TRANSACTIONAL EFFECTS OF DEPRESSION IN TRANSDIAGNOSTIC GROUP COGNITIVE BEHAVIOR THERAPY FOR ANXIETY

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A Dissertation

Presented to

The Faculty of the Department

of Psychology

University of Houston

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In Partial Fulfillment

Of the Requirements for the Degree of

Doctor of Philosophy

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By

Alexander M. Talkovsky

December, 2014

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Abstract

Anxiety and depressive disorders co-occur at high rates, and their comorbidity typically creates a more severe clinical presentation than when either occurs alone. Depression shares risk factors with anxiety disorders such as negative affectivity (NA), or the predisposition to experience negative emotions. NA is a higher-order factor that subsumes many cognitive vulnerabilities, and may underlie this comorbidity. Secondary depression affects the treatment of anxiety and is associated with poorer treatment outcomes compared to an anxiety diagnosis alone. Transdiagnostic treatments present a promising option to improve both anxiety and depression by targeting shared risks. Research using transdiagnostic protocols suggests that comorbid and simple cases improve at comparable rates, and that secondary diagnoses significantly improve following treatment. This study aimed to examine the reciprocal effects of secondary depression in transdiagnostic group cognitive behavioral therapy for anxiety. This study also aimed to analyze the role of NA as a transdiagnostic mediator in the treatment of both anxiety and depression.

Depressed individuals scored more severely on measures of anxiety, depression, and negative affect, as well as clinician-rated severity of primary anxiety disorder and overall clinical presentation at pre-treatment. However, only the differences in self-reported depression symptoms and NA remained significant at post-treatment. When change in self-reported anxiety was modeled over treatment, the best-fitting model was such that depressed

individuals began treatment scoring more severely, improved at a greater rate, and reached a similar outcome to those without a depressive disorder. Analyses of variance revealed that all individuals improved on every measure; depression only had an effect on one self-report measure of anxiety, and the time x depression interaction did not have any effect.

All individuals improved in self-reported depression, but there was a main effect of depression and the time x depression interaction was not significant. Among depressed individuals, the clinician-rated severity of the depression diagnoses improved significantly and, on average, dropped from a mild to moderate score to one that is no longer clinically significant based on scoring conventions. The time x depression interactions was not significant in any analyses. Mediation analyses showed that NA fully mediated improvements in anxiety, but only partially mediated improvements in depression.

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Introduction

Epidemiology

Anxiety and depression are the two most common classes of psychological disorders, with specific phobia, social phobia, and major depressive disorder being the three most common diagnoses in epidemiological samples (Kessler, Chiu, Demler, & Walters, 2005). Diagnostic comorbidity is common amongst these classes of disorders. For instance, among 1127 individuals presenting for treatment for anxiety, Brown, Campbell, Lehman, Grisham, and Mancill (2001) found that at the time of evaluation, 57% had at least one comorbid Axis I diagnosis, 43% had a comorbid anxiety disorder, and 28% had a comorbid depressive disorder; the total rate of comorbid anxiety or depressive diagnoses was 55%. The principal diagnostic categories at greatest risk for comorbidity were major depressive disorder, generalized anxiety disorder (GAD), and panic disorder with agoraphobia. Comorbid depression diagnoses were most common amongst the principal diagnoses of posttraumatic stress disorder (PTSD), generalized anxiety disorder (GAD), and panic disorder with agoraphobia. If not for the DSM (American Psychiatric Association, 2013) hierarchy rule stating that GAD cannot be diagnosed exclusively during a major depressive episode, at least two thirds of individuals with a primary mood disorder diagnosis would also meet criteria for GAD. These numbers expanded when Brown and colleagues examined lifetime diagnoses, with 81% of individuals meeting criteria for at least one additional lifetime Axis I diagnosis. For individuals with a primary anxiety diagnosis, the most common additional lifetime diagnosis was major depression, with 50% of individuals meeting lifetime criteria. Social phobia, obsessive-compulsive disorder (OCD), GAD and PTSD all had significantly elevated risks of lifetime major depression (Brown et al., 2001). Further, in a Dutch epidemiological

survey (Lamers, et al., 2011), comorbidity rates of depressive disorder were 63% in the past 12 months and 81% over their lifetimes based on DSM-IV-TR (American Psychiatric Association, 2000) criteria. As comorbidity is often associated with greater severity and a more chronic course (Lamers, et al., 2011), this area requires further analysis.

Anxiety disorders typically precede the onset of depression, and the presence of an anxiety disorder may be the most significant risk factor for the development of depression (Hranov, 2007). Also, Belzer and Schneier (2004) found that comorbid anxiety and depression is typically associated with earlier onset, a chronic course, and greater functional impairment. Among individuals with comorbid anxiety and depression, Lamers and colleagues (2011) found that anxiety preceded depression in 57% of the cases and the opposite was true in only 18%; they also observed that the individuals for whom anxiety preceded depression showed a long duration of depression and/or anxiety symptoms and earlier age of onset. In a Dutch epidemiological survey (de Graaf, Bijl, Beekman, & Vollebergh, 2003), the majority of individuals with comorbid anxiety and depression reported a history of anxiety prior to the onset of depression with the exception of individuals with a primary diagnosis of generalized anxiety disorder (GAD), for whom the onset of the two conditions was most often simultaneous. Strong relationships were observed between depression and diagnoses of GAD, obsessive compulsive disorder (OCD), and panic disorder, and the relationship with social phobia and specific phobia were more modest although they were statistically significant as well (de Graaf, Bijl, Beekman, & Vollebergh, 2003).

Phenomenology by Anxiety Diagnosis

Consistent with the results of de Graaf and colleagues (2003), there appear to be varying relationships between depression and anxiety disorders. In an analysis of the National Comorbidity Survey, Kessler and colleagues (1998) found that 55.6% of respondents with lifetime panic disorder also met lifetime criteria for depression, and 11.2% of individuals with lifetime major depression met criteria for lifetime panic disorder. Depression also tended to predict the onset of panic attacks, but not panic disorder. Panic attacks, with or without panic disorder, predicted the onset of major depression. They concluded that primary panic is a marker of an underlying Axis I causal risk factor and that secondary panic is a severity marker of depression rather than a comorbid condition, or that there is a strong relationship between depression and panic in some latent pathological characteristic. Panic disorder patients with comorbid depression tend to be more afraid of future panic attacks and tend to be judged as more impaired by clinicians than non-depressed panic patients (McLean, Woody, Taylor, & Koch, 1998) suggesting unique clinical need in these individuals compared to those diagnosed with panic disorder alone.

Depression has a strong interpersonal component (Joiner & Coyne, 1999), so a relationship with social phobia is not surprising. Among social phobia patients, those with comorbid depression exhibit more severe social anxiety, greater impairment, lower current and past year global assessment of functioning (GAF) scores, and earlier age of onset than simple social phobia cases. This extends beyond comparisons against non-anxious controls, as compared to individuals with comorbid anxiety, social phobia patients with comorbid depression demonstrate a more chronic course, greater impairment, earlier age of onset, and lower current and past year GAF scores (Erwin, Heimberg, Juster, & Mindlin, 2002).

Major depression is the most frequently co-occurring disorder among OCD patients (Huppert, 2009). Compared to non-depressed OCD patients, individuals with comorbid OCD and major depression tend to have an earlier age of first diagnosis, more severe obsessive-compulsive symptoms, and a greater likelihood of a family history of depression, but only earlier age of OCD onset remains a significant predictor when controlling for the other variables (Overbeek, Schruers, Vermetten, & Griez, 2002). Further, comorbid depression is associated with more severe obsessions but not compulsions, and significantly lower quality of life related to physical health, psychological health, and social relationships (Besiroglu, Uguz, Saglam, Agargun, & Cilli, 2007).

Depression and PTSD frequently co-occur, as more than half of individuals with PTSD may demonstrate clinically significant depressive symptoms (Blanchard, Buckley, Hickling, & Taylor, 1998; Nixon, Resick, & Nishith, 2004). Among individuals recruited from Veterans Affairs (VA) primary care clinics, individuals meeting criteria for PTSD and major depression were younger, less likely to be Caucasian, had higher levels of education, and reported greater physical disability than those that did not meet criteria for depression. Patients with both PTSD and depression also reported lower perceived social support, greater levels of anxiety, were more likely to have had a panic attack in the past month, and reported greater levels of suicidal ideation. The comorbid individuals also sought more outpatient health care visits, and more mental health related visits. Depressed PTSD patients also were more likely to make an emergency room visit for an emotional problem than those without a depression diagnosis (Campbell et al., 2007).

Depression's relationship with GAD appears to be the strongest amongst the anxiety disorders. GAD is more likely to co-occur with depression than any of the other anxiety

disorders (Alloy, Kelly, Mineka, & Clements, 1990; Mineka, Watson, & Clark, 1998). The major depression and GAD pairing is the second most common diagnostic couplet behind only double-depression (Kessler, Chiu, Demler, & Walters, 2005). Watson (2009) argues that GAD is more strongly related to depression than to the other anxiety disorders. Evidence for a shared genetic diathesis is equivocal for anxiety in general, but appears to be strong for depression and GAD specifically (Hranov, 2007). Comorbid GAD and depression is associated with longer bouts of depression and lower likelihood of depression remission compared to depression alone (Fava, et al., 2000). Worry, typically regarded as the hallmark of GAD, demonstrates comparable elevations in depressed patients suggesting that these disorders may have similar underlying vulnerabilities despite the potential differences in the content of worry (Starcevic, 1995). These similarities have led some to claim that anxious worry and depressive rumination are different manifestations of the same process (Ehring & Watkins, 2008).

Mechanisms

There are various mechanistic explanations of comorbid anxiety and depression. The first is diagnostic overlap. The criteria for major depressive disorder share symptoms with the anxiety disorders, especially GAD and PTSD. For example, all three conditions share the criteria of sleep disturbance, and difficulty concentrating, GAD and depression share the symptom of fatigue, and PTSD and depression share the symptom of diminished interest in activities that the individual previously enjoyed and also altered cognitions, which overlap with depressive cognitions such as feelings of guilt, worthlessness, or blaming the self (American Psychiatric Association, 2013).

The amygdala is implicated in both depression and anxiety, as is underactivity in the prefrontal cortex, although it may be overactive during anxious and depressive cognitive processes (Heim & Nemeroff, 2001). Neurochemically, the catecholamine hypothesis of depression has expanded to include a hypothesized overlap of a serotonin deficiency in both anxiety and mood disorders (McNaughton & Corr, 2004). Also, although there are certain distinctions among disorders like PTSD, dysregulation of the hypothalamus-pituitary-adrenal (HPA) axis is a well-established marker of anxiety and depression (Heim & Nemeroff, 2001). Evidence for shared genetic diatheses is scant with the exception of the relationship between GAD and MDD. Some argue that this is due to the genetic conferral of risk via underlying personality traits, such as negative affect or neuroticism, rather than disorders themselves (Huppert, 2009).

