Intolerance of Uncertainty: A Common Factor in the Treatment of Emotional Disorders

James F. Boswell, Johanna Thompson-Hollands, Todd J. Farchione, and David H. Barlow

Boston University

Intolerance of uncertainty (IU) is a characteristic predominantly associated with generalized anxiety disorder (GAD); however, emerging evidence indicates that IU may be a shared element of emotional disorders. **Aims:** This study aimed to examine IU across diagnostic categories, change in IU during transdiagnostic treatment, and the relationship between change in IU and treatment outcome. **Method:** Patients diagnosed with heterogeneous anxiety and depressive disorders received up to 18 weeks of a transdiagnostic cognitive-behavioral therapy intervention. Patient self-reported IU and self-report and clinician-rated symptom/functioning measures were administered at pretreatment and posttreatment. **Results:** When controlling for negative affectivity, IU correlated with measures of depressive symptoms and worry severity at pretreatment. Patients with GAD and panic disorder exhibited the highest pretreatment IU scores, yet IU scores did not differ significantly based on the presence or absence of a specific diagnosis. A significant decrease in IU was observed, and change in IU was related to reduced anxiety and depressive symptom levels at posttreatment across diagnostic categories. **Discussion:** Change in IU can be observed across problem areas in transdiagnostic treatment and such change is correlated with treatment outcome. J. Clin. Psychol. 69:630–645, 2013.

Keywords: intolerance of uncertainty; anxiety; depression; psychotherapy outcome

Intolerance of uncertainty (IU) has been defined as "a dispositional characteristic that results from a set of negative beliefs about uncertainty and its implications and involves the tendency to react negatively on an emotional, cognitive, and behavioral level to uncertain situations and events" (Buhr & Dugas, 2009, p. 216). The IU construct has been associated predominantly (conceptually and empirically) with generalized anxiety disorder (GAD; Dugas, Freeston, & Ladouceur, 1997) and obsessive-compulsive disorder (OCD; Lind & Boschen, 2009; Steketee, Frost, & Cohen, 1998). However, recent evidence calls into question the "narrow specificity" of IU to GAD and OCD (Gentes & Ruscio, 2011).

The cardinal feature of GAD is excessive and uncontrollable worry regarding future events and outcomes (Borkovec, 2002). Worry can serve an experiential avoidance function (Borkovec, 1994; Borkovec, Ray, & Stober, 1998) insofar as engaging in worry reduces anxious arousal related to feared potential outcomes; thus, worry is at least partially maintained through negative reinforcement.

Furthermore, excessive worriers report that worrying helps them to problem solve, prepare for potential outcomes, prevent negative outcomes from occurring, and minimize emotional reactions to negative outcomes (Borkovec, Hazlett-Stevens, & Diaz, 1999; Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994). Worry, therefore, is thought to reduce distress over uncertainty and its associated affects (Greco & Roger, 2003) and increase, at least the perception of, control over future outcomes. Because the potential catastrophic outcomes are relatively low base-rate events, individuals often attribute their nonoccurrence to preventative worry, yet uncertainty remains and worry processes are strengthened as opportunities for disconfirmation are missed.

Please address correspondence to: James F. Boswell, 648 Beacon St., 6th floor, Department of Psychology, Boston University, Boston, MA 02216. E-mail: jboswell@bu.edu

Published online in Wiley Online Library (wileyonlinelibrary.com/journal/jclp). DOI: 10.1002/jclp.21965

In the case of OCD, doubting obsessions, compulsions, and ritualistic behaviors serve a similar experiential avoidance and control function (Steketee et al., 1997, 1998). For example, uncertainty regarding leaving the stove on leads to negatively valenced arousal that is diminished, if only momentarily, through checking behaviors; mental compulsions are thought to decrease or eliminate the chances of a potential catastrophic event occurring (e.g., death of a loved one), and elaborate cleaning rituals prevent future illness that could potentially lead to death (Grayson, 2004). Similar to worry in GAD, obsessions and compulsions in OCD reduce experiences of uncertainty and increase perceptions of control, and, similarly, opportunities for disconfirmation and new learning are missed.

It is not surprising that GAD and OCD have received most of the attention in the IU literature. Distress related to uncertainty and the use of cognitive and behavioral strategies to reduce or manage anxious arousal related to feared potential outcomes and increase one's perception of certainty and control are core elements of GAD and OCD that are implicated in the development and maintenance of both disorders. However, upon closer inspection, these features are also present in other emotional disorders.

An emotion itself is a construct that is inferred by the presence of physiological, behavioral, and cognitive components, and each component is likely to be activated when confronted with uncertainty. Information processing biases toward *potentially* threatening stimuli are present across the spectrum of anxiety disorders (Barlow, 2002; Eysenck, 1997). In panic disorder, for example, while information processing biases are common, it may be the physiological component that is often most pronounced in the face of uncertainty.

Additionally, a diagnosis of panic disorder requires the presence of excessive worry regarding future panic attacks or changes in behavior to prevent future panic attacks and related symptoms (American Psychiatric Association, 2000). In the case of a panic attack, a high degree of distress is associated with uncertainty regarding the nature and consequences of internal somatic sensations (Barlow, 2002; Rachman, 1984). Despite avoidance (e.g., avoiding exercise or caffeine) and the use of safety signals, patients with panic disorder are also unable to predict reliably whether or when they will face a situation that will cause their uncomfortable physical feelings to increase (McNally, 2002). The heightened arousal associated with this uncertainty (Greco & Roger, 2003) may itself become a trigger for experiencing a panic attack and lead to further avoidance.

In social phobia, individuals fear negative evaluation or performing poorly in social situations. Anxiety is experienced before (anticipatory), during, and after social situations, which involves a sense of uncertainty related to current or future social evaluation. Furthermore, individuals with social phobia often make thinking errors such as "mind reading" and "fortune telling" (Hope, Heimberg, & Turk, 2010), which represent attempts to decrease uncertainty regarding others' judgments and future social outcomes.

