Cognitive-Behavioural Therapy for Generalized Anxiety Disorder: Examining the Patterns of Symptom Change and the Role of Intolerance of Uncertainty

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Abstract

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Generalized anxiety disorder (GAD) is characterized by excessive worry and somatic symptoms of anxiety (e.g., restlessness, muscle tension) (*DSM-IV-TR,* 2000). Although efficacious treatments have been developed, little is known about the nature and predictors of symptom change during treatment. Dugas and colleagues have developed a cognitive-behavioural therapy (CBT) for GAD which has been found to be efficacious in reducing worry and somatic anxiety in pre-to-posttreatment analyses (e.g., Dugas et al., 2010). This CBT is based on a cognitive model (Dugas et al., 1998) which implicates intolerance of uncertainty in the development and maintenance of GAD. The first goal of this study was to examine the nature of GAD symptom change during CBT. The second goal was to examine the role of intolerance of uncertainty, and its two component factors, in GAD symptom change. The results indicated that there was a bidirectional relationship between changes in worry and changes in somatic anxiety during treatment. In addition, intolerance of uncertainty was found to partially mediate GAD symptom change over time. However, different mediational roles were identified for the two factors, which represent distinct sets of beliefs about uncertainty. Specifically, Factor 2 (i.e., Uncertainty is unfair and spoils everything), was a stronger mediator of GAD symptom change than Factor 1 (i.e., Uncertainty has negative self-referential and behavioural implications). The theoretical and clinical implications of these findings are discussed.

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Table of Contents

List of Figures

List of Tables

Cognitive-Behavioural Therapy for Generalized Anxiety Disorder: Examining the Patterns of Symptom Change and the Role of Intolerance of Uncertainty

Generalized anxiety disorder (GAD) is a chronic condition characterized by excessive worry and somatic symptoms of anxiety (e.g., irritability, muscle tension) (American Psychiatric Association [*DSM-IV-TR*], 2000). GAD is also associated with substantial personal and societal costs, including impairment in social and occupational functioning (Hoffman, Dukes, & Wittchen, 2008). Fortunately, several psychological treatments have been developed and are efficacious in reducing GAD symptoms (e.g., Borkovec & Costello, 1993; Roemer & Orsillo, 2007). However, recovery rates of only 50-60% at posttreatment are common (Fisher, 2006) and there is clearly a need to improve treatment efficacy.

In order to refine existing treatment protocols, an understanding of how and why symptoms change during treatment is essential. However, studies examining the efficacy of psychological treatments have typically involved pre-to-posttreatment mean comparisons of symptoms or comparisons between treatment conditions at a given point in time (Laurenceau, Hayes, & Feldman, 2007). Although these analyses can reveal whether a particular treatment is associated with significant changes in symptoms by posttreatment, we know surprisingly little about what occurs *during* efficacious treatment programs. Specifically, we know little about typical patterns of symptom change or about the extent to which theoretically relevant variables predict symptom change.

Treatment Process Research

A growing body of research has begun to address this gap in our knowledge by relying on multi-wave longitudinal data in analyses designed to address treatment process

questions. This research is beginning to reveal the interrelationships between constellations of symptoms as they change during psychological treatments (e.g., Moskovitch, Hofmann, Suvak, & In-Albon, 2005; Roelofs, Huibers, Peters, Arntz, & Os, 2008), the rates of symptom change over time (e.g., Nishith, Resick, & Griffin, 2002; Penava, Otto, Maki, & Pollack, 1998), and the relationships between potential mechanisms of change and changes in targeted symptoms (e.g., Hoffart, Sexton, Hedley, & Martinsen, 2008; Hofmann et al., 2007; Teachman, Smith-Janik, & Marker, 2008).

A recent study by Hofman et al. (2007) provides an example of a multi-wave study in which analyses were conducted to address process-related questions. The authors wished to determine whether changes in catastrophic beliefs about panic symptoms served as a mechanism of symptom change in two different treatments for panic disorder (i.e., cognitive-behavioural therapy and pharmacotherapy). Statistical models of mediation (Baron & Kenny, 1986) were computed for each treatment condition to determine the extent to which change over time in panic symptoms (measured at pretreatment, posttreatment, and six months following treatment) were mediated by changes over the same period of time in panic-related beliefs. A partial mediation effect was found in the cognitive-behavioural therapy (CBT) condition, with reductions in panic-related beliefs accounting for approximately 20-30% of the observed changes in panic symptoms. In contrast, no mediation effect was found in pharmacotherapy. These results are consistent with theoretical models of the role played by cognitive factors in symptom change during treatments that specifically target negative cognitions. However, as the authors cautioned, their analyses did not test the reverse mediation effect (i.e., that

changes in panic symptoms might have mediated change in panic-related beliefs). Thus, the hypothesized mediating effects could not be established conclusively.

In other treatment process research, Moscovitch, Hofmann, Suvak, and In-Albon (2005) examined the patterns of symptom change during a 12-week CBT for social anxiety disorder. Prior research using pre-to-posttreatment mean comparisons had demonstrated that this CBT produced significant reductions in both social anxiety and depressive symptoms. Given that the treatment targeted only social anxiety explicitly, however, the authors argued that, during treatment, reductions in social anxiety would lead to subsequent reductions in depressive symptoms. Similar to Hofman et al. (2007), the authors computed a series of statistical mediation models to examine how these two constellations of symptoms, which were measured on a weekly basis, changed as a function of time and in relation to one another during CBT. The results indicated that while both social anxiety and depressive symptoms decreased significantly over time, decreases in social anxiety fully mediated (and accounted for 91% of) decreases in depressive symptoms. The authors also tested the reverse mediation relationship and found that decreases in depressive symptoms only partially mediated (and accounted for only 6% of) decreases in social anxiety. It was therefore argued that during CBT for social anxiety, depression improved during treatment because social anxiety improved, whereas social anxiety improved largely via mechanisms unrelated to changes in depression.

The multi-wave research designs and data analysis strategies described here have the potential to help researchers identify the nature and predictors of symptom change during efficacious psychological treatments. Given the potential value of treatment

process research, the overarching goal of the present study was to attempt to add to this growing literature by examining the patterns of symptom change, and a potential mechanism of this change, during an efficacious cognitive-behavioural therapy for GAD.

A Cognitive-Behavioural Therapy for GAD

The CBT protocol to be examined here was developed by Dugas and colleagues (Dugas & Ladouceur, 2000) and was designed to target GAD specifically. To date, four treatment outcome studies have demonstrated that this protocol leads to significant reductions in GAD-specific and associated symptoms. When compared to a waitlist condition, for example, this CBT was associated with higher rates of GAD diagnostic remission and greater change on measures of worry, somatic anxiety, and depressive symptoms (Ladouceur et al., 2000). Similar results were found when CBT was administered in a group format (Dugas et al., 2003). When compared to a non-directive treatment, CBT was associated with higher rates of diagnostic remission at posttreatment (65% vs. 20%) (Gosselin, Ladouceur, Morin, Dugas, & Baillargeon, 2006). Finally, when compared to applied relaxation training, although CBT was approximately equivalent to applied relaxation in symptom reduction at posttreatment, CBT was superior on a clinician-rated measure of global clinical improvement. Furthermore, although treatment gains were maintained in the applied relaxation condition over a 2-year follow-up period, only CBT was associated with continued improvement on measures of worry, trait anxiety, and global clinical improvement over the follow-up period (Dugas et al., 2010).

The studies described above have relied predominantly on pre-to-posttreatment mean comparisons of GAD symptoms at specific points in time and have helped to establish the efficacy of the CBT protocol developed by Dugas and colleagues. However,

establishing the patterns of symptom change that occur *during* treatment is an important first step in understanding the nature of typical symptom change (Laurenceau, Hayes, & Feldman, 2007). To date, only one study has examined patterns of symptom change in the current CBT protocol. Dugas, Francis and Bouchard (2009) used time-series analysis to examine the temporal precedence of changes in worry and somatic anxiety in a previous clinical trial of the CBT examined here. The authors tested whether changes in daily ratings of worry preceded and predicted changes in daily ratings of somatic anxiety, or whether the reverse was true. Significant effects in *both* directions were found for eight out of the ten participants in their sample and the authors concluded that a bidirectional relationship exists between changes in worry and changes in somatic anxiety during CBT. However, a sample size of 10 is small by conventional standards. Thus, further research is needed to determine the precise interrelationships between changes in GAD-specific symptoms in the current treatment protocol.

Intolerance of Uncertainty and GAD

Refining existing treatments for GAD requires knowledge not only of the nature of symptom change, but also of the variables that bring about this change. The CBT protocol examined here was derived from a theoretical model which implicates intolerance of uncertainty in the development and maintenance of GAD symptoms (Dugas, Gagnon, Ladouceur, & Freeston, 1998). In this context, intolerance of uncertainty is understood as a dispositional characteristic resulting from a set of negative beliefs about uncertainty (e.g., "Uncertainty is upsetting and should be avoided") (Dugas & Robichaud, 2007). Consistent with cognitive models of psychopathology (e.g., Beck & Clark, 1997), these negative beliefs are thought to lead to biased information processing

in situations with uncertain outcomes. For example, individuals who are intolerant of uncertainty might be more likely to make threatening interpretations of ambiguous situations, leading to elevated levels of worry and anxiety (see Dugas & Robichaud, 2007, for a discussion of possible alternate causal paths from intolerance of uncertainty to GAD symptoms).

Research using the *Intolerance of Uncertainty Scale* (IUS; Buhr & Dugas, 2002; Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994; see *Measures*) has provided some support for both the specificity of intolerance of uncertainty to GAD symptoms and for the causal role that this cognitive variable might play in the onset and maintenance of excessive worry and GAD diagnostic status. For instance, intolerance of uncertainty and worry have been found to be highly related among non-clinical individuals, and to remain significantly related even when controlling for scores on measures of anxiety and depressive symptoms (Buhr & Dugas, 2002; Dugas, Freeston, & Ladouceur, 1997; Freeston et al., 1994). In clinical samples, individuals with GAD have been found to have higher levels of intolerance of uncertainty than individuals with other anxiety disorders (e.g., obsessive-compulsive disorder, social anxiety disorder, panic disorder) (Dugas, Marchand, & Ladouceur, 2005; Ladouceur et al., 1999). Among individuals with GAD, scores on the IUS have been found to distinguish individuals with moderate and severe levels of GAD symptoms from individuals with mild GAD symptoms (Dugas et al., 2007). Finally, Ladouceur, Gosselin, and Dugas (2000) manipulated levels of intolerance of uncertainty experimentally among non-clinical individuals and found that those with higher manipulated levels of intolerance of uncertainty experienced higher levels of worry than individuals with lower manipulated levels of intolerance of uncertainty.

Given the evidence of a strong and specific relationship between intolerance of uncertainty and GAD symptoms, and given the potential causal role of intolerance of uncertainty in the etiology of excessive worry and GAD diagnostic status, one would expect that changes in intolerance of uncertainty during a psychological treatment that targets this variable would lead to subsequent reductions in GAD symptoms. Consistent with this assumption, when Dugas and Ladouceur (2000) conducted time-series analyses on the data from four individuals who underwent 16 sessions of CBT, they found that changes in intolerance of uncertainty preceded and predicted changes in daily ratings of the time spent worrying for three of the four individuals in the study. This effect almost reached statistical significance for the fourth individual. Changes in time spent worrying, in contrast, did not predict change in intolerance of uncertainty in any of the four individuals. In a larger sample of individuals with GAD ($N = 52$), Dugas et al. (2003) found that scores on the IUS accounted for a significant proportion of variance in change scores in a composite measure of GAD symptoms. This effect was found even when scores on measures of non-specific therapy factors were controlled (e.g., therapist characteristics, client expectations for treatment outcome, and client motivation).

The studies conducted thus far provide support for the hypothesis that intolerance of uncertainty may be implicated in the maintenance of GAD symptoms and, in theory, in their reduction during treatment. However, the potentially important mediational role of intolerance of uncertainty during CBT has not yet been established. The study by Dugas and Ladouceur (2000), for instance, was conducted with only four individuals and involved an analysis of a single item from the IUS and its relation to daily ratings of worry for each participant. Although the study by Dugas et al. (2003) involved a larger

sample, the temporal relationships between changes in intolerance of uncertainty and GAD symptoms were not assessed. Thus, further research is required to establish the specific role that intolerance of uncertainty might play in GAD symptom reduction during treatment.

One additional reason to examine the role of intolerance of uncertainty in GAD symptom reduction is that recent research examining this construct suggests that it may be composed of two distinct but related factors. In two large and recent factor analyses, Sexton and Dugas (2009) found that the first factor of the IUS was based on beliefs relating to the idea that uncertainty has negative self-referential and behavioural implications (e.g., "Being uncertain means that I lack confidence" and "When it's time to act, uncertainty paralyses me"). The second factor of the IUS appeared to be based on beliefs relating to the idea that uncertainty is unfair and spoils everything (e.g., "It's unfair not having any guarantees in life" and "A small unforeseen event can spoil everything, even with the best of planning"). If intolerance of uncertainty is found to mediate symptom change during treatment, it would be of interest to establish whether the two factors of the IUS play an equally important role in bringing about this symptom change.

The Current Study: Goals and Hypotheses

Dugas and colleagues began a fifth clinical trial of the CBT protocol for GAD in 2006. In this ongoing five-year study, participants with GAD receive CBT during 14 weekly individual treatment sessions. During the treatment program, participants complete ratings of GAD symptoms on a daily basis from pre to posttreatment. Participants also complete symptom and cognitive variable measures on a periodic basis

throughout the treatment program (i.e., pre, mid, and posttreatment, as well as at 3 and 6 months following treatment). All participants for the current analyses were recruited from this larger clinical trial.

The nature of daily symptom change from pre to posttreatment. Our first goal was to examine the nature of symptom change during the pre-to-posttreatment interval of the CBT protocol and our analyses tested two hypotheses. First, we examined the mean rates of change in GAD-specific and associated symptoms (i.e., excessive worry, somatic anxiety, and depressive symptoms). The daily symptom ratings generated by participants during treatment assessed the percentage of each day they spent worrying, experiencing somatic anxiety, and experiencing feelings of depression (hereinafter referred to as "daily symptoms;" see *Measures*). Given that this CBT protocol had been shown to lead to significant decreases in GAD and associated symptoms in prior pre-to-posttreatment analyses, we hypothesized that it would also lead to significant mean decreases in the amount of time that participants spent worrying, experiencing somatic anxiety, and experiencing depressive symptoms on a daily basis during treatment (*Hypothesis 1a*).

Our second hypothesis was related to the pattern of GAD-specific symptom changes that might be expected from pre to posttreatment (i.e., the interrelationships in the changes in daily ratings of worry and somatic anxiety). The results of the time-series analyses conducted by Dugas, Francis, and Bouchard (2009) suggested that the observed changes in GAD symptoms were bidirectional. However, this finding does not preclude the possibility that the bulk of symptom change occurs in a particular direction. In both the theoretical model developed by Dugas and colleagues (Dugas et al., 1998) and in current conceptualizations of GAD from a diagnostic point of view (e.g., Andrews et al.,

2010), excessive worry is understood to be the central feature of GAD. In the CBT protocol examined here, worry is explicitly targeted from the outset of treatment (whereas somatic anxiety is not) on the understanding that reductions in worry will nonetheless lead to subsequent reductions in somatic anxiety. Furthermore, there is evidence from experimental studies that when individuals reduce the time they spend worrying each day, they experience subsequent reductions in somatic complaints (e.g., muscle pain, dizziness) (Brosschot & van der Doef, 2006). As a result, we expected that the bulk of changes in GAD symptoms would occur in a particular direction. Specifically, we hypothesized that changes in daily ratings of worry would mediate change over time in daily ratings of somatic anxiety during treatment to a greater extent than the reverse mediational relationship (*Hypothesis 1b*).

Intolerance of uncertainty as a mechanism of GAD symptom change. Given the potential importance of intolerance of uncertainty in the maintenance (and reduction) of GAD symptoms, our second goal was to examine the extent to which changes in intolerance of uncertainty mediated GAD symptom change during the CBT program. For these analyses, we examined change in symptom and cognitive measures (i.e., WAQ and IUS, see *Measures*), which were administered periodically from pretreatment to six months following treatment. These analyses tested two hypotheses. First, we examined the mediational role of intolerance of uncertainty as assessed by the full-scale IUS. We hypothesized that change in intolerance of uncertainty would mediate change over time in GAD symptoms from pretreatment to six months following treatment (*Hypothesis 2*).