In the tripartite model of anxiety and depression (Clark & Watson, 1991), depression and anxiety share a nonspecific negative affect, or general distress, component while hyperarousal is specific to anxiety and low positive affect, or dysphoria is specific to depression; this model implies that a complete description of an affective requires the assessment of both specific and nonspecific factors. This model was an important step in reconciling the comorbidity and distinctions between anxiety and depression, but it was not perfect. For instance, Brown, Chorpita, and Barlow (1998) found that the anxious arousal component was not broadly characteristic of the anxiety disorders, but was specific to panic disorder. They also found that the positive affect dimension was associated with social anxiety and was not entirely specific to depression. Similarly, the DSM-5 criteria set for PTSD includes the persistent inability to experience positive emotions, or a disruption to

positive affect (American Psychiatric Association, 2013). Despite the advancements provided by this model, there are clear shortcomings.

Acknowledging this evidence, Mineka, Watson, and Clark (1998) incorporated Zinbarg and Barlow's hierarchical organization of anxiety disorders (1996). In this model, they posited that negative affect was a higher order factor common to anxiety and mood disorders, and that each individual disorder has its own unique component to distinguish it from the others. The contributions of the general specific components varied across disorders in this model. Substantiating this model, individuals with comorbid anxiety and depression show higher levels of neuroticism, which corresponds to negative affect (Tellegen, 1985), than individuals with either anxiety or depression alone (Lamers, et al., 2011). Watson (2005) expanded upon these results by proposing a quantitative hierarchical model of anxiety and mood disorders. In his proposed model, the anxiety and mood disorders fall under a category that he called the Emotional Disorders, which rests at the top level of the hierarchy. At the second level of the hierarchy, there are three categories: Bipolar Disorders (bipolar I disorder, bipolar II disorder, and cyclothymia), Distress Disorders (major depressive disorder, dysthymia, GAD, and PTSD), and Fear Disorders (panic disorder, agoraphobia, social phobia, and specific phobia). Notably, in this model, depression and dysthymia are more related to GAD and PTSD than the bipolar disorders and all of these disorders appear to be characterized by elevated negative affect.

Treatment Outcomes

Regardless of diagnoses or mechanisms, the presence of a comorbid depression diagnosis presents problems in the anxiety disorder treatment process. There have been

several studies that sought to evaluate this difficulty, but there are gaps in the literature that leave some questions unanswered. Wittchen, Essau, and Krieg (1991) compared individuals with anxiety disorders in the general population to former psychiatric inpatients in treatment outcomes. They found that the majority of individuals in both groups (69% and 95% respectively) had any comorbid diagnosis, and when the diagnosis was a depressive disorder, it was typically temporally secondary to anxiety. Further, the outcomes for individuals with both anxiety and depression tended to be worse than they were among individuals with anxiety alone, even if the depression remitted at follow-up. Long-term, the presence of major depression nearly halves the likelihood that an individual with panic disorder, social phobia, or GAD recovers, and increases the likelihood of relapse for those that do remit from panic disorder with agoraphobia (Bruce, et al., 2005).

Joorman, Kosfelder, and Schulte (2005) analyzed a sample of individuals with panic disorder (PD) or social phobia presenting for individual manualized CBT. Their results showed that comorbid cases endorsed more severe self-report anxiety and depression, and that comorbid and simple cases improved in parallel such that the rates of change were the same despite greater scores for comorbid patients. They only included individuals with comorbid major depression, but excluded individuals with any other comorbid diagnoses, thus limiting the generalizability of their findings to other settings. Additionally, they called their sample a mixed anxiety sample despite the fact that they only included two primary diagnoses rather than the full range of DSM-IV-TR anxiety disorders. Further, the average participant attended 25.6 sessions (SD = 14.1). There is a clear need for treatment that can deliver faster results.

Campbell-Sills and colleagues (2012) conducted a multi-site clinical trial of a multifaceted anxiety intervention using primary care referrals that included computer-assisted CBT, medication management, or a combination per the patient's request; the intervention lasted between three and twelve month depending on randomization. Patients were randomly assigned to either a "step up" condition when they received a larger dose of their current treatment, or a "step over" assignment in which they switched to or added a new modality. Patients continued in treatment until they met remission criteria; if the patient remitted before twelve months elapsed, then they received monthly phone calls to reinforce CBT skills, medication management, or both. They found that depressed patients displayed larger decreases in anxiety symptoms regardless of treatment assignment at 12- and 18-month follow-up, but not at 6-month follow-up. Depressed patients also displayed larger decreases in anxiety-related disability at all three follow-ups. Although depression predicted lower response rates at 6-month follow-up, this difference was not significant at 12- or 18-month follow-up. Despite these improvements, depressed patients continued to display elevated symptom levels and anxiety-related disability relative to individuals with anxiety alone out to 18-month follow-up. In this study, though, depressed patients were more likely at pre-treatment to be receiving adequate medication management in terms of type, duration, and dose at baseline, although not at follow-up. The depressed patients also received an average of one more CBT contact, and non-depressed patience endorsed relatively low levels of anxiety symptoms and anxiety-related disability at baseline. Using individuals seeking treatment for anxiety rather than primary care referrals may eliminate or at least ameliorate that concern.

Brown, Antony, and Barlow (1995) analyzed the effects of pre-treatment comorbidity on cognitive-behavioral treatment outcomes in individuals with a primary diagnosis of panic disorder. They found that comorbid GAD or social phobia did not affect short-term treatment gains, but those with comorbid depression were significantly less likely to have remitted panic disorder at post-treatment. In a large-scale, multi-site clinical trial for CBT for panic disorder, comorbid depression was not associated with a differential rate improvement on self-report panic disorder severity although individuals with a depressive diagnosis were more severe at both pre- and post-treatment; rates of comorbid depression did not change significantly following treatment (Allen, et al., 2010).

Steketee, Chambless, and Tran (2001) examined the effects of comorbid axis I diagnoses and axis II traits on symptom change following behavior therapy for patients with panic disorder with agoraphobia or OCD. Fourteen percent of OCD patients and 19% of panic disorder with agoraphobia patients also received diagnoses of comorbid major depressive disorder. They found that secondary major depressive disorder predicted worse outcomes at post-treatment for those that completed treatment. It predicted worse social adjustment for panic patients, but not OCD patients. Depression also predicted worse outcomes at 6-month follow-up. McLean and colleagues (1998) found that both depressed and non-depressed panic disorder patients improve comparably following CBT for panic, but the depressed patients tend to be more severe and have more functional impairment following treatment.

Chambless, Tran, and Glass (1997) examined predictors of response to group CBT for social phobia. They found that those that scored highest on measures of depression were less likely to improve or to maintain gains on self-reported anxiety. This was a robust

finding, as level of depression was the single best predictor of pre-post change in their analyses. Erwin and colleagues (2002) compared treatment outcomes between patients with simple social phobia, comorbid anxiety, and comorbid depression. After 12 weeks of group CBT and at 12-month follow-up, the three groups showed comparable rates of improvement, but independent assessors rated the comorbid depression cases as more severe than simple social phobia patients across treatment. Fracalanza, McCabe, Taylor, and Antony (2014) obtained similar results, finding that individuals with comorbid social phobia and major depression scored significantly higher on measures of social anxiety symptoms at pre-treatment than individuals with either social phobia and a comorbid anxiety disorder or individuals diagnosed with social phobia alone, and individuals with either a comorbid mood or anxiety disorder finished group CBT with greater scores on measures of social anxiety; all groups significantly improved following CBT for social phobia, including small to moderate improvements in depressive symptoms. However, this study used the depression subscale of the Depression Anxiety Stress Scales 21 item version (Lovibond & Lovibond, 1995) and individuals in their anxiety and comorbid depression group (post-treatment M_{76} = 10.31, *SD* = 6.34) scored at the 93rd percentile established in a psychometric investigation (Henry & Crawford, 2005); despite promise, a more potent intervention appears necessary. The preponderance of evidence suggests that anxiety patients with comorbid depression present with more severe symptoms for their principal disorder than those without comorbid depression (Davis, Barlow, & Smith, 2010).

Although there is quite a bit of research on the effects of comorbidity of panic disorder and depression on CBT outcomes, there is less on depression with the other anxiety disorders. Depression and GAD are frequently comorbid. Borkovec, Abel, and Newman

(1995) sought to evaluate if group CBT for GAD lead to reductions in comorbid conditions. They found that treatment significantly reduced all comorbidity at post-treatment, as 78.2% received a comorbid diagnosis at pre-treatment, but only 25.5% maintained a comorbid diagnosis at post-treatment; they did not observe a moderator effect of diagnosis. Further, only 3 of the original 43 patients met criteria for a comorbid diagnosis at 12-month follow-up.

Depression also co-occurs frequently with PTSD. In a sample of individuals who had been in motor vehicle accidents, Blanchard and colleagues (2003) found that CBT for PTSD led to greater reductions in PTSD symptoms and those in the CBT condition were less likely to meet criteria for depression following the intervention than individuals in either a supportive psychotherapy or wait-list control conditions. Forty-nine percent of the sample met criteria for major depressive disorder at pre-treatment. The CBT condition led to significantly greater reductions in the frequency of a major depression diagnosis and greater reductions in scores on measures of depression than either the support or wait-list conditions. Thus, the presence of depression did not impair the treatment process, and depression also responded to treatment targeting another diagnosis.

Abramowitz and Foa (2000) compared exposure and response prevention treatment outcomes among OCD patients with and without major depression. The two groups did not differ in pretreatment self-report OCD severity. At post-treatment, OCD symptoms improved significantly in both groups, there were no differences in treatment response rates, and gains were maintained at follow-up in both groups. Despite comparable rates of responding, those with comorbid depression scored significantly higher on self-report measures of OCD and depression at both post-treatment and follow-up. In a similar study, individuals with severe

depression at pre-treatment improved significantly less than non-depressed or less depressed OCD patients over the course of exposure and response prevention, but these individuals still demonstrated moderate gains (Abramowitz, Franklin, & Street, 2000). In summary, the overall body of research regarding the response of comorbid depression on anxiety, trauma, and obsessive-compulsive disorders treatment is mixed at best suggesting a need for treatments that can effectively and efficiently treat anxiety despite the presence of depression and also can simultaneously reduce the co-occurring depression.

Transdiagnostic Treatment

Brown and Barlow (1992) hypothesized on why treating a principal anxiety disorder may reduce the severity or eliminate the presence of a comorbid condition. Their first potential explanation was that treatment gains may generalize as the patient learns to apply skills gained in treatment to other problems. The second is that anxiety and mood disorders share certain overlapping diagnostic criteria and improvement on those particular symptoms would necessarily affect those same symptoms in additional diagnoses. A third possibility is that disorders share psychological processes and treatments that target these shared processes and mechanisms would affect all subsumed diagnoses (Borkovec, Abel, & Newman, 1995). This underlies the theories of transdiagnostic psychotherapy modalities.

Barlow, Allen, and Choate (2004) argued for a unified approach to treatment of anxiety and depression that expanded beyond the simplicity of dissemination and training. They emphasized the commonalities of these disorders, arguing that commonalities in etiology and latent structure outweigh the differences. They interpreted these similarities to mean that it is possible to distill a package of psychotherapy modalities that target emotion

disorders broadly by targeting these shared variables. Norton (2006) provided support for transdiagnostic interventions, highlighting that the anxious and depressive disorders respond to similar techniques, modalities, and medications. He also noted that improvements in comorbid conditions are not functions of symptom overlap between conditions. Because observations resulting from pharmacotherapy are similar to those following psychotherapy, it is likely that gains come from modifications of shared underlying vulnerabilities rather than the generalization of gains.

