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Intolerance of uncertainty as a mediator of reductions in worry in a cognitive behavioral treatment program for generalized anxiety disorder

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ABSTRACT

Growing evidence suggests that intolerance of uncertainty (IU) is a cognitive vulnerability that is a central feature across diverse anxiety disorders, including generalized anxiety disorder (GAD). Although cognitive behavioral therapy (CBT) has been shown to reduce IU, it remains to be established whether or not reductions in IU mediate reductions in worry. This study examined the process of change in IU and worry in a sample of 28 individuals with GAD who completed CBT. Changes in IU and worry, assessed bi-weekly during treatment, were analyzed using multilevel mediation models. Results revealed that change in IU mediated change in worry (ab = −0.20; 95% CI [−.35, −.09]), but change in worry did not mediate change in IU (ab = −0.16; 95% CI [−.06, .12]). Findings indicated that reductions in IU accounted for 59% of the reductions in worry observed over the course of treatment, suggesting that changes in IU are not simply concomitants of changes in worry. Findings support the idea that IU is a critical construct underlying GAD.

1. Introduction

Intolerance of uncertainty (IU), the dispositional tendency to experience fear of the unknown, is considered to be an important factor in the development and maintenance of anxiety disorders (Carleton, 2012). IU includes beliefs that uncertainty is threatening, stressful, and anxiety provoking, as well as the desire to avoid situations where uncertainty and ambiguity may be present (Buhr & Dugas, 2002; Dugas, Gosselin, & LaDoucuer, 2001). Although IU likely contributes to multiple anxiety disorders (e.g., Carleton, 2012), the most comprehensive conceptual model of the relationships between IU and anxiety psychopathology was designed primarily to account for symptoms of generalized anxiety disorder (GAD; Dugas, Gagnon, Ladouceur, & Freeston, 1998). GAD features worry, defined as "repetitive, uncontrollable thoughts about negative life events" (Segerstrom, Tsao, Alden, & Craske, 2000), as a predominant symptom (American Psychiatric Association, 2013). For those high in IU, the possibility of negative outcomes is proposed to trigger maladaptive behavioral and cognitive reactivity (e.g., biased interpretations of the situation, increased need for information during decision-making) that serve to increase worry and anxiety (Dugas et al., 2005; Dugas & Robichaud, 2007; Ladouceur, Gosselin, & Dugas, 2000). Moreover, IU contributes to other problematic cognitive processes, including poor problem orientation and cognitive avoidance, which conjointly and paradoxically maintain worry and anxiety (Dugas & Robichaud, 2007).

Data from several treatment outcome studies indicate that anxiety interventions impact IU, and suggest that IU may play a role in maintaining anxiety. Dugas and colleagues (e.g., Dugas et al., 2003, 2010; Dugas & Ladouceur, 2000) have developed a cognitive-behavioral intervention specifically to address IU as part of a comprehensive treatment for GAD, which has been shown to effectively decrease IU and other symptoms (e.g., worry,

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depression). Other types of CBT interventions that do not feature an explicit focus on IU also appear to reduce IU in GAD (e.g., Boswell, Thompson-Hollands, Farchione, & Barlow, 2013; Hewitt, Egan, & Rees, 2009; van der Heiden, Muris, & van der Molen, 2012). Thus, preliminary evidence suggests that IU is malleable with CBT interventions in individuals with GAD. Establishing IU as a process relevant to symptom reduction is critical to validate cognitive theories and to identify treatment strategies to optimize therapeutic outcomes (Kazdin, 2007; Smits, Julian, Rosenfield, & Powers, 2012). The aforementioned treatment outcome studies provide evidence that IU changes from pre to post treatment, but the process by which IU changes relative to other symptoms has not been empirically established. For example, it is possible that reducing IU lessens worry, or that IU levels are lower at the end of treatment because worry or general anxiety symptoms have decreased. One case-controlled study suggests that reductions in IU precede reductions in worry in treatment for GAD (Dugas & Ladouceur, 2000). An analogue study of exposure-based treatment components also suggested that reductions in IU predicted subsequent reductions in worry (Goldman, Dugas, Sexton, & Gervais, 2007). Further support for a causal relationship between IU and worry comes from experimental psychopathology studies indicating that manipulating IU appears to impact worry (Ladouceur, Gosselin, & Dugas, 2000; Meeten, Dash, Scarlet, & Davey, 2012). However, models suggesting reduction in IU as a mediator of reductions in worry longitudinally over the course of treatment have yet to be confirmed empirically. Evaluating whether or not changes in IU precede and account for symptom change during treatment provides a more rigorous test of the hypothesis that IU is a core construct that perpetuates worry and anxiety (Kazdin, 2007).