Given that the two factors of the IUS appeared to represent different subsets of uncertainty-related beliefs, our second hypothesis regarding intolerance of uncertainty

related to the potential mediational role played by the IUS factors. In their factor analyses, Sexton and Dugas (2009) provided empirical support for the validity of the IUS factors. Both factors were found to be similarly and highly correlated with pathological worry. However, Factor 1 was more strongly related to GAD analogue status, trait anxiety, somatic anxiety, and depressive symptoms than Factor 2. Given these findings, we expected that Factor 1 might serve as a stronger mediator of GAD symptom change than Factor 2. In addition, it seemed likely that the beliefs represented by Factor 1 (e.g., "When it's time to act, uncertainty paralyses me") were more internally-oriented and might be more directly amenable to change via the hypothesis testing that participants engaged in during CBT. The beliefs associated with Factor 2, in contrast, appeared to be more externally-oriented, reflecting longer-standing assumptions about the consequences of uncertainty (e.g., "A small unforeseen event can spoil everything, even with the best of planning"). It therefore seemed that the treatment would be more likely to bring about changes in Factor 1 than Factor 2 beliefs and that we would be more likely to observe a mediation effect of Factor 1 on GAD symptom change. Consequently, we hypothesized that IUS Factor 1 would mediate GAD symptom change to a greater extent than would IUS Factor 2 from pretreatment to six months following treatment (*Hypothesis 3*).

Method

Participants

The sample for the current study ($N = 51$) consisted of 40 women and 11 men, all of whom had a primary diagnosis of GAD. The average age of participants was 44.57 years ($SD = 12.69$) and they had completed an average of 16.04 years of education ($SD = 4.10$). Of the 51 participants, 49.02% were employed full-time, 11.76% were employed

part-time, 9.81% were full-time students, 1.96% were part-time students, and 27.45% were either not engaged in paid employment or were retired. The ethnic composition of the sample was as follows: 94.12% of participants identified as White/European, 3.92% as Middle Eastern, and 1.96% as Other. All participants were Francophone.

GAD symptom severity was assessed at pretreatment using the 9-point (0-8) Clinician's Severity Rating scale of the *Anxiety Disorders Interview Schedule for DSM-IV* (ADIS-IV; Di Nardo, Brown, & Barlow, 1994). The mean GAD severity at pretreatment was 5.91 ($SD = 0.75$) and the mean duration of GAD was 11.30 years ($SD = 12.66$). When assessing comorbidity at pretreatment, 78.43% of the sample met diagnostic criteria for at least one other diagnosis, with 43.14% of the sample meeting criteria for one secondary condition, 29.41% meeting criteria for two secondary conditions, and 5.88% meeting criteria for three secondary conditions. Secondary conditions were panic disorder with or without agoraphobia ($n = 14$), specific phobia ($n = 14$), major depressive disorder ($n = 13$), social anxiety disorder ($n = 12$), obsessive-compulsive disorder ($n = 5$), posttraumatic stress disorder ($n = 1$), substance dependence ($n = 1$), and an eating disorder ($n = 1$). In terms of psychoactive medication, 50.98% were taking anxiolytic or antidepressant medication at intake. Finally, 35.29% of participants had previously received cognitive-behavioural treatment for an anxiety or mood disorder, and an additional 15.69% had received previous psychotherapy which included at least some cognitive-behavioural elements (see Table 1 for a summary of additional clinical characteristics in the current sample).

Procedure

Participants were recruited from the Anxiety Disorders Clinic of the *Hôpital du Sacré-Cœur de Montréal,* as part of a larger ongoing clinical trial of cognitive-behavioural therapy for GAD (PI: Michel J. Dugas). To maximize the validity of initial diagnoses, each potential participant was interviewed by independent assessors using different structured diagnostic interviews. Initial assessments were conducted by a team psychiatrist using the *Mini International Neuropsychiatric Interview, Version 5.0* (MINI; Sheehan et al., 1994). Individuals who met GAD diagnostic criteria on the MINI were then assessed by a team psychologist using the ADIS-IV (see *Measures*). At the end of each diagnostic interview, assessors rated the severity of each diagnosed condition on the 9-point (0-8) Clinician's Severity Rating scale. The final severity rating for each disorder was determined by consensus during a team meeting with the Principal Investigator (M.J.D.).

Inclusion criteria for the study were as follows: (a) a primary diagnosis of GAD with a score of at least 4/8 on the Clinician's Severity Rating scale, derived by consensus from the MINI and ADIS-IV initial interviews; (b) a difference of at least 1 point on the Clinician's Severity Rating scale between GAD and all secondary conditions; (c) between 18 and 64 years of age; (d) no change in medication type or dose 4 to 12 weeks prior to intake assessment (4 weeks for benzodiazepines and 12 weeks for antidepressants and hypnotics); (e) willingness to maintain a stable dose and type of psychoactive medication during treatment; (f) no evidence of suicidal intent; (g) no evidence of current substance abuse; and (h) no evidence of current or past schizophrenia, bipolar disorder, or organic mental disorder.

A total of 73 individuals were assessed for eligibility for the clinical trial between March 2007 and January 2009. Of these individuals, 13 were excluded following the initial assessments because GAD was not diagnosed at intake ($n = 8$); GAD was not the primary diagnosis ($n = 2$); the severity of a secondary disorder was not sufficiently below the GAD severity rating on the Clinician's Severity Rating scale ($n = 2$); or another condition (i.e., attention deficit/hyperactivity disorder) appeared to be interfering with the completion of study measures ($n = 1$). An additional 7 participants withdrew their consent to participate after the initial assessment ($n = 5$) or after starting treatment ($n = 2$) due to difficulties meeting the time commitment for the study. Finally, two participants were excluded from the analyses due to difficulties completing the questionnaires ($n = 1$) or because of a change in psychoactive medication during treatment ($n = 1$).

The remaining 51 participants received cognitive-behavioural therapy for GAD, based on the protocol developed by Dugas and colleagues (Dugas & Ladouceur, 2000). Treatment consisted of 14 individual weekly sessions with a clinical psychologist and included five treatment components. Participants first received (a) *psychoeducation and worry awareness training*, in which it was explained that by identifying and reducing worries, participants would experience a subsequent decrease in somatic anxiety. Participants also learned to monitor their GAD symptoms on a daily basis. In the second component, participants worked with their therapist to (b) *re-evaluate the usefulness of worry*, including challenging positive beliefs about worry (e.g., "If I worry I will be less disturbed when unforeseen events occur"). The next component consisted of (c) *uncertainty recognition and behavioural exposure*, in which the therapist explained the role of intolerance of uncertainty in maintaining worry and somatic anxiety. Participants

also began to identify and enter into uncertainty-inducing situations. (d) *Problem-solving training* was then applied to help clients resolve current problems and to learn how intolerance of uncertainty can interfere with the problem solving process. Finally, (e) participants learned to use repeated *imaginal exposure* for worries about hypothetical situations, exposing themselves to the imaginal scenarios until they no longer experienced anxiety (see Dugas & Robichaud, 2007, for a detailed description of the protocol).

In addition to the initial structured diagnostic interviews, diagnostic assessments using the ADIS-IV were administered at posttreatment, and at 3 and 6-month follow-up. Participants also completed a battery of self-report questionnaires at pre, mid, and posttreatment, as well as at 3 and 6 months following treatment, and these batteries included several measures of GAD and associated symptoms (see *Measures*). Finally, therapists provided participants with a daily self-monitoring booklet during the first treatment session. In this booklet, participants indicated on a 0-100% scale the percentage of each day that they spent worrying, experiencing somatic anxiety, and experiencing feelings of depression during treatment. Participants also used this booklet to monitor their daily use of psychoactive medication. However, the use of medication was not part of the present analyses and only the daily symptom ratings are discussed here.

In order to enhance the validity of the daily symptom ratings, clinicians gave their clients a simple definition of each symptom in the first treatment session. Worry was defined as "a chain of upsetting thoughts about something bad that could happen to you or to others." Somatic anxiety was defined as "a physiological reaction that includes responses such as muscle tension, restlessness, and feeling keyed up or on edge."

Depression was defined as "a feeling of sadness, depression, or low mood." Although the somatic symptoms of GAD also include fatigue, difficulties concentrating, irritability, and sleep disturbance (*DSM-IV-TR*, 2000), these symptoms were excluded from the present definition of somatic anxiety because they are not specific to GAD (see Andrews et al., 2010).

Measures

Diagnostic interviews. The *Mini International Neuropsychiatric Interview, Version 5.0* (MINI; Sheehan et al., 1994) is a brief and structured diagnostic interview that assesses mood and anxiety disorders, substance use disorders, psychotic disorders, eating disorders, and suicidal risk. The MINI was designed for use in clinical settings and assesses the presence of current psychological problems. Clients provide yes/no answers to screening questions for each disorder. The MINI was designed to be used by a broad range of clinicians, including general medical practitioners, and has adequate psychometric properties (Sheehan et al., 1997). The 9-point Clinician's Severity Rating scale from the ADIS-IV (see below) was used to obtain severity ratings for MINI diagnoses.

The *Anxiety Disorders Interview Schedule for DSM-IV* (ADIS-IV; Di Nardo et al., 1994) assesses anxiety disorders and screens for other Axis I conditions, including mood and somatoform disorders, substance use and psychotic disorders, and medical problems. The severity of Axis I disorders is assessed using the Clinician's Severity Rating scale which ranges from 0 (Absent or none) to 8 (Very severe or very severely disturbing/disabling). A score of 4 (Moderate or definitely disturbing/disabling) indicates a clinically significant level of symptom severity. The ADIS-IV has been found to have

good to excellent interrater reliability for anxiety disorders (κ = .67 to .86) (Brown, Di Nardo, Lehman, & Campbell, 2001).

Symptom measures. The *Penn State Worry Questionnaire* (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item self-report measure of the tendency to engage in excessive and uncontrollable worry. The measure was designed to assess the intensity and excessiveness of worry regardless of the specific worry content (e.g., "I'm always worrying about something"). Items are rated on a 5-point Likert scale, ranging from 1 (Not at all typical of me) to 5 (Very typical of me). The PSWQ has high internal consistency (α = .86 to .95) and good 4-week test-retest reliability (r = .74 to .93) (Molina & Borkovec, 1994). The internal reliability for the PSWQ in the current sample was α = .83.

The *Worry and Anxiety Questionnaire* (WAQ; Dugas et al., 2001) is an 11-item self-report measure that assesses the presence and severity of GAD symptoms according to the DSM-IV diagnostic criteria. Overall, the WAQ has been found to have satisfactory test-retest reliability and diagnostic validity (i.e., it can be used to distinguish between individuals who do and do not meet GAD diagnostic criteria) (Dugas et al., 2001). The WAQ contains a subscale that assesses the six somatic symptoms associated with GAD (e.g., restlessness, muscle tension, fatigue, irritability, difficulties concentrating and difficulties sleeping), as well as questions regarding the frequency, intensity, and uncontrollability of worry. Items are rated on a 5-point Likert scale, ranging from 1 (Not at all) to 5 (Very severe). The internal reliability for the WAQ in the current sample was α = .65.

The *Beck Depression Inventory-II* (BDI-II; Beck, Steer, & Brown, 1996) is a self-report measure that assesses the severity of depressive symptoms (e.g., sadness, pessimism, and loss of interest) over the previous two weeks. The measure contains 21 groups of 4 items that reflect different degrees of depressive symptoms. Scores range from 0 to 3 on a Likert scale. The BDI-II has excellent internal consistency ($\alpha = .92$) and test-retest reliability over a 1-week period ($r = .93$) (Beck et al., 1996). The internal reliability for the BDI-II in the current sample was $\alpha = .90$.

The *State-Trait Anxiety Inventory-Trait version* (STAI-T; Spielberger, 1977) is a 20-item self-report measure used to assess the degree to which individuals have the stable tendency to experience anxiety (e.g., "I feel nervous and restless"). Items are rated on a 4-point Likert scale, ranging from 1 (Almost never) to 4 (Almost always). The internal consistency has been found to be high ($\alpha = .89$) in a sample of individuals diagnosed with a variety of anxiety disorders, and the measure has been found to correlate highly with other commonly-used measures of anxiety (e.g., *The Beck Anxiety Inventory*; Beck & Steer, 1990) and depressive symptoms (e.g., *The Beck Depression Inventory*; Beck, Rush, Shaw, & Emery, 1979). The internal reliability of the STAI-T was $\alpha = .84$ in the present sample.

The *Daily Self-Monitoring Booklet* is a 4-page booklet used to record daily symptom levels and psychoactive medication use during treatment. To assess symptoms, participants use a 0-100% scale to record the percentage of each day spent experiencing worry, somatic anxiety, and feelings of sadness or depression (e.g., "what proportion of the day did you spend worrying?"). This booklet is similar to those used in previous clinical trials (e.g., Dugas & Ladouceur, 2000; Gosselin et al., 2006) and comparable

daily ratings of worry have been shown to correlate significantly with scores on other well-established and valid measures of worry. Verkuil, Brosschot, and Thayer (2001) found, for example, that scores on the PSWQ were moderately correlated with the frequency of daily worries reported for a non-clinical sample during a 1-week period (r = .44, $p < .01$). Other researchers have found that PSWQ scores correlate moderately with daily ratings of the amount of time spent worrying over a 2-week period among both non-clinical participants (r = .59, $p < .01$) and individuals with GAD (r = .42, $p < .01$) (Dupuy, Beaudoin, Rhéaume, Ladouceur, & Dugas, 2001).

Cognitive process measure. The *Intolerance of Uncertainty Scale* (IUS; Freeston et al., 1994) is a 27-item scale that assesses negative beliefs about uncertainty. Scores on the IUS can range from 27 to 135, with higher scores indicating higher levels of intolerance of uncertainty. Recent and large factor analyses (N = 2,451) conducted by Sexton and Dugas (2009) have shown that the IUS has a two-factor structure. Factor 1 represents the belief that uncertainty has negative behavioural and self-referential implications and Factor 2 represents the belief that uncertainty is unfair and spoils everything. The IUS has been shown to have excellent internal consistency (α = .94), and good test-retest reliability when assessed over a 5-week period (Buhr & Dugas, 2002). In the current sample, the internal reliability of the full-scale IUS was α = .97. The internal reliability scores for Factor 1 and Factor 2 were α =.97 and α = .94 respectively. The two subscales were highly correlated with each other (r = .86, $p < .01$) and with the full-scale IUS (r = .97 and r = .96 respectively with $p < .001$).

Data Analysis Strategy

Coinciding with the recent increased interest in treatment process research, there has been a corresponding interest in data analytic strategies that allow researchers to model change over time in a continuous rather than a cross-sectional manner (Gueorguieva & Krystal, 2004; Willett, Singer, & Martin, 1998). Multilevel statistical modeling is a data analysis strategy that is becoming increasingly popular in studies involving longitudinal research designs (Kenny, Korchmaros, & Bolger, 2003; Krull & MacKinnon, 1999). In this approach, it is assumed that there are at least two levels in a data set. In the case of repeated measures designs, the lower level, or Level 1, consists of repeated measures of a variable of interest. Units at Level 1 are said to be nested within Level 2 units, or individuals. Multilevel modeling has several advantages over more traditional data analysis strategies. This strategy can, for example, effectively manage missing data in repeated measures, which is a common problem in longitudinal research. Multilevel models can also take into account (and adjust for) bias in standard errors and statistical tests that might result from the non-independence of observations nested within individuals (Gueorguieva & Krystal, 2004; Krull & MacKinnon, 1999; Raudenbush & Bryk, 2002).

The data in the current study were longitudinal, with each participant providing two types of repeated symptom measures, including daily symptom ratings during treatment and periodic symptom and cognitive measures at pre, mid, and posttreatment, as well as at 3 and 6 months following treatment. Given that the longitudinal nature of the study produced a multilevel or nested data structure, with repeated measures nested within individuals, all main analyses were carried out using *Hierarchical Linear*

Modeling, which is a computer software program that can be used to conduct multilevel statistical modelling (HLM 6.06; Raudenbush, Bryk, & Congdon, 2006).

