A meta-analysis of the relation of intolerance of uncertainty to symptoms of generalized anxiety disorder, major depressive disorder, and obsessive–compulsive disorder

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ABSTRACT

Intolerance of uncertainty (IU) has been suggested to reflect a specific risk factor for generalized anxiety disorder (GAD), but there have been no systematic attempts to evaluate the specificity of IU to GAD. This meta-analysis examined the cross-sectional association of IU with symptoms of GAD, major depressive disorder (MDD), and obsessive–compulsive disorder (OCD). Random effects analyses were conducted for two common definitions of IU, one that has predominated in studies of GAD (56 effect sizes) and another that has been favored in studies of OCD (29 effect sizes). Using the definition of IU developed for GAD, IU shared a mean correlation of .57 with GAD, .53 with MDD, and .50 with OCD. Using the alternate definition developed for OCD, IU shared a mean correlation of .46 with MDD and .42 with OCD, with no studies available for GAD. Post-hoc significance tests revealed that IU was more strongly related to GAD than to OCD when the GAD-specific definition of IU was used. No other differences were found in the magnitude of associations between IU and the three syndromes. We discuss implications of these findings for models of shared and specific features of emotional disorders and for future research efforts.

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Several early psychological theories proposed that the experience of uncertainty and the tendency to avoid uncertain states may play a central role in the development and maintenance of anxiety and mood psychopathology (Hammen & Cochran, 1981; McFall &
Wollersheim, 1979). Current theories continue to highlight the relationship between uncertainty and psychopathology (Dugas, Freeston, & Ladouceur, 1997; Steketee et al., 1997) but many now distinguish between the state experience of uncertainty and an individual difference that has been termed intolerance of uncertainty (IU).

In 1995, the Obsessive Compulsive Cognitions Working Group formed to identify domains of beliefs that contribute to the development or maintenance of obsessive-compulsive disorder (OCD). Intolerance of uncertainty, defined as “the belief that uncertainty, newness, and change are intolerable because they are potentially dangerous” (Steketee et al., 1997, p. 669), was one of six beliefs identified as central to the disorder. Studies in the OCD literature have most commonly assessed IU using the 87-item or the 44-item Obsessive Beliefs Questionnaire (OBQ) developed by the working group, which include subscales for IU and IU/perfectionism, respectively (OCCWG, 2003; 2005).

While the Obsessive Compulsive Cognitions Working Group was beginning this work of identifying OCD-relevant cognitions, a separate group of investigators identified IU as a key process variable in generalized anxiety disorder (GAD; Dugas, Ladouceur, Boisvert; & Freeston, 1996; Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994; Ladouceur, Talbot, & Dugas, 1997). Definitions of IU within the GAD literature have evolved slightly over the past 13 years, with IU most recently 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. 218). GAD investigators have most commonly assessed IU using the 27-item Intolerance of Uncertainty Scale (IUS; Buhr & Dugas, 2002; Freeston et al., 1994) developed by this group of researchers.

Although these two groups were the first to define IU in relation to psychopathology, IU shares a number of similarities with the earlier concept of intolerance of ambiguity (IA). Introduced by Frenkel-Brunswik, 1949, IA describes a tendency to interpret ambiguous situations as a threat or a source of discomfort and to react to such situations with rigidity, anxiety, and avoidance (Grenier, Barrette, & Ladouceur, 2005). Correlations between measures of IA and IU have been reported to range from .42 (Buhr & Dugas, 2006) to .50 (Dugas et al., 2005). In a recent review, Grenier et al. (2005) discussed the relation of IU to IA. They noted that while both traits are characterized by discomfort in the absence of certainty and clarity, IU refers explicitly to uncertain future events, while IA refers only to ambiguity in the present. Perhaps because of this difference in temporal focus, IA has been studied primarily in management, industrial–organizational, and accounting settings (Grenier et al., 2005). Only two papers have examined IA in relation to anxiety or mood symptoms, so IA is not considered further here.

IU, in contrast, has been studied extensively in relation to emotional disorders, particularly the anxiety disorders (Grenier et al., 2005). Etiological theories suggest several mechanisms through which IU may relate to symptoms of OCD and GAD. First, core features of these disorders have been linked to efforts to attain certainty or to reduce the anxiety associated with uncertain outcomes. In OCD, rituals and compulsions are hypothesized to serve the function of reducing the distress resulting from uncertainty about a potential feared outcome (Steketee, Frost, & Cohen, 1998; Tolin, Abramowitz, Brigidi, & Foa, 2003). Similarly, individuals with GAD have been hypothesized to engage in worry as an attempt to control feelings of uncertainty and anxiety about future events (Freeston et al., 1994). Second, high IU is thought to lead to over-identification of potential problems and to a negative problem orientation (Freeston et al., 1994), both of which have been associated with GAD (Dugas et al., 2007; Rusco & Seitchik, 2007). Although IU has primarily been studied in relation to anxiety disorders, recent theoretical accounts suggest that IU may lead to major depressive disorder (MDD) through pathways similar to those proposed for GAD. Over-identification of problems and negative problem orientation associated with IU have been hypothesized to result in depressive as well as anxiety symptoms (Yook, Kim, Suh, & Lee, 2010). Furthermore, persons who experience discomfort with uncertainty may prefer to live with pessimistic certainty about future events (Dupuy & Ladouceur, 2008; Yook et al., 2010) which may predispose them to depression.