The present study examined the process of change in IU and worry in a sample of individuals with GAD who completed a CBT program. The goal of the analyses was to test the proposed mediational relationship outlined in models of GAD and worry—specifically that reductions in IU would account for reductions in worry over the course of treatment. Using data from an open trial of a transdiagnostic CBT treatment protocol for anxiety, changes in IU and worry assessed at pre-treatment and bi-weekly during treatment were analyzed using multilevel mediation procedures. We hypothesized that reductions in IU would mediate subsequent reductions in worry across sessions.

2. Method

2.1. Design

Data were drawn from a trial examining neural differences between healthy and anxious individuals and the relationship between neural activity and treatment response to 10 sessions of cognitive behavioral therapy (clinicaltrials.gov Identifier NCT00947570). Analyses utilized symptom measures collected from participants over the course of CBT. Each assessment in the mediation models was collected at baseline and sessions 2, 4, 6, 8, and 10. The institutional review board of the University of California San Diego approved all procedures, and all participants provided written informed consent.

2.2. Participants

Participants were 28 individuals aged 18–55 recruited from the San Diego community, who met current diagnostic criteria for primary GAD and had at least a high school level education. Because of the specific theoretical link between IU and GAD, individuals were selected based on GAD diagnosis from a larger sample of individuals with anxiety disorders who were enrolled in the clinical trial. Exclusion criteria included a diagnostic history of a psychotic disorder, bipolar I disorder, organic mental disorder, substance dependence (past 12 months) or abuse (past month), use of psychotropic or anti-epileptic medication (past 6 weeks), consumption of more than six caffeinated beverages daily or cigarette use, and unwillingness or contraindications to completing fMRI scanning (e.g., potential pregnancy, history of claustrophobia, metal in body). Individuals with comorbid depression were eligible provided the level of depression was not in the severe range (operationalized as Quick Inventory of Depression Symptomology $-6 \leq 18$ and neither of depressed mood or anhedonia most of the day nearly every day during the past two weeks; Rush et al., 2006). Eligibility for the study was determined with structured diagnostic interviewing using the MINI Neuropsychiatric Interview (MINI) conducted by masters- and doctoral-level interviewers. Potential participants were referred to the study from the University of California San Diego (UCSD) and UCSD-affiliated outpatient clinics, other local clinical facilities, and through community-based advertisements.

2.3. Intervention

The CBT program consisted of 10 1 h individual in-person sessions delivered over 10–12 weeks. The intervention was adapted from the protocol developed as part of a multi-site trial of evidence-based treatment for anxiety in primary care (Coordinated Anxiety Learning and Management program [CALM]; for a detailed review of treatment components see Craske et al., 2009). The intervention included generic modules of anxiety treatment (self-monitoring, psychoeducation, breathing retraining, and relapse prevention). The intervention also included disorder-specific modules addressing exposure to internal and external cues (e.g., imaginal exposure to worry themes) and cognitive restructuring (e.g., generating alternative thoughts in response to catastrophic negative predictions). Modules did not specifically aim to target IU; however, in the course of therapy participants completed activities or discussed thoughts that may have addressed concerns about uncertainty (see Mahoney & McEvoy, 2012, and van der Heiden et al., 2012 for examples of general CBT's effects on IU). Modules were presented in a computer-assisted, interactive module format that directed the clinician and patient. Clinicians for the study were Ph.D.-level psychologists with specialized training in cognitive behavioral treatment of anxiety. To assure treatment fidelity, all sessions were audiotaped (with patients' permission), and for each patient one session was randomly chosen to be rated on protocol adherence and clinician competence by a licensed clinical supervisor (AJL). The highest adherence rating, a 2 on a 0–2 scale, indicated that the therapist "Presented all materials per CALM Tools program;" the highest competence rating, also a 2 on a 0–2 scale, indicated that the therapist "Facilitated application of the materials. Appropriately fielded patients' questions and elicited relevant examples." In all cases, therapists received ratings of "2", indicating they adhered to the treatment protocol, and appropriately delivered the components of the CALM intervention.

2.4. Assessments

2.4.1. Demographic variables

Demographic information was collected via a self-report assessment administered at the time of the in-person screening for study eligibility. This assessment included questions regarding ethnicity, gender, and socioeconomic status.

2.4.2. Diagnostic status

To determine diagnostic status and other initial eligibility criteria, a study clinician administered the MINI (Sheehan et al., 1998). Interviewers were individuals with prior clinical diagnostic