Davis, Barlow, and Smith (2010) sought to test the effects Barlow's Unified Protocol (Barlow, Allen, & Choate, 2004) and the transactional effects of anxiety and comorbid disorders. The unified protocol consists of weekly individual treatment sessions focusing on core treatment modules targeting, among other factors, emotional awareness, cognitive flexibility, and emotional and behavioral avoidance. They found that patients with comorbid disorders begin treatment with a more severe presentation, but comorbidity does not impede rate of improvement across principal anxiety diagnoses. Further, comorbid disorders did not impede patients' rates of progress. However, patients with comorbid conditions attended an average of three additional sessions, suggesting a greater demand from individual therapy.

Norton, Hayes, and Hope (2004) conducted a preliminary analysis on 23 anxious treatment seekers, 8 of whom had a secondary depressive diagnosis, using the protocol to be used in this proposal. They found that individuals in the transdiagnostic group cognitive behavioral therapy (TGCBT) condition improved significantly in anhedonic depression over the course of treatment, but those in the wait-list control condition did not improve (this study lacked the power to assess changes in clinician-rated depression severity). This study provided a promising potential line of research for analyses in a larger dataset. Furthermore,

this study did not analyze the effects of depression on treatment outcomes in anxious patients.

Transdiagnostic group cognitive behavioral therapy (TGCBT) has since demonstrated effectiveness in treating comorbid conditions. Norton and colleagues (2013) found that anxious treatment seekers with more than one diagnosis, whom constituted the majority of the sample, were significantly more severe than those with a single diagnosis before treatment, but there were no differences in rates of improvement in overall clinical severity or broad anxiety severity. About two thirds of those with comorbid diagnoses that completed treatment no longer met criteria for a secondary diagnosis, which may be greater than rates observed in previous studies. The effect of depression on response to TGCBT for anxiety and those of anxiety treatment on comorbid depression remain unstudied despite its promise as a a novel and effective treatment method. This study posits TGCBT as a promising, parsimonious, and easily disseminated option to improve individuals carrying comorbid diagnoses. In this study, depressive disorders were the most common comorbid conditions (observed in 23 of the 79 individuals in the study), but the total sample had heterogeneous profiles of comorbidity and these profiles were collapsed together in order to sufficiently power the analyses.

Statement of Problem, Purpose, and Hypotheses

Transdiagnostic group cognitive behavioral therapy (TGCBT) appears to be a good candidate for the treatment of comorbid Axis I conditions, and as such, its popularity is increasing. However, there is a lack of research that examines the reciprocal effects of depression on anxiety treatment and vice versa. Given the high rate of comorbid depression

with anxiety disorders, the present study aims to address a gap in the literature by examining the transactional effects of comorbid depressive disorders on the treatment of anxiety disorders in transdiagnostic group cognitive-behavioral therapy (TGCBT), as well as the effect of TGCBT for anxiety on comorbid depression. Further, this study aims to extend this line of research by analyzing the role of negative affect, a higher-order risk factor, as a transdiagnostic variable worthy of targeting not only in anxiety treatment, but in treatment of the emotional disorders more broadly. The first hypothesis is that anxious individuals with a comorbid diagnosis of major depressive disorder would improve significantly less on measures of severity of overall anxiety, primary anxiety disorders, and overall clinical presentation than those without a depression diagnosis. Second, it is hypothesized that all individuals will improve on measures of depression, and individuals with comorbid depression will improve a significantly greater amount than those without a depression diagnosis. The third hypothesis is that depression diagnoses will respond to TGCBT for anxiety and post-treatment depression diagnosis severity will be significantly reduced compared to pre-treatment levels. The fourth hypothesis is that negative affect will significantly mediate improvement in overall anxiety, and depression among all individuals.

Methods

Participants

This study used data collected from the Anxiety Disorder Clinic (ADC) at the University of Houston. The ADC dataset includes individuals seeking treatment for anxiety whose data has been collected as part of previous clinical trials (Norton, 2006, 2008, 2012a), and also as part of ongoing data collection to evaluate treatment outcomes. To be included in ADC research, consenting individuals must be age 18 years or older, have a primary DSM-IV-TR diagnosis of an anxiety disorder, be proficient in the English language, show no suicidality, substance misuse, or other condition requiring immediate clinical intervention, and demonstrate no cognitive decline. For this particular study, individuals diagnosed with bipolar disorder were excluded due to its differences compared to other mood disorders (Watson, 2005).

The dataset includes 120 individuals; demographic statistics are depicted in Table 1. The mean age of the sample was 33.30 years (*S.D.* = 11.30), and there was not a significant difference in age between the depressed (DEP; $M = 32.49$, $S.D. = 11.14$) and the non-depressed groups (NDEP; $M = 33.70$, $S.D. = 11.48$; $F_{1,103} = .47$, $p = .496$). In the DEP group, 42.8% self-identified as male, 52.4% as female, and 4.8% chose not to respond; there were no significant differences in gender ($\chi^2_1 = 1.38$, $p = .240$). More than half (57.1%) self-identified as European American, 9.1% as African American, 1.3% as Native American, 10.4% as Asian American, 14.3% as Hispanic, 7.8% as another race or ethnicity, and 1.3% did not respond. More than half (51.2%) reported being single, 42.3% reported that they are married or cohabitating with a partner, 2.6% reported being divorced, 1.3% reported being

separated, and 3.8% did not respond. Two thirds (66.7%) reported either being employed or enrolled in classes full-time, 3.8% reported being employed part-time, 15.4% reported being unemployed, and 14.1% reported either another occupational status or did not respond. Nearly a third (29.5%) reported that they had a graduate or professional degree, 2.6% reported that they did not complete high school, 1.3% that they graduated from high school, 28.2% that they had completed some undergraduate courses or an associate's degree, 28.2% that they had a bachelor's degree, 5.1% that they had completed some graduate courses, and 5.1% did not respond.

In the NDEP group, 56.4% self-identified as male and 43.6% as female. Nearly two thirds (64.9%) self-identified as European American, 2.7% as African American, 5.4% as Asian American, 21.6% as Hispanic, 2% as another race or ethnicity, and 11.9% chose not to respond. In the depressed group, 33.3% reported that they are married or cohabitating with a partner, 57.1% reported being single, 4.8% reported being divorced, 2.4% reported being widowed, and 2.4% did not respond. Regarding employment status, 61.9% reported being either employed or enrolled in classes full-time, 4.8% reported being employed part-time, 19.0% reported being unemployed, 2.3% reported being a homemaker, and 11.9% endorsed either another occupational status or did not respond. When asked about education, 4.8% reported that they did not complete high school, 7.1% that they graduated from high school, 33.3% that they had completed some undergraduate courses or an associate's degree, 21.4% that they had a bachelor's degree, 11.9% that they had completed some graduate courses, 14.3% that they had a graduate or professional degree, and 7.1% did not respond. There were no significant differences in gender ($\chi^2 = 1.38$, $p = .240$), racial identification ($\chi^2 = 3.90$, $p =$

.564), marital status ($\chi^2 = 4.14$, $p = .529$), occupational status ($\chi^2 = 5.20$, $p = .393$) or education across the DEP and NDEP groups ($\chi^2 = 8.26$, $p = .143$).

Frequencies of primary diagnoses are shown in Table 2. The most frequent diagnoses in this sample were social phobia (45.8%), panic disorder (20.0%), and GAD (18.3%). In the NDEP group, these percentages were 47.4%, 17.9%, and 15.4% respectively, and they were 42.9%, 23.8%, and 23.8% respectively among the DEP group. All DSM-IV-TR anxiety disorders were represented except for PTSD and acute stress disorder. There were no significant differences in the frequency of diagnoses between groups ($\chi^2 = 5.04$, $p = .655$).

This sample displays significant comorbidity, as 76 (63.3%) individuals met criteria for a second Axis I disorder, 37 (30.8%) for a third, 11 (9.2%) for a fourth, and 2 (1.7%) for a fifth. The rank order of the diagnoses was determined by the clinical judgment of the assessor. Among the 78 individuals in the NDEP group, 44 (56.4%) met criteria for at least two disorders. A depressive disorder was the secondary diagnosis for 23 (19.2%) individuals, the tertiary diagnosis for 14 (11.7%) individuals, the quaternary diagnosis for 4 (3.3%) individuals, and the quinary diagnosis for 1(0.8%) individual, thus 42 individuals (27.2%) with a primary anxiety disorder have a comorbid depression diagnosis. Twenty eight (66.7%) of individuals in the DEP group met criteria for at least one additional disorder compared to 42.3% of those without a depressive diagnosis, and this proportion was significantly different ($\chi^2 = 6.48$, $p = .011$). As comorbidity is often associated with increased severity (Lamers, et al., 2011), the presence of comorbidity was included as a covariate in statistical models to account for differences due to comorbidity rather than depression.

Measures

When individuals opened a request for services from the ADC, they received a large battery of self-report questionnaires before they completed a structured interview with a clinician. They repeated the self-report measures at mid-treatment and post-treatment, and they were assessed by a clinician following treatment as well.

Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV). The ADIS-IV (Brown, Di Nardo, & Barlow, 1994) is a clinician-administered assessment tool that screens primarily for anxiety disorders, but also contains diagnostic sections for mood disorders, substance use disorders, somatoform disorders, and psychosis. It allows the clinician to provide an Axis I diagnosis with severity ratings as well as assessment on Axes III, IV, and V; Axis II was deferred for all individuals. Additionally, assessors provide a Clinical Global Impressions (CGI) score, which is a rating of the overall severity of the client's clinical presentation. Brown, Di Nardo, Lehman, and Campbell (2001) found inter-rater reliability (kappa coefficients) ranging from .67 to .86 (except for dysthymic disorder, which had a coefficient of .22) for principal diagnoses, and .56 to .81 for any diagnosis (except for dysthymic disorder, which had a coefficient of .31). Previous research from this group has found high inter-rater reliability ($\kappa = 0.759$, $p < .001$; Chamberlain & Norton, 2013).

State-Trait Anxiety Inventory – State Version (STAI). The STAI (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1993) is a 20-item self-report measure designed to assess state and trait levels of anxiety. Only the state version was used for the purposes of this investigation. Items are scored from 1 ("not at all") to 4 ("very much so") to indicate how much each statement reflects the subject's present feelings; total scores range from 20 to 80. The original manual reported mean scores of 35.72 (*SD* = 10.40) for men and 35.20 (*SD* = 10.61) for women. The original manual reported strong psychometric properties in several

populations. This measure is also sensitive to treatment effects (Fisher & Durham, 1999; (Norton, 2008). In this sample, its internal consistency was excellent at session 1 (Cronbach's $\alpha = .931$).