Theories linking IU to multiple emotional disorders suggest that IU may constitute a shared feature for these disorders. Consistent with this notion, several studies have found IU to be significantly related to symptoms of GAD, MDD, and OCD (Clark, 2002; Dugas et al., 2007; Freeston et al., 1994; Steketee et al., 1998; Tolin et al., 2003). Other studies, in contrast, have revealed a more specific relationship of IU to GAD, in line with proposals that IU contributes to the unique development and clinical presentation of this disorder. These apparently discrepant findings may be reconciled by considering that specificity may be defined broadly or narrowly in etiological models (Garber & Hollon, 1991). Broad specificity asks whether a model is specific to a given disorder relative to its general higher-order set (e.g., Does IU distinguish GAD patients from a heterogeneous group of anxious patients?) while narrow specificity asks whether a model is specific to a given disorder relative to each other disorder belonging to the same higher-order set (e.g., Does IU distinguish GAD patients from those with OCD, or those with MDD?). Several papers have concluded that IU demonstrates broad specificity to GAD compared to other anxiety disorders (Ladouceur et al., 1999) and that it is more strongly related to worry than to obsessions and compulsions, depression, and panic sensations (Dugas, Gosselin, & Ladouceur, 2001; Dugas, Schwartz, & Francis, 2004). At the same time, a substantial body of research across separate literatures shows strong and significant associations of IU with symptoms of OCD and MDD (e.g., Buhr & Dugas, 2002; Crittendon & Hopko, 2006; OCCWG, 2003; Boelen & Reijntjes, 2009; Fergus & Wu, 2010), hinting that IU may not demonstrate narrow specificity to GAD.

A more systematic investigation of the specificity of IU-disorder relations has been complicated by the fact that independent groups of researchers use different measures to assess IU. Further, the most commonly used measures (the IUS and the OBQ) were created specifically to assess IU in relation to a given disorder (GAD and OCD, respectively). Thus, the IUS may always be more strongly related to symptoms of GAD while the OBQ may always be more strongly related to symptoms of OCD, simply because they were originally designed by GAD or OCD researchers to tap into key features of these disorders. Although the two measures are correlated, they are not redundant (r = .59; Fergus & Wu, 2010). The IUS 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). In comparison, the OBQ assesses beliefs that (a) certainty is necessary, (b) unpredictable change cannot be coped with, and (c) adequate functioning is difficult in inherently ambiguous situations (Steketee et al., 1997).

Given the varied definitions and measures of IU, it is not surprising that the literature is characterized by conflicting findings. However, at a time when transdiagnostic approaches (e.g., Harvey, Watkins, Mansell, & Shafran, 2004) and the preparation of DSM-5 are encouraging examination of shared and specific features across disorders (e.g., Brown & Barlow, 2009), these conflicting findings hamper efforts to understand the role of IU in GAD, MDD, and OCD. Increased knowledge of IU as a shared versus specific feature of these disorders may have several benefits. First, it may provide insight into issues of comorbidity, classification, and etiology. For instance, if IU is shared by these disorders, it may help explain their common features and frequent comorbidity (Grant et al., 2005). In contrast, if IU is specific to one or a subset of these disorders, it may help account for
the unique clinical presentation of each disorder despite shared features, or explain why a given vulnerable individual develops one disorder versus another. Second, a central aim of transdiagnostic models is to facilitate the transfer of treatment advances between disorders that are typically studied in isolation, and to aid in the development of fewer, more parsimonious treatments for multiple disorders. Some clinicians have already begun to target IU as a point of intervention for multiple anxiety disorders, including GAD and OCD (e.g., Grayson, 2004; Ladouceur et al., 2000). Increased knowledge of the relationship of IU to GAD and OCD, as well as to MDD, may encourage further development and utilization of transdiagnostic interventions (if shared), or help researchers and clinicians tailor interventions to the features most relevant to each disorder (if specific).

The current paper sought to advance understanding of IU through a quantitative review of its relation to symptoms of GAD, MDD, and OCD. The paper had three objectives. The first objective was to estimate the mean association of IU with each syndrome, both as it is observed in the literature and after correcting for attenuation due to unreliability of measurement. This association was estimated using two definitions of IU: a GAD-specific definition operationalized by scores on the IUS, and an OCD-specific definition operationalized by scores on the OBQ. Independent examination of the IUS and the OBQ was a priority because these measures are the most commonly used in the GAD and OCD literatures, respectively, and it was hypothesized that the relationship of IU to each syndrome may vary systematically by measure. The second objective was to examine differences in the magnitude of the relationship of IU to each syndrome. The third objective was to test whether, across syndromes, the association of IU with symptoms is moderated by IU definition (GAD-specific versus OCD-specific) or by the population sampled (student versus treatment-seeking).

1. Method

1.1. Literature search

Relevant studies were identified via PsycINFO searches through August 2010 using combinations of the following keywords: uncertainty, intolerance of ambiguity, tolerance for ambiguity, tolerance of ambiguity WITH generalized anxiety disorder, generalised anxiety disorder, GAD, worry, depression, major depressive disorder, rumination, obsessive compulsive disorder, obsessive-compulsive disorder, OCD, and obsession. Searches were limited to studies that were written in English, sampled from adult populations (age 18 years and older), and published in peer-reviewed journals or edited book chapters. Unpublished dissertations were also included to reduce publication bias. This initial search strategy yielded 731 abstracts, which were reviewed for relevance.