The nature of daily symptom change from pre to posttreatment. To determine the mean rate of change in GAD and associated symptoms (i.e., worry, somatic anxiety, and depressive symptoms) from pre to posttreatment, we calculated a series of lower-level growth curve models representing change over time in the daily symptom ratings that participants completed between their first and final treatment sessions. In each model, the repeated symptom measures were predicted by time at level 1 of the analysis (with number of days from the start of treatment as the indicator of time). Following recommendations by Willett, Singer, and Martin (1998), we initially used power polynomials to test whether change over time in daily symptoms was best represented by a linear time function (i.e., time coded as 0, 1, 2...) or a quadratic time function (i.e., time coded as 0^2, 1^2, 2^2...).

We then examined the patterns of change in daily ratings of GAD-specific symptoms (i.e., worry and somatic anxiety) from pre to posttreatment. A series of lower-level mediation models (Kenny, Korchmaros, & Bolger, 2003) were computed to determine whether changes in worry mediated changes in somatic anxiety during treatment to a greater extent than the reverse mediational relationship. Time was again used as the initial predictor variable. Two mediation models were tested (see Figure 1). Model 1a consisted of time predicting somatic anxiety, with worry as the mediator. This model was the hypothesized model for the treatment protocol, in which changes in worry were expected to mediate change over time in somatic anxiety to a greater extent than the

reverse. Model 1b tested the reverse mediation relationship and consisted of time predicting worry, with somatic anxiety as the mediator.

Because these and all subsequent main analyses involved an exploration of mediation effects, several general comments should be made here about the methods we used to assess mediation. Our approach to mediation analysis was derived from Baron and Kenny (1986) and adapted to a multilevel context by Kenny, Korchmaros, and Bolger (2003). Conceptually, demonstrating that mediation has occurred involves establishing that a mediating variable partially or fully accounts for the relation between an initial predictor and an outcome variable. Three regression equations are computed. In the first equation, the total effect of the initial predictor (i.e., time) on the outcome is estimated (i.e., the *c* paths depicted in Figure 1). Two mediational equations are then computed, including estimates of the effect of the initial predictor on the mediator (i.e., the *a* paths in Figure 1), and the effect of the initial predictor on the outcome when the effect of the mediator is added to the final regression model (i.e., the *c'* and *b* paths, respectively).

Traditional tests of mediation effects have relied on quantifying the reduction in the total effect of the initial predictor on the outcome once the mediator is included in the model (i.e., the reduction from *c* to *c'*). However, to test for the presence of mediation effects in the current study, we used two statistical methods that assess the magnitude of the indirect effects directly (i.e., the product of the *a* and *b* paths). Specifically, we conducted the Aroian version of the Sobel test (Baron & Kenny, 1986) for each indirect effect, which is recommended for lower level fully-random mediation analyses because it allows for the possibility that the components of the indirect effects co-vary across

individuals. However, although the Sobel test is widely-recognized, it has also been found to be conservative in small samples (Krull & MacKinnon, 1999). As a result, we also used a test of the distribution of the *ab* products in each model which is a more powerful test of mediation (Fritz & MacKinnon, 2007). To do so, we constructed the 95% confidence intervals around each *ab* product using a statistical software program, *Product Confidence Limits for Indirect Effects* (PRODCLIN; MacKinnon, Fritz, Williams, & Lockwood, 2007). Because the distribution of *ab* products tends to be asymmetrical (MacKinnon et al., 2007), PRODCLIN produces asymmetric confidence limits, consistent with the non-normal distribution of indirect effects. Finally, we also wished to compare the magnitude of mediation effects across mediation models. As a result, we also computed the percentage of mediation for each model, which is the proportion of the total effect of the initial predictor on the outcome that is accounted for by the mediator (Shrout & Bolger, 2002).

The type of mediation analysis described here has allowed process researchers to identify the interrelationships between changes in variables over time (e.g., Hofmann et al., 2007; Moscovitch, Hofmann, Suvak, & In-Albon, 2005; Smits, Rosenfield, McDonald, & Telch, 2006). However, statements regarding mediation presume both a particular statistical relationship between the mediator and outcome variable as well as a temporal one. In order to establish that mediation has occurred, the mediating variable must be shown to *temporally precede* the outcome variable (Baron & Kenny, 1986). To date, most analyses examining mediation have relied on estimates of mediator and outcome variables that were assessed *at the same point in time* (e.g., Hofmann et al., 2007; Moscovitch et al., 2005). As a result, researchers typically conduct follow-up

analyses in which a temporal lag is created between the mediator and outcome variables to determine the temporal precedence of observed changes (e.g., Meuret, Rosenfield, Hofmann, Suvak, & Roth, 2009; Smits, Rosenfield, McDonald, & Telch, 2006).

Newer methods of testing mediation are beginning to incorporate a temporal lag directly into mediation analyses in a variety of ways (MacKinnon, 2008). As a result, in these and all subsequent mediation analyses, we chose to incorporate this temporal relationship by creating a time lag between mediating and outcome variables such that scores for each mediating variable preceded scores for each outcome variable by one assessment point.[1] For instance, in the mediation models examining the patterns of change in daily symptom ratings, the *a* paths depicted in Figure 1 represent change over time in the mediating variable from the first day of treatment to the second-to-last day of treatment. In contrast, the *c* and *c'* paths represent change in the outcome variable from the second day of treatment to the final day of treatment (i.e., there is a lag of one day between mediator and outcome variables).

Intolerance of uncertainty as a mechanism of GAD symptom change. To determine whether changes in intolerance of uncertainty served as a mechanism of symptom change, we examined the effect of intolerance of uncertainty on GAD symptoms assessed at pre, mid, and posttreatment, and at 3 and 6-month follow-up. We again computed lower-level mediation models (see Figure 2). We began by examining the mediational role of the full-scale IUS (see *Measures*) on GAD symptom change. Two mediation models were tested using a procedure similar to the one described for the daily symptom ratings. Model 2a consisted of time predicting GAD symptoms (i.e., WAQ

scores), with intolerance of uncertainty (i.e., IUS full-scale scores) as the mediator. This was the hypothesized model for the full-scale IUS.

Model 2b (see Figure 2) tested the reverse mediation relationship and consisted of time predicting intolerance of uncertainty (i.e., IUS full-scale scores), with GAD symptom scores as the mediator (i.e., WAQ scores). A time lag was again created between mediating and outcome variables. Specifically, scores on the mediator (e.g., full-scale IUS scores in Model 2a) preceded scores on the outcome variable (e.g., WAQ scores) by one assessment point. Thus, in the mediation models depicted in Figure 2, the paths representing change over time in each mediator (i.e., the *a* paths) represent change over time in the mediator from pretreatment to 3-month follow-up. In contrast, change over time in the outcome variable in each model (i.e., the *c* and *c'* paths) represents change from mid-treatment to 6-month follow-up. To examine the role of the IUS factors in GAD symptom change, these mediation models were computed again with each of the IUS factors as potential mediators (see Models 3a to 3d in Figure 2).

Results

Participant Selection for the Current Sample

The goal in this study was to examine some of the potentially important processes of change during CBT. Because of the preliminary nature of these analyses (i.e., this was the first attempt at formally establishing intolerance of uncertainty as a mediator and the data represent only a portion of the full sample that will eventually be obtained), we included only individuals who had completed the treatment portion of the CBT program and the 6-month follow-up period in the present sample. Individuals who drop out of treatment may differ in important ways from those who complete treatment and although

an analysis of the change processes among individuals who drop out of treatment is an important next step, we wished to first establish the rate and mechanism of symptom change that can be expected for those who completed the treatment program.

Interrater Agreement on Diagnostic Status

We assessed the degree of interrater agreement on GAD diagnoses at pretreatment in two ways. First, we calculated the percentage of agreement between the MINI and ADIS interviews for GAD diagnoses in the total sample of individuals who were initially assessed for the treatment study ($N = 73$). Agreement between assessors was met when they agreed on both the presence of GAD and the severity of GAD symptoms (i.e., scores on the Clinician's Severity Rating scale were equal to 4 or above on the 9-point scale and there was a difference of no more than one point in severity between assessors' ratings). The percentage of agreement for the assessed sample was 68.50%. We then calculated the interrater agreement on GAD symptom severity for the sample of individuals who were included in the present study ($N = 51$), all of whom met diagnostic criteria for primary GAD. Scores on both the MINI and ADIS had to be within one point of each other on the Clinician's Severity Rating scale for assessors to be considered in agreement. The percentage of agreement for GAD severity ratings in the treated sample was 89.04%.

The Nature of Daily Symptom Change from Pre to Posttreatment

Preliminary analyses. The current treatment protocol was designed to be administered over approximately 14 weekly individual treatment sessions and participants were asked to complete ratings of the time spent worrying, experiencing somatic anxiety, and experiencing feelings of depression on a daily basis from the first to the last treatment session. The mean number of treatment sessions completed by

participants was 14.63 (*SD* = 1.37), although the number of sessions that participants actually received ranged from 11 to 18 sessions. The expected number of ratings per person was approximately 91 daily ratings over the 13-week treatment period. However, given variability in scheduling weekly sessions, participants actually completed an average of 110.77 (*SD* = 21.74) daily ratings. There was also considerable variability in the number of ratings completed during treatment, ranging from 73 to 192 ratings.

To assess participants' daily symptom ratings at the start of treatment, we calculated the mean percentage of each symptom for the first week of the treatment program. On average, participants spent 35.19% (*SD* = 16.86) of each day worrying, 34.04% (*SD* = 17.63) of each day experiencing somatic anxiety, and 15.19% (*SD* = 13.76) of each day experiencing feelings of depression during the first week of treatment. Thus, although participants were spending an equal amount of time worrying and experiencing somatic symptoms, they were experiencing feelings of depression for a smaller proportion of the day at the start of treatment.

Mean rates of symptom change. Before examining mean rates of change in daily symptom ratings during treatment, we first examined the raw daily ratings obtained from the sample. As shown in Figure 3, considerable fluctuations were observed throughout the treatment program. Although this appeared to be true for most participants, there also appeared at first glance to be a dramatic increase in the amount of time participants spent worrying, experiencing somatic anxiety, and experiencing depressive symptoms towards the end of treatment. However, it should be noted here that only a few participants provided a considerably larger number of daily symptom ratings than the majority of individuals in the sample. In particular, five of the 51 participants in the sample provided

more than 135 daily symptom ratings. Of these individuals, only one participant actually showed a substantial increase in symptom ratings during the final days of treatment. The other four participants experienced either small decreases or no change in symptoms over treatment. However, these participants began and continued to experience high levels of daily symptom ratings relative to other participants throughout the treatment program. In fact, it was for this reason that these participants were offered additional treatment sessions. In Figure 3, the cumulative effect of these participants, who made up only 9.80% of the total sample, was the apparent dramatic increase in daily symptom severity at the end of treatment. Nonetheless, the analyses described below were conducted both with and without these individuals. Their removal had little effect on the parameters that were estimated. As a result, their daily ratings were included in these analyses to maintain the representativeness of the sample data.

We then calculated a series of lower-level growth curve models to determine the mean rate of change in daily symptoms, with each of the daily symptom measures predicted by time at level 1 of the analyses. Time was coded as both a linear and a quadratic function in each regression model. In order to assess the relative amount of within-person variance accounted for by each time variable, these variables were entered into each regression model in a hierarchical manner. Consistent with our first hypothesis (*Hypothesis 1a*), we found that there was a significant decrease in the daily ratings of time spent worrying ($B = -0.15, p < .001$), experiencing somatic anxiety ($B = -0.15, p < .001$), and experiencing feelings of depression ($B = -0.07, p < .001$) during the treatment program (see Table 2). We also found that nonlinear change accounted for a significant proportion of within-person variance above and beyond the effect of linear time on

ratings of worry and somatic anxiety, but not for ratings of depression. However, the additional amount of variance accounted for by nonlinear time was small for each daily symptom measure (i.e., accounting for only an additional 4.68%, 4.07%, and 3.47% of the within-person variance in ratings of worry, somatic anxiety, and feelings of depression respectively). As a result, we used linear time as a predictor in all subsequent analyses involving the daily symptom ratings.

When examining the linear rates of change for each daily symptom rating, we found that participants experienced an average decrease of approximately 0.15 of a percentage point per day in time spent worrying and experiencing somatic anxiety, which translated into a decrease of approximately 13.65% on the 0-100% scale over 91 days of treatment. Participants experienced a decrease of only 0.07 of a percentage point per day on average in time spent feeling depressed during treatment (or a decrease of approximately 6.37% on the 0-100% scale).

Patterns of symptom change. We examined the patterns of change in daily ratings of GAD-specific symptoms (i.e., worry and somatic anxiety) from pre to posttreatment. For this analysis, two lower-level mediation models were computed to determine whether changes in worry mediated changes in somatic anxiety during treatment, and/or whether the reverse was true (see Figure 1). Linear time was the initial predictor variable in each model. Model 1a consisted of time predicting somatic anxiety, with worry as the mediator and this was the hypothesized model for the CBT program (*Hypothesis 1b*). Model 1b tested the reverse mediation relationship. A time lag of one day was introduced between mediating and outcome variables in each model (see *Data Analysis Strategy*), so

that the temporal relationship between changes in each variable could be directly assessed.

The results from the two mediation analyses are presented in Table 3. In Model 1a, there was a significant direct effect of time on somatic anxiety (B = -0.15, p < .001). However, when worry was entered as the mediator, the magnitude of the direct effect of time on somatic anxiety was reduced (B = -0.08, p < .001) and the indirect effect was significant (Sobel test statistic = -6.99, p < .001). In Model 1b, there was a significant direct effect of time on worry (B = -0.15, p < .001). However, with somatic anxiety entered as the mediator, the direct effect of time on worry was also reduced (B = -0.10, p < .001) and this indirect effect was also significant (Sobel test statistic = -6.24, p < .001). We then calculated the percentage of mediation for each model and found that the percentage of mediation was greater in the expected direction. Changes in worry mediated (and accounted for 47.67% of) changes in somatic anxiety, whereas changes in somatic anxiety mediated (and accounted for 36.40%) of changes in worry.

Intolerance of Uncertainty as a Mechanism of GAD Symptom Change

Preliminary analyses. Before examining whether changes in intolerance of uncertainty mediated change over time in GAD symptoms, we computed lower-level growth curve models to assess the mean rates of change from pretreatment to 6-month follow-up. In these and all subsequent analyses, GAD symptoms were assessed using the *Worry and Anxiety Questionnaire* (WAQ) and intolerance of uncertainty was assessed using the *Intolerance of Uncertainty Scale* (IUS). It should also be noted here that because the two-factor structure of the IUS had not yet been confirmed in a clinical sample, we first conducted a preliminary confirmatory factor analysis in a sample of

individuals with GAD ($N = 271$) before proceeding with the mediation analyses described below. The results of this confirmatory factor analysis provided support for the two IUS factors originally identified by Sexton and Dugas (2009). A summary of the findings are presented in Table 6 and a more detailed description of the analysis is presented in Appendix A.

An initial examination of the nature of change in intolerance of uncertainty and GAD symptoms from pretreatment to 6-month follow-up suggested that this change was likely not linear (see Figure 4). As a result, we began by including both linear and quadratic time variables as initial predictors when assessing mean rates of change. The mean rates of change in GAD symptoms, intolerance of uncertainty, and in its two factors are presented in Table 4. In order to determine the proportion of within-person variance accounted for by each time variable, the linear and quadratic time functions were again added to each model in a hierarchical manner. On average, significant mean linear decreases were observed in each variable of interest from pretreatment to 6-month follow-up. However, non-linear time accounted for a significant amount of within-person variability in each variable above and beyond the effect of linear time. Thus, on average, participants not only showed mean decreases in GAD symptoms and intolerance of uncertainty (and its factors), but also a significant degree of deceleration in this change from pretreatment to 6-month follow-up. An inspection of the graphs presented in Figure 4 suggested that this deceleration likely occurred during the 3 to 6-month follow-up period.

In terms of the rate of change in each variable, a significant mean linear decrease was found on the WAQ ($B = -2.14$, $p < .001$), with linear time accounting for 65.30% of

the within-person variance from pretreatment to 6-month follow-up. Nonlinear time only accounted for an additional 6.01% of the total within-person variance in WAQ scores. The mean linear decrease in the full-scale IUS was also significant ($B = -5.04$, $p < .001$), with linear time accounting for 47.58% of the within-person variance in IUS scores, and nonlinear time accounting for an additional 17.32%. Significant mean linear decreases were also observed in both IUS factors (Factor 1: $B = -2.42$, $p < .001$; Factor 2: $B = -2.63$, $p < .001$) from pretreatment to 6-month follow-up. Non-linear time accounted for an additional 13.28% of the within-person variance in IUS Factor 1 and an additional 18.97% of the within-person variance in IUS Factor 2.