Thus, conceptual analysis indicates that difficulty tolerating uncertainty is present in various emotional disorders characterized by negative affect (Boelen & Reijntes, 2009; Deacon & Abramowitz, 2008; Dugas, Gosselin, & Ladouceur, 2001; Foa, Zinbarg, & Rothbaum, 1992), as are cognitive and behavioral attempts to reduce uncertainty and enhance perceptions of control (over both internal and external experiences and outcomes). The specific nature or focus of this uncertainty and the specific methods developed to manage it, however, may differ between specific problem areas (e.g., worry in GAD, avoiding drinking coffee in panic disorder). This conceptualization is consistent with the triple vulnerabilities model of emotional disorders (Barlow, 2002), which posits that specific disorders may represent different (and sometimes trivial) manifestations of the same generalized, underlying mechanism. If this is the case for IU, then it should be present as a shared factor in not just GAD and OCD, but other emotional disorders as well, which may help to explain the high degree of comorbidity observed across different emotional disorders (Brown & Barlow, 2009).

Although there is evidence that IU is more strongly associated with GAD severity compared with other anxiety disorders (Dugas et al., 2001), accumulating evidence brings into question IU's narrow specificity (Garber & Hollon, 1991) to GAD. For example, Boelen and Reijntes (2009) found that IU explained a significant portion of the variance in social anxiety severity. Furthermore, recent evidence has indicated that IU may be an important factor in the etiology and maintenance of major depressive disorder (MDD; Yook, Kim, Suh, & Lee, 2010). MDD

is characterized by rumination, which is hypothesized to serve a similar function to that of worry (i.e., experiential avoidance; McLaughlin, Borkovec, & Sibrava, 2007). Although often past rather than future oriented, rumination involves repeated attempts to better understand the nature of past outcomes (e.g., guilt over things that one should have done, should not have done, or could have done differently) and has been found to mediate the relationship between IU and depressive symptoms (Liao & Wei, 2011). In addition, a recent meta-analysis found strong associations between IU and general symptom levels of GAD, OCD, and MDD (Gentes & Ruscio, 2011), providing further evidence that IU may be a shared feature of emotional disorders.

Given that distress related to experiences of uncertainty has been associated with levels of symptom severity in several emotional disorders, and strategies utilized to avoid uncertainty and increase perceived control are closely tied with diagnostic factors and sequelae that are hypothesized to strengthen and maintain psychopathology, IU may represent an important factor in treatment. In one of the few treatment studies to target IU and examine changes in IU over the course of psychotherapy (Ladouceur et al. 2000; Dugas & Ladouceur 2000) found a significant reduction in IU in a cognitive-behaviorally oriented treatment for principal GAD; similar reductions in IU were observed in a more recent study examining the efficacy of an IU-targeted treatment (Intolerance of Uncertainty Treatment [IUT]; van der Heiden, Muris, & van der Molen, 2011) for GAD. However, with the exception of OCD (see Grayson, 2004, and Wilhelm & Steketee, 2006), IU has yet to be examined in the treatment of many anxiety and mood disorders, or across multiple emotional disorders in the context of a single treatment study. More research is needed to increase understanding of the role of IU in emotional disorders and their treatment (Gentes & Ruscio, 2011). If IU represents a common mechanism of psychopathology in emotional disorders, then changes in this factor should be associated with positive outcomes across different disorders, thus, representing a common change factor.

Transdiagnostic cognitive-behavioral therapy (CBT) treatments are being developed in response to the proliferation of single-diagnosis treatment protocols that may actually hinder the dissemination and adoption of evidence-based psychological treatments (see McHugh & Barlow, 2010), and to address shared features and mechanisms of change in emotional disorders (Norton & Philipp, 2008). Decades of research has shown considerable comorbidity (Brown, Campbell, Lehman, Grisham, & Mancill, 2001) and overlap among anxiety and mood disorders (Barlow, Allen, & Choate, 2004; Moses & Barlow, 2006), and interest in the identification of relevant, underlying factors to aid in conceptualization and treatment has grown (Brown & Barlow, 2009). IU appears to have such transdiagnostic implications (McEvoy & Mahoney, 2011). However, we are unaware of previous research that has examined IU in the context of a single transdiagnostic treatment for heterogeneous disorders.

The Unified Protocol for the Transdiagnostic Treatment of Emotional Disorders (UP; Barlow et al., 2011) is an emotion-focused CBT treatment that distills and incorporates common evidence-based treatment strategies (e.g., restructuring maladaptive appraisals, changing maladaptive action tendencies, prevention of experiential avoidance, exposure), and is designed to be applied to anxiety and unipolar mood disorders, as well as other problem areas with strong emotional components. Conceptually, the UP is particularly well-suited to address a shared feature such as IU, due to the treatment's focus on commonly employed experiential control strategies that have been linked with this dispositional characteristic (e.g., worry, ritualistic behavior, rumination, safety signals), as well as identification of and exposure to situations that are avoided because they may trigger experiences of uncertainty (e.g., situations that may elicit panic sensations).

Specific Aims and Hypotheses

The present study had several aims, with the overall goal being to advance knowledge of IU as a shared feature of emotional disorders and its relationship with posttreatment symptom levels in a transdiagnostic treatment study focused on patients presenting with heterogeneous principal anxiety disorder diagnoses and comorbid depression (Farchione et al., 2012). The first aim was to examine the association between IU scores and disorder specific (GAD, OCD, panic disorder,

social phobia, and depressive disorder) and general measures of symptom severity prior to treatment. We hypothesized that IU scores would be significantly correlated with both disorder specific and general measures of symptom severity prior to treatment. Given that IU appears to be associated with disorders that are also characterized by negative affect, we conducted a second, exploratory analysis within this aim to test whether these relationships (between IU and symptom levels) held when controlling for levels of negative affect. This was important because heightened levels of negative affect could explain most of the variance in IU that is associated with diverse symptomatology, rather than distress regarding uncertainty per se. Thus, if associations between IU and symptom severity hold when controlling for negative affect, this rival hypothesis (e.g., that IU is simply a proxy for distressing negative affect), becomes less plausible.