After relevant self-report measures were identified from eligible papers, additional PsycINFO searches were conducted combining the disorder- and symptom-related keywords listed above WITH self-report measures of IU and IA, including: Intolerance of Uncertainty Scale, IUS, Obsessional Beliefs Scale, Obsessive Beliefs Questionnaire, OBQ, Obsessive Compulsive Disorder Cognitive Schemata Scale, Obsessive Compulsive Beliefs Questionnaire, Obsessional Beliefs Questionnaire, Need for Cognitive Closure Scale, Tridimensional Personality Scale, Personal Need for Structure, Temperament Character Inventory, Typical Interpretation of Thoughts, Irrational Beliefs Regarding Obsessions, Responsibility Questionnaire, Responsibility Scale, Intolerance of Ambiguity Scale, Walk's A, Ambiguity Tolerance, AT-20, Measure of Ambiguity Tolerance, Situational Test of Intolerance of Ambiguity, Scale of Tolerance–Intolerance of Ambiguity, TIA, and Kischkel Scale. This search resulted in an additional 254 abstracts for review.

Finally, the reference sections of relevant studies were reviewed to identify additional studies that might meet inclusion criteria, and unpublished data were requested via email from 14 researchers who had published extensively (i.e., >3 publications) on IU. Raw datasets were obtained from three of these researchers and an additional six replied that they did not have any unpublished data.

1.2. Selection of studies

All papers that mentioned empirical data on IU or IA and GAD, MDD, or OCD in the abstract were obtained for further review (n = 159). Papers were considered for inclusion if they (a) assessed IU or IA, (b) assessed GAD, MDD, or OCD or core symptoms of these disorders, and (c) reported either correlations or between-groups (i.e., disorder versus normal control) data on relationships between these constructs. Of the 159 studies obtained for review, 46 were excluded because they did not include a measure of IU or IA and six were excluded because they did not include a measure of GAD, MDD, or OCD. An additional 26 studies were excluded because they either did not report data on the relationship between IU and GAD, MDD, or OCD or reported data in a format that could not be converted to an r-type effect size (e.g., cluster analysis). Further, six studies were excluded because they reported the relationship between variables of interest only after a manipulation or intervention. Finally, studies that administered self-report measures in a language other than English were included only if the paper reported that the translated versions of these measures were validated through independent translation and back-translation procedures. Five studies were excluded for inadequate translation. See Fig. 1 for a flow diagram of study inclusion and exclusion decisions and Tables 1 and 2 for a summary of included studies.

Most studies assessed the relationship of IU to continuous measures of disorder symptoms rather than to a diagnosed anxiety or mood disorder. These two research designs address slightly different questions. Correlational research examines the relationship of IU to the experience of disorder symptoms on a continuum within normal or clinical samples. Between-groups research, in contrast, examines whether individuals diagnosed with a particular disorder experience different levels of IU than normal controls, or than individuals diagnosed with a different disorder. Because the goal of the current study was to fully represent the research on the relationship of IU to each syndrome, both correlational and between-groups studies were included. Further, studies were not excluded on the basis of population sampled. If participants were selected for a study on the basis of criteria other than GAD, MDD, or OCD diagnosis but provided data relevant to one or more of these syndromes, the study remained eligible for inclusion.

1.2.1. Eligible disorder measures

Diagnostic (DSM-IV) and syndrome measures of GAD, MDD, or OCD were considered eligible for inclusion. Tables 1 and 2 show all included measures of GAD, MDD, and OCD. A great deal of research has examined IU in relation to worry, rather than to GAD symptomatology more generally. Studies that only assessed worry without including a measure of GAD symptoms were not included. Although worry is a core feature of GAD, it is not by itself sufficient for a diagnosis, nor representative of the full set of diagnostic criteria (Ruscio, 2002). Further, core features of MDD and OCD that might be considered to parallel the worry found in GAD (e.g., rumination and obsessions) were not studied in isolation relative to IU, which led to concerns that inclusion of worry as a proxy for GAD symptoms may
bias the magnitude of effect sizes and the comparison across syndromes.

1.3. Effect size coding

Application of the previously described inclusion and exclusion criteria resulted in a set of 70 studies and 87 independent effect sizes (57 for the IUS and 30 for the OBQ). The variables that were coded for each study included correlations between IU and each syndrome as well as the sample size for each correlation. In addition, reliability coefficients (alpha) for the IU and syndrome measures were recorded when reported in order to correct for measurement unreliability. As mentioned above, between-groups (i.e., mean difference type) effect sizes were included along with r-type effect sizes. Mean difference-type effect sizes were converted to r-type effect sizes using either Cohen’s d or the reported M(SD) using the formula:

$$r_{pb} = \frac{ES_{mm}}{\sqrt{\left(\frac{1}{p(1-p)}\right) + ES_{mm}^2}}$$

where $r_{pb}$ is the point-biserial correlation, $ES_{mm}$ is the standardized mean difference effect size, $p$ is the proportion of subjects in Group 1 and $1-p$ is the proportion of subjects in Group 2.