The graphs in Figure 4 also suggested that the rate of change in Factor 2 might have been greater during the interval from pre to midtreatment than the rate of change in Factor 1. In fact, when we conducted paired-samples *t* tests on the two factors, we found that while the mean difference in Factor 2 scores from pre to midtreatment was statistically significant (Factor 2 Mean $\text{Difference}_{\text{pre-mid}} = 5.35$, $t(50) = 4.30$, $p < .001$), the difference in Factor 1 scores was not (Factor 1 Mean $\text{Difference}_{\text{pre-mid}} = 12.77$, $t(50) = 1.74$, $p = .09$). In contrast, the mean differences within each factor between mid and posttreatment were both statistically significant (Factor 1 Mean $\text{Difference}_{\text{mid-post}} = 5.72$, $t(50) = 4.80$, $p < .001$; Factor 2 Mean $\text{Difference}_{\text{mid-post}} = 4.64$, $t(50) = 5.39$, $p < .001$).

The full-scale IUS as a mechanism of GAD symptom change. We next examined whether change over time on the full-scale IUS mediated change over time in GAD symptoms from pretreatment to 6-month follow-up. Two lower-level mediation models were computed (see Figure 2). Although the initial regression analyses described above suggested that mean changes over time on all variables of interest were non-linear, a

linear time function nonetheless accounted for the greatest proportion of within-person variance in each variable. A linear time function is also more straightforward to interpret in the context of mediation analyses. As a result, we chose to conduct these analyses using linear time as the initial predictor.

The results from all IUS mediation analyses are presented in Table 5. Models 2a and 2b describe the mediation analyses conducted with the full-scale IUS and WAQ. Our expectation was that change over time on the full-scale IUS would mediate change over time on the WAQ (*Hypothesis 2*). As can be seen in Table 5, change over time on the full-scale IUS was found to partially mediate change over time on the WAQ (Sobel test statistic = -4.28, $p < .001$) from pretreatment to 6-month follow-up. The percentage of mediation indicated that the indirect effect of the full-scale IUS accounted for 23.13% of the total effect of time on the WAQ. However, we also found that the reverse mediation relationship was also statistically significant. In other words, change over time on the WAQ was also found to mediate change over time on the full-scale IUS (Sobel test statistic = -4.13, $p < .001$), and the percentage of mediation in this model was 73.22%. Thus, these first analyses suggested that it was primarily change in GAD symptoms which mediated change over time in intolerance of uncertainty.

The IUS factors as mechanisms of GAD symptom change. We next examined the role of IUS Factor 1 in GAD symptom reduction (i.e., Models 3a and 3b in Figure 2). Our expectation was that change over time in IUS Factor 1 would mediate change over time on the WAQ to a greater extent than would IUS Factor 2 (*Hypothesis 3*). However, although change in IUS Factor 1 was indeed found to mediate change over time on the WAQ (i.e., Model 3a; Sobel test statistic = -3.26, $p < .001$), the percentage of mediation

for Model 3a was only 15.87% (see Table 5). Model 3b describes the reverse mediation effect (i.e., change in GAD symptoms mediating change in IUS Factor 1), which was also found to be statistically significant (Sobel test statistic = -5.46, $p < .001$). The percentage of mediation for Model 3b was 96.78%. Thus, an examination of the mediating effects of both the full-scale IUS and IUS Factor 1 suggested that change in intolerance of uncertainty was not a strong mediator of GAD symptom change. More specifically, Factor 1 appeared to be a relatively weak mediator of symptom change, and instead was almost fully mediated by change in GAD symptoms.

When we examined the role of IUS Factor 2 on change over time on the WAQ, however, we found a different pattern. As shown in Model 3c, change over time in Factor 2 of the IUS partially mediated change over time on the WAQ from pretreatment to 6-month follow-up (Sobel test statistic = -4.87, $p < .001$) and the percentage of mediation in this model was 37.15%. Model 3d examined the reverse mediation effect and it was found that change on the WAQ also partially mediated change over time on IUS Factor 2 (Sobel test statistic = -1.94, $p = .053$; 95% CI for ab = -0.71[-1.46, -0.01]). The percentage of mediation for this final model was 38.22%.

Discussion

The goal of the present study was to examine the processes of symptom change during an efficacious CBT program designed specifically for GAD. We began our analyses by examining the rate and patterns of symptom change during the 14-session CBT program developed by Dugas and colleagues. Our first hypothesis (*Hypothesis 1a*) was that, consistent with past clinical trials of this CBT protocol, we would observe significant symptom decreases from pre to posttreatment in daily ratings of worry,

somatic anxiety, and depressive symptoms. This hypothesis was confirmed. On average, participants experienced significant decreases in the amount of time they spent worrying and experiencing somatic anxiety from pre to posttreatment. In fact, in the first week of treatment, participants were spending an average of 34.04% to 35.19% of each day worrying and feeling anxious (i.e., 5.12 to 5.23 hours, respectively, in a 15-hour day). An average decrease of 13.64% was observed during treatment, suggesting that participants were experiencing GAD symptoms for only 3.06 to 3.23 hours in a 15-hour day on average by the end of the 14-session treatment program.

Although participants were only experiencing feelings of depression for 15.19% of each day on average at the start of treatment, they nonetheless also showed a significant mean reduction of 6.37% during treatment (i.e., a reduction from 2.28 to 1.32 hours in a 15-hour day). Thus, although participants were not asymptomatic by the end of treatment, they nonetheless experienced observable decreases in both GAD and associated symptoms. These changes are encouraging, particularly given that the treatment protocol primarily targets worry, without explicit attempts to decrease either somatic anxiety or depressive symptoms.

Our next analyses examined the patterns of symptom change in daily ratings of GAD symptoms during treatment (i.e., worry and somatic anxiety). Given that worry is understood as the central feature of GAD (Andrews et al., 2010; *DSM-IV-TR*, 2000), and given that this CBT protocol targets worry (but not somatic anxiety) explicitly, we expected that changes in worry would mediate changes in somatic anxiety from pre to posttreatment to a greater extent than the reverse mediational relationship (*Hypothesis 1b*). This hypothesis was also confirmed. Changes in the amount of time that participants

spent worrying partially mediated changes in the amount of time spent experiencing somatic anxiety. The reverse mediational relationship was also found, in which changes in time spent experiencing somatic anxiety also partially mediated changes in the amount of time participants spent worrying. However, although the percentage of mediation was greater in the expected direction (47.67% vs. 36.40%), the difference in the magnitude of each indirect effect was only 11.27%. As a result, it may be more meaningful from a clinical point of view to consider this relationship as essentially bidirectional.

Although unexpected, the bidirectional nature of GAD symptom change, even in a treatment that targets only worry explicitly, makes sense given current assumptions that anxiety disorders consist of interacting cognitive, physiological, affective, and behavioural sets of symptoms, and that changes in one set of symptoms may result in changes in another (e.g., Beck & Clark, 1997). In terms of why changes in worry might lead to subsequent change in anxiety, some researchers have suggested that the process of worrying may prolong physiological stress responses to a particular stressor, even beyond the actual presence of that stressor (Brosschot, Gerin, & Thayer, 2006). It seems possible, given our findings, that this process might actually work in both directions and, in fact, the typical relationship between changes in worry and somatic anxiety during treatment may be cyclical in nature. If this is the case, it may be that this cyclicality is in fact the typical pattern of symptom change and it may be clinically meaningful in the future to identify participants for whom this cyclicality does not occur. Future research is needed to determine whether different patterns of symptom change than those presented here can be used by clinicians as a guide to the efficacy of CBT for individual clients.

The finding that the CBT protocol examined here leads to reductions in both worry and somatic anxiety is encouraging in light of current theories of information processing. It appears, for instance, that individuals with high levels of both worry and anxiety have greater difficulty disengaging their attention from threat-related stimuli (e.g., angry faces) than individuals who experience high levels of worry or anxiety alone (Verkuil, Brosschot, Putman, & Thayer, 2009). Thus, clinicians may take heart in the fact that focusing on one set of symptoms for GAD may result in positive and pervasive changes in the way individuals with GAD process information. It should also be noted, however, that the effects of mediation in the analyses in the current study were not complete in either direction, and it may be that during treatment changes in each GAD symptom are also brought about by changes in other phenomena (e.g., changes in symptoms of depression).

In our next analyses, we wished to determine whether changes in a theoretically-relevant cognitive variable, intolerance of uncertainty, served as a mechanism of GAD symptom change during the CBT program. In addition, we wished to examine the role that specific beliefs about uncertainty played in symptom reduction during treatment. To this end, we first examined the factor structure of the *Intolerance of Uncertainty Scale* (IUS) by conducting a confirmatory factor analysis of the IUS in a Francophone clinical sample ($N = 271$). This analysis allowed us to confirm the two-factor structure identified previously by Sexton and Dugas (2009) (see Table 6). Specifically, Factor 1 appeared to be represented by beliefs that are consistent with the idea that uncertainty has negative self-referential and behavioural implications (e.g., "Being uncertain means that I lack confidence" and "When it's time to act, uncertainty paralyses me"). Factor 2 appeared to

be represented by beliefs regarding the fact that uncertainty is unfair and spoils everything (e.g., "It's unfair not having any guarantees in life" and "A small unforeseen event can spoil everything, even with the best of planning"). Thus, in addition to examining the role that intolerance of uncertainty plays generally in GAD symptom change, we were able to examine the role played by two distinct sets of beliefs about uncertainty.

Given that previous research had demonstrated a close relationship between full-scale scores on the IUS and both excessive worry and GAD diagnostic status (e.g., Buhr & Dugas, 2002; Ladouceur, Gosselin, & Dugas, 2000), we expected that changes in intolerance of uncertainty, as assessed by the full-scale IUS, would mediate GAD symptom change during the CBT program (i.e., from pretreatment to 6 months following treatment) (*Hypothesis 2*). This hypothesis was partially confirmed. Although change in intolerance of uncertainty did appear to partially mediate symptom change, the reverse mediation effect was also found. In other words, change over time in GAD symptoms as assessed by the WAQ also partially mediated change over time in intolerance of uncertainty and this indirect effect was in fact greater than in the hypothesized model for the full-scale IUS.

Furthermore, when we examined the role that each of the two IUS factors played in GAD symptom reduction, a somewhat more complex pattern emerged. We had expected that changes in Factor 1 would mediate GAD symptom change to a greater extent than would changes in Factor 2 (*Hypothesis 3*). Contrary to our expectations, however, it was found that Factor 1 was not a strong mediator of GAD symptom change during the CBT program, with only 15.87% of the direct effect of time on GAD

symptoms accounted for by change over time in Factor 1. In contrast, Factor 1 was found to be almost fully mediated by change over time in GAD symptoms, with GAD symptoms accounting for 96.78% of the direct effect of time on Factor 1. When we examined the hypothesized and alternate models for Factor 2, however, we found that change in Factor 2 accounted for 37.15% of the direct effect of time on GAD symptoms, and change in GAD symptoms accounted for 38.22% of the direct effect of time on Factor 2.

Consistent with the theoretical model on which the current treatment protocol was based, our analyses revealed that changes in intolerance of uncertainty did partially mediate change in GAD symptoms. However, the different effects of the IUS factors on symptom change suggest that not all negative beliefs about uncertainty are equally effective in reducing GAD symptoms. Specifically, if we compare the mediation models in which the IUS factors served as mediators of GAD symptom change, we can see that the percentage of mediation for Factor 2 (i.e., Model 3c; 37.15%) was approximately twice that of Factor 1 (i.e., Model 3a; 15.88%). In addition, when the reverse mediation effects were tested, GAD symptom change clearly (and almost fully) mediated change in Factor 1, with a percentage of mediation (i.e., Model 3b; 96.78%) that was more than twice that of Factor 2 (i.e., Model 3d; 38.22%). These findings are of interest here because, in addition to helping to clarify the role that intolerance of uncertainty plays in reducing GAD symptoms during treatment, it also has the potential to help clinicians identify the specific negative beliefs about uncertainty that, if targeted during treatment, are most likely to lead to symptom change.

Although the reasons for the different roles that the two IUS factors appear to play in GAD symptom reduction are not yet known, there are several potentially intriguing explanations. The first possible explanation relates to the fact that the IUS factors, which represent two distinct sets of beliefs about uncertainty, might also be differently related to GAD-specific and associated symptoms. Factor 1 consists of items that describe, primarily, the negative self-related implications of uncertainty. When taken at face value, one might also argue that some of the Factor 1 items reflect or are at least consistent with features of depression, including low mood (e.g., "Uncertainty makes me vulnerable, unhappy, or sad"), negative views of the self (e.g., "Being uncertain means that I am not first rate"), and low self-esteem (e.g., "Being uncertain means that I lack confidence"). The factor analyses conducted by Sexton and Dugas (2009) did in fact find that Factor 1 was more strongly associated with a measure of depressive symptoms than Factor 2. In the current study, although the relationship between change over time in intolerance of uncertainty and depressive symptoms was not examined, we did calculate partial correlations between scores on the BDI-II and each IUS factor at pretreatment. Although none of the partial correlations between pretreatment BDI-II and IUS factor scores were statistically significant, we nonetheless found a trend in the relationship between BDI-II pretreatment scores and Factor 1 of the IUS ($pr = .27$, $p = .06$), when controlling for Factor 2. It may be that changes in Factor 1 are therefore less likely to mediate GAD-specific symptom changes directly, and instead might partially mediate change over time in depressive symptoms. However, further research would of course be required to test this possibility empirically.

When examining the relationship between the IUS factors and worry, Sexton and Dugas (2009) found that both factors were equally and highly related to scores on a measure of worry (i.e., PSWQ). In the current sample, however, although partial correlations between scores on the PSWQ and each IUS factor were not statistically significant at pretreatment, we found a trend in the relationship between PSWQ pretreatment scores and Factor 2 of the IUS ($pr = .27$, $p = .06$), when controlling for Factor 1. That Factor 2 items might be more closely related to worry seems plausible given that many of the items appear, at face value, to reflect a future-oriented set of concerns (e.g., "I always want to know what the future has in store for me") and a view of the self situated in an inherently uncertain world (e.g., "One should always look ahead so as to avoid surprises" and "A small unforeseen event can spoil everything even with the best planning"). Thus, although we did not examine the potential mediating effect of changes in intolerance of uncertainty on worry specifically, it seems possible that changes in Factor 2 might be more likely to lead directly to changes in worry during treatment due to its future-oriented content.

A second possible explanation for the different effects of the IUS factors on GAD symptom change might lie in the distinct nature of the negative affect represented within the IUS factors. As discussed, the affect described by Factor 1 items is consistent with the feelings of sadness and low mood associated with depression. Several of the items in Factor 2 also assess negative affect. However, the affect represented in Factor 2 appears to be characterized primarily by feelings of anxiety, stress or tension, a sense of being upset or uneasy, and feelings of frustration (e.g., "Uncertainty makes me uneasy, anxious or stressed" and "It frustrates me not having all the information I need"). Items on this

factor also appear to be more externally-oriented, reflecting somewhat rigid beliefs about the degree of certainty that should be attainable (e.g., "I should be able to organize everything in advance" and "I can't stand being undecided about my future") and about the unfairness of not being able to achieve a desired degree of certainty (e.g., "It's unfair having no guarantees in life"). One might argue, as a result, that the affect and beliefs reflected in Factor 2 are more consistent with feelings of frustration or even anger than with sadness. Although measures of frustration or anger were not included in the present study, there is evidence that individuals with anxiety disorders generally (Moscovitch, McCabe, Antony, Rocca, & Swinson, 2008), and with GAD specifically (Erdem, Celik, Yetkin, & Ozgen, 2008) do experience higher levels of anger than non-anxious individuals. Thus, it is interesting to speculate about the potential implications of high levels of these emotions among individuals with GAD.

One potential implication of the presence of anger among individuals with GAD can be seen in studies examining the effect of negative affect on information processing. There is evidence, for example, that individuals with high levels of trait anxiety are more likely to interpret threat/ambiguous stimuli in a threatening manner than individuals with low trait anxiety (Richards & French, 1992). Angry and anxious responses may share a number of similarities (e.g., over-responsiveness to stress, negative affect) and might have similar effects on information processing. In fact, Wenzel and Lystad (2005) found that individuals high on either self-reported anger or anxiety rated negative outcomes in ambiguous scenarios as more likely than non-angry/anxious individuals. Angry individuals also rated positive outcomes as less likely to occur and anger-related outcomes as more likely to occur than did high-anxiety or non-angry/anxious individuals.