The second aim was to examine baseline levels of IU across different diagnoses. We pursued this aim in two steps. First, we examined the effect of the presence or absence of specific principal anxiety disorder diagnoses on pretreatment levels of IU. Based on findings from the preponderance of research to-date, we hypothesized that the presence of a principal GAD or OCD diagnosis would have a significant effect on IU (i.e., participants with principal GAD would evidence significantly higher pretreatment IU scores than participants with nonprincipal GAD), while the presence or absence of a different principal anxiety disorder would not have a statistically significant effect on IU. Second, because of the high degree of diagnostic comorbidity in our sample, which could affect interpretation of differences between principal diagnoses if such differences are observed, we explored whether the presence or absence of any specific diagnosis (e.g., any GAD—principal or comorbid, any comorbid depression) differentially related to baseline IU scores.

The third aim was to examine change in IU scores over the course of treatment and in comparison to a waitlist control, and to examine such changes in relation to diagnosis. If IU is most relevant to GAD or OCD, then we might expect a greater amount of change in IU during treatment in individuals with these diagnoses. Alternatively, demonstrating a similar magnitude of change during treatment across different problem areas would provide additional support for the transdiagnostic relevance of IU. To our knowledge, this question has not been addressed empirically within the context of a single treatment. We hypothesized that a significant reduction in IU scores would be observed between pretreatment and posttreatment, while no change in IU would be observed during the waitlist period for patients in a delayed-treatment condition. In addition, we hypothesized that the magnitude of pre-post change in IU would be unrelated to diagnosis.

The fourth aim was to examine associations between change in IU scores and posttreatment symptom levels on the Hamilton Anxiety (Hamilton, 1959) and Hamilton Depression Rating Scales (Hamilton, 1960). We chose to focus on these two commonly used outcome indicators for the sake of statistical parsimony and because they were not designed to be diagnosis-specific. Similar to the implications of aim three, if the relationship between change in IU and posttreatment outcome is not tied to the presence or absence of a specific diagnosis, this would provide additional support for the transdiagnostic relevance of IU in treatment. We hypothesized that change in IU scores over the course of treatment would be correlated with posttreatment symptom levels, regardless of diagnosis.

Method

Data for this study were derived from a randomized control trial (RCT; Farchione et al., 2012) investigating the efficacy of the UP compared to a 16-week waitlist/delayed-treatment condition. Although depressive disorders were represented as comorbid problem areas, this RCT was primarily focused on individuals presenting with diverse principal anxiety disorders. A university-based institutional review board (IRB) approved all measures and procedures in the treatment study prior to its initiation. Patients initially randomized to the waitlist condition subsequently received the same active treatment (UP). No significant differences in initial severity, motivation, or IU scores were observed between the immediate and delayed treatment groups. Furthermore, no posttreatment symptom differences were observed between the immediate and

delayed treatment groups (see Farchione et al., 2012). As such, the sample in the present study included patients from both conditions.

Participants

Participants in the original RCT were recruited from a pool of individuals seeking routine treatment at a large university-based community mental health center in the Northeast United States specializing in the treatment of anxiety disorders. To be eligible for participation, patients had to receive a principal (most interfering and severe) diagnosis of an anxiety disorder, be 18 years or older, be fluent in English, able to complete all study-related tasks, and provide informed consent. Individuals were excluded if they required a higher level of care or if they completed a course of CBT (eight or more sessions) in the past 5 years. This was done to help ensure that treatment effects could be more clearly attributable to the current dose of therapy.

A total of 37 patients consented to treatment and were randomized to either the immediate-treatment ($n = 26$) or delayed-treatment (waitlist, $n = 11$) conditions. The combined sample included 15 males and 22 females, with a mean age of 29.76 years (standard deviation [*SD*] = 9.54, range = 19 to 52 years). The study sample was primarily Caucasian 94.6% ($n = 35$). Principal diagnoses were as follows: GAD ($n = 7$), social phobia ($n = 8$), OCD ($n = 8$), panic disorder with agoraphobia ($n = 8$), anxiety disorder NOS ($n = 2$), and posttraumatic stress disorder (PTSD; $n = 1$). Three participants had a coprincipal anxiety diagnosis (a diagnosis of equal severity): social phobia and anxiety disorder not otherwise specified (NOS), GAD, and social phobia, and OCD and panic disorder with agoraphobia. The majority of the treatment initiator sample was assigned multiple clinical diagnoses ($n = 24$; 69%, mode = 2). Of the 35 patients who fully initiated treatment, $n = 15$ were assigned a clinical diagnosis of GAD (principal or comorbid), $n = 15$ were assigned a clinical diagnosis of social phobia, $n = 12$ were assigned a clinical diagnosis of panic disorder with agoraphobia, $n = 11$ were assigned a clinical diagnosis of OCD, and $n = 12$ were assigned a clinical diagnosis of a depressive disorder (MDD or dysthymia).

A total of 32 patients were labeled treatment completers (operationalized by completing at least eight sessions; see below for further details). No statistical differences in initial severity or demographic variables were observed between treatment completers and noncompleters, and baseline evaluation indicated that no significant differences in the type or severity of psychopathology existed between eligible participants and those excluded from the trial (e.g., refused potential randomization to waitlist/delayed treatment condition; see Farchione et al., 2012, for more detailed information). In addition, there was no difference in baseline Intolerance of Uncertainty Scale (IUS) score between treatment completers and noncompleters, $F(1, 34) = 0.36$, $p = .55$.

Therapists for the study were three doctoral students with 2 to 4 years of clinical experience and one licensed doctoral-level psychologist with 7 years of experience. All therapists underwent extensive training and certification prior to treating study patients, and therapists produced statistically equivalent outcomes across their patients.

