



Mediators of transdiagnostic group cognitive behavior therapy



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ABSTRACT

The efficacy of cognitive-behavioral therapy (CBT) for anxiety is well established. Investigations into the mechanisms of change in CBT report changes in cognitive vulnerabilities mediating improvements over the course of treatment. As anxiety disorders share certain risk factors, there is a trend toward CBT emphasizing these vulnerabilities, including negative affectivity (NA) and also more specific constructs such as anxiety sensitivity (AS) and intolerance of uncertainty (IU). The purpose of this investigation was to analyze potential mediators of anxiety reduction over the course of transdiagnostic group CBT. NA, AS, and IU all decreased over the course of treatment. Among the potential mediators, change in NA had a significant relationship with change in anxiety but change in AS and change in IU did not. Neither the main effect of primary diagnosis nor the interactions between potential mediators and primary diagnoses were significant, indicating that there were no differential changes in anxiety or the potential mediators across primary diagnoses. Results strongly point toward NA as an overarching mediator of anxiety reduction during transdiagnostic group CBT.

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1. Introduction

The most common class of psychological disorders is anxiety, as roughly a quarter to a third of all people will meet criteria for an anxiety disorder in their lifetimes (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012; Kessler et al., 2005). A large body of research supports the use of cognitive behavioral therapy (CBT) for the treatment of anxiety (Norton & Price, 2007). CBT-based interventions have robust effects on panic (Barlow, Gorman, Shear, & Woods, 2000), social phobia (Heimberg, 2002) generalized anxiety disorder (GAD; Covin, Ouimet, Seeds, & Dozois, 2008), posttraumatic stress disorder (PTSD; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010), and obsessive compulsive disorder (OCD; Whittal, Thordarson, & McLean, 2005), and clinically significant gains are maintained long-term.

Despite the large body of empirical support for CBT, practitioners often struggle to deliver care to all those that would benefit from it (Shafraan et al., 2009), partially because it is time-consuming, costly, and inefficient to disseminate multiple disorder-specific manualized CBT protocols (Clark, 2009). There is a trend toward anxiety treatment that emphasizes vulnerability shared across the anxiety disorders, as there is a large amount of overlap (Barlow, 2000; Norton, 2006). In Clark and Watson's (1991) tripartite

model of anxiety and depression, depression and anxiety share the underlying vulnerability of negative affectivity, or a dispositional penchant for responding to negative stimuli with feelings of fear, anxiety, depression, guilt and self-dissatisfaction (Clark, Watson, & Mineka, 1994). Negative affectivity serves as a higher-order factor subsuming lower-order risk factors that confer more specific risk for certain disorders. Brown, Chorpita, and Barlow (1998) observed a model in which negative affectivity had a direct causal influence on GAD, panic disorder, OCD, and social phobia and this model had an excellent fit to their data. Watson (2005) suggested that a model of anxiety disorders should not only include factors that cut across diagnoses (e.g., negative affectivity), but also those that are more specific to particular conditions. The most heavily studied constructs proposed to be specific factors may be anxiety sensitivity and intolerance of uncertainty.

Anxiety sensitivity, or the fear of fearful and/or anxious symptoms due to the belief that they may have adverse consequences (Reiss & McNally, 1985) is heavily implicated in panic disorder (McNally, 2002) and PTSD (Taylor, 1993), but scores are also elevated among individuals with GAD, social phobia, and OCD compared to non-anxious controls (Taylor, Koch, & McNally, 1992). Furthermore, Hazen, Walker, and Eldridge (1996) found that anxiety sensitivity (AS) is responsive to anxiety treatment, and this improvement is related to the reduction of anxiety symptoms. In motor vehicle accident victims, Fedoroff, Taylor, Asmundson, and Koch (2000) found, using regression analyses, that AS and pain severity were significant predictors of both trauma symptoms prior to treatment and symptom reduction following treatment.

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Smits, Powers, Cho, and Telch (2004) used the Baron and Kenny (1986) method to test whether a reduction in fear of fear underlies improvement resulting from CBT for panic disorder. They observed full mediation of the relationship between the effects of CBT and changes in global impairment, and partial mediation on agoraphobia, anxiety, and panic frequency. Hofmann et al. (2007) substantiated these results, finding that panic-related catastrophic cognitions mediated treatment change in CBT.

Few studies analyze the role of anxiety sensitivity in treatment of other anxiety disorders, but rather they focus on panic disorder (Smits et al., 2004) or PTSD (Fedoroff et al., 2000), leaving a need to analyze the rest of the anxiety disorders. Boswell et al. (2013) observed elevated AS in patients with primary diagnoses of panic disorder, social phobia, GAD, and OCD, and all groups demonstrated AS improvements following interoceptive exposures, and lower levels of AS were associated with lower levels of clinical severity in analyses collapsed across principal diagnoses. Thus, AS may represent a specific vulnerability to panic, but it may confer risk to the other anxiety disorders as well.

