determine whether the changes in emodiversity after treatment are independent of depressive symptoms (Aim 2). In Study 1, some significant findings lost their significance after controlling for mean depressive symptom severity. This was not surprising, as more covariates can reduce power. The increase in positive emodiversity and decrease in negative emodiversity within the MDD-CBT group were not significant, unlike the results for Aim 1. The MDD group still had significantly higher negative emodiversity, showing that some results did still retain significance. In Study 2, all results
remained robust even after controlling for mean depressive symptom severity. These results indicate that emodiversity may track changes above and beyond symptom improvement, even though it is still affected by different factors.

**Differential Efficacy of CBT versus SSRIs on Emodiversity.** Although not part of our a priori predictions, it is noteworthy that we found differential effects on CBT versus SSRIs in Study 1. There is wide consensus that antidepressant medications are highly effective in treating depression. Today, antidepressants such as selective serotonin reuptake inhibitors (SSRIs) are commonly used. Neurotransmitters like serotonin are released at synapses, the area between two neurons used for transmitting signals. The neurotransmitters then bind to receptors, with different neurotransmitters having different effects. Serotonin contributes to feelings of well-being and happiness. Extra serotonin still in the synaptic cleft that were not taken up by receptors are taken back by the neuron that released them. SSRIs block this reuptake of the serotonin, leaving more serotonin that is available. There is a well-established association between depression and levels of serotonin (Stockmeier, 2003).

Researchers have found that the relationship between serotonin and depression is a bit more complicated than this, but there is still a lot of interest in using serotonin as treatment for depression because SSRIs have been shown to be effective as antidepressants (Cowen & Browning, 2015). Studies have shown that administration of SSRIs may shift responses to emotionally-valenced stimuli in a positive way in both healthy and depressed patients. This seems to be due to serotonergic innervation in the amygdala, the area of the brain responsible for basic emotional responses (Cowen & Browning, 2015).

This existing data shows that antidepressants, specifically SSRIs, are an important option for treatment for depression because they are proven to be very effective. However, as mentioned
before, studies have shown that perhaps antidepressant medications only treat symptoms of depression, and while they may be effective in doing that, they do not seem to prevent against relapse. This may suggest that pharmacotherapy might not cause any significant changes that can help depression long-term without having to depend on medication. A gap in our understanding is whether or not antidepressants may have the potential to also impact emotional relevant processes such as emodiversity as well.

Because only Study 1 had a different group with antidepressants as well as a healthy control, it is hard to draw conclusions about the effectiveness of SSRIs as a treatment for depression and if it impacts emodiversity. The results from Study 1 indicate that participants taking SSRIs had changes in emodiversity that trended in the same direction as those receiving CBT. However, the changes in the MDD-CBT were more robust than those in the MDD-SSRI group, showing that CBT may elicit these changes in a way that goes beyond symptom improvement and supports literature suggesting CBT may be more effective in preventing relapse of depression.

**Limitations and Future Directions**

We acknowledge several limitations in this investigation. First, one challenge in this multi-study analysis was obtaining datasets studying CBT on depression that included emotion measures. With so many studies on CBT and antidepressants as treatments for depression, it was surprising that so few actually collected data measures. Having more data sets to include in addition to the two included in this analysis would give clearer insight on the questions we ask. Second, we were limited to examining a single disorder (e.g., depression) and were not able to draw more transdiagnostic conclusions about the efficacy of CBT to enhance emodiversity across disorders. Future research should also explore the effects of CBT on emodiversity for
other mood disorders, such as bipolar disorder. Third, as a first step we focused specifically on CBT and were not able to examine other well-studied interventions for mood disorders. It would also be interesting to investigate other established treatments for mood disorders and their effect on emodiversity, such as Mindfulness Based Cognitive Therapy. Future research could also include a follow-up study with a large community sample with a range of depression severity, from mild to severe, to address inconsistencies in the analyses of these two studies.

Taken together, our findings suggest that there might be a different interaction between emodiversity and well-being and that more emodiversity may not necessarily always a good thing, especially for depressed patients. However, the results show that emodiversity is significant in assessing improvement for depressed patients.
References


Table 1. Demographics and Clinical Characteristics Across Study 1 and Study 2.