Beck Anxiety Inventory (BAI). The BAI is a 21-item self-report measure designed to measure the severity of general anxiety symptoms. It shows good internal consistency ($\alpha = .92$) and test-retest reliability (correlation of .75 between administrations) in clinical populations (Beck, Epstein, Brown, & Steer, 1988). The original manual reports the following means by diagnosis: panic disorder with agoraphobia, 27.27 (*SD* = 13.11); panic disorder without agoraphobia, 28.21 (*SD* = 13.46); SP, 17.77 (*SD* = 11.64); OCD, 21.96 (*SD* = 12.42); and GAD, 18.83 (*SD* = 9.08). In a community sample with demographics closely matching the 1990 U.S. Census, the mean for individuals ages 18-44 was 6.6 (*SD* = 8.1) and the mean for individuals ages 45-65 was 4.4 (*SD* = 6.3) with no significant sex, race, or income differences; in the entire sample, a score of 3 fell at the 50th percentile, 10 at the 80th percentile, and 17 at the 90th percentile (Gillis, Haaga, & Ford, 1995). The internal consistency at pre-treatment in this sample, measured with Cronbach's alpha, is 0.937.

Beck Depression Inventory – Second Edition (BDI-II). The BDI-II (Beck, Steer, & Brown, 1996) is a widely-used 21-item measure of depression symptomatology that generally shows three factors reflecting negative attitudes toward the self, performance impairment, and somatic disturbance. Items are scored from 0 to 3 in intensity. The Center for Cognitive Therapy suggested the following cut-off scores: minimal depression is less than 10; mild to moderate depression is 10-18; moderate to severe depression is 19-29; and severe depression is 30-63; however, the appropriateness of cut-off scores is variable depending on the sample (Beck, Steer, & Garbin, 1988). In a community sample of individuals ages 18 to

64, Lasa, Ayusa-Mateos, Vásquez-Barquero, Diéz-Manrique, and Dowrick (2000) found 100% sensitivity and 99% specificity for a cut-off score of 13 in diagnosing a depressive disorder. It has good reliability and validity. In clinical samples, Cronbach's alphas ranging from 0.76 to 0.95 have been observed; in community samples, the range is from 0.73 to 0.92. It shows strong correlation with other measures of depression, such as 0.61 to 0.86 with the Hamilton Rating Scale for Depression, and a mean correlation coefficient with clinical severity rating of 0.72. Depressed individuals score significantly higher than non-clinical controls (Beck, Steer, & Garbin, 1988). The internal consistency at pre-treatment in this sample is 0.897.

Anxiety Disorder Diagnostic Questionnaire (ADDQ). The ADDQ (Norton & Robinson, 2010) was developed as a screening tool for the presence of clinical fear and anxiety irrespective of diagnoses. It demonstrates high internal consistency, and evidences convergent and discriminant validity. In the original publication, anxious treatment-seekers (M = 34.53, $S.D.$ = 8.81) scored significantly higher than non-anxious controls (M = 20.60, $S.D.$ = 10.03), and individuals with primary diagnoses of panic disorder, GAD, and social phobia did not score significantly differently after controlling for severity. It demonstrates high sensitivity to treatment, as the ADDQ pre-post change score correlates very strongly (r = .81, $p < .001$) with clinician-rated improvement following treatment. The internal consistency at pre-treatment in this sample is 0.793.

Positive Affect Negative Affect Schedule – Negative Affect (PANAS). The PANAS (Watson, Clark, & Tellegen, 1988) is a 20-item self-report measure designed to assess positive affect (PA) and negative affect (NA). PA and NA are considered orthogonal measures of independent affective dimensions (Watson, Clark, & Carey, 1988), but weak

correlations are sometimes observed (Vittengl & Holt, 1998). This measure can assess either state or trait affect depending on the specific phrasing of the instructions (Watson, Clark, & Tellegen, 1988). For this study, the instructions were phrased to measure trait affect ("Indicate to what extent you generally feel this way, that is, how you feel on average") rather than state affect ("Indicate to what extent you feel this way right now, that is, at the present moment," or "Indicate to what extent you have felt this way today"). The PANAS has demonstrated acceptable psychometric properties in clinically anxious and depressed samples (Brown, Chorpita, & Barlow, 1998; Watson, Clark, & Carey, 1988). The reliability of the 10 negative affect items in this sample is acceptable (Cronbach $\alpha = .781$).

Procedures

Participants called the Psychological Research Services Center (PRSC) at the University of Houston to open a request for services through the ADC. They then received a large battery of self-report questionnaires which they brought to their initial evaluation if they consented to participate in research. In their first appointment, individuals completed the ADIS-IV. They returned approximately one week later for their feedback session, during which they received a copy of their assessment report and were presented with their treatment options and recommendations. This study included individuals that opted to participate in transdiagnostic group cognitive behavioral therapy through the ADC and satisfied all the inclusionary criteria.

The treatment program consisted of 12 weekly 2-hour long sessions following a manualized protocol (Norton, 2012b). The facilitators in each group were two doctoral student clinicians trained in CBT for anxiety that were under weekly individual and group supervision from the author of the treatment manual. During the first 9 weeks of treatment,

the primary components were psychoeducation, self-monitoring, cognitive restructuring of anxious thoughts specific to the individual's referral concerns (e.g., "When my heart races, I must be having a heart attack and I will die"), and graduated exposure based on each individual's fear hierarchy. Exposures were conducted in session and were also assigned to clients as homework. The final component of treatment consisted of restructuring underlying core beliefs and perceptions such as perceived threat and lack of control (e.g., "I am not safe anywhere"). Every week, subjects completed the STAI. After weeks 6 and 12, subjects completed the same questionnaire battery that they did at their initial assessment.

Following the completion of treatment, participants completed a second ADIS, and the assessor rated the post-treatment severity of the diagnoses provided at pre-treatment. The assessor also provided a post-treatment CGI score and a score of global improvement (CGI-Improvement). Neither of the individual's group facilitators is allowed to conduct the post-treatment assessment in order to reduce bias.

Data Analysis

Because one of the outcome measures was a clinician-administered interview following the completion of treatment, only data collected from individuals that completed the full course of therapy were analyzed.

Descriptive Statistics. Frequencies, means, and standard deviations for all relevant demographic, diagnostic, and anxiety variables were calculated for the total sample, the DEP group, and the NDEP group. Analyses of variance (ANOVAs) were used to compare the DEP and NDEP groups. Further ANOVAs were used to determine if there are differences

across primary diagnoses in depression prevalence in this sample, overall severity, or depression severity.

Effects of Depression Diagnosis on Anxiety Reduction. To determine the effects of depression on anxiety reduction following TGCBT for anxiety, a mixed effects linear regression model was used to compare the DEP and NDEP groups. STAI scores were modeled over time and the two groups were compared on intercepts (pre-treatment scores), rates of change over the course of treatment (slopes), and session 12 (end state). Unconstrained, partially constrained, and fully constrained regression models were compared in order to determine if restricting the intercept, slope, or end-state to be invariant across groups improves model fit. Further, repeated measures ANOVAs were used to examine differential improvement during treatment using pre/post clinician severity rating, and also ADDQ and BAI scores taken at pre-, mid-, and post-treatment as repeated measures factors and the presence of depression at pre-treatment diagnosis as the between-subjects factor.

Effects of TGCBT for Anxiety on Depression. To determine if depression affected response to TGCBT in anxious individuals, a mixed analysis of variance (ANOVA) of BDI-II scores was conducted with depression diagnosis (comorbid depression vs. no comorbid depression) serving as a between-subjects variable and time (pre-treatment vs. post-treatment) serving as a within-subjects variable. A repeated measures ANOVA was conducted in the comorbid depression group using the ADIS Clinician Severity Rating (CSR) of the depression diagnosis to determine the effect of anxiety treatment on a depression diagnosis.

Mediation of Improvements. To determine if negative affect mediated improvements in anxiety and depression, data was analyzed using multilevel modeling. Multilevel modeling allows for analysis of both individual-level and group-level (Krull & MacKinnon, 2001). The criterion variables, BAI and BDI-II, were modeled over time using a mixed-effect linear regression using a Maximum Likelihood estimator. Similarly, the potential mediator variable, PANAS, was modeled at pre-treatment, and post-treatment. The three steps to this procedure are 1) determine if there is a significant effect for condition, 2) determine if the magnitude of that effect is attenuated by the mediator variable, 3) determine the effect of condition on the mediator variable using mixed effects linear regressions (Krull & MacKinnon, 2001).

Results

Descriptive Statistics

Means and standard deviations for relevant clinical variables are displayed in Table 3. The NDEP group was rated significantly lower on Clinician Severity Rating (CSR; $M = 5.27$, $SD = 0.91$) than the DEP group ($M = 5.86$, $SD = 1.02$; $F_{1,117} = 10.21$, $p = .002$) at pre-treatment but not at post-treatment ($M = 3.16$, $SD = 1.60$ vs. $M = 3.52$, $SD = 2.22$; $F_{1,118} = 1.07$, $p = .304$). On CGI, the NDEP group ($M = 4.55$, $SD = 0.88$) scored significantly lower (they were rated as less severe) at pre-treatment than the DEP group ($M = 5.00$, $SD = .86$; $F_{1,118} = 7.26$, $p = .008$), but there were no differences at post-treatment ($M = 2.91$, $SD = 1.22$ vs. $M = 3.19$, $SD = 1.57$; $F_{1,118} = 1.18$, $p = .280$) or on CGI – Improvement, the clinician-rated assessment of improvement following therapy ($M = 2.19$, $SD = 0.81$ vs. $M = 2.13$, $SD = 0.79$; $F_{1,116} = 0.19$, $p = .667$).

On the PANAS, the NDEP group (M = 27.85, SD = 6.68) scored significantly lower than the DEP group (M = 33.38, SD = 7.47; $F_{1,102}$ = 14.53, p < .001) at pre-treatment and at post treatment (M = 20.64, SD = 6.17 vs. M = 24.42, SD = 6.87; $F_{1,98}$ = 7.47, p = .007). On the ADDQ, the NDEP group (M = 31.53, SD = 8.15) scored significantly lower than the DEP group (M = 37.89, SD = 7.21; $F_{1,114}$ = 16.88, p < .001) at pre-treatment but not at post treatment (M = 21.03, SD = 8.74 vs. M = 24.40, SD = 10.88; $F_{1,98}$ = 2.84, p = .095). Similarly, on the BAI, the NDEP group (M = 21.90, SD = 12.21) scored significantly lower than the DEP group (M = 30.39, SD = 14.79; $F_{1,100}$ = 9.40, p = .003) at pre-treatment but not at post treatment (M = 14.56, SD = 9.85 vs. M = 12.20, SD = 8.89; $F_{1,26}$ = .39, p = .536). As expected, on the BDI-II, the NDEP group (M = 15.74, SD = 9.70) scored significantly lower than the DEP group (M = 27.79, SD = 9.70; $F_{1,102}$ = 9.05, p = .003) at pre-treatment and at post treatment (M = 8.03, SD = 8.29 vs. M = 14.32, SD = 12.25; $F_{1,98}$ = 9.403, p = .003). The majority of values were consistent with expectations, as the NDEP group was less severe at pre-treatment but most of these differences were no longer significant at post-treatment.

Effect of Depression on Anxiety Reduction

Linear Regression Model. The DEP group (M_1 = 50.46, SD_1 = 8.06; M_{12} = 42.20, SD_{12} = 9.78) scored higher than the NDEP group (M_1 = 47.30, SD_1 = 9.38; M_{12} = 39.12, SD_{12} = 10.45) on the STAI at both sessions 1 and 12. Models were examined using confirmatory factor analysis. Model fit was examined using the chi-square test of model fit (Muthén & Muthén, 2013). Following recommendations of Byrne (1998), structural invariance across groups was evaluated using a multi-step procedure. First, the model was evaluated independently to determine absolute fit. Second, if the data showed acceptable fit, the full data were fit using multigroup analysis constraining y-intercepts or slopes to be

equal. All analyses were conducted using Mplus (version 7.1; Muthén & Muthén, 2013) using a maximum likelihood (ML) estimator. These analyses were first conducted using session 1 as the y-intercept and repeated using session 12 as the y intercept.