Interestingly, both angry and anxious individuals rated anxiety-related outcomes as more likely than did non-angry/anxious individuals, suggesting that the effects of anger on information processing may be pervasive. Barazzone and Davey (2008) also demonstrated a unique causal effect of manipulated levels of anger on interpretation biases. Specifically, individuals who received either an anger or anxious mood induction were more likely to make threat interpretations of threat/neutral stimuli than those receiving positive or neutral mood inductions and the effect of anger remained significant even when levels of anxiety were controlled.

Further research is required to determine whether there are specific and important relationships between anger, anxiety, and interpretational biases among individuals with GAD, and whether and how these variables are related to the specific beliefs in Factor 2 of the IUS. In the cognitive model of GAD developed by Dugas and colleagues, one proposed pathway from intolerance of uncertainty to GAD symptoms is that beliefs related to uncertainty lead to interpretational biases, which in turn lead to elevated levels of anxiety. It might be that these biases also lead to elevated levels of anger. However, the information processing literature described above suggests that this effect may be bidirectional, with elevated levels of anxiety and anger also leading to increased interpretational biases. Whether anger plays a direct role, a mediating role, or a moderating role in producing these biases remains to be seen.

One final explanation that might account for the different effects of the IUS factors on GAD symptom change during treatment is perhaps the most straightforward. We had expected that changes in Factor 1 of the IUS would be more likely to occur than changes in Factor 2, given that Factor 1 items reflect beliefs about uncertainty that could

be directly challenged in between-session exposure exercises. In particular, we expected that beliefs about the behavioural implications of uncertainty (e.g., "When it's time to act, uncertainty paralyses me") might be difficult to maintain while clients were actively entering into uncertainty-inducing situations. In contrast, exposure exercises seemed less likely to have an effect on the more external and future-oriented beliefs reflected in Factor 2 (e.g., "It's not fair not having any guarantees in life"). As a result, Factor 1 was expected to be a stronger mediator of change in GAD symptoms than Factor 2.

If the above reasoning is correct, this same reasoning also implies that an active commitment to behaviour change would be necessary before changes in the beliefs associated with Factor 1 could occur. We also know, however, that exposure to uncertainty-related situations can be stressful for participants (Dugas & Robichaud, 2007). It might therefore be more difficult to bring about changes in Factor 1 beliefs than in those associated with Factor 2, which might require less behavioural change. Consistent with this possibility, the rate of change in Factor 1 of the IUS appears to have been slower during the first half of the treatment program (i.e., pre to midtreatment) than was the case for Factor 2. There is still the possibility that the treatment protocol might be further refined to bring about changes in Factor 1 beliefs earlier on, with the possibility that changes in Factor 1 might therefore contribute to the overall mediational effect of intolerance of uncertainty to a greater extent.

Whatever the reasons for the different impact of the IUS factors on GAD symptoms during treatment, this study provides a preliminary glimpse into the nature of GAD symptom change during the pre-to-posttreatment interval and a clearer understanding of the specific ways in which a theoretically relevant cognitive variable

might serve as a mechanism of symptom change. However, despite its potential to fill a gap that currently exists in the treatment process literature, this study also involved several limitations. For instance, the current study did not include a control or comparison treatment group. It would have been of interest, however, to determine whether the interrelationships between daily ratings of worry and somatic anxiety observed here also occur among non-clinical individuals, or whether intolerance of uncertainty plays the same mediating role in psychological treatments in which it is not explicitly targeted.

Another limitation relates to the limited number of assessments taken during the treatment program. Increasing the number of assessment points during treatment can allow researchers to examine the nature of mediation with greater precision, in particular because this may allow researchers to capture a mediation effect closer to the time at which it occurs. As Laurenceau, Hayes, and Feldman (2007) point out, the ability to identify the precise action of a mechanism of symptom change during treatment might be decreased if assessment of a potential mediator occurs either too early or too late in the treatment program. In the present study, it is possible that changes in the beliefs associated with Factor 1 on the IUS do mediate symptom change, but perhaps they do so at a time that was not captured by the assessments administered here.

Another limitation in the present study is that we examined the potential mediating effect of intolerance of uncertainty on GAD-specific symptoms only. There is evidence, however, that intolerance of uncertainty is also related to depressive symptoms (Dugas, Schwartz, & Francis, 2004). Given that changes in depressive symptoms also occurred, although to a lesser degree than changes in GAD symptoms, future research

should examine the possibility that changes in intolerance of uncertainty might also mediate changes in depressive symptoms during treatment for GAD. In fact, we cannot yet rule out the possibility that changes in intolerance of uncertainty actually mediate changes in depressive symptoms, which might then lead to subsequent reductions in GAD-specific symptoms. Ideally, future studies would assess the relative magnitude of the indirect effects of changes of intolerance of uncertainty on depressive symptoms, worry, and somatic anxiety individually. In addition, it would be interesting to determine whether the magnitude of the indirect effects in these models was moderated by the presence of co-morbid anxiety or mood disorders.

The study described here examined only one of four cognitive variables that are proposed to play a role in the maintenance of GAD symptoms in the cognitive model developed by Dugas and colleagues (1998). However, when mediated effects were found in the current study, these effects only accounted for some of the total effect of time on the outcome variable. Future research is needed to determine whether the other three cognitive variables in the theoretical model also mediate GAD symptom change during treatment (i.e., positive beliefs about worry, negative problem orientation, and cognitive avoidance). Multilevel modeling techniques can also be used to determine whether there is significant inter-individual variability in the extent to which particular mediators function to reduce GAD symptoms. In addition, future studies would also benefit from including measures of the mechanisms of symptom change proposed within other empirically-supported theoretical models of GAD (e.g., Mennin, Heimberg, Turk, & Fresco, 2005; Wells, 2005). Comparisons of the interrelations between theoretical constructs and their impact on symptom change might allow us to identify the theoretical

constructs that are similarly or differently related to GAD symptom change, and might also lead to further refinements of the current CBT protocol.

One final limitation relates to the size of the sample in the analyses presented here. The decision to include 51 individuals in the sample was based on an effort to maximize the number of assessment points available for the IUS mediation analyses, while also maximizing the number of participants in the sample. Although this decision was made primarily for pragmatic reasons, it should be noted that a sample of 51 individuals is small by some conventional standards. Fritz and MacKinnon (2007) provide, for instance, guidelines to researchers when selecting sample sizes for mediation analyses. These guidelines are based on effect size estimates of the *a* and *b* paths that are estimated in mediation models and on the assumption that a power of .80 is desired. They suggest that if moderate effect sizes are expected for the paths of the mediation models (i.e., 13% variance explained according to Cohen's (1988) suggestions for moderate effects), a sample of 74 individuals would be required when using asymmetric confidence intervals to detect the presence of mediation, and a sample of 90 participants would be required when using the less powerful Sobel test to detect mediation.

Given the small sample size in the current study, we therefore suggest that the results presented here be considered preliminary in nature. However, it should also be noted that the suggestions made by Fritz and MacKinnon were not made specifically for clinical process studies. In fact, when one examines the sample sizes typical for researchers conducting longitudinal multilevel mediation analyses within a clinical context, a sample of 51 is well within the reported range of sample sizes, which tend to have samples ranging from approximately 30 or 40 participants (e.g., Meuret, Rosenfield,

Hofmann, Suvak, & Roth, 2009; Teachman, Marker, Smith-Janik, & Shannon, 2008) to 90 participants (Hofmann, Meuret, Rosenfield, Suvak, Barlow, Gorman et al., 2007). While many of these studies may have been underpowered, it is also the case that significant mediation effects were found, as was the case in the current study. Nonetheless, it would be of interest to determine whether the findings in the current study could be replicated in larger samples of individuals with GAD.

The goal of the current study was to add to the growing volume of treatment process literature in which researchers are identifying the nature and predictors of symptom change during psychological treatments. The CBT protocol developed by Dugas and colleagues is efficacious. However, we are just beginning to identify the processes that lead to symptom change, and many questions remain. Nonetheless, it is hoped that the analyses described here will provide a step towards a better understanding of the nature of GAD symptom change during a treatment that targets worry primarily, and insight into the precise role that specific beliefs about uncertainty play in bringing about symptom change during CBT for GAD.

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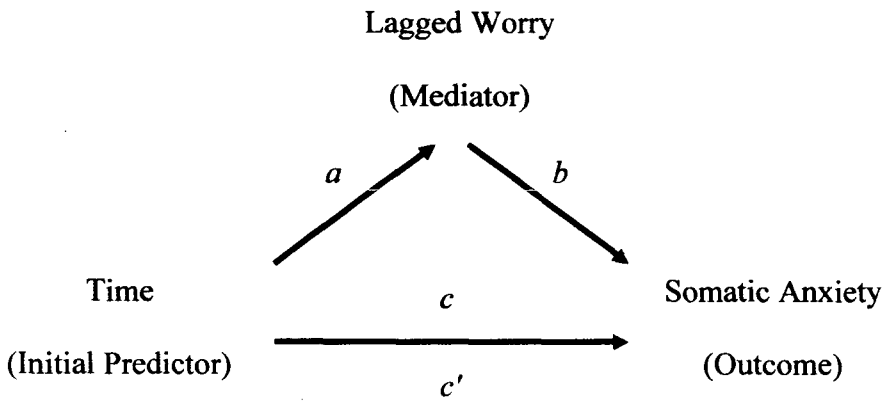
Footnotes

[1]We wish to thank Michael Suvak, PhD, Boston University, for his suggestions about how best to combine a time-lagged and mediation analysis in order to model all aspects of a mediation relationship statistically in a single analysis.

[2]The graph lines presented in Figure 3 include daily symptom ratings from all 51 participants in the sample. Note that only 5 of these 51 individuals provided more than 135 daily ratings from pre to posttreatment. As a result, the portion of Figure 3 that represents daily ratings beyond this point is based on the data provided by only these 5 individuals (i.e., by less than 10% of the sample). The apparent increase in the proportion of time spent worrying, experiencing somatic, and symptoms of depression from day 135 on is therefore not representative of the full sample.

Model 1a: Worry mediating change in somatic anxiety (hypothesized model)



Model 1b: Somatic anxiety mediating change in worry (alternate model)

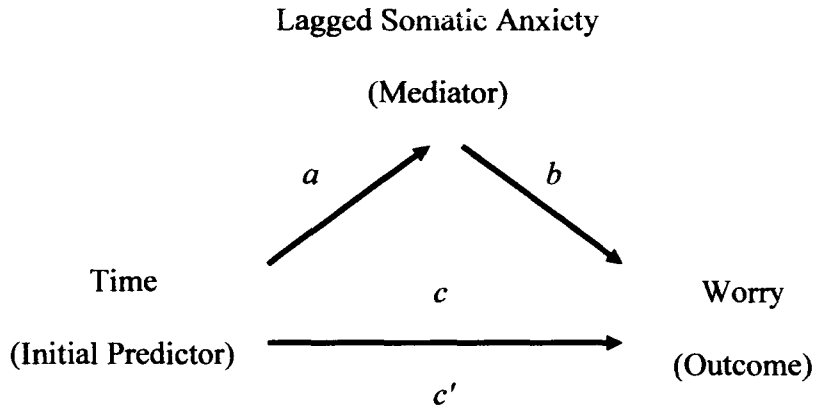
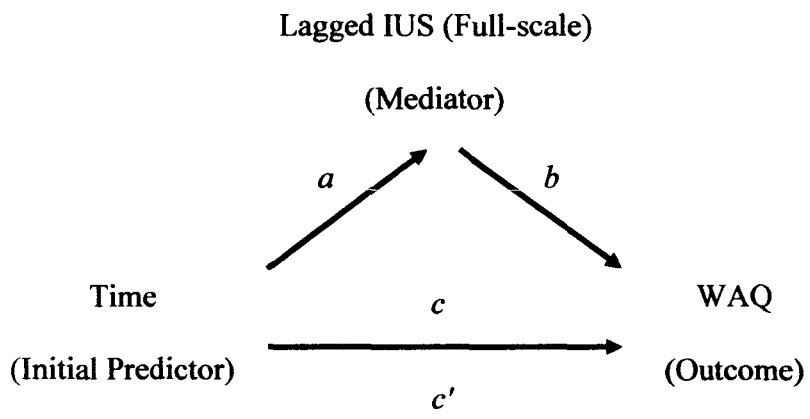


Figure 1. Patterns of change in daily symptoms during CBT: hypothesized and alternate mediation models. CBT = Cognitive-behavioural therapy.

Model 2a: IUS (Full-scale) mediating change in the WAQ (hypothesized model)



Model 2b: WAQ mediating change in the IUS (Full-scale) (alternate model)

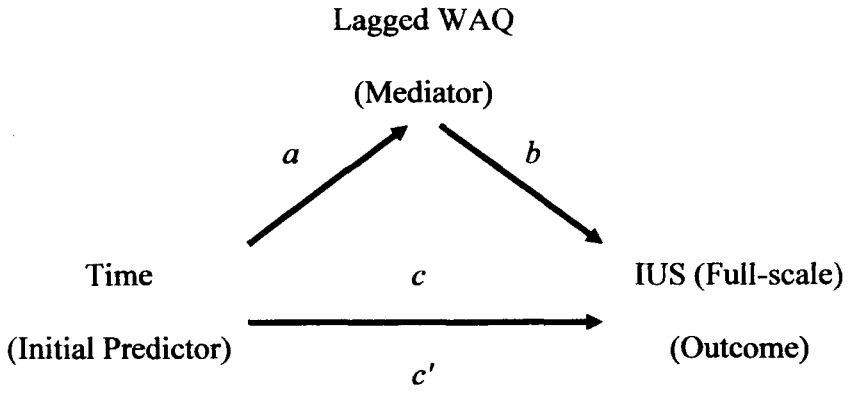
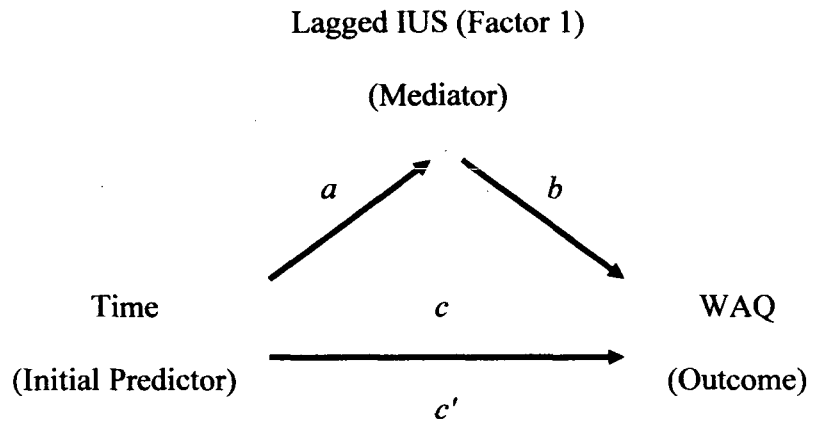


Figure 2. IU (Full-scale) as a mediator of symptom change from pretreatment to 6-month follow-up: hypothesized and alternate mediation models. IU = Intolerance of uncertainty; CBT = Cognitive-behavioural therapy; IUS = Intolerance of Uncertainty Scale; WAQ = Worry and Anxiety Questionnaire. **Figure continues on next page.**

Model 3a: IUS (Factor 1) mediating change in the WAQ (hypothesized model)



Model 3b: WAQ mediating change in the IUS (Factor 1) (alternate model)