<table>
<thead>
<tr>
<th>Demographics</th>
<th>CTL (n=35)</th>
<th>MDD-CBT (n=70)</th>
<th>MDD-SSRI (n=22)</th>
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<th>All (n=41)</th>
<th>Statistic</th>
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<td>$\chi^2=3.644$</td>
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<td>Education (Yrs)</td>
<td>16.03 (2.18)</td>
<td>15.24 (2.21)</td>
<td>13.86 (1.78)</td>
<td>F=6.980</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>BDI (BDI-II for Study 1)</td>
<td>1.44 (2.62)</td>
<td>31.95 (8.95)</td>
<td>30.10 (10.04)</td>
<td>--</td>
<td>17.38 (9.20)</td>
<td>--</td>
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</tbody>
</table>

*Note: CTL = Healthy control group; MDD-CBT = Participants with Major Depressive Disorder who received CBT treatment; MDD-SSRI = Participants with Major Depressive Disorder who received SSRI treatment; BDI = 21-item self-report questionnaire to measure severity of depression severity; BDI-II = revision of the BDI, still a 21-item self-report questionnaire that also accounts for increases in sleep and appetite in addition to decreases.*
Table 2. Full list of “top 15” researchers in the field of mood disorders and CBT not already contacted using data solicitation approach (i.e., “Approach 1”)

<table>
<thead>
<tr>
<th></th>
<th>Name</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>Greg Siegle</td>
</tr>
<tr>
<td>2</td>
<td>Jutta Joorman</td>
</tr>
<tr>
<td>3</td>
<td>Lauren Alloy</td>
</tr>
<tr>
<td>4</td>
<td>Mark Whisman</td>
</tr>
<tr>
<td>5</td>
<td>Jacqueline Persons</td>
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<tr>
<td>6</td>
<td>Stefan Hofmann</td>
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<td>7</td>
<td>David Barlow</td>
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<td>8</td>
<td>David Miklowitz</td>
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<td>9</td>
<td>Michael Otto</td>
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<td>10</td>
<td>Sheri Johnson</td>
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<td>11</td>
<td>Roz Shafran</td>
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<tr>
<td>12</td>
<td>Michael Thase</td>
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<tr>
<td>13</td>
<td>Greg Murray</td>
</tr>
<tr>
<td>14</td>
<td>Ellen Frank</td>
</tr>
<tr>
<td>15</td>
<td>Allison Harvey</td>
</tr>
</tbody>
</table>
Figure 1. Overview of Data Acquisition Methods.
Flow charts demonstrating steps taken to acquire articles according to a three-prong strategy. A) Approach 1: Authors from articles within top five meta-analyses contacted. B) Approach 2: Top 15 researchers in the field of CBT and mood disorders. C) Approach 3: Authors of relevant articles within the last 15 years contacted.
Figure 2. Sample solicitation email for data acquisition.

**Email template for authors of articles:**

Subject Line: Emotion Data for Inclusion in a Meta-Analysis?

cc: Stephanie, June, Mike, Jordi

Dear (Author),

We are a group of psychology researchers (from Harvard, CU Boulder, and ESADE) examining the concept of “emotional diversity” (e.g., [www.emodiversity.org](http://www.emodiversity.org)) in the context of cognitive behavioral treatment for mood disorders. This project examines whether CBT is effective in changing the variety and abundance of different emotions that participants report experiencing, as previous work has linked greater emotional diversity with reduced symptoms of depression.

We have reviewed the most highly-cited meta-analyses on CBT for mood disorders and came across your interesting (year) article titled: “*(Title of article.)*” We wondered if you collected any self-reports of emotion at baseline (pre-treatment) and once treatment was completed (post-treatment)? If so, assuming you have not already published on this data in particular, would you be willing to share your self-reported emotion data with us so that we could compute a validated index of emotional diversity and examine changes as a function of treatment? We also imagine it would be useful to have information on participant demographics and associated clinical characteristics too.

We would be delighted to work together as collaborators should any of the analyses yield interesting results for publication.

Sincerely,
Stephanie Yu, Jordi Quoidbach, Michael Norton, & June Gruber
**Figure 3:** Global Emodiversity Controlling for Mean Emotion Across Groups Pre-Treatment and Post-Treatment in Study 1

*Note:* There were no significant differences between groups at Pre- or Post-Treatment. There were also no significant differences within groups at Pre- or Post-Treatment.
**Figure 4.** Positive Emodiversity Controlling for Mean Emotion Across Groups Pre-Treatment and Post-Treatment in Study 1

![Positive Emodiversity With Controlling for Mean Emotion](image)

*Note:* There was no difference across groups Pre-Treatment. The MDD-SSRI group had lower levels of positive emodiversity than the MDD-CBT group Post-Treatment. Within the MDD-CBT group, there was a significant increase in positive emodiversity after CBT treatment.
**Figure 5.** Negative Emodiversity Controlling for Mean Emotion Across Groups Pre-Treatment and Post-Treatment in Study 1