The first author performed all coding for this study. To assess interrater reliability, a research assistant was trained on the procedure and independently coded data for 10 randomly selected studies. This reliability check was considered adequate because the coding procedure involved extracting only information that was explicitly reported in the studies and therefore required minimal judgment on the part of the coder. Observed agreement was 10/10 or 100%.

1.4. Analytic procedure

1.4.1. Publication bias

Publication bias is a concern in meta-analysis because nonsignificant findings typically are not published and therefore are excluded, resulting in an inflation of mean effect sizes. In addition to our efforts to reduce publication bias through inclusion of unpublished dissertations and acquisition of raw data, we assessed for the presence of publication bias in two ways. First, a funnel plot was created for each analysis by plotting a measure of sample size (standard error) as a function of reported effect size (Fisher’s z) for each study. In the absence of publication bias, such a plot would appear as a funnel shape, with large studies at the top distributed around the mean effect size and a greater amount of variability observed in the bottom of the plot (which represents smaller sample sizes). In the presence of publication bias, however, the bottom of the plot would appear skewed, with a higher concentration of studies on one side of the calculated mean (usually higher) than the other, indicating under-representation of small studies with nonsignificant effect sizes. Each funnel plot created for the current analyses appeared symmetrically distributed, suggesting an absence of publication bias. However, because most effect sizes in the current analyses are from studies with large sample sizes, the resulting plots are difficult to interpret. Therefore, Orwin’s fail-safe N (Orwin, 1983) was also calculated for each mean effect size to indicate the number of unpublished studies with a negligible effect size that would be necessary to reduce the mean observed effect size to a zero magnitude. Orwin’s fail-safe N is defined by:

$$k*(ES−ES_c)/ES_c$$

where $k$ is the number of studies contributing to the effect size, $ES$ is the observed effect size, and $ES_c$ is the criterion effect size that is judged to be negligible. The criterion effect size of $r = .10$ was used for all analyses in the current study, as recommended by Orwin (1983) and Naragon-Gainey (2010).

1.4.2. Independence of effect sizes

Like most common forms of statistical analysis, meta-analysis assumes the independence of each observation, meaning that a given analysis may include only one effect size per construct per sample. Several steps were taken to ensure the independence of coded effect sizes in the current analyses. For studies that reported data on multiple independent samples within a single report, data from each sample were coded. For studies that administered measures to the same sample at more than one time point, only the first time point was included. Twenty-five studies reported data on multiple syndromes from the same sample (e.g., a correlation between IU and OCD and a correlation between IU and GAD in the same sample). Syndrome was therefore coded as a moderator and all analyses were conducted separately by syndrome. Further, five studies reported data for multiple measures of a single syndrome (e.g., a correlation between IU and the Beck Depression Inventory and between IU and the Center for Epidemiologic Studies Depression Scale in the same sample). In these cases, the effect sizes were averaged and a single mean effect size was entered for each sample. Finally, 12 pairs of papers reported data from the same or overlapping samples. In these cases, effect sizes were coded from the original publication, with information from subsequent papers coded only if data for a given syndrome were not reported in the original paper. Analyses were conducted on a final set of 58 studies yielding 87 independent effect sizes.
sizes (57 for the IUS and 30 for the OBQ). In order to maintain independence of effect sizes while examining three outcomes (GAD, MDD, OCD) in relation to two definitions of IU (IUS, OBQ), this paper reports a total of six analyses.

### 1.4.3. Outliers

Before conducting each of these analyses, the sample-adjusted meta-analytic deviance (SAMD) statistic (Huffcut & Arthur, 1995) was calculated to test for the presence of statistical outliers. The values of the SAMD statistic approximate a normal $t$ distribution, with absolute values greater than 2 considered large. The SAMD statistic identified one outlier in the IUS-MDD analysis (Gentes et al., 2008) and one outlier in the OBQ-MDD analysis (Wu, Aardema, & O'Connor, 2009). All analyses were conducted with and without these two outliers to examine whether they had a significant impact on conclusions. Both outliers affected mean effect size estimates for MDD but their influence the overall pattern of results nor the effect size magnitude according to Cohen’s (1992)
conventions. Therefore, all analyses are reported with outliers included.

The SAMD statistic was also used to evaluate whether r-type effect sizes that were calculated from between-groups designs were comparable to the average r-type effect sizes calculated from studies with correlational designs. None of the six converted effect sizes (three for IUS and three for OBQ) was identified by the SAMD statistic as an outlier, so all were included in the analyses.

1.4.4. Meta-analytic model

Calculations of weighted mean effect sizes, heterogeneity, and moderator analyses were conducted using Comprehensive Meta-Analysis version 2.2.0.46 (Borenstein, Hedges, Higgins, & Rothstein, 2005). Because correlation coefficients have a skewed standard error formulation (Rosenthal, 1991), effect sizes were transformed using Fisher’s Zr-transform (Hedges & Olkin, 1985), which is defined as:

\[ ES_{Zr} = 5 \log_\left( \frac{1 + r}{1 - r} \right) \]

where \( ES_{Zr} \) is the Fisher’s Zr transformed correlation and \( r \) is the reported correlation.