Measures

Anxiety Disorders Interview Schedule for the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) Lifetime Version (ADIS-IV-L; Di-Nardo, Brown, & Barlow, 1994). Baseline diagnoses were assessed with the ADIS-IV-L. This semistructured, diagnostic clinical interview focuses on DSM-IV diagnoses of anxiety disorders, mood disorders, and their accompanying mood states, somatoform disorders, and substance and alcohol use. Principal (most interfering and severe) and additional diagnoses are assigned a clinical severity rating (CSR) on a scale from 0 (*no symptoms*) to 8 (*extremely severe symptoms*), with a rating of 4 or above (*definitely disturbing/disabling*) marking the clinical threshold for DSM-IV diagnostic criteria. The condition with the highest rated CSR is considered principal. All independent evaluators in the trial were trained to a high level of reliability and underwent a rigorous certification process (see Brown et al., 2001). The ADIS-IV-L has demonstrated

good (e.g., GAD $\kappa = 0.67$; MDD and dysthymia $\kappa = 0.72$) to excellent (e.g., panic disorder with agoraphobia and social anxiety disorder $\kappa = 0.77$; OCD $\kappa = 0.85$) interrater reliability for the anxiety and mood disorders (Brown et al., 2001). In addition to the training procedures, staff held weekly meetings during which all initial diagnostic interviews were discussed and a consensus diagnosis was reached.

IUS (Freeston et al., 1994). The IUS is a 27-item patient self-report measure that assesses beliefs that (a) uncertainty is stressful and upsetting, (b) uncertainty leads to the inability to act, (c) uncertain events are negative and should be avoided, and (d) being uncertain is unfair (Buhr & Dugas, 2002). Items also focus on emotional and behavioral reactions to ambiguous situations, the consequences of uncertainty, and attempts to control future events. Item responses are based on a 5-point scale (range = 1 – 5). The IUS has excellent internal consistency ($\alpha = .91$; Freeston et al., 1994; *alpha* = .95 in the present sample) and good test-retest reliability ($r = .78$; Dugas et al., 1997).

Positive and Negative Affect Schedule – Negative Affect (PANAS-NA; Watson, Clark, & Tellegen, 1988). Negative affect was measured using the 20-item PANAS. Items describe either positive or negative affect; however, only the negative affect scale was used in this study. Participants are asked to indicate how often they feel a particular way on a 5-point Likert scale. In the validation sample, internal consistency and test-retest reliability were high ($\alpha = .90$; $r = .71$). Additionally, the PANAS-NA was significantly correlated with the Hopkins Symptoms Checklist (Derogatis, Lipman, Ulenhuth, & Covi, 1976), which has been shown to measure general distress ($r = .74$).

Hamilton Anxiety Rating Scale (HARS; Hamilton, 1959). The HARS was used to assess anxiety symptoms and was administered in accordance with the Structured Interview Guide for the Hamilton Anxiety Rating Scale (SIGH-A; Shear, Vander Bilt, & Rucci, 2001). This commonly used measure has demonstrated good levels of interrater and test-retest reliability, as well as convergent validity with similar clinician rated and self-report measures of anxiety symptoms (Shear et al., 2001). Independent clinical evaluators received extensive training on the SIGH-A and had to demonstrate acceptable levels of reliability prior to their participation in the trial.

Hamilton Depression Rating Scale (HDRS; Hamilton, 1960). The HDRS was used to evaluate depressive symptoms and was administered in accordance with the Structured Interview Guide for the Hamilton Depression Rating Scale (SIGH-D; Williams, 1988). This commonly used measure has demonstrated good levels of interrater and test-retest reliability (Williams, 1988), as well as concurrent validity with similar clinician rated and self-report measures of depression symptoms (Bech et al., 1992). Clinical raters received extensive training on the SIGH-D and had to demonstrate acceptable levels of reliability prior to their participation in the trial.

Diagnosis specific measures. Several self-report measures with established reliability and validity were used to assess diagnosis-specific symptoms. GAD symptom severity was assessed using the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990; *alpha* = .93 in the present sample). Social anxiety symptoms were assessed using the Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998; *alpha* = .94 in the present sample). Symptoms related to panic were assessed using the Panic Disorder Severity Scale – Self-Report Version (PDSS-SR; Houck, Spiegel, Shear, & Rucci, 2002) (*alpha* = .91 in the present sample). OCD symptom severity was assessed using the self-report version of the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Baer, 1991; Goodman et al., 1989; *alpha* = .93 in the present sample). The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) was also used as a self-report of depressive symptoms (*alpha* = .88 in the present sample).

Treatment

Treatment with the UP comprised a maximum of 18, 60-minute individual psychotherapy sessions. The UP comprises five core treatment modules designed to target key aspects of emotional processing and regulation of emotional experiences, including decreasing maladaptive avoidance and control processes. The five core modules are preceded by a module focused on enhancing motivation, readiness for change, and treatment engagement, as well as an introductory module educating patients on the nature of emotions and providing a framework for understanding their emotional experiences. A final module comprises reviewing progress over treatment and developing relapse prevention strategies.

The UP modules are flexibly linked to sessions in that depending on the needs of the individual, more or less time can be spent on a given module. Each module could conceivably be covered in a single session, which would result in a treatment that is less than 18 weeks in duration. A participant was considered a treatment completer after eight sessions because all treatment modules could have been covered in this duration. For the overall sample, the mean number of sessions completed was 15.26 ($SD = 4.60$). The average number of sessions in the trial for individuals with a principal diagnosis of GAD was 14.88 sessions ($SD = 6.36$), social phobia = 15.67 sessions ($SD = 3.14$), panic disorder = 15.00 sessions ($SD = 2.71$), OCD = 17.88 sessions ($SD = 0.35$), and other = 15.60 sessions (2.88). All treatment completers received all treatment modules. Ongoing adherence ratings were not conducted in this trial. All study cases were closely supervised by a senior treatment developer. Adherence was monitored during weekly supervision and by reading required session notes. A sample of treatment sessions were recorded and reviewed as part of this supervision process.

Procedure

As noted, patients were randomized to two conditions, immediate or delayed treatment. Participants assigned to the immediate UP condition were assessed at pretreatment and posttreatment. Delayed treatment participants were assessed at the beginning and end of the 16-week waitlist period. Following the post-waitlist assessment, patients were assigned to the active treatment protocol. Initiation of therapy with the UP for the waitlist/delayed treatment participants began immediately after the post-waitlist assessment at Week 16, with an average of 2 weeks between the post-waitlist assessment and the first UP session. An additional assessment was then conducted at posttreatment. The IUS, PANAS, and symptom measures were completed by patients at their baseline assessment and at posttreatment. For the delayed treatment group, the baseline assessment was post-waitlist, immediately prior to the onset of treatment.