Model 1a – No invariance. The first SEM multigroup model estimated session 1 and 12 intercepts, slopes, and covariances freely across groups to establish a base model. Results indicated that this unconstrained model showed excellent fit to the data ($\chi^2_{146} = 322.12$, $p < .001$). Subsequent models were sequentially evaluated for change in fit using the chi square difference test.

Model 2 – full invariance. The second model held slopes, session 1 intercepts, and session 12 intercepts invariant across groups. This model showed excellent fit as well ($\chi^2_{148} = 335.370$, $p < .001$), although modeling the groups to be fully invariant resulted in a significant decrease in model fit ($\chi^2 \Delta_2 = 13.253$, $p = .001$).

Model 3 – slope invariance. The third model, holding slopes invariant across groups with freely varying y-intercepts showed excellent fit ($\chi^2_{147} = 326.29$, $p < .001$). Specifying the slopes to be invariant in this model resulted in a significant decrease in model fit ($\chi^2\Delta_1 = 4.171$, $p = .041$).

Model 4a – session 1 intercept invariance. The fourth model held session 1 y-intercepts invariant across groups while allowing slopes to vary freely. This model showed excellent fit ($\chi^2_{147} = 323.97$, $p < .001$) but differed significantly from the freely estimated model ($\chi^2\Delta_1 = 13.091$, $p < .001$).

Model 4b – session 12 intercept invariance. The fifth model held session 12 y-intercepts invariant across groups while allowing slopes to vary freely. This model showed

excellent fit (χ^2 (578) = 1185.08, $p < .001$) and did not differ significantly from the freely estimated model ($\chi^2\Delta_1 = 1.81$, $p = .179$). Based on Byrne's (1998) criteria, this model, in which the two groups reached similar end state outcomes despite differences in pre-treatment scores and slopes, was deemed acceptable.

Analyses of Variance (ANOVAs). Initially, mixed ANOVAs were conducted using the degree of the depressive diagnosis (secondary vs. all others) as a between-subjects factor and time as a within-subjects factor to determine if there was an effect of depression primacy on the outcome variables. If the between-subjects factor was degree of depression diagnosis, then there would only be one cell with a sample size greater than 20 and the analyses would be underpowered. The presence of additional comorbid diagnoses was included in the model as a covariate in order to account for the fact that the DEP group was inherently more likely to have comorbid diagnoses. Simply controlling for the presence of comorbidity would cause this variable to become collinear with depression, therefore only additional comorbidity was included as covariate.

Time had a significant main effect but neither the degree of the depressive diagnosis nor the time x degree interaction had a significant effect on any of the following: CGI (Time: $F_{1,39} = 72.49$, $p < .001$; Degree: $F_{1,39} = .009$, $p = .322$; Time x Degree: $F_{1,39} = 2.36$, $p = .133$), primary diagnosis CSR (Time: $F_{1,39} = 48.87$, $p < .001$; Degree: $F_{1,39} = .426$, $p = .518$; Time x Degree: $F_{1,39} = 2.76$, $p = .105$), PANAS (Time: $F_{1,23} = 19.64$, $p < .001$; Degree: $F_{1,23} = 1.68$, $p = .208$; Time x Degree: $F_{1,23} = .51$, $p = .485$), ADDQ (Time: $F_{1,31} = 31.21$, $p < .001$; Degree: $F_{1,31} = .47$, $p = .499$; Time x Degree: $F_{1,31} = 1.62$, $p = .212$), or BDI-II (Time: $F_{1,23} = 13.01$, $p = .001$; Degree: $F_{1,23} = .009$, $p = .322$; Time x Degree: $F_{1,23} = .51$, $p = .133$). There were no significant effects at all on BAI (Time: $F_{1,6} = 1.86$, $p = .222$; Degree: $F_{1,6} =$

..02, $p = .907$; Time x Degree: $F_{1,6} = .52$, $p = .499$), although this may be due to low sample sizes as a consequence of missing data. Because there was no effect of the degree of the depressive diagnosis, it was not included as a covariate in subsequent analyses.

A 2x2 ANOVA with depression diagnosis (presence of comorbid depression vs. absence of comorbid depression) serving as a between-subjects factor and ADDQ score (pre-treatment vs. post-treatment) serving as a within-subjects factor revealed significant main effects of time ($F_{1,94} = 128.33$, $p < .001$, $\eta^2_p = .58$) and depression diagnosis ($F_{1,94} = 5.68$, $p = .019$, $\eta^2_p = .06$), but the time x depression diagnosis interaction was not significant ($F_{1,94} = 1.30$, $p = .258$, $\eta^2_p = .01$). Both groups improved significantly on the ADDQ following treatment and the rates of change did not differ significantly, but the depressed group scored significantly higher. Further, a 2x2 ANOVA with depression diagnosis (presence of comorbid depression vs. absence of comorbid depression) serving as a between-subjects factor and BAI score (pre-treatment vs. post-treatment) as a within-subjects factor was conducted. These analyses showed a significant main effect of time ($F_{1,23} = 43.48$, $p < .001$, $\eta^2_p = .65$); neither the main effect of depression diagnosis ($F_{1,23} = 0.24$, $p = .633$, $\eta^2_p = .01$) nor the time x depression diagnosis interaction ($F_{1,23} = 1.50$, $p = .234$, $\eta^2_p = .06$) were significant. Thus, on the BAI, both groups improved significantly, there was no significant difference in observed change, and there were no significant differences in scores.

These analyses were repeated using pre- and post-treatment clinician severity ratings (CSRs) for the primary anxiety diagnosis as the outcome measures. These analyses yielded a significant main effect of time ($F_{1,116} = 217.53$, $p < .001$, $\eta^2_p = .65$), no significant main effect of a depressive diagnosis ($F_{1,116} = 2.23$, $p = .138$, $\eta^2_p = .02$), and no significant time x depression interaction ($F_{1,116} = .62$, $p = .432$, $\eta^2_p = .01$). Therefore, CSR for the primary

diagnosis decreased in both groups following treatment, and there were no significant differences in change or total scores. Additionally, a series of ANOVAs were conducted to compare the depressed and non-depressed groups on their changes in overall clinical presentation. A 2x2 ANOVA with depression diagnosis (presence of comorbid depression vs. absence of comorbid depression) serving as a between-subjects factor and CGI score (pre-treatment vs. post-treatment) serving as a within-subjects factor revealed a significant main effect of time ($F_{1,117} = 250.10$, $p < .001$, $\eta^2_p = .68$) suggesting that both groups improved following treatment. Neither the main effect of depression diagnosis ($F_{1,117} = 1.57$, $p = .213$, $\eta^2_p = .01$) nor the time x depression diagnosis was significant ($F_{1,117} = 1.02$, $p = .315$, $\eta^2_p = .01$), so there was no difference in observed change or in clinician ratings across groups. A two-way ANOVA to compare the two groups (DEP vs. NDEP) on the CGI improvement score revealed that the main effect of a depressive diagnosis was not significant ($F_{1,115} = 0.14$, $p = .871$, $\eta^2_p < .01$) such that clinician-rated improvement following treatment was not different between the DEP and NDEP groups.

Effect of TGCBT for Anxiety on Depression

ANOVAs. A 2x2 ANOVA with depression diagnosis (presence of comorbid depression vs. absence of comorbid depression) serving as a between-subjects factor and BDI-II score (pre-treatment vs. post-treatment) serving as a within-subjects factor revealed significant main effects for time ($F_{1,86} = 70.22$, $p < .001$, $\eta^2_p = .45$) and depression diagnosis ($F_{1,86} = 28.89$, $p < .001$, $\eta^2_p = .25$); the time x depression diagnosis interaction was not significant ($F_{1,86} = 3.34$, $p = .071$, $\eta^2_p = .04$). Thus, both groups improved significantly on self-reported depression, the depressed group scored significantly higher, and there was no difference between the groups in change over time.

Focusing on the group of individuals with a depressive diagnosis, a repeated measures ANOVA with the CSR of the depression diagnosis as the within-subjects variable, was conducted to determine the effect of TGCBT for anxiety on depression. There was a significant main effect of time ($F_{1,41}$ = 46.57, p < .001, η^2_p = .53) suggesting that the severity of the depression diagnosis decreased significantly following treatment.

Mediation of Improvements

Preliminary Analyses. Initially, a 2x2 ANOVA with depression diagnosis (DEP vs. NDEP) serving as a between-subjects factor and PANAS score (pre-treatment vs. post-treatment) serving as a within-subjects factor was conducted in order to determine if there were differential changes in the potential mediator. Analyses showed significant main effects of time ($F_{1,86}$ = 93.91, p < .001, η^2_p = .52) and depression diagnosis ($F_{1,86}$ = 10.37, p = .002, η^2_p = .11), but not the time x depression diagnosis interaction ($F_{1,86}$ =.45, p = .505, η^2_p = .01).

Linear Regression Model. Mixed effects analyses showed that depression had a significant effect on STAI scores (B = -3.99, *SE* = 1.70, $t_{102.97}$ = -2.35, p = .021). In the second model, the effect of depression was not significant (B = -.04, *SE* = 1.48, $t_{105.75}$ = .027, p = .98), while the effect of PANAS was significant (B = .90, *SE* = .09, $t_{155.97}$ = 10.56, p < .001). Finally, the effect of depression on negative affect (NA) was significant (B = -4.86, SE = 1.19, $t_{109.88}$ = -4.07, p < .001). These results yielded a significant full mediation effect (z = -4.39, p < .001).

These analyses were repeated with BDI-II as the outcome variable. Mixed effects analyses showed that depression had a significant effect on BDI-II scores (B = -9.46, *SE* =

1.63, $t_{108.90}$ = -5.79, p < .001). In the second model, the effect of depression was significant (B = -4.61, *SE* = 1.33, $t_{111.43}$ = -3.48, p = .001), as was the effect of PANAS (B = .98, *SE* = .07, $t_{198.41}$ = 13.76, p < .001). Finally, the effect of depression on NA was significant (B = -4.86, SE = 1.19, $t_{109.88}$ = -4.07, p < .001). These results yielded a significant effect (z = -4.78, p < .001) suggesting that changes in NA partially mediated the improvement in BDI-II scores.

Finally, these analyses were repeated with the ADDQ as the outcome variable due to low sample size for the BAI. Mixed effects analyses showed that the effect of depression had a significant effect on ADDQ scores (B = -4.96, *SE* = 1.48, t_{214} = -3.35, p = .001). In the second model, the effect of depression was not significant (B = -93, *SE* = 1.25, $t_{111.02}$= -.75, p = .457), but the effect of PANAS was (B = .93, *SE* = .07, $t_{187.90}$ = 12.897, p < .001). Finally, the effect of depression on NA was significant (B = -4.86, SE = 1.19, t109.88 = -4.07, p < .001). These results yielded a significant effect (z = -4.56, p < .001) suggesting that changes in NA fully mediated the improvement in ADDQ scores.