*Note:* Both the MDD-CBT and MDD-SSRI groups had significantly higher levels of negative emodiversity compared to the CTL group at baseline (i.e., pre-treatment). Within the MDD-CBT group, there was a significant decrease in negative emodiversity from pre-treatment to post-treatment time points.
Figure 6. Global Emodiversity Controlling for Mean Positive and Negative Emotion Pre-Treatment and Post-Treatment in Study 2

Note: There was no significant difference in global emodiversity from Pre- to Post-Treatment.
Figure 7. Positive Emodiversity Controlling for Mean Positive Emotion Pre-Treatment and Post-Treatment in Study 2

Note: There was a significant decrease in positive emodiversity between pre-treatment and post-treatment time points.
Note: There was a significant decrease in negative emodiversity between pre-treatment and post-treatment time points.
Acknowledgements

The past two years that I have been in the PEP Lab has been a wonderful experience for me. I would like to thank the lab and everybody in the lab for the opportunity to write an undergraduate honors thesis and for providing me with resources. I would especially like to thank Dr. June Gruber for being my mentor and for her support throughout the entire project. Her mentorship has been fundamental to my thesis project.

I would like to thank Jordi Quoidbach and Michael Norton for their collaboration on this project. They were part of the original conception of emodiversity and have provided tremendous support in formulating the project and analyzing the data.

I would also like to thank Dr. Greg J. Siegle and the other authors of the Siegle et al., 2012 paper as well as Dr. Ann M. Kring, Dr. Jacqueline Persons, and Dr. Cannon Thomas for providing their data sets for analysis. Without their valuable datasets, we would not have had data to analyze the impact of CBT on emodiversity, and we are extremely grateful for their generosity in sharing their hard-earned data with us. I look forward to hopefully collaborating with them on the subsequent manuscript arising from this initial thesis project.

Lastly, I want to thank Dr. Jennifer Martin and Dr. Sona Dimidjian for serving on my committee and for giving me thoughtful feedback for my project.
**Supplementary Materials**

For interested readers, we also include results without controlling for mean emotion intensity below organized by Study 1 and Study 2 separately.

**Study 1 Results**

**Results for Aim 1**

**Global Emodiversity.** For Global-ED without controlling for the mean intensity of emotion, there was no significant main effect of Time, $F(1, 99) = 0.011, p = 0.918, \eta^2_p = 0.000$. In other words, across all participants there was no change in global emodiversity. For Group, there was no significant main effect, $F(2, 99) = 0.192, p = 0.825, \eta^2_p = 0.004$. In other words, the three groups did not differ across both time points (CTL $M = 2.201$, SE $= 0.059$; MDD-CBT $M = 2.239$, SE $= 0.046$; MDD-SSRI $M = 2.190$, SE $= 0.087$). For the Group x Time interaction, there was no significant interaction, $F(2, 99) = 1.087, p = 0.341, \eta^2_p = 0.021$ (See Figure 9).

**Positive Emodiversity.** For Positive-ED without controlling for the mean intensity of emotion, there was a significant trend for the main effect of Time, $F(1, 99) = 16.392, p < 0.001, \eta^2_p = 0.142$. In other words, across all participants there was a significant increase in positive emodiversity (Pre-Treatment $M = 1.594$, SE $= 0.073$; Post-Treatment $M = 1.911$, SE $= 0.044$). For Group, there was also a significant main effect, $F(2, 99) = 13.394, p < 0.001, \eta^2_p = 0.213$. In other words, the three groups did differ across both time points, with the CTL group having the highest positive emodiversity (CTL $M = 2.064$, SE $= 0.070$; MDD-CBT $M = 1.656$, SE $= 0.055$; MDD-SSRI $M = 1.538$, SE $= 0.104$). For the Group x Time interaction there was a significant trend, $F(2, 99) = 6.258, p = 0.003, \eta^2_p = 0.112$. To identify the source of the Time x Group interaction, two separate one-way ANOVAs were run for each group to compare the Pre-
Treatment and Post-Treatment separately. Results suggest the CTL group differed from the MDD groups in positive emodiversity Pre-Treatment, having higher scores compared to both MDD-CBT and MDD-SSRI, $F(2, 124) = 11.701, p < 0.001, \eta^2_p = 0.161, (CTL M = 2.049, SE = 0.108; MDD-CBT M = 1.431, SE = 0.075; MDD-SSRI M = 1.452, SE = 0.138)$. The CTL group only differed from the MDD-SSRI group in positive emodiversity Post-Treatment, $F(2, 101) = 4.180, p = 0.018, \eta^2_p = 0.078, (CTL M = 2.074, SE = 0.069; MDD-CBT M = 1.936, SE = 0.053; MDD-SSRI M = 1.725, SE = 0.101)$. The mean scores for positive emodiversity for the CTL group was still higher but less so because the mean scores for positive emodiversity Post-Treatment for the MDD-CBT and MDD-SSRI groups were raised (See Figure 10).