Results

Baseline IU and Pretreatment Symptom Severity

Descriptive statistics for the IUS and symptom measures are reported in Table 1. A series of zero-order correlations were conducted to examine the associations between baseline IU (sample mean = 40.35, $SD = 22.29$) and initial symptom levels across disorder-specific (GAD, OCD, social phobia, panic disorder, depression) and general symptom measures (see Table 2). Baseline IUS scores were significantly correlated with higher pretreatment HARS and HDRS scores. However, the PSWQ and BDI-II were the only diagnosis-specific self-report symptom measures that correlated significantly with IUS. A second series of partial correlations were then conducted specifically with IUS, this time controlling for negative affectivity. Results generally remained consistent, yet the correlation between baseline IUS and the HARS was no longer significant ($p = .06$).

Baseline IU and Diagnosis

A univariate analysis of variance (ANOVA) was conducted to investigate the effect of the presence or absence of principal diagnoses on baseline IUS scores. The presence or absence of

Table 1
Descriptive Statistics for Intolerance of Uncertainty Scale and Symptom Measures

	Baseline			Posttreatment		
Measure	n	Mean (*SD*)	Range	n	Mean (*SD*)	Range
IUS	36	40.35 (22.29)	2.00–85.00	29	27.07 (19.04)	2.00–88.00
HARS	35	15.34 (7.44)	3.00–34.00	29	7.97 (6.17)	0.00–24.00
HDRS	35	9.74 (5.61)	1.00–21.00	29	6.07 (5.71)	0.00–21.00
BAI	35	17.46 (10.04)	0.00–46.00	30	7.47 (6.65)	0.00–24.00
BDI-II	35	11.76 (8.53)	0.00–29.00	29	4.79 (5.72)	0.00–25.00
PSWQ	35	61.34 (12.65)	36.00–78.00	29	49.48 (13.67)	26.00–80.00
PDSS-SR	35	7.46 (5.97)	0.00–21.00	29	2.66 (3.13)	0.00–10.00
SIAS	35	29.94 (18.47)	4.00–70.00	29	23.17 (16.72)	4.00–72.00
YBOCS	35	8.73 (9.29)	0.00–28.00	29	6.14 (7.69)	0.00–23.00

Note. SD = standard deviation; IUS = Intolerance of Uncertainty Scale; HARS = Hamilton Anxiety Rating Scale; HDRS = Hamilton Depression Rating Scale; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; PDSS-SR = Panic Disorder Severity Scale-Self Report; SIAS = Social Inhibition and Anxiety Scale; YBOCS – Yale-Brown Obsessive-Compulsive Scale.

Table 2
Zero Order (Partial) Correlations Between Baseline Intolerance of Uncertainty Scale Scores and Symptom Levels

		1.IUS	2.	3.	4.	5.	6.	7.	8.
1.	IUS	–							
2.	BDI-II	.55** (.49**)	–						
3.	HARS	.38* (.32)	.62**	–					
4.	HDRS	.43* (.35)*	.75**	.88**	–				
5.	PSWQ	.49** (.39*)	.36*	.28	.41*	–			
6.	SIAS	.30 (.25)	.18	.09	.08	.22	–		
7.	PDSS-SR	.13 (.13)	.27	.56**	.40*	−.26	.23	–	
8.	YBOCS	.05 (−.02)	.04	−.10	−.04	.24	−.03	−.14	–

Note. IUS = Intolerance of Uncertainty Scale; BDI = Beck Depression Inventory-II; BAI = Beck Anxiety Inventory; HARS = Hamilton Anxiety Rating Scale; HDRS = Hamilton Depression Rating Scale; PSWQ = Penn State Worry Questionnaire; SIAS = Social Inhibition and Anxiety Scale; PDSS = Panic Disorder Severity Scale; YBOCS – Yale-Brown Obsessive-Compulsive Scale.
Correlations in parentheses are partial correlations when controlling for negative affectivity with the PANAS-NA.
* $p < .05$. ** $p < .01$. $n = 35$.

each diagnosis was coded as a 0 or 1 for each participant, for the following principal diagnoses represented in the sample: GAD, OCD, social phobia, panic disorder, and "other" (anxiety disorder NOS, PTSD, and coprincipal disorders). PTSD was included in the "other" group because only one participant presented with this as a principal diagnosis. Contrary to our hypothesis, univariate ANOVA results indicated that the presence or absence of a particular principal diagnosis did not have an effect on baseline IUS scores, univariate $F(4, 35) = 1.11$, $p = .37$, $\eta_{p2} = .13$ (see top half of Table 3).

Because of the high degree of comorbidity in the sample, a second univariate ANOVA was conducted to investigate the effect of the presence of absence of *any* (principal or comorbid) clinical diagnosis on baseline IUS scores. Once again, the presence or absence of each diagnosis was coded as a 0 or 1 for each participant, for the following disorders represented in the sample: GAD, OCD, social phobia, panic disorder, depressive disorder, and "other" (anxiety disorder NOS, PTSD, and specific phobia). Univariate ANOVA results indicated that the presence or

Table 3
Univariate ANOVAs Testing Effect of Diagnosis on Baseline Intolerance of Uncertainty Scores