Discussion

The main goal of this study was to examine the effects of a comorbid depressive disorder on outcomes of transdiagnostic group cognitive behavioral therapy for anxiety (TGCBT) on anxiety symptoms and overall clinical presentation. It sought to accomplish this by comparing individuals with a comorbid depressive disorder diagnosis to those without one on measures of anxiety symptoms, anxiety severity, and clinician-rated clinical presentation. Results showed that individuals in the comorbid depressive disorder diagnosis (DEP) group began treatment with significantly more severe anxiety, both self-report symptoms and clinician-rated diagnosis, than those in the no comorbid depression (NDEP) group, but these

differences diminished following treatment and were no longer significant following 12 weeks of TGCBT. On average, the NDEP group dropped from moderate to mild anxiety, and individuals in the DEP group dropped from severe to mild anxiety based on BAI scores (Beck & Steer, 1993), and the treatment had a large effect size on the severity of the primary diagnosis (η^2_p = .65), overall clinical presentation (η^2_p = .68), BAI (η^2_p = .65) and ADDQ (η^2_p = .58). Although the presence of depression had an effect on change in ADDQ score, the effect was fairly small (η^2_p = .06; Cohen, 1988) and was not significant at post-treatment. Despite this pre-treatment difference, comorbid depression did not impair treatment changes nor affect end states on the ADDQ score.

This study also aimed to examine the response of depression to TGCBT despite the fact that it is not directly targeted by the intervention. It did so by both comparing anxious treatment-seekers with depression to those without depression on self-reported depression and by examining the degree of improvement in depression diagnosis. Despite beginning treatment with clinically-significant depression, self-report and clinician-rated depression decreased following treatment. These effects were robust (BDI-II η^2_p = .45); on average individuals in the DEP group dropped from moderate-to-severe depression to minimal-to-mild depression, and the average individual without a depressive diagnosis dropped from mild to minimal depression (Beck, Steer, & Brown, 1996). Also, on average, depression diagnoses dropped from moderate, and clinically significant (rated “Definitely disturbing/disabling” or more severe by an assessor), to mild, and not clinically significant (rated “Slightly disturbing/not really disabling” by an assessor; (CSR η^2_p = .53); Brown, Di Nardo, & Barlow, 1994; Hope, Laguna, Heimberg, & Barlow, 1996/1997).

Third, this study sought to determine if negative affect (NA) mediated improvements in depression consistent with the findings of Talkovsky and Norton (In Press) regarding anxiety following this protocol. The DEP group scored significantly higher on a self-report measure of NA both before and after treatment. Results showed that NA significantly fully mediated improvements in self-reported anxiety, consistent with Talkovsky and Norton, but partially mediated improvements in self-reported depression following TGCBT. That is, change in anxiety symptoms occurred via modification of NA, but changes in NA only partially explained improvement in depressive symptoms.

These results are largely consistent with previous research that found a significant response to CBT for anxiety for both those with and without a comorbid depressive diagnosis. For instance, Fracalanza and colleagues (2014) found that individuals seeking treatment for social phobia with comorbid major depression scored significantly higher than individuals without comorbid depression on measures of social anxiety and depression, but still improved significantly following a manualized CBT protocol for social phobia. These results are comparable to those of Allen and colleagues in their investigation of panic disorder (PD) and comorbid conditions (2010), who found that the presence of a comorbid depressive disorder did not impede the rate of gains following manualized CBT for anxiety. However, in their sample, rates of major depression did not improve following eleven weeks of treatment. In the present study, not only was there a robust effect on depression diagnoses, but the average participant's depression diagnosis was also no longer clinically significant. Individuals in the present study also attended half the number of sessions observed in the Joorman, Kosfelder, and Schulte study (2005), whose participants attended approximately 25 sessions on average, suggesting that this treatment protocol may be more parsimonious that

others. Like the study by Campbell-Sills and colleagues (2012), individuals with a depressive diagnosis improved more in their anxiety and severity of their overall presentation than those without one in this study. However, unlike their methodology, this study included anxious treatment seekers, limited treatment to twelve sessions of CBT, and also did not increase therapeutic dosage at any point. The present findings may be more externally valid and applicable for clinical practice.

These results are also consistent with literature concerning anxiety and comorbidity more broadly. This study replicated the results Davis, Barlow, and Smith (2010), as comorbid patients in this study began treatment with a more severe presentation but did not suffer any impairment in their rate of change. The present investigation used a time-limited group protocol though, so all patients attended a maximum of twelve sessions unlike the Davis, Barlow, and Smith study in which comorbid patients attended an average of three additional individual therapy session compared to simple cases. This study provided an extension of the results of Norton and colleagues (2013). Similar to their study, in this investigation, comorbid patients reported more severe anxiety at pre-treatment, but did not have worse rates of change than simple cases. The present study focus specifically on specific depressive diagnoses rather than any Axis I disorder, which was most commonly anxiety in their study. For those with comorbid anxiety disorders, the comorbid conditions are targeted in treatment as this particular treatment protocol (Norton, 2012b) aims to affect anxiety beyond the individual's diagnosed anxiety disorder; depression improved in both the DEP and NDEP groups despite that it was not targeted. Thus, this study provides evidence that TGCBT modifies something other than specific manifestations of the primary disorder.

There are several theories about the mechanisms of Axis I comorbidity (Brown & Barlow, 1992). There is the possibility that the skills learned in CBT (e.g., self-monitoring, and cognitive restructuring) may generalize to other conditions besides the individual's primary diagnosis and are similar to interventions used for other disorders. Another potential explanation is that reducing symptoms of the primary diagnosis may alleviate a causal factor in secondary conditions and necessarily reduce the symptoms and intensity of those secondary conditions (Allen, et al., 2010). A third hypothesis is that therapy targets a more broad risk factor that is endemic to multiple conditions; this hypothesis is the crux of transdiagnostic theory (e.g. Barlow, Allen, & Choate, 2004; Norton, 2006). If true, then therapy should reduce the intensity of all aversive emotional symptoms and not just the symptoms of the primary disorder. The results of this study mostly substantiate this theory. Consistent with previous work from this research group (Talkovsky & Norton, In Press), improvement in anxiety occurred via modification of NA, which is a higher order risk factor for Axis I pathology (Clark & Watson, 1991). Further, NA partially mediated improvements in depression. NA is heavily implicated in the course of depression, as it is associated with depression severity and non-remission following psychotherapy for major depressive disorder (Vrieze, et al., 2014). However, the indirect effect was only partial. It is possible that the specific treatment for anxiety did not give attention to specific components of depression related to NA, such as rumination (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Despite mediation only being partial, the indirect effect was significant. Overall, these results lend credence to transdiagnostic models of emotional disorders (Barlow, Allen, & Choate, 2004; Norton, 2006).

This study had several strengths. First, this study asks a novel research question in a burgeoning area. To the best of the author's knowledge, this is the first investigation to use a large sample size and to examine the reciprocal effects of depression in transdiagnostic treatment. This study used structural equation modeling to determine its primary outcome. This statistical procedure reduces the possibility of spurious findings and strongly points towards correct conclusions. Also, this study included a large sample size of clinically-diagnosed treatment seekers with liberal inclusionary criteria in a mixed anxiety sample. Consistent with transdiagnostic theory, individuals with a primary diagnosis of any anxiety disorder were included and were not excluded based on the presence of comorbidity. This study also relied on multiple assessment methods and incorporated both self-report and clinician ratings. These help to provide confidence in the conclusions drawn from the data.

This study had a few limitations that may limit its impact. First, this study excluded individuals diagnosed with bipolar disorder due to the unique nature of mania (Watson, 2009). Understanding bipolar disorder's relation to TGCBT is a potentially important question that warrants future investigation. Further, this study did not include any individuals with a primary diagnosis of PTSD due to sampling. Because of the high co-occurrence rates of PTSD and depression (Nixon, Resick, & Nishith, 2004), understanding the effect of depression on PTSD treatment is an important clinical question that requires further research. Also, to accommodate missing data, the last obtained values for self-report measures were measures were carried forward and treated as post-treatment scores. Thus, there were only two time points available. This limits this study's ability to draw temporal inference in mediational analyses. More frequent assessment of potential mediators would be useful to improve the ability to reach conclusions about indirect effects in TGCBT. Many argue that

the BAI and BDI-II are collinear and lack discriminant validity in the assessment of anxiety and depression (Lovibond & Lovibond, 1995). However, they are the measures that clinicians most often endorse using when assessing comorbid anxiety and depression (Collimore & Rector, In Press) and demonstrated convergence with clinician ratings, increasing confidence in the validity of their use for the purposes of this investigation. Despite the external validity of these measures, use of measures with greater discriminant validity may improve internal validity. Some would also argue that the treatment plan targets schema-level cognitions, which may target depression in addition to anxiety. Thus, the treatment may actually target depression, even if this is not the explicit goal. Future research should evaluate the effects of each component of the treatment plan on depression; this would determine if any components affect depression more than others and demonstrate whether or not schema-level cognitive restructuring does, in fact, target depression more so than other components. This study also lacked a waitlist control group limiting its internal validity.

As comorbid depression is quite common in anxious treatment-seekers, this study provides important information for researchers and clinicians. Seventy-two percent of clinicians report that they would treat comorbid anxiety and depression sequentially in some fashion compared to 9% who would treat them by focusing on shared underlying vulnerabilities or key symptoms (Collimore & Rector, In Press). Based on these data as well as the results of previous research from this group (Norton, et al., 2013), TGCBT is an effective option for simultaneously targeting comorbid conditions including depression. This time-limited protocol is parsimonious and could reduce burdens on both clinicians and treatment-seekers. Future research should continue to investigate the relationships between

transdiagnostic treatments, comorbidity, and mechanisms of improvement to continue to refine this modality and potentially expand its utility in the treatment of Axis I disorders.

References

Abramowitz, J. S., & Foa, E. B. (2000). Does comorbid major depressive disorder influence outcome of exposure and response prevention in OCD? *Behavior Therapy, 31*(4), 795-800. doi:10.1016/S0005-7894(00)80045-3

Abramowitz, J. S., Franklin, F. E., & Street, G. P. (2000). Effects of comorbid depression on response to treatment of obsessive-compulsive disorder. *Behavior Therapy, 31*(3), 517-528. doi:10.1016/S0005-7894(00)80028-3

Allen, L. B., White, K. S., Barlow, D. H., Shear, M. K., Gorman, J. M., & Woods, S. W. (2010). Cognitive-behavior therapy (CBT) for panic disorder: Relationship of anxiety and depression comorbidity with treatment outcome. *Journal of Psychopathology and Behavioral Assessment, 32*(2), 185-192. doi:10.1007/s10862-009-9151-3

Alloy, L. B., Kelly, K. A., Mineka, S., & Clements, C. M. (1990). Comorbidity of anxiety and depressive disorders: A helplessness-hopelessness perspective. In J. D. Masser, & C. R. Cloninger (Eds.), *Comorbidity of mood and anxiety disorders* (pp. 499-543). Arlington, VA: American Psychiatric Association.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, fourth edition, text revision (DSM-IV-TR).* Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.).* Arlington, VA: American Psychiatric Publishing.

Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified treatment for emotional disorders. *Behavior Therapy, 35*(2), 205-230. doi:10.1016/S0005-7896(04)80036-4

Beck, A. T., & Steer, R. G. (1993). *Beck Anxiety Inventory manual.* San Antonio, TX: Psychological Corporation.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56*(6), 893-897. doi:10.1037/0022-006X.56.6.893

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory Manual (2nd ed.).* San Antonio, TX: Psychological Corporation.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8*(1), 77-100. doi:10.1016/0272-7358(88)90050-5

Belzer, K., & Schneier, F. R. (2004). Comorbidity of anxiety and depressive disorders: Issues in conceptualization, assessment, and treatment. *Journal of Psychiatric Practice, 10*(5), 296-306. doi:10.1097/00131746-200409000-00003

Besiroglu, L., Uguz, F., Saglam, M., Agargun, M. Y., & Cilli, A. S. (2007). Factors associated with major depressive disorder occurring after the onset of obsessive-compulsive disorder. *Journal of Affective Disorders, 102*(1-3), 73-79. doi:10.1016/j.jad.2006.12.007

Blanchard, E. B., Buckley, T. C., Hickling, E. J., & Taylor, A. E. (1998). Posttraumatic stress disorder and comorbid major depression: Is the correlation an illusion? *Journal of Anxiety Disorders, 12*(1), 21-37. doi:10.1016/S0887-6185(97)00047-9

Blanchard, E. B., Hickling, E. J., Devineni, T., Veazey, C. H., Galovski, T. E., Mundy, E., . . . Buckley, T. C. (2003). A controlled evaluation of cognitive behavioral therapy for posttraumatic stress in motor vehicle accident survivors. *Behaviour Research and Therapy, 41*(1), 79-96. doi:10.1016/S0005-7967(01)00131-0

Borkovec, T. D., Abel, J. L., & Newman, H. (1995). Effects of psychotherapy on comorbid conditions in generalized anxiety disorder. *Journal of Consulting and Clinical Psychology, 63*(3), 479-483. doi:10.1037/0022-006X.63.3.479

Brown, T. A., & Barlow, D. H. (1992). Comorbidity among anxiety disorders: Implications for treatment and DSM-IV. *Journal of Consulting and Clinical Psychology, 60*, 835-844.

Brown, T. A., Antony, M. M., & Barlow, D. H. (1995). Diagnostic comorbidity in panic disorder: Effect on treatment outcome and course of diagnoses following treatment. *Journal of Consulting and Clinical Psychology, 63*(3), 403-418. doi:10.1037/0022-006X.63.3.408

Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology, 110*(4), 585-599. doi:10.1037/0021-843X.110.4.585

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology, 107*(2), 179-192. doi:10.1037/0021-843X.107.2.179

Brown, T. A., Di Nardo, P. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV (ADIS-IV).* New York, NY: Graywind.

Brown, T. A., Di Nardo, P. A., Lehman, C. L., & Campbell, L. A. (2001). Reliability of DSM-IV anxiety and mood disorders: Implications for the classification of emotional disorders. *110*(1), 49-58. doi:10.1037/0021-843X.110.1.49

Bruce, S. E., Yonkers, K. A., Otto, M. W., Eisen, J. L., Weisberg, R. B., Pagano, M., . . . Keller, M. B. (2005). Influence of psychiatric comorbidity on recovery and recurrence in generalized anxiety disorder, social phobia, and panic disorder: A 12-year prospective study. *American Journal of Psychiatry, 162*(6), 1179-1187. doi:10.1176/appi.ajp.162.6.1179

Campbell, D. G., Felker, B. L., Liu, C., Yano, E. M., Kirchner, J. E., Chan, D., . . . Chaney, E. F. (2007). Prevalence of depression-PTSD comorbidity: Implications for clinical practice guidelines and primary care-based interventions. *Journal of General Internal Medicine, 22*(6), 711-718. doi:10.1007/s11606-006-0101-4

Campbell-Sills, L., Sherbourne, C. D., Roy-Byne, P., Craske, M. G., Sullivan, G., Byrstritsky, A., . . . Stein, M. B. (2012). Effects of co-occurring depression on treatment for anxiety disorders: Analysis of outcomes from a large primary care

effectiveness trial. *Journal of Clinical Psychiatry, 73*(12), 1509-1516. doi:10.4088/JCP.12m07955

Chamberlain, L. D., & Norton, P. J. (2013). An evaluation of the effects of diagnostic composition on individual treatment outcome within transdiagnostic cognitive-behavioral group therapy for anxiety. *Cognitive Behaviour Therapy, 42*(1), 56-63. doi:10.1080/16506073.2012.748090

Chambless, D. L., Tran, G. Q., & Glass, C. R. (1997). Predictors of response to cognitive-behavioral group therapy for social phobia. *Journal of Anxiety Disorders, 11*(3), 221-240. doi:10.1016/S0887-6185(97)00008-X

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*(3), 316-336. doi:10.1037/0021-843X.100.3.316

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates.

Collimore, K. C., & Rector, N. A. (In Press). Treatment of anxiety disorders with comorbid depression: A survey of expert CBT clinicians. *Cognitive and Behavioral Practice*. doi:10.1016/j.cbpra.2014.01.007

Davis, L., Barlow, D. H., & Smith, L. (2010). Comorbidity and the treatment of principal anxiety disorders in a naturalistic sample. *Behavior Therapy, 41*(3), 296-305. doi:10.1016/j.beth.2009.09.002

de Graaf, R., Bijl, R. V., Beekman, A. T., & Vollebergh, W. A. (2003). Temporal sequencing of lifetime mood disorders in relation to comorbid anxiety and substance use disorders. *Social Psychiatry & Psychiatric Epidemiology, 38*(1), 1-11.

de Graaf, R., Bijl, R. V., ten Have, M., Beekman, A. T., & Vollebergh, W. A. (2004). Pathways to comorbidity: The transition of pure mood, anxiety, and substance use disorders into comorbid conditions in a longitudinal population-based study. *Journal of Affective Disorders, 82*(3), 461-467. doi:10.1016/j.jad.2004.03.001

Ehring, T., & Watkins, E. R. (2008). Repetitive Negative Thinking as a Transdiagnostic Process. *International Journal of Cognitive Therapy*, *1*(3), 192-205. doi:10.1521/ijct.2008.1.3.192

Erwin, B. A., Heimberg, R. G., Juster, H., & Mindlin, M. (2002). Comorbid anxiety and mood disorders among persons with social anxiety disorder. *Behaviour Research and Therapy, 40*(1), 19-35. doi:10.1016/S0005-7967(00)00114-5

Fava, M., Rankin, M. A., Wright, E. C., Alpert, J. E., Nierenberg, A. A., Pava, J., & Rosenbaum, J. F. (2000). Anxiety disorders in major depression. *Comprehensive Psychiatry, 41*(2), 97-102. doi:10.1016/S0010-440X(00)90140-8

Fisher, P. L., & Durham, R. C. (1999). Recovery rates in generalized anxiety disorder following psychological therapy: An analysis of clinically significant change in the STAI-T across outcome studies since 1990. *Psychological Medicine, 29*, 1425-1434. doi:10.1017/S0033291799001336

Fracalanza, K., McCabe, R. E., Taylor, V. H., & Antony, M. M. (2014). The effect of comorbid major depressive disorder or bipolar disorder on cognitive behavioral therapy for social anxiety disorder. *Journal of Affective Disorders, 162*, 61-66. doi:10.1016/j.jad.2014.03.015

Gillis, M. M., Haaga, D. F., & Ford, G. T. (1995). Normative values for the Beck Anxiety Inventory, Fear Questionnaire, Penn State Worry Questionnaire, and Social Phobia and Anxiety Inventory. *Psychological Assessment, 7*(4), 450-455. doi:10.1037/1040-3590.7.4.450

Heim, C., & Nemeroff, C. B. (2001). The role of childhood trauma in the neurobiology of mood and anxiety disorders: Preclinical and clinical studies. *Biological Psychiatry, 49*(12), 1023-1039. doi:10.1016/S0006-3223(01)01157-X

Henry, J. D., & Crawford, J. R. (2005). The short-form version of the Depression Anxiety Stress Scales (DASS-21): Construct validity and normative data in a large non-clinical sample. *British Journal of Clinical Psychology, 44*(2), 227-239. doi:10.1348/014466505X29657

Hope, D. A., Laguna, L. B., Heimberg, R. G., & Barlow, D. H. (1996/1997). Relationship between ADIS clinician's severity rating and self-report measures among social phobics. *Depression and Anxiety, 4*(3), 120-125. doi:10.1002/(SICI)1520-6394(1996)4:3<120::AID-DA4>3.0.CO;2-K

Hranov, L. G. (2007). Comorbid anxiety and depression: Illumination of a controversy. *International Journal of Psychiatry in Clinical Practice, 11*(3), 171-189. doi:10.1080/13651500601127180

Huppert, J. D. (2009). Anxiety disorders and depression comorbidity. In M. M. Antony, & M. B. Stein (Eds.), *Oxford handbook of anxiety and related disorders* (pp. 576-586). New York, NY: Oxford University Press.

Joiner, T., & Coyne, J. C. (Eds.). (1999). *The interactional nature of depression: Advances in interpersonal approaches.* Washington, DC: American Psychological Association.

Joorman, J., Kosfelder, J., & Schulte, D. (2005). The impact of comorbidity of depression on the course of anxiety treatments. *Cognitive Therapy and Research, 29*(5), 569-591. doi:10.1007/s10608-005-3340-5

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of twelve-month DSM-IV disorders in the National Comorbidity Survey Replication (NCS-R). *Archives of General Psychiatry, 62*(6), 617-627. doi:10.1001/archpsyc.62.6.617

Kessler, R. C., Stang, P. E., Wittchen, H.-U., Ustun, T. B., Roy-Byrne, P. P., & Walters, E. E. (1998). Lifetime panic-depression comorbidity in the National Comorbidity Survey. *Archives of General Psychiatry, 55*(9), 801-808. doi:10.1001/archpsyc.55.9.801

Krull, J. L., & MacKinnon, D. P. (2001). Multilevel modeling of individual and group level mediated effects. *Multivariate Behavioral Research, 36*(2), 249-277. doi:10.1207/S15327906MBR3602_06

Lamers, F., van Oppen, P., Comijs, H. C., Smit, J. H., Spinhoven, P., van Balkom, A. J., . . . Penninx, B. W. (2011). Comorbidity patterns of anxiety and depressive disorders in a

large cohort study: the Netherlands Study of Depression and Anxiety (NESDA). *Journal of Clinical Psychiatry, 72*, 341-348. doi:341-348

Lasa, L., Ayuso-Mateos, J. L., Vásquez-Barquero, J. L., Diéz-Manrique, F. J., & Dowrick, C. F. (2000). The use of the Beck Depression Inventory to screen for depression in the general population: a preliminary analysis. *Journal of Affective Disorders, 57*, 261-265. doi:10.1016/S0165-0327(99)00088-9

Leary, M. R., Koch, E. J., & Hechenbleikner, N. R. (2001). Emotional responses to interpersonal rejection. In M. R. Leary (Ed.), *Interpersonal Rejection* (pp. 145-166). New York, NY: Oxford University Press.