Similarly, the construct of intolerance of uncertainty (IU) has been operationalized in many different ways (Starcevic & Berle, 2006). For the purposes of this investigation, it will be defined as a characteristic tendency to find uncertainty regarding outcomes of an event aversive (Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994). Treatment specifically targeting IU in individuals with GAD leads to clinically significant change immediately following treatment and gains are maintained at twelve-month follow up whether treating individuals (Ladouceur et al., 2000) or groups (Dugas et al., 2003). Although it was originally hypothesized that IU is specific to GAD (Dugas, Gagnon, Ladouceur, & Freeston, 1998), Carleton et al. (2012) found elevated scores in social anxiety disorder (SAD), panic disorder (PD), GAD, OCD, and depression patients, and concluded that IU may represent a transdiagnostic vulnerability. McEvoy and Mahoney (2012) found that IU mediates the relationship between neuroticism, a higher order vulnerability, and social anxiety, panic, and depression; also, they found that the prospective anxiety factor of IU mediated the relationship between neuroticism and worry, and obsessive-compulsive symptoms. Belloch et al. (2011) found that IU decreased over the course of cognitive therapy for OCD. McEvoy and Mahoney (2012) observed reductions in IU specific to social situations following group CBT for SAD, but it is possible that this situation-specific assessment captures variance overlapping with diagnostic criteria for SAD and it would be worth replicating results with a more general assessment tool. IU is clearly relevant to the treatment process, but presently lacks empirical support as a mediator of treatment outcomes for anxiety. Despite its associations with GAD, IU is a promising transdiagnostic marker for anxiety in light of its associations with a number of other disorders.

Sexton, Norton, Walker, and Norton (2003) tested a hierarchical model in a sample of undergraduates with neuroticism serving as the first-order factor subsuming AS and IU. They found that AS acted as a risk for panic, obsessive-compulsive symptoms, and health anxiety, IU acted as a risk for worry, and neuroticism was an overarching risk for all symptom types. Norton, Sexton, Walker, and Norton (2005) partially replicated these results in a sample of treatment-seekers. They observed that negative affectivity served as a global risk factor and influenced AS and IU, AS affected panic, and health anxiety, and IU influenced worry and depression; however, the expected relationship between negative affectivity and obsessive-compulsive symptoms was not observed. Norton and Mehta (2007) used structural equation modeling to compare potential hierarchical models. They improved the fit of previous models by including a path from IU to obsessive-compulsive and social anxiety symptoms. These studies combine to provide support for hierarchical models of anxiety with AS and IU acting as

second-order risk factors that show a degree of specificity, but not as much as some originally thought.

As IU and AS are oft-acknowledged risk factors, and they decrease across treatment, it would be enlightening to determine their relationships to symptom reduction and to do so across diagnoses. Because of their demonstrated relationships with anxiety disorders and their response to treatment, this study posits AS and IU as potential mediators of anxiety reduction. Mediator variables explain the way an antecedent exerts its effects on a dependent variable, thus allowing a more complete understanding of how the criterion and outcome variables relate to one another (Mackinnon & Fairchild, 2009). Mediation analyses are important for treatment and prevention, as interventions are often designed to change an outcome by targeting mediators hypothesized to have a causal relationship with the outcome (MacKinnon, Fairchild, & Fritz, 2007). It is important to understand what makes therapy work because understanding mechanisms of change can improve the identification of appropriate, better, different, and new treatments (Kazdin, 2007, 2009). Much of the extant literature suggests that cognitive variables mediate the outcomes of diagnosis-specific anxiety treatment (Arch, Wolkaty-Taylor, Eifert, & Craske, 2012; Hofmann, 2004; Hofmann et al., 2007; McEvoy & Mahoney, 2012; Smits et al., 2004).

As interest in transdiagnostic psychotherapy grows, it is important to establish the role of shared vulnerabilities, such as AS and IU, in the course of psychotherapy. In a mixed anxiety disorder sample of 35 individuals randomized to receive CBT that included components shared across the anxiety disorders, Arch et al. (2012) found that AS significantly mediated post-treatment worry and they observed no differential effects by diagnosis. However, they failed to observe significant mediation for four of their five outcome measures. Updating these results with a larger sample would contribute to the growing body of transdiagnostic literature. We attempt to improve upon cross-sectional analysis by investigating simultaneous change over the entire course of a manualized treatment protocol. This study aims to identify potential mediators by observing the change in potential criterion, mediator, and outcome variables.

The purpose of this study is to analyze potential mediators of anxious symptom reduction in transdiagnostic cognitive behavioral group therapy. Norton (2008) established the effectiveness of transdiagnostic CBGT, but the mechanisms of change have not yet been established. This study seeks to identify potential mediators of overall anxiety reduction across anxiety diagnoses. The first hypothesis is that reductions in negative affectivity, a higher-order risk for anxiety, will be a significant mediator of symptom improvement across treatment in all diagnostic categories. Second, it is hypothesized that AS and IU will each act as specific mediators, with AS significantly mediating improvement in panic symptoms and social anxiety, and IU mediating the improvement in worry.

2. Methods

The data used in this study was collected during three previous clinical trials (Norton, 2008, 2012a; Norton & Barrera, 2012) that share methodologies and treatment protocols (Norton, 2012b). These results have not been previously published. All procedures were approved for human subjects research by the Institutional Review Board at the University of Houston.

2.1. Participants

Participants included 256 individuals with principal diagnoses of panic disorder (PD), social phobia (SP) or generalized anxiety disorder (GAD) seeking treatment at the Anxiety Disorder Clinic

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