Principal diagnosis	Not present Mean (*SE*)	Present Mean (*SE*)	*F*	*p*	η_{p2}
GAD (n = 7)	37.42 (4.26)	49.50 (7.83)	2.24	.15	.07
SOC (n = 8)	41.40 (4.26)	33.57 (7.83)	0.06	.82	.01
PDA (n = 8)	37.51 (4.20)	49.14 (8.37)	2.05	.16	.06
OCD (n = 8)	40.70 (4.26)	36.38 (7.83)	0.21	.65	.01
Other (n = 6)[a]	30.60 (4.02)	41.92 (9.50)	0.21	.65	.01
Any diagnosis	**Not present Mean (*SE*)**	**Present Mean (*SE*)**	***F***	***p***	η_{p2}
GAD (n = 15)	31.71 (6.90)	48.68 (7.33)	1.90	.18	.07
SOC (n = 15)	41.59 (7.00)	38.80 (7.24)	0.12	.74	.01
PDA (n = 12)	38.72 (6.24)	42.27 (8.35)	0.01	.94	.00
OCD (n = 11)	39.27 (6.08)	41.49 (8.57)	0.02	.88	.01
DEP (n = 12)	35.14 (4.65)	46.50 (8.83)	1.30	.26	.04
Other (n = 11)[b]	45.61 (4.84)	36.03 (8.73)	0.92	.34	.03

Note. ANOVA = analysis of variance; SE = standard error; GAD = generalized anxiety disorder; SOC = social phobia; PDA = panic disorder with agoraphobia; OCD = obsessive compulsive disorder; DEP = major depressive disorder, dysthymic disorder, or depressive disorder not otherwise specified (NOS).
[a]Other = PTSD, anxiety disorder NOS, and individuals with co-principal diagnoses.
[b]Other = PTSD, anxiety disorder NOS, specific phobia, and individuals with co-principal diagnoses.

absence of any particular diagnosis did not have an effect on baseline IUS scores, univariate F (5, 35) = 0.57, $p = .83$, $\eta_{p2} = .31$ (see bottom half of Table 3).

Changes in IU During Treatment

Consistent with our hypothesis, results from a t test demonstrated a significant decrease in IUS scores during UP treatment, t (28) = 3.49, $p < .01$, $d = 0.73$, confidence interval [CI] = 0.22: 1.23, with a medium-to-large effect size between pretreatment (mean [M] = 41.92, $SD = 21.57$) and posttreatment ($M = 27.07$, $SD = 19.04$), across diagnoses. Conversely, a nonsignificant difference in IUS scores was observed in the delayed-treatment condition, t (9) = −0.43, $p = .68$, $d = -0.05$, CI = −0.98: 0.87, with a small, negative effect size between pretreatment ($M = 38.56$, $SD = 23.69$) and post-waitlist ($M = 40.11$, $SD = 30.09$) scores, across diagnoses. Utilizing Jacobson and Truax's (1991) formula for calculating a reliable change index (RCI), the mean pre-post treatment difference exceeded the RCI criterion (13.82 IUS points). A total of 14 out of 29 (48.3%) treatment completers with pre-IUS and post-IUS scores evidenced a reliable decrease in IUS scores. No patients in the delayed-treatment condition evidenced reliable change in IUS from baseline to postwaitlist. A patient's statistical likelihood of exhibiting reliable change on the IUS was unrelated to principal diagnosis, χ^2 (4) = 6.84, $p = .15$, yet, descriptively, more people with principal panic disorder demonstrated reliable change than any other principal diagnosis (see Table 4).

Using the same approach as the analysis comparing baseline IU levels (presence or absence of each diagnosis coded as a 0 or 1 for each participant), consistent with our hypothesis, results from a univariate ANOVA showed no statistical difference in the magnitude of pre-post treatment change in IUS scores among principal diagnoses, univariate F (4, 28) = 1.83, $p = 16$, $\eta_{p2} = .23$. Additionally, the presence or absence of any specific principal or comorbid diagnosis was unrelated to magnitude of change in IU, univariate F (5, 28) = 1.11, $p = .43$, $\eta_{p2} = .60$.

Table 4
Number of Patients Demonstrating Reliable Change on the Intolerance of Uncertainty Scale by Principle Diagnosis

	IUS Pre *M (SD)*	IUS Post *M (SD)*	# Met RCI cutoff	# Did not meet RCI cutoff
Principal diagnosis				
GAD	50.50 (11.34)	31.50 (16.08)	3	3
SOC	40.71 (21.27)	39.60 (28.47)	2	3
PDA	53.50 (19.65)	20.83 (15.28)	5	1
OCD	36.38 (26.44)	24.25 (18.59)	4	4
Other	24.25 (18.24)	19.75 (14.27)	0	4

Note. RCI = reliable change index; IUS = Intolerance of Uncertainty Scale; SD = standard deviation; GAD = generalized anxiety disorder; SOC = social anxiety disorder; PDA = panic with agoraphobia; OCD = obsessive-compulsive disorder; other = anxiety disorder NOS and co-principals.
Includes participants ($n = 29$) with pre (IUS Pre) and posttreatment (IUS post) data. RCI = 13.82.

Change in IU and Posttreatment Symptom Severity

Two multiple regression models were then tested to see if pre-post change in IU was related to posttreatment symptom levels on the HARS and HDRS. Pretreatment symptom scores were included in each model and entered simultaneously with pre-post change in IUS scores. Results showed that change in IU was a significant predictor of posttreatment symptom severity on the HARS ($\beta = .60$, SE = .05, $t = 3.53$, $p < .01$, CI = 0.07:0.25, $pr = .57$) and HDRS ($\beta = .58$, SE = .04, $t = 3.67$, $p < .01$, CI = 0.06:0.22, $pr = .59$), such that greater reductions in IU were associated with reduced anxiety and depression symptom severity. We then tested two additional sets of multiple regression models, this time including presence or absence of any specific (principal or comorbid) diagnosis as a potential moderator of the relationship between change in IU and outcome. Thus, the only difference in these models was the addition of an interaction term (presence or absence of GAD, panic disorder, OCD, social phobia, depressive disorder, or "other" [anxiety disorder NOS, PTSD, or specific phobia] by change in IUS). IUS change remained significant in each model, yet none of the diagnosis by IUS change interaction terms was significantly associated with posttreatment severity on either the HARS or the HDRS (see Table 5). This indicated that the relationship between change in IU and posttreatment symptom severity was not influenced by the presence or absence of a specific diagnosis.