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy, 33*, 335-343. doi:10.1016/0005-7967(94)00075-U

McEvoy, P. M., Watson, H., Watkins, E. R., & Nathan, P. (2013). The relationship between worry, rumination, and comorbidity: Evidence for repetitive negative thinking as a transdiagnostic construct. *Journal of Affective Disorders, 151*(1), 313-320. doi:10.1016/j.jad.2013.06.014

McLean, P. D., Woody, S., Taylor, S., & Koch, W. J. (1998). Comorbid panic disorder and major depression: Implications for cognitive-behavioral therapy. *Journal of Consulting and Clinical Psychology, 66*(2), 240-247. doi:10.1037/0022-006X.66.2.240

McNaughton, N., & Corr, P. J. (2004). A two-dimensional neuropsychology of defense: Fear/anxiety and defensive distance. *Neuroscience & Biobehavioral Reviews, 28*(3), 285-305. doi:10.1016/j.neurbiorev.2004.03.005

Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology, 49*, 377-412.

Muthén, L. K., & Muthén, B. (2006). *Mplus user's guide* (Fourth ed.). Los Angeles, CA: Muthén & Muthén.

Muthén, L. K., & Muthén, B. O. (2013). *Mplus user's guide.* (Seventh ed.). Los Angeles, CA: Muthén & Muthén.

Nixon, R. D., Resick, P. A., & Nishith, P. (2004). An exploration of comorbid depression among female victims of intimate partner violence with posttraumatic stress disorder. *Journal of Affective Disorders, 82*(2), 315-320. doi:10.1016/j.jad.2004.01.008

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3*(5), 400-424. doi:10.1111/j.1745-6924.2008.00088.x

Norton, P. J. (2006). Toward a clinically-oriented model of anxiety disorders. *Cognitive Behaviour Therapy, 35*(2), 88-105. doi:10.1080/1650670500441561

Norton, P. J. (2008). An open trial of a transdiagnostic cognitive-behavioral group therapy for anxiety disorder. *Behavior Therapy, 39*(3), 242-250. doi:10.1016/j.beth.2007.08.002

Norton, P. J. (2012). *Group cognitive-behavioral therapy of anxiety: A transdiagnostic treatment manual.* New York, NY: Guilford Press.

Norton, P. J., & Barrera, T. L. (2012). Transdiagnostic versus diagnosis-specific CBT for anxiety disorders: A preliminary randomized controlled noninferiority trial. *Depression and Anxiety, 29*(10), 874-882. doi:10.1002/da.21974

Norton, P. J., & Robinson, C. M. (2010). Development and Evaluation of the Anxiety Disorder Diagnostic Questionnaire. *Cognitive Behaviour Therapy, 39*(2), 137-149. doi:10.1080/1650607090314030

Norton, P. J., Barrera, T. L., Mathew, A. R., Chamberlain, L. D., Szafranski, D. D., Reddy, R. R., & Smith, A. H. (2013). Effect of transdiagnostic CBT for anxiety disorders on comorbid diagnoses. *Depression and Anxiety, 30*(2), 168-173. doi:10.1002/da.22018

Norton, P. J., Hayes, S. A., & Hope, D. A. (2004). Effects of a transdiagnostic group treatment for anxiety on secondary depression. *Depression and Anxiety, 4*, 198-202. doi:10.1002/da.20045

Overbeek, T., Schruers, K., Vermetten, E., & Griez, E. (2002). Comorbidity of obsessive-compulsive disorder and depression: prevalence, symptom severity, and treatment effect. *Journal of Clinical Psychiatry, 63*(12), 1106-1112.

Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1993). *State-trait anxiety inventory for adults.* Palo Alto, CA: Mind Garden.

Starcevic, V. (1995). Pathological worry in major depression: a preliminary report. *Behaviour Research and Therapy, 33*(1), 55-56. doi:10.1016/0005-7967(93)E0028-4

Steketee, G., Chambless, D. L., & Tran, G. Q. (2001). Effects of axis I and II comorbidity on behavior therapy outcome for obsessive-compulsive disorder and agoraphobia. *Comprehensive Psychiatry, 42*(1), 76-86. doi:10.1053/comp.2001.19746

Talkovsky, A. M., & Norton, P. J. (In Press). Mediators of Transdiagnostic Group Cognitive Behavior Therapy. *Journal of Anxiety Disorders*. doi:10.1016/j.janxdis.2014.09.07

Tellegen, A. (1985). Structures of mood and personality and their relevance to assessing anxiety, with an emphasis on self-report. In A. Tuma, & D. Maser (Eds.), *Anxiety and the anxiety disorders* (pp. 681-706). Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc.

Vittengl, J. R., & Holt, C. S. (1998). Positive and negative affect in social interactions as a function of partner familiarity, quality of communication, and social anxiety. *Journal of Social and Clinical Psychology, 17*, 196-208. doi:10.1521/jscp.1998.17.2.196

Vrieze, E., Demyttenaere, K., Bruffaerts, R., Hermans, D., Pizzagalli, D. A., Sienaert, P., . . . Claes, S. (2014). Dimensions in major depressive disorder and their relevance for treatment outcome. *Journal of Affective Disorders, 155*, 35-41. doi:10.1016/j.jad.2013.10.020

Watkins, E., Moulds, M., & Mackintosh, B. (2005). Comparisons between rumination and worry in a non-clinical population. *Behaviour Research and Therapy, 43*(12), 1577-1585. doi:10.1016/j.brat.2004.11.008

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology, 114*(4), 522-536. doi:10.1037/0021-843X.114.4.522

Watson, D. (2009). Differentiating the mood and anxiety disorders: A quadripartite model. *Annual Review of Clinical Psychology, 5*, 221-247. doi:10.1146/annurev.clinpsy.032408.153510

Watson, D., Clark, L. A., & Carey, G. (1988). Positive and negative affectivity and their relations to anxiety and depressive disorders. *Journal of Abnormal Psychology, 97*(3), 346-353. doi:10.1037/0021-843X.97.3.346

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 103-107. doi:10.1037/0022-3514.54.6.1063

Wittchen, H., Essau, C. A., & Krieg, J. (1991). Anxiety disorders: Similarities and differences of comorbidity in treated and untreated groups. *British Journal of Psychiatry*, 23-33.

Zinbarg, R. E., & Barlow, D. H. (1996). Structure of anxiety and the anxiety disorders: A heirarchical model. *105*(2), 181-193. doi:10.1037/0021-843X.105.2.181

Table 1

Demographics.

	Total (n = 120)	No Depression (n = 42)	Comorbid Depression (n = 78)
Age	M = 33.30 (*S.D.* = 11.30)	M = 33.70 (*S.D.* = 11.48)	M = 32.49 (*S.D.* = 11.14)
% Female	51.7	43.6	52.4
% European American	56.7	56.4	57.1
% Married or Cohabitating	39.2	42.3	33.3
% Employed Full-Time	65.0	66.7	61.9
% Graduated College or Greater	57.5	62.8	47.6

Notes. For percentage values, the significance represents the results of chi-square analyses for the proportions of all categories. % Employed full-time = proportion of individuals that either work or take classes full time. Based on chi-square analyses, there were no significant differences between the two groups on any of these demographics (ps $\geq$ .143).

Table 2

Frequency of DSM-IV-TR Anxiety Diagnoses.

	Total		No Depressive Diagnosis		Depressive Diagnosis	
	n	%	*n*	%	*n*	%
Panic Disorder	24	20.0	14	17.9	10	23.8
Generalized Anxiety Disorder	22	18.3	12	15.4	10	23.8
Social Phobia	55	45.8	37	47.4	18	42.9
Agoraphobia	1	0.8	1	1.3	0	0.0
Specific Phobia	5	4.2	5	6.4	0	0.0
Obsessive-Compulsive Disorder	6	5	4	5.1	2	4.8
Posttraumatic Stress Disorder	0	0.0	0	0.0	0	0.0
Anxiety Disorder NOS	7	5.8	5	6.4	2	4.8

Notes. Panic disorder = both panic disorder and panic disorder with agoraphobia. Agoraphobia = Agoraphobia without a history of panic disorder.

Table 3.

Means for Relevant Clinical Variables at Pre- and Post-Treatment.

		Total Sample		No Depression		Comorbid Depression	
		M	*S.D.*	*M*	*S.D.*	*M*	*S.D.*
CGI	Pre-Treatment	4.70	.89	4.54[a]	0.87	5.00[b]	0.86
	Post-Treatment	3.00	1.35	2.90[a]	1.22	3.19[a]	1.57
	Improvement	2.17	0.80	2.19[a]	0.80	2.13[a]	0.79
Primary Diagnosis Severity	Pre-Treatment	5.48	0.97	5.28[a]	0.91	5.86[b]	1.03
	Post-Treatment	3.29	1.83	3.16[a]	1.59	3.52[a]	2.22
PANAS	Pre-Treatment	29.70	7.36	27.94[a]	6.67	33.38[b]	7.47
	Post-Treatment	21.80	6.57	20.63[a]	6.12	24.42[b]	6.87
ADDQ	Pre-Treatment	33.75	8.32	31.58[a]	8.10	37.78[b]	7.21
	Post-Treatment	22.11	9.63	20.89[a]	8.74	24.40[a]	10.88
BAI	Pre-Treatment	24.77	13.61	22.11[a]	12.25	30.39[b]	14.79
	Post-Treatment	13.41	9.39	14.05[a]	9.82	12.20[a]	8.89
BDI-II	Pre-Treatment	19.66	11.15	15.76[a]	9.64	27.79[b]	9.70
	Post-Treatment	9.90	10.04	7.94[a]	8.26	14.32[b]	12.25

Depression Diagnosis CSR	Pre-Treatment	-	-	-	-	4.43	1.35
	Post-Treatment	-	-	-	-	2.10	2.18

Notes. Means in the same row with different superscripts are significantly different ($p \leq .05$). CGI = Clinical Global Impressions. PANAS-NA = Positive Affect Negative Affect Schedule – Negative Affect. ADDQ = Anxiety Disorder Diagnostic Questionnaire. BAI = Beck Anxiety Inventory. BDI-II = Beck Depression Inventory. Depression CSR = Clinician Severity Rating of the individual's depressive diagnosis.

Table 4

Results From Linear Modeling of STAI Over the Course of Treatment.

	d.f.	χ^2	$\Delta\chi^2$	Δd.f.	Δp
Intercept Set at Session 1					
Freely Estimated Model	146	322.117			
Slope Invariant Model	147	326.288	4.171	1	.0411
Intercept Invariant Model	147	335.208	13.091	1	.0003
Fully Invariant Model	148	335.370	13.253	2	.0013
Intercept Set at Session 12					
Freely Estimated Model	146	322.117			
Slope Invariant Model	147	326.288	4.171	1	.0411
Intercept Invariant Model	147	323.927	1.810	1	.1785
Fully Invariant Model	147	335.370	13.253	2	.0013

Notes. Models were run when setting the intercept at session 1 to determine differences in pre-treatment scores, and at session 12 to determine differences in post-treatment scores. $\Delta p > .05$ signifies no significant decrements in model fit due to restrictions. d.f. = Degrees of freedom; χ^2 = Chi squared; $\Delta\chi^2$ = Change in chi squared from the freely estimated model; Δd.f. = Change in degrees of freedom from the freely estimated model; Δp = significance of the change in model fit from the freely estimated model.