**Negative Emodiversity.** For Negative-ED without controlling for the mean intensity of emotion, there was a significant trend for the main effect of Time, $F(1, 99) = 19.459, p < 0.001, \eta^2_p = 0.164$. In other words, across all participants there was a significant decrease in negative emodiversity (Pre-Treatment $M = 1.147, SE = 0.074$; Post-Treatment $M = 0.738, SE = 0.087$). For Group, there was a significant main effect as well, $F(2, 99) = 21.945, p < 0.001, \eta^2_p = 0.307$. In other words, the three groups did differ across both time points (CTL $M = 0.387, SE = 0.102$; MDD-CBT $M = 1.196, SE = 0.080$; MDD-SSRI $M = 1.244, SE = 0.151$). For the Group x Time interaction there was a significant trend, $F(2, 99) = 8.553, p < 0.001, \eta^2_p = 0.147$. To identify the source of the Time x Group interaction, two separate one-way ANOVAs were run for each group to compare the Pre-Treatment and Post-Treatment separately. Results suggest the CTL group differed from the MDD groups in negative emodiversity Pre-Treatment, $F(2, 123) = 42.405, p < 0.001, \eta^2_p = 0.412, (CTL M = 0.348 SE = 0.110; MDD-CBT M = 1.531, SE = 0.078; MDD-SSRI M = 1.576, SE = 0.141)$. The groups also differed in negative emodiversity Post-Treatment, $F(2, 101) = 4.127, p = 0.019, \eta^2_p = 0.077, (CTL M = 0.435, SE = 0.134; MDD-CBT M = 0.914, SE =
0.105; MDD-SSRI \( M = 0.863, SE = 0.199 \). In other words, the mean scores for negative emodiversity for the MDD-CBT group were higher than those of the MDD-SSRI group, which were higher than those for the CTL group (MDD-CBT > MDD-SSRI > CTL) (See Figure 11).

**Study 2 Results**

**Results for Aim 1**

**Global Emodiversity.** For Global-ED without controlling for the mean intensity of emotion, there was no significant main effect of Time, \( F(1, 33) = 2.434, p = 0.128, \eta^2_p = 0.069 \) (Pre-Treatment \( M = 2.544, SE = 0.052 \); Post-Treatment \( M = 2.435, SE = 0.070 \)).

**Positive Emodiversity.** For Positive-ED without controlling for the mean intensity of emotion, there was no significant main effect of Time, \( F(1, 34) = 2.423, p = 0.129, \eta^2_p = 0.067 \) (Pre-Treatment \( M = 1.868, SE = 0.080 \); Post-Treatment \( M = 1.820, SE = 0.125 \)).

**Negative Emodiversity.** For Negative-ED without controlling for the mean intensity of emotion, there was no significant main effect of Time, \( F(1, 35) = 1.752, p = 0.194, \eta^2_p = 0.048 \) (Pre-Treatment \( M = 1.817, SE = 0.071 \); Post-Treatment \( M = 1.467, SE = 0.116 \)).
Figure 9: Global Emodiversity Without Controlling for Mean Emotion Across Groups Pre-Treatment and Post-Treatment in Study

Note: There were no significant differences between groups at Pre- or Post-Treatment. There were also no significant differences within groups at Pre- or Post-Treatment.
Figure 10. Positive Emodiversity Without Controlling for Mean Emotion Across Groups Pre-Treatment and Post-Treatment in Study 1

Note: The MDD groups had significantly lower positive emodiversity than the CTL group at baseline (i.e., pre-treatment). The MDD-SSRI group had lower levels of positive emodiversity than the CTL group Post-Treatment. Within the CBT and SSRI groups, there is a significant increase in positive emodiversity after treatment.
Figure 11. Negative Emodiversity Without Controlling for Mean Emotion Across Groups Pre-Treatment and Post-Treatment in Study

Note: Both the MDD-CBT and MDD-SSRI groups had significantly higher levels of negative emodiversity compared to the CTL group at baseline (i.e., pre-treatment). The MDD-CBT had higher negative emodiversity than the CTL post-treatment. Within the MDD-CBT and MDD-SSRI groups, there was a significant decrease in negative emodiversity from pre-treatment to post-treatment time points.