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Secondary depression in transdiagnostic group cognitive behavioral therapy among individuals diagnosed with anxiety disorders

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ABSTRACT

Anxiety and depression co-occur at high rates, and their comorbidity typically creates a more severe clinical presentation then either alone. The effect of comorbid depression appears to vary across anxiety and related disorders. Transdiagnostic treatments present a promising option to improve comorbid conditions by targeting shared factors (e.g., information processing biases). The purpose of this study was to examine the reciprocal effects of secondary depression in transdiagnostic group cognitive behavioral therapy for anxiety (TGCBT). 120 individuals diagnosed with a primary anxiety disorder, 42 of whom had a depressive diagnosis, were enrolled in 12 weeks of TGCBT. Depressed individuals were compared to those without a depressive diagnosis on both clinician-rated and self-reported anxiety and depression following TGCBT. Although depressed individuals scored higher on most indices of anxiety at pre-treatment, both groups improved similarly with some evidence of greater improvement among those with comorbid depression. All individuals improved in self-reported depressive symptoms and comorbid depression improved to subclinical levels. These results posit TGCBT as an effective, efficient option for treating patients with anxiety and comorbid depression.

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1. Introduction

Anxiety and depression are the two most common classes of psychological disorders (Kessler, Chiu, Demler, & Walters, 2005). Among individuals diagnosed with anxiety disorders, depression co-occurs at rates ranging from 28% to 63% (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Lamers et al., 2011). This comorbidity is associated with greater severity and chronicity of illness (Lamers et al., 2011). There are various potential explanations of the high rate of comorbidity of anxiety and depression. For instance, the criteria set for major depressive disorder shares symptoms with the anxiety disorders, especially generalized anxiety disorder (GAD) and posttraumatic stress disorder (PTSD) (American Psychiatric Association, 2013). There are also similar biological and neurochemical mechanisms (Heim & Nemeroff, 2001; McNaughton & Corr, 2004), likely due to the genetic conferral of risk via underlying personality traits, such as negative affectivity (NA) rather than disorders themselves (Huppert, 2009).

NA, or neuroticism (Tellegen, 1985), is a shared dispositional risk factor for anxiety and mood disorders (Clark & Watson, 1991; Mineka, Watson, & Clark, 1998) and may be the factor underlying emotional disorder comorbidity. Models of anxiety and depression posit NA as the common factor for both classes of disorders, with specific or lower-order vulnerabilities conferring risk for disorders themselves. For instance, the original tripartite model (Clark & Watson, 1991) presented NA subsuming low positive affect as the unique risk for depression and physiological hyperarousal as the unique risk for anxiety. This model was subsequently refined such that each disorder had its own specific component, such as anxious arousal being specific to panic disorder, and the contributions of the general NA factor and each specific factor varying across disorders (Mineka et al., 1998). Other models have suggested there are broad factors lower-order factors, such as anxiety sensitivity or intolerance of uncertainty acting as mediators between NA and specific disorders (Paulus, Talkovsky, Heggeness, & Norton, 2015) or that these constructs may better conceptualized as types of disorders such as fear disorders versus anxious-misery disorders (Watson,

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2005). Shared features of these models include NA as the overarching factor and some include shared lower-order features between anxiety and depression. The co-occurrence of anxiety and depression appears to be a clinical reality rather than an artifact of the current system of classification.

The presence of this comorbidity confers a poorer prognosis to the affected individual than anxiety alone. Individuals diagnosed with comorbid anxiety and depression had poorer outcomes than those with anxiety alone, even if the depression remits at follow-up (Wittchen, Essau, & Krieg, 1991). Long-term, the presence of major depression nearly halves the likelihood that an individual with panic disorder (PD), social anxiety disorder (SAD), or GAD recovers, and also increases the likelihood of relapse for those that do remit from PD with agoraphobia (Bruce et al., 2005). That comorbid depression increases the severity of clinical presentation appears to be a consistent finding.

There are divergent results regarding the effect of secondary depressive diagnoses on anxiety-focused therapy (see Bauer, Wilansky-Traynor, & Rector, 2012). Those presenting with anxiety and comorbid depression tend to demonstrate more severe psychopathology at pre-treatment and often at post-treatment; despite these impairments, many find that the presence of a depressive diagnosis does not prevent the treatment-seeker from benefitting from treatment compared to those with only an anxiety diagnosis, or present with increased needs in care (Blanchard, Buckley, Hickling, & Taylor, 1998; Besiroglu, Uguz, Saglam, Agargun, & Cilli, 2007; Bauer et al., 2012; Campbell-Sills et al., 2012; Fracalanza, McCabe, Taylor, & Antony, 2014). The overall body of research regarding the response of comorbid depression on anxiety, trauma, and obsessive-compulsive disorders treatment is mixed suggesting a need for treatments that can efficiently and effectively treat anxiety despite the presence of depression and can simultaneously reduce the co-occurring depression. Taken together, previous research suggests CBT can remain effective despite the presence of comorbid depression and positively affect the depressive symptoms through anxiety-focused treatment.