Each effect size was then weighted by the inverse of its squared standard error value (inverse variance weight) in order to account for its precision using the formula:

\[ w_{Zr} = \frac{1}{SE_{Zr}^2} \]

where \( w_{Zr} \) is the inverse variance weight and \( SE^2 \) is the squared standard error of the Z-transformed correlation.

The heterogeneity of effect sizes was examined using the Q statistic and the \( I^2 \) statistic. The Q statistic is distributed as a chi-square and provides a significance test indicating whether the distribution of effect sizes around their mean is greater than expected from sampling error alone. The \( I^2 \) statistic supplement the Q statistic and represents the percentage of total variance that is attributed to between-study variance, with 25, 50, and 75% typically considered benchmarks for low, medium, and high heterogeneity (Higgins, Thompson, Deeks, & Altman, 2003).

Significant heterogeneity of effects was predicted in all analyses because studies included different symptom measures and sampled from different populations. Therefore, a random effects model, which includes an estimate for between-study variance in addition to sampling error, was judged to be most appropriate for the current analyses.

1.4.5. Correction for attenuation

In addition to calculating observed mean effect sizes, corrected mean effect sizes were estimated by correcting each correlation individually using the reported coefficient alpha (\( \alpha \)) for the IU and symptom measures. When a study did not report coefficient alpha for a measure, alpha was estimated using either the mean reliability of that measure (derived from other studies in the meta-analysis that administered the measure to a comparable sample) or the reliability reported for that measure in the original scale development article (if no other studies in the meta-analysis used the measure, and if the original article used a comparable sample). Each correlation was then corrected for attenuation using the following formula (Lipsey & Wilson, 2001):

\[ \rho = \frac{r}{\sqrt{JU \text{ measure}} \times \sqrt{\alpha \text{ IU measure}}} \]

where \( \rho \) is the correlation corrected for attenuation, \( r \) is the reported correlation, and \( \alpha \) is the coefficient alpha (reliability coefficient) for each measure included in the correlation.
Because this correction increases the sampling error correspondingly, the inverse variance weights were also corrected using the following formula (Lipsey & Wilson, 2001):

\[ w' = w \left( \frac{\alpha_{\text{IU measure}}}{\alpha_{\text{symptom measure}}} \right) \]

where \( w' \) is the corrected inverse variance weight, \( w \) is the uncorrected inverse variance weight, and \( \alpha \) is the coefficient alpha (reliability coefficient) for each measure included in the correlation. Random effects analyses were then repeated using the adjusted correlations and inverse variance weights to estimate the corrected mean effect sizes for GAD, MDD, and OCD.

### 1.4.6. Moderation and tests for significant differences between correlations

In order to test for significant differences in the magnitude of IU-syndrome relations, syndrome (GAD, MDD, OCD) was coded as a categorical moderator of effect size. Moderation was then examined using the Qb test, which is an analog to the Analysis of Variance (ANOVA). The Qb test groups effect sizes into mutually exclusive categories on the basis of a categorical independent variable (moderator), then tests the homogeneity among effect sizes within each category as well as the differences between the categories. Significant between-category variance (Qb) indicates that the mean effect sizes across groups (i.e., levels of the moderator) differ by more than sampling error. Because many studies reported data on multiple syndromes from the same sample, comparisons of effect size estimates across syndromes included comparisons across dependent samples. Each set of moderator analyses was therefore conducted twice. First, the assumption of independence was upheld by using a random selection procedure to determine which syndrome effect size estimate each sample would contribute to in each pairwise comparison. These analyses were then repeated with the assumption of independence relaxed so that each sample could contribute an effect size to each syndrome. Relaxation of the independence assumption in conducting moderator analyses actually provides a more conservative test of significance. For this reason, and because these two approaches produced identical patterns of results, results are reported only for the second set of analyses with the independence assumption relaxed.

Additional moderator analyses were performed to test whether the mean effect size estimates varied as a function of IU definition (GAD-specific versus OCD-specific) or population sampled (student versus treatment-seeking). To maximize statistical power, we tested for moderation in the full sample, collapsing across the three syndromes.

### 2. Results

Effect sizes have been converted from Fisher’s Z values back to \( r \) values for clarity of presentation.

#### 2.1. GAD-specific definition of IU: The Intolerance of Uncertainty Scale (IUS)

Table 3 shows the results of three meta-analyses summarizing the relationship of the IUS to symptoms of GAD, MDD, and OCD. The table includes observed mean \( r_s \) as well as \( r_p \) in which the correlations are corrected for unreliability. All confidence intervals presented in this table exclude zero; therefore, the observed correlations of the IUS with all three syndromes are statistically significant (\( p < .01 \)). Mean observed correlations range from .50 to .57, meeting or exceeding Cohen’s convention for a large effect size (.50; Cohen, 1992). Mean corrected correlations range from .54 to .65. Effect size distributions for each syndrome are significantly heterogeneous, as indicated by significant Q values (all \( p < .01 \)). \( I^2 \) values also indicate substantial heterogeneity, with over 72% of total variance in effect sizes for each syndrome attributable to between-study variance.

Post hoc Qb tests were conducted to test for differences in the magnitude of mean observed correlations between syndromes. These tests indicated that IU, operationalized by the IUS, was more strongly associated with GAD symptoms than with OCD symptoms, \( Q(1) = 4.80, p = .03 \). IU was no more strongly correlated with GAD symptoms than with MDD symptoms, \( Q(1) = 2.75, p = .10 \) nor more strongly correlated with MDD symptoms than with OCD symptoms, \( Q(1) = 0.42, p = .52 \).