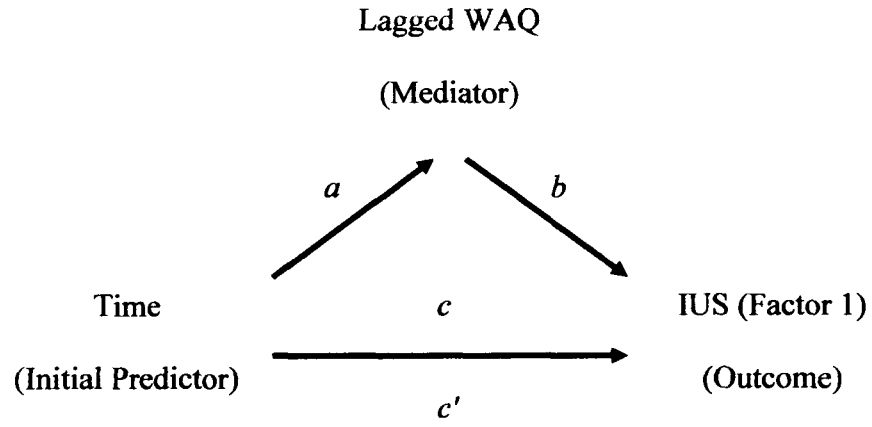
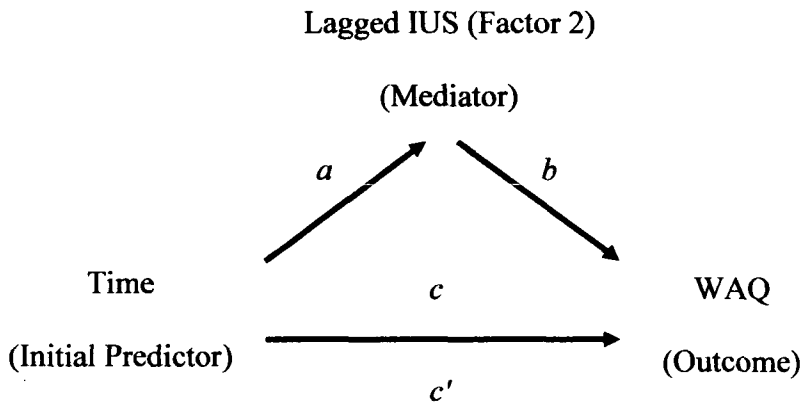


Figure 2. IU (Factor 1) as a mediator of symptom change from pretreatment to 6-month follow-up: hypothesized and alternate mediation models. IU = Intolerance of uncertainty; CBT = Cognitive-behavioural therapy; IUS = Intolerance of Uncertainty Scale; WAQ = Worry and Anxiety Questionnaire. **Figure continues on next page.**

Model 3c: IUS (Factor 2) mediating change in the WAQ (hypothesized model)



Model 3d: WAQ mediating change in the IUS (Factor 2) (alternate model)

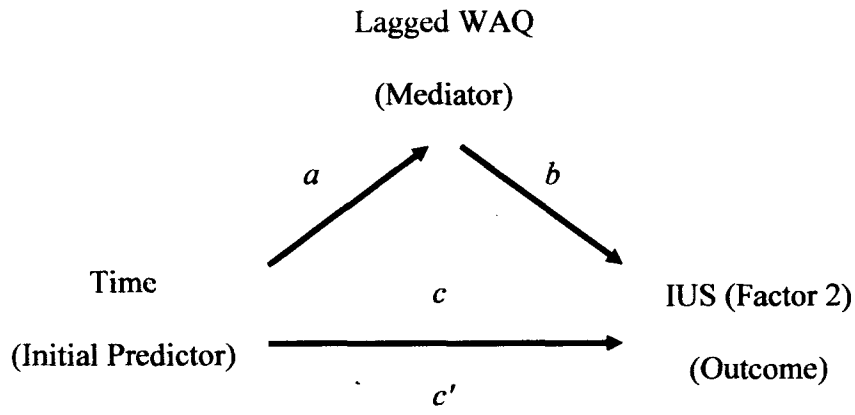


Figure 2. IU (Factor 2) as a mediator of symptom change from pretreatment to 6-month follow-up: hypothesized and alternate mediation models. IU = Intolerance of uncertainty; CBT = Cognitive-behavioural therapy; IUS = Intolerance of Uncertainty Scale; WAQ = Worry and Anxiety Questionnaire. **Continued from previous page.**

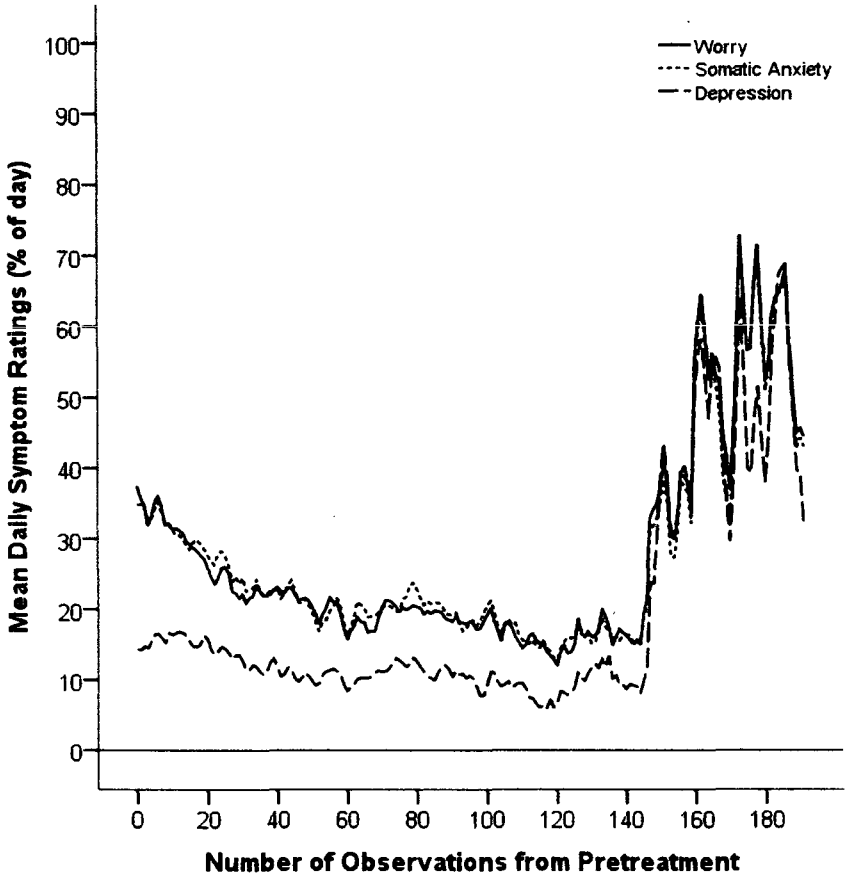


Figure 3. Percentage of each day spent experiencing worry, somatic anxiety, and feelings of depression during CBT. For ease of visual inspection, graph lines represent mean 3-day overlapping averages.[2]

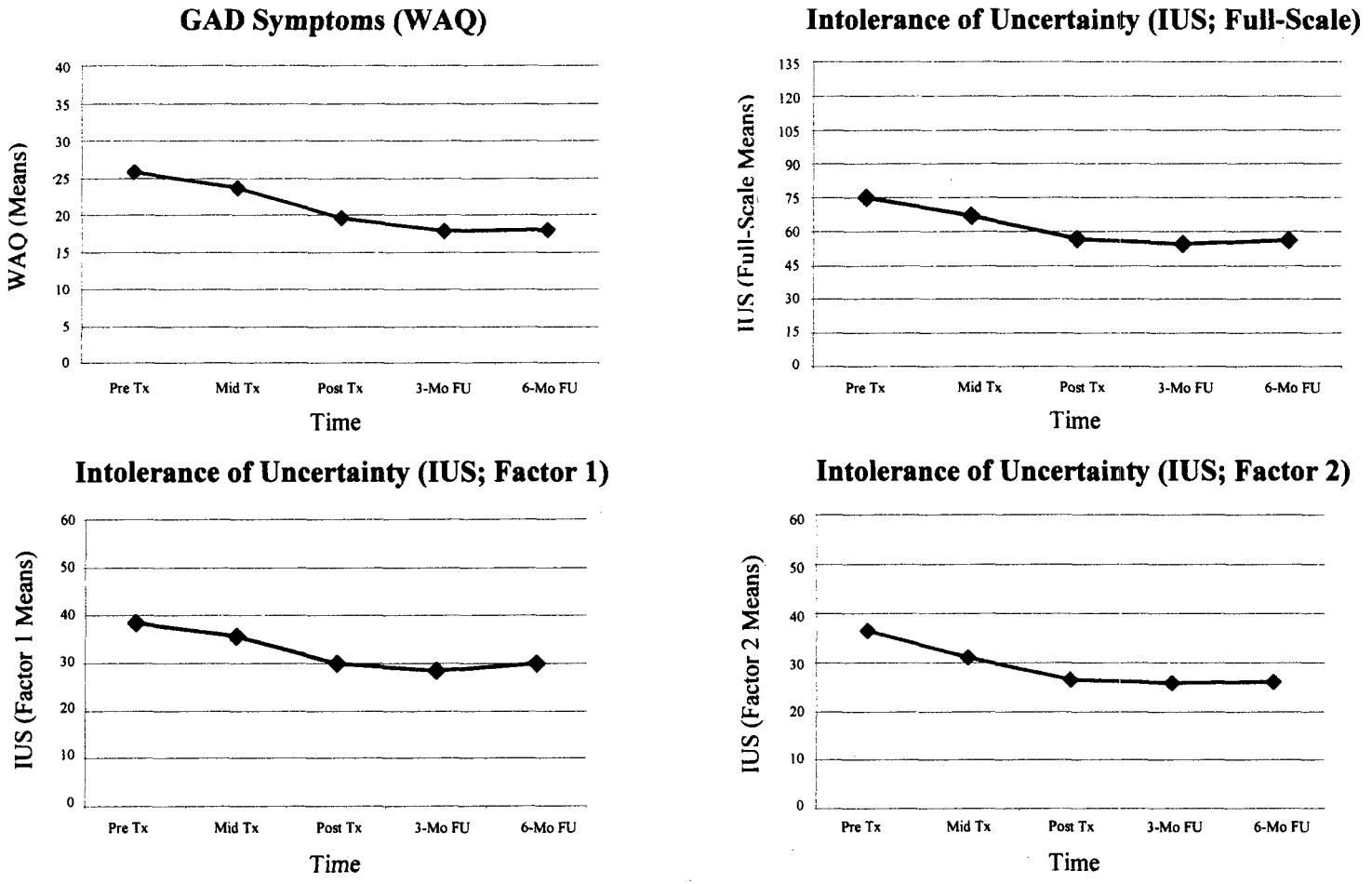


Figure 4. Mean GAD symptom and intolerance of uncertainty scores from pretreatment to 6-month follow-up. GAD = Generalized anxiety disorder; WAQ = Worry and Anxiety Questionnaire; IUS = Intolerance of Uncertainty Scale; Factor 1 = Uncertainty has negative self-referential and behavioural implications; Factor 2 = Uncertainty is unfair and spoils everything.

Table 1

Sample Clinical Characteristics at Pretreatment (N = 51)

	M	*SD*
ADIS (CSR)	5.91	0.75
Duration of GAD (years)	11.30	12.66
WAQ	25.77	3.10
PSWQ	63.63	7.46
BDI-II	17.13	10.13
STAI-T	54.27	7.05
IUS (Full-Scale)	75.02	22.12
IUS (Factor 1)	38.53	12.56
IUS (Factor 2)	36.50	10.36

Note. ADIS (CSR) = Clinician's Severity Rating of the Anxiety Disorders Interview Schedule for DSM-IV; GAD = Generalized anxiety disorder; WAQ = Worry and Anxiety Questionnaire; PSWQ = Penn State Worry Questionnaire; BDI-II = Beck Depression Inventory - II; STAI-T = State-Trait Anxiety Inventory - Trait Version; IUS = Intolerance of Uncertainty Scale.

Table 2

Regression Slopes Describing Change Over Time in Daily Ratings of Worry, Somatic Anxiety, and Depressive Symptoms during CBT

	Predictor	*B*	SE_B	*t*-ratio	*df*	Within-Person Variance (%)
Daily symptom ratings:						
Worry (% of day)						
	Time	-0.150*	0.02	-8.26	50	16.54
	$Time^2$	0.002*	0.00	3.50	50	+ 4.68
Somatic anxiety (% of day)						
	Time	-0.150*	0.02	-7.39	50	13.96
	$Time^2$	0.002*	0.00	3.17	50	+ 4.07
Symptoms of depression (% of day)						
	Time	-0.070*	0.02	-4.73	50	9.35
	$Time^2$	-0.001	0.00	1.16	50	+ 3.47

Note. CBT = Cognitive-behavioural therapy. *$p < .05$.

Table 3

Patterns of Change in Daily Symptoms during CBT: Hypothesized and Alternate Mediation Models

Step	Path	Predictor	Outcome	*B*	SE_B	*t*-ratio	*df*
Model 1a: Mediator = Lagged Worry; Outcome = Somatic Anxiety							
1	*c*	Time	Som. Anx.	-0.15*	0.02	-7.39	50
2	*a*	Time	Worry (L)	-0.15*	0.02	-8.16	50
3	*b*	Worry (L)	Som. Anx.	0.45*	0.03	13.73	50
	c'	Time	Som. Anx.	-0.08*	0.02	-5.80	50
Sobel test statistic = -6.99 (*SE* = 0.01), $p < 0.001$; **95% CI for *ab*** = -0.07 [-0.09, -0.05]; **Percent Mediation = 47.67%**							
Model 1b: Mediator = Lagged Somatic Anxiety; Outcome = Worry							
1	*c*	Time	Worry	-0.15*	0.02	-8.26	50
2	*a*	Time	Som. Anx. (L)	-0.15*	0.02	-7.28	50
3	*b*	Som. Anx. (L)	Worry	0.37*	0.03	13.78	50
	c'	Time	Worry	-0.10*	0.01	-7.37	50
Sobel test statistic = -6.24 (*SE* = 0.01), $p < 0.001$; **95% CI for *ab*** = -0.05 [-0.07, -0.04]; **Percent Mediation = 36.40%**							

Note. CBT = Cognitive-behavioural therapy; (L) indicates that a variable was lagged by one day. *$p < .05$.

Table 4

Regression Slopes Describing Change Over Time in GAD Symptoms and IU from Pretreatment to 6-Month Follow-up

	Predictor	*B*	SE_B	*t*-ratio	*df*	Within-Person Variance (%)
WAQ						
	Time	-2.14*	0.16	-13.09	50	65.30
	$Time^2$	0.46*	0.09	4.93	50	+ 6.01
IUS (Full-scale)						
	Time	-5.04*	0.73	-6.88	50	47.58
	$Time^2$	1.98*	0.43	4.58	50	+ 17.32
IUS Factor 1						
	Time	-2.42*	0.44	-5.46	50	42.92
	$Time^2$	0.91*	0.25	3.59	50	+ 13.28
IUS Factor 2						
	Time	-2.63*	0.31	-8.35	50	48.27
	$Time^2$	1.07*	0.20	5.43	50	+ 18.97

Note. IU = Intolerance of uncertainty; WAQ = Worry and Anxiety Questionnaire; IUS = Intolerance of Uncertainty Scale. *$p < .05$.

Table 5

IU as a Mediator of Symptom Change from Pretreatment to 6-Month Follow-Up: Hypothesized and Alternate Mediation Models

Step	Path	Predictor	Outcome	*B*	SE_B	*t*-ratio	*df*
Model 2a: Mediator = Lagged IUS (Full-scale); Outcome = WAQ							
1	*c*	Time	WAQ	-1.94*	0.18	-10.89	50
2	*a*	Time	IUSfs (L)	-7.16*	0.92	-7.76	50
3	*b*	IUSfs (L)	WAQ	0.08*	0.02	5.17	50
	c'	Time	WAQ	-1.43*	0.21	-6.65	50
Sobel test statistic = -4.28 (*SE* = 0.13), $p < 0.001$; **95% CI for *ab*** = -0.56 [-0.86, -0.31]; **Percent Mediation = 23.13%**							
Model 2b: Mediator = Lagged WAQ; Outcome = IUS (Full-scale)							
1	*c*	Time	IUSfs	-3.50*	0.86	-4.08	50
2	*a*	Time	WAQ (L)	-2.78*	0.22	-12.77	50
3	*b*	WAQ (L)	IUSfs	1.18*	0.27	4.37	50
	c'	Time	IUSfs	-0.58*	0.27	-2.15	50
Sobel test statistic = -4.13 (*SE* = 0.78), $p < 0.001$; **95% CI for *ab*** = -3.28 [-4.97, -1.74]; **Percent Mediation = 73.22%**							

Note. IU = Intolerance of uncertainty; IUS = Intolerance of Uncertainty Scale; WAQ = Worry and Anxiety Questionnaire. (L) indicates that a variable was lagged by one assessment point. *$p < .05$. **Table continues on next page.**

Table 5 (Continued from previous page)

IU as a Mediator of Symptom Change from Pretreatment to 6-Month Follow-Up: Hypothesized and Alternate Mediation Models

Step	Path	Predictor	Outcome	*B*	SE_B	*t*-ratio	*df*
Model 3a: Mediator = Lagged IUS (Factor 1); Outcome = WAQ							
1	c	Time	WAQ	-1.94*	0.18	-10.89	50
2	a	Time	IUSf1 (L)	-3.55*	0.55	-6.43	50
3	b	IUSf1 (L)	WAQ	0.09*	0.02	3.83	50
	c'	Time	WAQ	-1.66*	0.21	-7.99	50
Sobel test statistic = -3.26 (*SE* = 0.10), $p < 0.001$; **95% CI for *ab*** = -0.32 [-0.52, -0.14]; **Percent Mediation = 15.87%**							
Model 3b: Mediator = Lagged WAQ; Outcome = IUS (Factor 1)							
1	c	Time	IUSf1	-1.88*	0.54	-3.49	50
2	a	Time	WAQ (L)	-2.78*	0.22	-12.77	50
3	b	WAQ (L)	IUSf1	0.91*	0.15	6.06	50
	c'	Time	IUSf1	0.40	0.66	0.61	50
Sobel test statistic = -5.46 (*SE* = 0.46), $p < 0.001$; **95% CI for *ab*** = -2.53 [-3.55, -1.62]; **Percent Mediation = 96.78%**							

Note. IU = Intolerance of uncertainty; IUS = Intolerance of Uncertainty Scale; WAQ = Worry and Anxiety Questionnaire. (L) indicates that a variable was lagged by one assessment point. *$p < .05$.