Treating a principal anxiety disorder may reduce the severity or lead to the remittance of a comorbid condition via shared psychological processes; treatments that target shared processes likely affect all subsumed diagnoses according to transdiagnostic theories of psychotherapy (Brown & Barlow, 1992). An approach that emphasizes symptoms or processes, such as NA (Clark & Watson, 1991) rather than syndromes could likely treat comorbidity effectively (Brown & Barlow, 1992). Norton (2006) identified support for the notion that anxious and depressive disorders respond to similar techniques, modalities, and medications, and noting that improvements in comorbid conditions are not simply functions of symptom overlap. The emphasis on core processes of pathology may make transdiagnostic CBT more efficacious in treating comorbidity due to its emphasis on identifying and targeting these common processes (Erickson, Janeck, & Tallman, 2009) and transdiagnostic CBT explicitly targets these processes.

In one study of manualized individual CBT using a diagnostically heterogeneous sample, individuals diagnosed with comorbid disorders began treatment with a more severe presentation, but comorbidity did not hinder the rate of improvement across principal anxiety diagnoses. Primary diagnoses in this sample included (from most to least frequent) SAD, PD, GAD, MDD and dysthymia collapsed together, OCD, and specific phobia. Over half (58.7%) were diagnosed with at least one comorbid condition, with depression being the most common. However, individuals diagnosed with comorbid conditions attended an average of three additional sessions, suggesting a greater level of need in individual therapy (Davis, Barlow, & Smith, 2010). Similarly, in an analysis of 23 anxious treatment seekers, 8 of whom had a secondary depressive diagnosis, individuals receiving transdiagnostic group cognitive behavioral therapy (TGCBT) improved significantly in depression over the course of treatment, but those in a control condition did not improve (Norton, Hayes, & Hope, 2004). These data presented promising early results, but a need remains to demonstrate the effectiveness of transdiagnostic therapy in its ability to confer improvements across diagnostic classes while maintaining parsimony and efficiency in a large treatment-seeking sample.

TGCBT has demonstrated effectiveness in treating individuals with broad profiles of comorbid diagnoses. Norton et al. (2013) found that individuals diagnosed with anxiety and at least one additional diagnosis (64.6% of their sample) were significantly more severe than those with a single diagnosis before treatment, but there were no differences in rates of improvement in overall or anxious severity. About two thirds of those with comorbid diagnoses who completed treatment no longer met criteria for a secondary diagnosis, which was greater than rates observed in diagnosis-specific treatment studies. Although depressive disorders were among the comorbid conditions (representing 27.4% of all comorbid diagnoses), the sample had heterogeneous profiles of comorbidity, which were combined to sufficiently power diagnostic analyses. As anxiety and depressive disorders vary in their rates of comorbidity as principle and additional diagnoses, (Brown, Campbell et al., 2001), inspecting particular diagnostic profiles could guide treatment and allow clinicians to target specific profiles. As such, the specific effect of depression on response to TGCBT for anxiety remains unclear. Furthermore, demonstrating improvements across diagnostic classes following TGCBT would not only substantiate transdiagnostic theory, but would also add to the line of research that posits TGCBT as a strong option to treat individuals diagnosed with depressive comorbidity, which appears to be the norm rather than the exception for clinicians treating anxiety and depression. Because diagnosis-specific protocols appear to maintain a fair level of effectiveness in treating anxiety and comorbid depression, transdiagnostic treatments have both theoretical and preliminary empirical support for maintaining effectiveness in treating anxiety. They appear to do so while simultaneously improving depression, with empirical support for treating primary anxiety and comorbid conditions, transdiagnostic treatment is a good candidate for treating primary anxiety while simultaneously improving comorbid depression.

Given the high rate of comorbid depression with anxiety disorders, the need to evaluate specific patterns of comorbidity, and the relative infancy and transdiagnostic therapy, we aimed to examine the transactional effects of comorbid depressive disorders on the treatment of primary anxiety disorders in TGCBT, as well as the effect of TGCBT for anxiety on comorbid depression. Based on the theory and existing research for transdiagnostic treatment supporting its effectiveness in comorbid samples (Norton et al., 2004, 2013; Talkovsky & Norton, 2014), the first hypothesis was that there would be no significant differences in improvement on measures of overall anxiety severity, primary anxiety disorder severity, severity of overall clinical presentation between, and NA anxious individuals with a comorbid diagnosis of a depressive disorder (DEP) and those without a comorbid depression diagnosis (NDEP). Second, it was hypothesized that all individuals would improve on measures of depression, and individuals with comorbid depression will improve a significantly greater amount than those without a depression diagnosis. Whether related to regression to the mean of the depressive disorder, treatment improving NA rather than a vulnerability broadly specific to anxiety (Talkovsky & Norton, 2014), the individual diagnosed with depression desiring to generalize therapeutic skills to depressive symptoms (Brown & Barlow, 1992), we believed this milieu of factors would result in the DEP group demonstrating greater improvements in self-reported depression symptoms. For similar reasons, it was expected that depression diagnoses would respond to TGCBT for anxiety such that post-

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