Post hoc Qb tests performed on the corrected correlations revealed a similar pattern of results. The corrected IUS-GAD correlation was significantly stronger than the corrected IUS-OCD correlation, \( Q(1) = 6.94, p = .01 \). There were no significant differences in magnitude between the corrected IUS-MDD and IUS-OCD correlations, \( Q(1) = .96, p = .33 \). However, the difference between the corrected IUS-GAD and IUS-MDD correlations approached significance, \( Q(1) = 3.77, p = .05 \).

#### 2.2. OCD-specific definition of IU: The Obsessive Beliefs Questionnaire (OBQ)

Table 4 shows the results of two meta-analyses summarizing the relationship of the OBQ to symptoms of MDD and OCD. Symptoms of GAD were not included in this set of analyses because no study included measures of GAD along with the OBQ. All confidence intervals presented in this table exclude zero; therefore, the observed correlations of IU with both OCD and MDD symptoms are statistically significant (\( p < .01 \)). Mean observed correlations are in the range .41 to .42, falling between Cohen’s conventions for medium (.30) and large (.50) effects (Cohen, 1992). Mean corrected correlations are in the range .46 to .49. Effect size distributions for both syndromes are significantly heterogeneous, as indicated by significant Q values (all \( p < .01 \)). Values for \( I^2 \) indicate substantial heterogeneity, with over 75% of total variance in effect sizes for each syndrome attributable to between-study variance.

The Qb test revealed no significant differences between OCD and MDD symptoms in the strength of their association with IU, defined by the OBQ. Neither the observed \( Q(1) = 0.04, p = .84 \) nor the corrected \( Q(1) = 0.14, p = .70 \) correlations with OBQ scores differed significantly for the two syndromes.

#### 2.3. Moderator analyses

Table 5 shows the results of moderator analyses examining variation in effect size as a function of IU definition (GAD-specific versus OCD-specific) and population sampled (student versus...
treatment-seeking). Across syndromes, IU definition emerged as a significant moderator of effect size; $Q(1) = 13.82, p < .01$. The GAD-specific definition of IU (IUS) was more strongly associated with the set of three clinical syndromes than the OCD-specific definition (OBQ). Population sampled was also a significant moderator of effect size; $Q(1) = 5.12, p = .02$. IU was more strongly associated with the set of three clinical syndromes in student samples than in treatment-seeking samples.

3. Discussion

The current paper estimated the relationship of IU to symptoms of GAD, MDD, and OCD using two separate definitions of IU, one developed in relation to GAD and operationalized by scores on the IUS, and a second developed in relation to OCD and operationalized by scores on the OBQ. Using both definitions, IU was found to be significantly related to symptoms of GAD, MDD, and OCD. Thus, according to established guidelines for etiological specificity (Garber & Hollon, 1991), IU did not demonstrate narrow specificity to any of the syndromes studied here.

The significant association of IU with symptoms of both GAD and OCD was expected given the central role of IU in theories of these disorders (e.g., Dugas et al., 1996; Freeston et al., 1994; Ladouceur et al., 1997; Steketee et al., 1997). The association of IU with symptoms of MDD was more surprising and is at odds with theories of IU and its definitions. However, IU has been shown to correlate with disorders characterized by negative affect, including social anxiety (Boelen & Reijntjes, 2009), hypochondriasis (Deacon & Abramowitz, 2008), and panic (although IU is less strongly associated with symptoms of panic than with GAD; Dugas et al., 2001). However, IU has been shown to explain significant variance in anxiety symptoms beyond that contributed by negative affect (Boelen & Reijntjes, 2009) and to mediate the association of negative affect with symptoms of worry, MDD, and OCD (Norton & Mehta, 2007; Norton, Sexton, Walker, & Norton, 2005; Sexton, Norton, Walker, & Norton, 2003). These findings underscore the potential utility of IU as a correlate of emotional disorders and their comorbidity while leaving questions open about the disorders in which it may play a role and the mechanisms by which it is related to these disorders.

One mechanism through which IU may be linked to emotional disturbance is suggested by a shared feature of the three syndromes studied here. GAD, MDD, and OCD all have in common the experience of intrusive or repetitive negative thought. Research on the relationship of IU to GAD (Dugas et al., 2001) has suggested that IU may contribute to symptoms of GAD by increasing repetitive negative thought (i.e., worry) in which individuals engage as an attempt to control feelings of uncertainty and anxiety about future events (Freeston et al., 1994). This raises the possibility that ruminative thoughts (in MDD) and obsessive thoughts (in OCD) may similarly mediate the relationship of IU to symptoms of these disorders. Consistent with this possibility, a recent study found rumination to fully mediate the association between IU and MDD (Yook et al., 2010). Future research should examine whether obsessions similarly account for the relationship between IU and OCD and whether interventions targeting IU in all three disorders reduce repetitive negative thought as well as affective symptoms.