Discussion

Although IU has been most commonly associated (both conceptually and empirically) with GAD and OCD in particular (Dugas et al., 2001; Wilhelm & Steketee, 2006), and anxiety disorders generally (Dugas, Schwartz, & Francis, 2004), emerging evidence suggests that IU may be a shared factor (or general correlate) of emotional disorders (Gentes & Ruscio, 2011; McEvoy & Mahoney, 2011). The present study was aimed at investigating IU, measured with the IUS, in the context of a transdiagnostic treatment for emotional disorders. Baseline IUS scores were associated with higher baseline scores on the HDRS, PSWQ, and BDI-II when controlling for levels of negative affect. Patients with a principal diagnosis of GAD or panic disorder demonstrated the highest IUS scores. However, the presence or absence of any specific diagnosis (principal or comorbid) was not statistically associated with IUS scores. Across diagnoses, patients demonstrated a significant, reliable decrease in IU between pretreatment and posttreatment, while no change in IU was observed during the 16-week waitlist period. Similar magnitudes of change in IU were observed across diagnoses (principal and comorbid). When controlling for initial severity, change in IU was a significant predictor of posttreatment symptom severity on the

Table 5
Results From Multiple Regression Models Predicting Posttreatment Outcome

	HARS					HDRS				
	β	*SE*	*t*	95% CI	Partial *r*	β	*SE*	*t*	95% CI	Partial *r*
GAD			(HARS, $R^2 = .34$)					(HDRS, $R^2 = .41$)		
Pretreatment severity	.32	.14	1.82	−.03 : .55	.34	.46	.17	2.83**	.13 : .81	.49
IUS change	.62	.05	3.24**	.06 : .27	.54	.54	.05	3.01**	.43 : .23	.52
IUS*GAD	−.06	.08	−0.32	−.19 : .14	−.06	.07	.07	0.39	−.12 : .17	.08
OCD			(HARS, $R^2 = .35$)					(HDRS, $R^2 = .40$)		
Pretreatment severity	.36	.14	2.04*	−.01 : .59	.33	.46	.17	2.78**	.12 : .82	.43
IUS change	.68	.06	3.28**	.07 : .30	.53	.60	.05	3.12**	.05 : .25	.48
IUS*OCD	−.14	.08	−0.72	−.22 : .11	−.12	−.05	.07	0.25	−.16 : .13	−.04
SOC			(HARS, $R^2 = .34$)					(HDRS, $R^2 = .41$)		
Pretreatment severity	.32	.14	1.87	−.03 : .55	.30	.45	.16	2.81**	.12 : .79	.43
IUS change	.54	.06	2.59*	.03 : .26	.42	.50	.05	2.60*	.03 : .23	.40
IUS*SOC	.08	.08	−0.42	−.13 : .19	.07	.12	.07	0.63	−.10 : .18	.10
PDA			(HARS, $R^2 = .34$)					(HDRS, $R^2 = .42$)		
Pretreatment severity	.33	.14	1.90	−.02 : .57	.31	.48	.17	2.95**	.15 : .83	.45
IUS change	.57	.06	2.68*	.04 : .27	.44	.48	.05	2.40*	.02 : .22	.37
IUS*PDA	.04	.08	0.20	−.14 : .17	.03	.17	.07	0.82	−.08 : .19	.13
DEP			(HARS, $R^2 = .35$)					(HDRS, $R^2 = .47$)		
Pretreatment severity	.39	.16	2.01	−.01 : .64	.32	.58	.17	3.49**	.24 : .95	.51
IUS change	.53	.05	2.67*	.03 : .25	.43	.40	.04	2.24*	.01 : .19	.33
IUS*DEP	.15	.08	0.67	−.12 : .23	.11	.36	.07	0.08	−.02 : .27	.27
Other			(HARS, $R^2 = .34$)					(HDRS, $R^2 = .41$)		
Pretreatment severity	.33	.14	1.90	−.02 : .55	.36	.44	.16	2.78*	.12 : .79	.49
IUS change	.59	.05	3.44**	.06 : .26	.57	.58	.04	3.63**	.06 : .23	.59
IUS*other	−0.02	.47	−0.14	−1.03 : .90	−.03	.06	.41	0.36	−.70 : .99	.07

Note. HARS = Hamilton Anxiety Rating Scale; CI = confidence interval; HDRS = Hamilton Depression Rating Scale; IUS = Intolerance of Uncertainty Scale; GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder; SOC = social phobia; PDA = panic disorder with agoraphobia; DEP = depressive disorder; other = anxiety disorder not otherwise specified and posttraumatic stress disorder.
* $p < .05$. ** $p < .01$.

HARS and HDRS, and this relationship was not influenced by the presence of any particular diagnosis.

The significant correlation between baseline IU and baseline worry scores is consistent with previous research findings (e.g., Buhr & Dugas, 2002), yet other findings were less consistent or novel. For example, the lack of significant associations between baseline IUS scores and other disorder-specific symptom measures, with the exception of depression, is inconsistent with some recent findings (e.g., McEvoy & Mahoney, 2011). The lack of association with OCD symptoms was somewhat surprising, given results from previous studies (Gentes & Ruscio, 2011; Steketee et al., 1998). The small sample size of the present study is one potential explanation; however, regardless of statistical significance, the observed magnitude of association was quite small ($r = .05$). Another potential explanation could be that the Y-BOCS items do not relate as strongly to the IU construct as alternative measures of OCD symptomology (e.g., the Obsessive-Compulsive Inventory-Revised [OCI-R]; Abramowitz, & Deacon, 2006), that have also been used in previous research (see Gentes and Ruscio, 2011). Conversely, it has been suggested that the significant

associations observed between worry severity (as measured by the PSWQ, for example) and IU may be partially explained by the IU measure. The IUS was initially created to assess this hypothesized construct in GAD; thus, the scale may be particularly sensitive to worry-related phenomena (Gentes & Ruscio, 2011).