Table 5 (Continued from previous page)

IU as a Mediator of Symptom Change from Pretreatment to 6-Month Follow-Up: Hypothesized and Alternate Mediation Models

Step	Path	Predictor	Outcome	*B*	SE_B	*t*-ratio	*df*
Model 3c: Mediator = Lagged IUS (Factor 2); Outcome = WAQ							
1	c	Time	WAQ	-1.94*	0.18	-10.89	50
2	a	Time	IUSf2 (L)	-3.61*	0.40	-8.95	50
3	b	IUSf2 (L)	WAQ	0.22*	0.04	5.84	50
	c'	Time	WAQ	-1.21*	0.23	-5.28	50
Sobel test statistic = -4.87 (*SE* = 0.16), $p < 0.001$; **95% CI for *ab*** = -0.79 [-1.13, -0.49]; **Percent Mediation = 37.15%**							
Model 3d: Mediator = Lagged WAQ; Outcome = IUS (Factor 2)							
1	c	Time	IUSf2	-1.61*	0.35	-4.62	50
2	a	Time	WAQ (L)	-2.78*	0.22	-12.77	50
3	b	WAQ (L)	IUSf2	0.26†	0.13	1.97	50
	c'	Time	IUSf2	-1.02†	0.52	-1.99	50
Sobel test statistic = -1.94 (*SE* = 0.37), $p = 0.053$; **95% CI for *ab*** = -0.71 [-1.46, -0.01]); **Percent Mediation = 38.22%**							

Note. IU = Intolerance of uncertainty; IUS = Intolerance of Uncertainty Scale; WAQ = Worry and Anxiety Questionnaire. (L) indicates that a variable was lagged by 1 assessment point. *$p < .05$. †$p < .10$.

Table 6

Factor Loadings for the Confirmatory Factor Analysis of the IUS (N = 271)

Item	Factor 1	*E*
22. Being uncertain means that I lack confidence	0.88	0.84
17. Uncertainty makes me vulnerable, unhappy, or sad	0.87	0.49
20. The smallest doubt can stop me from acting	0.85	0.51
12. When it's time to act, uncertainty paralyses me	0.84	0.52
15. When I am uncertain I can't function very well	0.84	0.38
14. When I am uncertain, I can't go forward	0.83	0.42
13. Being uncertain means that I am not first rate	0.82	0.58
9. Uncertainty keeps me from living a full life	0.79	0.65
16. Unlike me, others always seem to know where they are going with their lives	0.78	0.90
1. Uncertainty stops me from having a firm opinion	0.73	0.75
23. I think it's unfair that other people seem sure about their future	0.71	0.14
24. Uncertainty keeps me from sleeping soundly	0.69	1.03
25. I must get away from all uncertain situations	0.69	0.63
3. Uncertainty makes life intolerable	0.64	0.73
2. Being uncertain means that a person is disorganized	0.54	0.76

Note. IUS = Intolerance of Uncertainty Scale; Factor 1 = Uncertainty has negative behavioural and self-referent implications; *E* = standardized error variance. All factor loadings are significant when $\alpha = .05$. **Table continues on next page.**

Table 6 (Continued from previous page)

Factor Loadings for the Confirmatory Factor Analysis of the IUS (N = 271)

Item	Factor 2	*E*
10. One should always look ahead so as to avoid surprises	0.89	0.73
18. I always want to know what the future has in store for me	0.87	0.64
19. I can't stand being taken by surprise	0.85	0.52
11. A small unforeseen event can spoil everything even with the best of planning	0.81	0.57
21. I should be able to organize everything in advance	0.81	0.68
5. My mind can't be relaxed if I don't know what will happen tomorrow	0.79	0.70
7. Unforeseen events upset me greatly	0.77	0.64
8. It frustrates me not having all the information I need	0.75	0.69
26. The ambiguities in life stress me	0.74	0.54
6. Uncertainty makes me uneasy, anxious, or stressed	0.71	0.51
27. I can't stand being undecided about my future	0.70	0.88
4. It's unfair not having any guarantees in life	0.57	1.07

Note. See Appendix A for a description of the IUS confirmatory factor analysis. IUS = Intolerance of Uncertainty Scale; Factor 2 = Uncertainty is unfair and spoils everything; *E* = standardized error variance. All factor loadings are significant when $\alpha = .05$.

Appendix A

A Confirmatory Factor Analysis of the *Intolerance of Uncertainty Scale*

in a Clinical Sample

The *Intolerance of Uncertainty Scale* (IUS) was developed by Freeston et al. (1994) to assess the negative beliefs about uncertainty that individuals with GAD appear to hold. Although the IUS has typically been administered in treatment outcome studies as a single-factor measure (e.g., Dugas et al., 2010; Ladouceur, Dugas et al., 2000), a number of factor analyses have been conducted since its development. These analyses suggest that the IUS may in fact assess distinct subsets of negative beliefs about uncertainty. For example, in an exploratory factor analysis conducted on the original French version of the measure, Freeston et al. (1994) identified five negative beliefs about uncertainty in a non-clinical sample of worriers. These beliefs included the idea that (a) uncertainty is unacceptable and should be avoided, (b) being uncertain reflects badly on a person, (c) uncertainty is frustrating, (d) uncertainty causes stress, and (e) uncertainty prevents action. A more recent exploratory factor analysis was conducted on an English translation of the IUS by Buhr and Dugas (2002). This analysis identified four distinct negative beliefs about uncertainty, including the idea that (a) uncertainty prevents a person from being able to act, (b) uncertainty is stressful and upsetting, (c) unexpected events are negative and should be avoided, and (d) being uncertain about the future is unfair. However, subsequent factor analyses have failed to replicate either a four or five-factor structure (e.g., Norton, 2005; Carleton, Norton, & Asmundson, 2007).

In order to clarify the factor structure of the IUS, Sexton and Dugas (2009) conducted exploratory and confirmatory factor analyses on the English version of the measure. These studies involved the largest non-clinical samples to date (i.e., $N = 1,230$ and $N = 1,221$, respectively) and were consistent in identifying, and confirming, a two-factor solution. The first factor was found to be represented by 15 of the 27 items on the

IUS, and appeared to reflect the belief that uncertainty has negative behavioural and self-referential implications (e.g., "When it's time to act, uncertainty paralyses me" and "Being uncertain means that I lack confidence"). The second factor appeared to be represented by the 12 remaining items on the IUS and seemed to reflect the belief that uncertainty is unfair and spoils everything (e.g., "It's unfair not having any guarantees in life" and "One should always look ahead so as to avoid surprises").

Despite the evidence of a two-factor structure for the IUS, however, the analyses conducted by Sexton and Dugas (2009) were conducted in non-clinical samples. The two-factor structure of the IUS has not yet been confirmed in a clinical sample. As a result, we conducted a preliminary confirmatory factor analysis on a clinical sample of individuals ($N = 271$) who met DSM-IV diagnostic criteria for GAD and who had volunteered to participate in one of several treatment outcome studies examining the efficacy of CBT for GAD. All participants had completed the French version of the IUS prior to the start of their first treatment sessions, which were conducted sometime between 2003 and 2009. The sample consisted of 86 males and 185 females, all of whom were Francophone, and who had an average age of 44.96 years ($SD = 11.78$).

The confirmatory factor analysis reported here was conducted using the structural equation modeling program EQS, Version 6.1 (Bentler, 1995; Bentler & Wu, 1995). Before proceeding with the analysis, the total IUS scores and all 27 IUS items were assessed for skewness and kurtosis. Although the total IUS scores were normally distributed, two of the 27 items were significantly skewed. There was also a significant degree of kurtosis observed among the IUS items (Mardia's coefficient of multivariate kurtosis = 70.68, normalized estimate $Z = 14.70$). As a result, the elliptical (ERLS)

method of factor extraction was employed, as this method is preferred for non-normally distributed data and is less prone to error in analyses involving small samples (Kline, 1998). Given that the two proposed factors were thought to represent different aspects of the same construct, they were allowed to co-vary in our analysis.

All items on the IUS loaded significantly onto their respective factor, with factor loadings ranging from 0.54 to 0.89, and the correlation between the factors was $r = 0.78$ (see Table 6). The two-factor solution generally met conventional standards for good model fit. The independence model chi square test was significant ($\chi^2(351) = 16{,}044.03$, $p < .001$), indicating that there was at least some relationship among the IUS items to be analyzed. However, the model chi square test, which assesses the degree of fit between the sample covariance matrix and the estimated population covariance matrix, was also significant (χ^2 (323) = 1191.19, $p < .001$). Ideally, this goodness of fit index should be non-significant. However, this index has also been found to be unreliable because it is closely associated with sample size (Tabachnick & Fidell, 2007). As a result, other fit indices were used to evaluate the two-factor model. The model produced a Bentler-Bonett normed fit index (NFI) of .93 (NFIs > .90 are indicative of good model fit; Tabachnick & Fidell, 2007), a comparative fit index (CFI) of .95 (CFIs > .95 indicate good fit; Tabachnick & Fidell, 2007), and a standardized root-mean-square residual (SRMR) of .078 (SRMRs < .08 are recommended; Hu & Bentler, 1999), although the root-mean-square error of approximation (RMSEA) was higher than ideal at .10 (RMSEAs < .06 are recommended; Bentler & Wu, 1999). However, this final index may produce values that are overestimates in small samples (Tabachnick & Fidell, 2007).

The preliminary confirmatory factor analysis described here was conducted on a small but adequately sized sample, by conventional standards (Tabachnick & Fidell, 2007). Despite the small sample size, however, a two-factor solution generally appeared to be a good fit for the data. The fit indices reported here are similar to those reported by Sexton and Dugas (2009) in their much larger non-clinical sample (i.e., $N = 1{,}221$; NFI = .96; CFI = .97; SRMR = .05; RMSEA = .07). For comparative purposes, however, and given that the IUS has most frequently been used as a single-factor measure, we also assessed a single-factor solution. The model chi square test of the single-factor solution indicated a poor model fit ($\chi^2(324) = 1876.98, p < .001$), and this solution also proved to be less than adequate on several additional indices of goodness-of-fit (i.e., Bentler-Bonett NFI = .88; CFI = .90; SRMR = .08; RMSEA = .13). When the model chi square estimates were compared directly, the two-factor solution provided a superior fit to the data, $\Delta\chi^2(1) = 685.79, p < .001$. On the basis of this preliminary analysis, we felt confident in proceeding with mediation analyses that examined the potential role that both the full-scale IUS, and its two factors, might play in bringing about change over time in GAD symptoms during cognitive-behavioural therapy.

Appendix B

RECRUITMENT ADVERTISEMENT

Êtes-vous une personne inquiète?

Le Laboratoire des troubles anxieux de l'Université Concordia en collaboration avec la Clinique des troubles anxieux de l'Hôpital du Sacré-Cœur de Montréal est à la recherche de personnes qui s'inquiètent de façon excessive ou exagérée pour participer à une étude évaluant un traitement psychologique ayant déjà fait preuve de son efficacité.

Si vous avez entre 18 et 65 ans et que vous êtes en bonne santé physique, vous pourriez être éligible pour participer à l'étude.

Pour plus d'information, veuillez téléphoner au : 514 848-2424, poste 5085

Laboratoire des troubles anxieux
Directeur : Michel Dugas, Ph.D., psychologue

www.concordia.ca

Appendix C

CONSENT FORMS FOR STUDY PARTICIPATION

HÔPITAL DU SACRÉ-COEUR
DE MONTRÉAL
HSCM

UNIVERSITÉ Concordia UNIVERSITY

Formulaire d'information et de consentement téléphonique

(1[e] partie : Évaluation de l'admissibilité)

Titre de l'étude : La thérapie cognitivo-comportementale pour le trouble d'anxiété généralisée : Impact du traitement de l'information sur l'efficacité thérapeutique à court et à long terme

Chercheur principal : Michel Dugas, Ph.D. Professeur titulaire, Université Concordia
Chercheur, Centre de recherche HSCM

INFORMATION

A. BUT DE L'ÉTUDE

Le but de cette étude est d'évaluer l'impact des biais de traitement de l'information sur l'efficacité à court et à long terme de la thérapie cognitivo-comportementale pour le trouble d'anxiété généralisée (TAG). La première partie de l'étude consiste à évaluer de façon préliminaire la nature et la sévérité de vos symptômes anxieux afin de déterminer si vous rencontrez les critères de sélection pour passer à la seconde étape d'évaluation et par la suite recevoir le traitement pour le trouble d'anxiété généralisée.

B. PROCÉDURES

Dans un premier temps, vous participerez à une entrevue d'évaluation téléphonique (durée 1h30) avec une psychologue de l'équipe.

S'il semble que vous rencontrez les critères de sélection de l'étude, vous serez référé(e) à la Clinique des troubles anxieux de l'Hôpital du Sacré-Cœur de Montréal, où vous serez évalué(e) à nouveau par un(e) psychiatre de notre équipe. Cette évaluation se déroule en personne et est d'une durée d'une heure trente environ. Après cette rencontre, les membres de l'équipe de recherche (psychologues, psychiatres et chercheur principal) se réunissent pour discuter et vérifier si vous rencontrez bien les critères requis pour l'étude. Nous vous ferons ensuite part de la décision de l'équipe.

Si vous rencontrez les critères pour être inclus(e) dans l'étude, vous aurez à signer un autre formulaire de consentement concernant la suite de l'étude.

C. RISQUES ET BÉNÉFICES

1. Risques, effets secondaires et désagréments

Il n'est pas impossible que certaines questions provoquent un léger malaise à court terme (possiblement en vous faisant réfléchir à vos difficultés). Par contre, cette entrevue a déjà été utilisée à plusieurs reprises auprès des personnes anxieuses et les malaises sont rares. Si cela vous arrive, nous vous prions d'en discuter avec nous.

2. Bénéfices et avantages

En participant à cette étude, vous bénéficierez d'une évaluation détaillée de votre état. Évidemment, si vous rencontrez les critères de sélection pour l'étude de traitement, vous recevrez une psychothérapie efficace pour le traitement du TAG. Parallèlement, vous pourrez contribuer à l'avancement des connaissances en participant à cette étude.

D. CONDITIONS DE PARTICIPATION

1. Versement d'une indemnité

Vous ne recevrez aucune rémunération pour votre participation à ce volet d'évaluation.

2. Confidentialité

Tous les renseignements recueillis à votre sujet demeureront strictement confidentiels, dans les limites prévues par la loi, et vous ne serez identifié(e) que par un code.

3. Indemnisation en cas de préjudice

En acceptant de participer à cette étude, vous ne renoncez à aucun de vos droits et vous ne libérez pas les chercheurs, l'organisme subventionnaire (Instituts de recherche en santé du Canada) ou les établissements impliqués de leurs responsabilités légales et professionnelles.

4. Participation volontaire et retrait de l'étude

Votre participation à cette étude est volontaire. Vous êtes donc libre de refuser d'y participer. Vous pouvez également vous retirer de l'étude à n'importe quel moment, sans avoir à donner de raisons, en faisant connaître votre décision au chercheur ou à l'un des membres de l'équipe de recherche.