Although IU did not exhibit narrow specificity for any of the three syndromes considered here, it was more strongly associated with symptoms of GAD than OCD when the GAD-specific definition of IU (based on the IUS) was used. This definition did not distinguish GAD from MDD, nor MDD from OCD, all of which showed associations of comparable magnitude with IU. Unfortunately, it was not possible to compare relative magnitude across all three syndromes for the OCD-specific definition (based on the OBQ) because no studies using this definition reported associations with symptoms of GAD. However, similar to results for IU defined by the IUS, IU defined by the OBQ did not distinguish OCD and MDD symptoms. These results are broadly consistent with suggestions that IU is more closely related to GAD than to other anxiety disorders (e.g., Dugas et al., 2001; Ladouceur et al., 1999). At the same time, the heightened association with GAD symptoms was found solely in analyses using the IUS, which may be more strongly related to symptoms of GAD than to symptoms of OCD simply because it was originally designed by GAD researchers to tap into key features of the disorder. This limitation highlights the need for future research to utilize disorder-neutral measures that allow unbiased comparisons of the relationship of IU to other disorders. The recently developed Intolerance of Uncertainty Index (Carleton, Gosselin, & Asmundsen, 2010), which includes a 15-item scale assessing general unacceptability of uncertainty in addition to a 30-item scale assessing manifestations of uncertainty relevant to common anxiety symptoms, may prove useful for future assessment of IU.

### Table 4

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>$k$</th>
<th>$N$</th>
<th>Mean $r$</th>
<th>95%CI</th>
<th>$Q$</th>
<th>$I^2$</th>
<th>$p$</th>
<th>FSN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depressive disorder</td>
<td>11</td>
<td>2395</td>
<td>.41</td>
<td>.29–.52</td>
<td>90.50</td>
<td>88.95</td>
<td>.46</td>
<td>34</td>
</tr>
<tr>
<td>Obsessive–compulsive disorder</td>
<td>19</td>
<td>3927</td>
<td>.42</td>
<td>.37–.48</td>
<td>72.25</td>
<td>75.09</td>
<td>.49</td>
<td>61</td>
</tr>
</tbody>
</table>

Note. OCD = obsessive–compulsive disorder; OBQ = Obsessive Beliefs Questionnaire; $k$ = number of correlations; $N$ = total sample size for each symptom type; mean $r$ = average uncorrected correlation; $95\%CI$ = lower–upper limits of 95% confidence interval for uncorrected correlations; $Q$ = $Q$ statistic for heterogeneity; $I^2$ = proportion of total variance attributable to between-study variance; $p$ = average corrected correlation; FSN = Orwin’s Fail-Safe N.

### Table 5

<table>
<thead>
<tr>
<th>Moderator</th>
<th>$k$</th>
<th>Mean $r$</th>
<th>95%CI</th>
<th>$Q$</th>
<th>$I^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>IUS definition</td>
<td>57</td>
<td>.53$^a$</td>
<td>.50–.56</td>
<td>273.69</td>
<td>79.54</td>
</tr>
<tr>
<td>OCD-specific (OBQ)</td>
<td>30</td>
<td>.42$^b$</td>
<td>.37–.47</td>
<td>162.92</td>
<td>82.20</td>
</tr>
<tr>
<td>Population sampled$^d$</td>
<td>43$^f$</td>
<td>.50$^a$</td>
<td>.46–.54</td>
<td>315.83</td>
<td>86.70</td>
</tr>
<tr>
<td>Student</td>
<td>21</td>
<td>.42$^a$</td>
<td>.35–.48</td>
<td>73.43</td>
<td>72.76</td>
</tr>
</tbody>
</table>

Note. IU = intolerance of uncertainty; GAD = generalized anxiety disorder; IUS = Intolerance of Uncertainty Scale; OCD = obsessive–compulsive disorder; OBQ = Obsessive Beliefs Questionnaire; $k$ = number of correlations; mean $r$ = average uncorrected correlation; $95\%CI$ = lower–upper limits of 95% confidence interval for uncorrected correlations; $Q$ = $Q$ statistic for heterogeneity; $I^2$ = proportion of total variance attributable to between-study variance. Mean correlations within moderator (IU definition and population sampled) that do not share the same superscript differ from one another ($p < .01$).

$^a$ Community samples were not included because a small number of studies ($k = 5$) made use of community samples.

$^b$ One effect size (Ladouceur et al., 1998 Study 2) was excluded from analyses because it used an analog (GAD-diagnosed) student sample.

### Notes

- The recently developed Intolerance of Uncertainty Index (Carleton, Gosselin, & Asmundsen, 2010), which includes a 15-item scale assessing general unacceptability of uncertainty in addition to a 30-item scale assessing manifestations of uncertainty relevant to common anxiety symptoms, may prove useful for future assessment of IU.
Results of moderator analyses suggested that the way in which IU is measured may have a significant effect on the observed strength of association of IU to anxiety and mood symptoms. Specifically, the relation of IU to these symptoms was stronger when IU was measured with the GAD-specific measure (IUS) than when it was measured with the OCD-specific measure (OBQ). The lack of available GAD–OBQ effect sizes prohibited a systematic test of whether effect sizes were largest when IU measure was matched to the syndrome for which it was developed. However, visual inspection reveals that the OCD–IUS effect size was actually slightly larger than the OCD–OBQ effect size (this difference did not reach statistical significance, Q(1) = 3.41, p = .07). These findings suggest that IUS items may be more broadly relevant than OBQ items to the concerns of persons reporting anxiety and depression symptoms. Inspection of the two scales reveals two possible reasons for this pattern. First, items on the OBQ tend to be worded more severely than IU items, which may make participants less likely to endorse these items in comparison to the more mildly worded IU items. Further, OBQ items relate specifically to the concerns relevant to OCD (e.g., responsibility to prevent harm), whereas the IUS taps into more general discomfort with uncertainty and anticipatory anxiety about future events. The IUS may therefore be more broadly relevant to the concerns of a broader cross-section of individuals, including the nonclinical samples that predominate in this literature.