Perhaps most unique to the present study was the finding that patients with a principal diagnosis of panic disorder exhibited comparably high IUS scores to those with a principal diagnosis of GAD, and were more likely to demonstrate reliable change in IUS scores. Panic disorder has not typically been associated with IU in the literature, yet it is recognized that patients with panic disorder spend a great deal of energy planning ahead to avoid potential panic and using safety signals to provide a sense of control over their feared physical sensations (Barlow, 2002; Rachman, 1984). Despite these behaviors, patients with panic disorder are not always able to predict whether or when they will face a situation that will cause their uncomfortable physical feelings to increase (McNally, 2002). Research has shown that uncertainty itself results in increased physiological arousal (Greco & Roger, 2003), so patients may notice stronger physical feelings when they are in this state. Individuals with panic disorder may also react more strongly to the physiological component of the emotional response to uncertainty, perhaps before reacting cognitively or behaviorally. It is, therefore, not surprising that the patients with panic disorder in our sample endorsed similarly high levels of IU.

When taken as a whole, this study's results are consistent with emerging evidence questioning the "narrow" specificity of IU to GAD (see Garber & Hollon, 1991). Although levels of IU may be more highly correlated with worry severity, and to some extent GAD, it does not appear to be pathognomonic of GAD. Baseline IU correlated significantly with clinician-rated and patient self-report measures of both anxiety and depressive symptomology, levels of IU were comparably high in individuals with principal panic disorder, and the presence or absence of GAD did not have a significant effect on baseline IUS scores or the degree of change in IU observed between pretreatment and posttreatment. The significant association between IU and depressive symptoms, including IU's association with change in depressive symptoms, supports the contention that IU is a potentially important factor in the etiology, maintenance, and treatment of depression. High levels of comorbidity observed between GAD and MDD (Simon, 2009) may be partially explained by IU and its related cognitive avoidance processes: worry and rumination.

Although emerging evidence questions the narrow specificity of IU to GAD, stronger associations with worry and GAD have been found in previous research (e.g., Dugas et al., 1997; Freeston et al., 1994). It is important to consider what might account for IU's stronger associations, yet lack of specificity. As noted, one potential explanation is a measure-effect–IUS items may be geared toward worry processes and GAD. However, IU may also have particular relevance to GAD because it represents a "slice" of a broader construct, such as *perceived control* (Barlow, 2002).

Dimensions of perceived control include whether or not the object of control is located in the past or the future, and whether or not control is related to an outcome or a specific behavior or experience (internal or external; Wallston, Strudler, Wallston, Smith, & Dobbins, 1987). Objects of control that are future and outcome oriented are more likely to elicit experiences of uncertainty (Chorpita & Barlow, 1998), which has been linked to psychopathology (Barlow, 2002; Dugas et al., 2004). Lack of perceived control may represent a generalized psychological vulnerability to develop emotional disorders (Chorpita, Brown, & Barlow, 1998), as well as influence their progression and maintenance (Barlow, 2002; Brown, White, Forsyth, & Barlow, 2004). IU may capture a manifestation of this broader construct that is particularly relevant to GAD, given that the inability to tolerate uncertainty regarding future outcomes is part and parcel of worry. If this is indeed the case, then IU should have some level of specificity to GAD, yet also evidence elevated levels in other emotional disorders where perceived control is a relevant underlying factor. Accumulating evidence appears to support this, and further consideration of the construct of perceived control may help to integrate and consolidate findings related to IU's concomitant lack of narrow specificity to GAD and strong relationship with measures of worry.

This study also builds on the work of Ladoucer et al. (2000) and van der Heiden et al. (2011), by demonstrating significant reductions in IU over the course of treatment. Unlike

the treatments examined in these studies (e.g., IUT), the UP is not exclusively focused on GAD or IU; rather, the target is emotion regulation and avoidance/control processes more broadly. The significant and reliable reduction in IU observed across diagnostic categories indicates that the UP may effectively affect control processes as an important element underlying emotional disorders. Change in IU was significantly related to posttreatment symptom levels across diagnoses, demonstrating a potential transdiagnostic change factor. Furthermore, this study may be the first to demonstrate that change in IU over the course of treatment not only affects symptoms of anxiety, but depressive symptoms as well.

Study limitations include the absence of individuals with principal depressive disorders, the lack of PTSD representation, the small sample size, and the high degree of ethnic homogeneity. Each of these factors may affect generalizability. Although the principal diagnosis models were underpowered, interpretation of the results was enhanced by the inclusion of effect sizes. Generalizability of these results is also limited to a primarily anxious population that did not require a higher level of care. Although more research is certainly needed, it is not known at this time or from this sample whether such a population with more severe pathology (including comorbid personality disorders) would be responsive to a short-term treatment such as the UP. In addition, the timing of observations precluded the testing of specific mediation hypotheses (e.g., worry or rumination as a mediator of the relationship between IU and outcome) within and between diagnostic categories, or examining relationships between early symptom change and change in IU. Consequently, causal relationships regarding the direct effects of the UP on IU or IU on symptom change cannot be determined, and future studies in this area should implement designs that allow for direct tests of mediation hypotheses.

Future research should continue to investigate IU's role in etiology, maintenance, and treatment across the scope of emotional disorders, including principal depressive disorders and PTSD. For example, increasing our understanding of the common, underlying information processing biases that emerge in the face of uncertainty (see Eysenck, 1997) could lead to improved transdiagnostic interventions. Experimental paradigms that measure behavioral responses, physiological reactivity, and cognitive biases during uncertainty inductions or exposures could be integrated into treatment studies; thus, increasing clinical utility. These results would also have implications for understanding IU in light of potentially related transdiagnostic constructs, such as perceived control. IU may represent a specific "slice" of distress related to perceptions of lack of control. Therefore, future research should investigate IU's relationship to the construct of perceived control, including more directly testing explanatory models (e.g., Chorpita & Barlow, 1998; Barlow, 2002), which would likely require longitudinal designs.

Clinically, although treatments have been developed to narrowly target IU, particularly in individuals with GAD, these results indicate that broader transdiagnostic treatments, such as the UP, may effectively and efficiently affect IU and its various manifestations across the spectrum of anxiety disorders. Regardless of the particular approach, clinicians might do well to attend to patients who present with distress related to uncertainty, as changes in this area appear to be related to treatment outcome. Future research in this area may also assist in tailoring such a focus to various manifestations of IU (e.g., appraisals of panic symptoms in panic disorder, social cost in social anxiety).

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