The present findings should be interpreted in the context of several limitations. First, a majority of effect sizes included in the current analyses were contributed by a small number of researchers and laboratories. It therefore remains possible that effect sizes are biased by researcher allegiance in addition to the use of disorder-specific measures.

Second, the heterogeneous nature of samples included in the current review represents a significant limitation. Mean effect sizes in the current analyses are based on data from student, community, and treatment-seeking samples as well as from several special populations (e.g., veterans with and without posttraumatic stress disorder). Due to extremely small sample sizes, we were unable to examine population as a moderator within each IU definition and within each syndrome. However, in the total sample, population was a significant moderator of the observed effects, with IU more strongly related to anxiety and mood symptoms in student samples than in treatment-seeking samples. This is perhaps unsurprising, as effect sizes in clinical samples are likely attenuated by uniformly high symptom (and possibly IU) levels compared with greater variability in student samples. Nevertheless, this finding has implications for future research on IU, most notably that the use of clinical samples will likely result in smaller effect sizes due to restriction of range. However, effect sizes in clinical samples were still in the moderate to large range. Therefore, researchers should continue to use clinical samples to address questions that are most appropriately tested in these samples (e.g., relationship of IU to clinically significant syndromes), but they should be aware that effect sizes may be smaller than those found in student samples and should power the study to detect these smaller effects.

Third, the high level of comorbidity between anxiety and mood disorders, and the fact that several papers contributed effect size data for multiple syndromes, suggests that many samples were characterized by symptoms of multiple disorders that may have complicated tests of specificity. For example, if a clinical sample was selected on the basis of GAD diagnosis but also experienced high rates of MDD, the patients’ GAD symptoms may have affected their reporting of IU or of MDD symptoms, which may then have biased the estimated relationship between IU and MDD symptoms. It was not possible in the current analyses to examine the relationship of IU to one syndrome while controlling for the effects of the other syndromes under study (e.g., an examination of the relationship of IU to MDD symptoms while controlling for symptoms of GAD and OCD). Consequently, the significant relationship of IU to symptoms of MDD may simply reflect the frequent comorbidity of MDD with anxiety disorders, rather than a unique association between IU and MDD symptoms over and above the variance explained by comorbid anxiety. This question will be an important agenda for future research studies. This question may be addressed in correlational research by assessing all three syndromes and reporting partial correlations between MDD and IU after removing the variance that is explained by anxiety. It may be further tested in between-groups research by comparing IU across samples diagnosed with “pure” (non-comorbid) GAD, MDD, and OCD. Regardless of the reason for the observed association, our results suggest that, at least in some cases, clinicians may need to take IU into account when working with depressed individuals.

Finally, the current paper is a quantitative review of cross-sectional data and cannot be used to infer or rule out a causal role of IU in GAD, MDD, and OCD. Several theories have proposed a causal role of IU, particularly in GAD (Freeston et al., 1994; Dugas et al., 2004). However, as Garber and Hollon (1991) note, nonspecificity only rules out “a simple univariate model; that is, that the variable being tested is not a sufficient cause of the disorder in question” (p. 129). The current results do not discount the possibility that IU is implicated in more complex causal processes in the development of GAD, MDD, and OCD, only that it is uniquely associated with the development of any one of these syndromes. Furthermore, the current results cannot rule out other possible relationships between IU and these syndromes. For instance, heightened anxiety and depression may lead to increased experience or reporting of IU, or a common third factor (such as intrusive or repetitive negative thought) may lead both to IU and to symptoms of GAD, MDD, and OCD. Such issues of causality are extremely difficult to disentangle, especially for syndromes that so often co-occur within individuals, as was the case for many of the samples represented in the current study. Nevertheless, given the abundance of correlational research already conducted, future energies may most productively be directed towards experimental and prospective longitudinal designs that can help illuminate the reasons for the observed correlations between IU and symptoms of GAD, MDD, and OCD.

In sum, the current findings establish IU as a shared feature of GAD, MDD, and OCD. They support the inclusion of IU in future research on the origins and comorbidity of all three syndromes. Further, they support the development and application of IU-focused interventions (Grayson, 2004; Ladouceur et al., 2000), both to help clarify the causal role of IU in these syndromes and to improve treatment outcomes. As this work proceeds, it will be important to ensure that IU is defined and assessed in a consistent fashion across disparate literatures, ideally using disorder-neutral measures. Greater consistency in operationalizing IU and other putative risk factors will help advance transdiagnostic models (e.g., Harvey et al., 2004) and classification debates (e.g., Brown & Barlow, 2009) that seek to clarify shared versus specific feature of anxiety and mood disorders. Improved understanding, in turn, will help foster the development of more parsimonious theoretical models and more effective and efficient treatments.

References

References marked with an asterisk indicate studies included in the meta-analysis.