CONSENTEMENT

- Je comprends que je donne mon consentement verbal pour que l'équipe de recherche évalue si je rencontre les critères de sélection de l'étude.
- Je comprends que je peux retirer mon consentement et interrompre ma participation à tout moment, sans conséquences négatives.
- Je comprends que ma participation à cette étude est CONFIDENTIELLE (c.-à-d. les membres de l'équipe connaissent mon identité mais ne la révéleront pas).

J'AI ÉCOUTÉ ATTENTIVEMENT CE QUI M'A ÉTÉ LU ET JE COMPRENDS LA NATURE DE CETTE ÉTUDE: OUI_____ NON_____

JE CONSENS DONC VERBALEMENT, DE FAÇON LIBRE ET VOLONTAIRE À PARTICIPER À L'ÉVALUATION TÉLÉPHONIQUE ET S'IL Y A LIEU À LA RENCONTRE AVEC UN(E) PSYCHIATRE DE L'ÉQUIPE :

OUI_____ NON_____

NOM DU PARTICIPANT : _____ **DATE :** _____

NOM DU MEMBRE DE L'ÉQUIPE : _____ **HEURE :** _____

SIGNATURE _____ **DATE** _____

Si vous avez des questions à poser au sujet de cette étude, vous pouvez contacter en tout temps la direction générale de l'Hôpital du Sacré-Cœur de Montréal au (514) 338-2222, poste 3581.

HÔPITAL DU SACRÉ-COEUR
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FORMULAIRE D'INFORMATION ET DE CONSENTEMENT

Titre de l'étude: La thérapie cognitivo-comportementale pour le trouble d'anxiété généralisée : Impact du traitement de l'information sur l'efficacité thérapeutique à court et à long terme

Chercheur: Michel Dugas, Ph.D. (psychologie)

Chercheur régulier, Centre de recherche, HSCM

Psychologue, Clinique des troubles anxieux, HSCM

Professeur titulaire, Département de psychologie, Université Concordia

Tél : 514-338-4201 ou 514-848-2424 (poste 2215)

Courriel : Michel.Dugas@concordia.ca

Co-chercheurs: Adam Radomsky, Ph.D. (psychologie)

Professeur adjoint, Département de psychologie, Université Concordia

Tél : 514-848-2424 (poste 2202)

Natalie Phillips, Ph.D. (psychologie)

Professeur agrégé, Département de psychologie, Université Concordia

Tél : 514-848-2424 (poste 2218)

William Bukowski, Ph.D. (psychologie)

Professeur titulaire, Département de psychologie, Université Concordia

Tél : 514-848-2424 (poste 2184)

Julie Turcotte, M.D. (psychiatrie)

Professeur adjoint, Département de psychiatrie,

Faculté de Médecine, Université de Montréal

Psychiatre, Clinique des troubles anxieux, HSCM

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Tél : 514-338-4201

Organisme

de subvention : Instituts de recherche en santé du Canada

410 avenue Laurier ouest, 9ème étage, indice de l'adresse 4209A,

Ottawa, Ontario, K1A 0W9

INFORMATION

1. Nature et objectif de l'étude

Nous savons aujourd'hui que les personnes atteintes de troubles anxieux ont certains biais dans leur façon de traiter l'information provenant de leur environnement. Par exemple, les personnes anxieuses tendent à porter leur attention plus rapidement à certains « signes de danger » et à interpréter certaines situations ambiguës de façon menaçante. Par contre, nous ne savons pas si l'ampleur de ces biais affecte la réponse à la psychothérapie. En d'autres mots, nous ne savons pas si les personnes anxieuses qui présentent des biais plus importants dans leur façon de traiter l'information répondent différemment aux interventions psychologiques.

Le but de cette étude est d'évaluer l'impact des biais de traitement de l'information sur l'efficacité à court et à long terme de la thérapie cognitivo-comportementale pour le trouble d'anxiété généralisée (TAG). Plus particulièrement, nous voulons : (1) évaluer l'impact des biais « pré-thérapie » sur la réponse à cette thérapie; et (2) évaluer l'impact des biais « post-thérapie » sur le maintien des gains thérapeutiques suite à la thérapie. Afin d'évaluer l'ampleur des biais de traitement de l'information, nous nous proposons d'utiliser trois tâches informatiques qui sont expliquées ci-dessous.

Cent dix (110) adultes avec un diagnostic principal de trouble d'anxiété généralisée participeront à cette étude. Les participants seront recrutés à la Clinique des troubles anxieux de l'Hôpital du Sacré-Cœur de Montréal ou par le biais d'annonces placées dans le quotidien *La Presse*.

2. Déroulement de l'étude et méthodes utilisées

Les grandes lignes pour la suite de l'étude sont les suivantes : (1) évaluation pré-thérapie en deux rencontres; (2) thérapie cognitivo-comportementale administrée en 14 rencontres hebdomadaires; (3) évaluation post-thérapie en huit rencontres sur une période de 18 mois.

Premier volet : Évaluation pré-thérapie

Suite à l'évaluation de vos symptômes d'anxiété – entrevues téléphoniques et entrevue psychiatrique à la Clinique des troubles anxieux – nous avons déterminé que vous rencontrez les critères d'inclusion de cette étude. Vous participerez maintenant à une rencontre d'environ deux heures avec une psychologue de notre équipe (Isabelle Geninet, Pascale Harvey ou Amélie Seidah) – le but de cette rencontre est d'évaluer vos traits de personnalité ou votre façon habituelle de réagir aux événements de tous les jours. Au cours de cette rencontre, vous aurez aussi à compléter des questionnaires portant sur vos symptômes d'anxiété. Par la suite, vous aurez à participer à une dernière rencontre d'évaluation pendant laquelle vous ferez trois tâches sur un ordinateur et répondrez à des questionnaires. En ce qui concerne les tâches informatiques, vous ferez une tâche évaluant votre façon de porter attention à certains mots et deux tâches évaluant votre façon de comprendre certaines situations. Chacune des trois tâches prend environ 20 minutes à compléter. Vous répondrez ensuite à des questionnaires qui ont pour but d'évaluer votre état général. Cela vous prendra

environ 20 minutes pour répondre aux questionnaires. La durée totale de cette rencontre (directives, tâches informatiques, pause et questionnaires) sera d'environ une heure et demie.

Deuxième volet : Thérapie cognitivo-comportementale

En participant à cette étude, vous recevrez une psychothérapie efficace pour le traitement du TAG. Cette thérapie, de type cognitivo-comportementale, pourrait vous aider à comprendre et à changer les comportements et pensées qui contribuent à vos difficultés. La durée de cette thérapie est de quatre mois (14 rencontres hebdomadaires de 50 minutes) et elle vous sera administrée par une des psychologues de notre équipe. Entre les rencontres, vous aurez des lectures à faire et des exercices à pratiquer.

Troisième volet : Évaluation post-thérapie

Afin d'évaluer les effets de la psychothérapie à long terme, vous serez évalué(e) à sept reprises, sur une période de 18 mois, suite à votre thérapie. Immédiatement après la thérapie, vous participerez à deux rencontres d'évaluation (rencontre 1 : entrevue diagnostique et questionnaires; rencontre 2 : tâches à l'ordinateur et questionnaires). Par la suite, vous participerez à une rencontre d'évaluation (entrevue diagnostique et questionnaires) à six reprises, c'est-à-dire aux relances de 3, 6, 9, 12, 15 et 18 mois.

3. Risques, effets secondaires et désagréments

Évaluations

Il n'est pas impossible que certaines tâches ou certains questionnaires provoquent un léger malaise à court terme (possiblement en vous faisant réfléchir à vos difficultés). Par contre, ces tâches et questionnaires ont déjà été utilisés à plusieurs reprises auprès des personnes anxieuses et les malaises sont rares. Si cela vous arrive, nous vous prions d'en discuter avec la professionnelle de recherche ou avec votre thérapeute.

Psychothérapie

Il est possible que quelques uns des exercices prescrits par votre psychologue provoquent certains malaises à court terme. Ceux-ci sont temporaires et disparaissent habituellement avec la pratique répétée de ces exercices.

Si vous recevez un médicament de votre médecin ou de votre psychiatre au moment du début de l'étude, cela demeure la responsabilité de ce dernier pendant la durée du traitement. Cependant, nous vous demandons seulement de ne pas augmenter le dosage de votre médication ou de modifier le type de médicament sans en avertir préalablement votre thérapeute.

4. Bénéfices et avantages

Tel que mentionné précédemment, en participant à cette étude, vous recevrez une psychothérapie efficace pour le traitement du TAG. De plus, cette thérapie vous sera offerte par des psychologues qui sont des experts dans son application. Vous profiterez aussi d'une évaluation plus poussée de votre état, avec un suivi sur une période de 18 mois après la fin de la psychothérapie. Parallèlement, vous allez nous aider à mieux évaluer les facteurs qui influencent l'efficacité de cette thérapie et ainsi contribuer à l'avancement des connaissances en participant à cette étude.

5. Versement d'une indemnité

Vous ne recevrez aucune rémunération pour votre participation à la première partie de cette étude (évaluation pré-thérapie, psychothérapie et évaluation immédiatement après la thérapie). Par contre, vous recevrez une compensation de 30$ pour chacune des six rencontres de relance (3, 6, 9, 12, 15 et 18 mois après la fin de la psychothérapie). Donc, si vous vous présentez pour toutes les rencontres de relances, vous recevrez une indemnité de 180$.

6. Confidentialité

Tous les renseignements recueillis à votre sujet au cours de l'étude demeureront strictement confidentiels, dans les limites prévues par la loi, et vous ne serez identifié(e) que par un code. Les rencontres avec les

psychologues seront enregistrées sur cassettes audio afin de nous permettre d'évaluer la qualité des interventions offertes par celles-ci (les cassettes seront aussi identifiées par un code). Immédiatement après l'étude, toutes les cassettes seront détruites. Aucune publication ou communication scientifique résultant de cette étude ne renfermera quoi que ce soit qui puisse permettre de vous identifier.

Cependant, à des fins de contrôle du projet de recherche, votre dossier pourra être consulté par une personne mandatée par le comité d'éthique de la recherche de l'Hôpital du Sacré-Cœur ainsi que par des représentants de l'organisme de subvention (Instituts de recherche en santé du Canada). Tous ces organismes adhèrent à une politique de stricte confidentialité.

7. Indemnisation en cas de préjudice

Si vous deviez subir quelque préjudice que ce soit résultant de votre participation à cette étude, vous recevrez tous les soins médicaux nécessaires, sans frais de votre part. Toutefois, ceci ne vous empêche nullement d'exercer un recours légal en cas de faute reprochée à toute personne impliquée dans l'étude.

En acceptant de participer à cette étude, vous ne renoncez à aucun de vos droits ni ne libérez les chercheurs, l'organisme subventionnaire (Instituts de recherche en santé du Canada) ou les établissements impliqués de leurs responsabilités légales et professionnelles.

8. Participation volontaire et retrait de l'étude

Votre participation à cette étude est volontaire. Vous êtes donc libre de refuser d'y participer. Vous pouvez également vous retirer de l'étude à n'importe quel moment, sans avoir à donner de raisons, en faisant connaître votre décision au chercheur ou à l'un des membres de l'équipe de recherche. Toute nouvelle connaissance acquise durant le déroulement de l'étude qui pourrait affecter votre décision de continuer d'y participer vous sera communiquée sans délai.

Votre décision de vous en retirer n'aura aucune conséquence sur les soins qui vous seront fournis par la suite ou sur vos relations avec votre médecin et les autres intervenants.

9. Personnes à contacter

Si vous avez des questions à poser au sujet de cette étude ou s'il survient un incident quelconque ou si vous désirez vous retirer de l'étude, vous pouvez contacter en tout temps le Dr Michel Dugas (le chercheur principal de l'étude) aux numéros de téléphone suivants :

Lundi, mardi, jeudi et vendredi : (514) 848-2424, poste 2215 (Département de psychologie, Université Concordia)
Mercredi : (514) 338-4201 (Clinique des troubles anxieux, Hôpital du Sacré-Cœur)

Si vous voulez poser des questions à un professionnel ou à un chercheur qui n'est pas impliqué dans cette étude, vous pouvez communiquer avec Dr. Normand Lussier, omnipraticien à la Clinique des troubles anxieux, au (514) 338-4201.

Si vous avez des questions à poser concernant vos droits en tant que participant à un projet de recherche, ou si vous avez des plaintes ou des commentaires à formuler, vous pouvez communiquer avec la direction générale de l'hôpital, au (514) 338-2222, poste 3581.

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CONSENTEMENT

La thérapie cognitivo-comportementale pour le trouble d'anxiété généralisée : Impact du traitement de l'information sur l'efficacité thérapeutique à court et à long terme

La nature de cette étude, les procédés à utiliser, les risques et les bénéfices que comporte ma participation à cette étude ainsi que le caractère confidentiel des informations qui seront recueillies au cours de l'étude m'ont été expliqués.

J'ai eu l'occasion de poser toutes mes questions concernant les différents aspects de cette étude et on y a répondu à ma satisfaction.

Je reconnais qu'on m'a laissé le temps voulu pour prendre ma décision.

J'accepte volontairement de participer à cette étude. Je demeure libre de m'en retirer en tout temps sans que cela ne nuise aux relations avec mon médecin ou les autres intervenants et sans préjudice d'aucune sorte.

Je recevrai une copie signée de ce formulaire d'information et de consentement.

Nom du sujet (en lettres moulées)	Signature	Date
Nom du chercheur ou de son représentant (en lettres moulées)	Signature	Date

Appendix D

ETHICS APPROVAL FORMS

Concordia
UNIVERSITY

CERTIFICATION OF ETHICAL ACCEPTABILITY FOR RESEARCH INVOLVING HUMAN SUBJECTS

Name of Applicant:	Michel J. Dugas
Department:	Psychology
Agency:	CIHR submitted fall '05
Title of Project:	Cognitive-Behavioural Treatment for Generalized Anxiety Disorder: Impact of Cognitive Processing on Short- and Long-Term Outcomes
Certification Number:	UH2005-093
Valid From:	4/22/2008 to 4/22/2009

The members of the University Human Research Ethics Committee have examined the application for a grant to support the above-named project, and consider the experimental procedures, as outlined by the applicant, to be acceptable on ethical grounds for research involving human subjects.

Dr. James Pfaus, Chair, University Human Research Ethics Committee

31/05/2007

HSCM

APPROBATION D'UN PROJET DE RECHERCHE

TITRE: La thérapie cognitivo-comportementale pour le trouble d'anxiété généralisée : Impact du traitement de l'information sur l'efficacité thérapeutique à court et à long terme
- *Version du 11 novembre 2005*

LIEU: Hôpital du Sacré-Cœur de Montréal, 5400, boul. Gouin Ouest, Montréal (Québec) H4J 1C5

CHERCHEUR(s): Michel Dugas, Ph. D., Adam Radomsky, Ph. D., Natalie Phillips, Ph. D., William Bukowski, Ph. D., Julie Turcotte, M.D., Pierre Savard, M.D., Ph. D., et Adrienne Gaudet, M.D.

PROVENANCE DES FONDS: Instituts de recherche en santé du Canada

PROBLÉMATIQUE et OBJECTIF DE L'ÉTUDE: Évaluer si les biais dans le traitement cognitif (attention et interprétation) prédisent une moins grande efficacité de la TCC pour le TAG à court et à long terme

TYPE DE RECHERCHE: Étude évaluative dans une population souffrant de problèmes de santé mentale

ADMISSIBILITÉ DES SUJETS: Adultes (entre 18 et 65 ans) ayant un diagnostic primaire de trouble d'anxiété généralisée. Les individus ayant des préoccupations suicidaires ou atteintes de schizophrénie, de trouble bipolaire ou de trouble mental organique seront exclus

LES CONSÉQUENCES ÉTHIQUES:

Liberté de participer:	oui	Consentement éclairé :	oui
Confidentialité:	oui	Liberté d'en sortir sans contrainte :	oui

FORMULAIRE DE CONSENTEMENT: requis: oui (version initiale du 11 novembre 2005)
approuvé: oui Le 21 novembre 2005

COMITÉ D'ÉTHIQUE: No de code: C.E. 2005-10-62

DATE DE L'ÉTUDE PAR LE COMITÉ : 24 octobre 2005 (séance plénière)
- **6 septembre 2006** (renouvellement)
- **1er octobre 2007** (renouvellement)

MEMBRES DU COMITÉ D'ÉTHIQUE DE LA RECHERCHE ET DE L'ÉVALUATION DES TECHNOLOGIES DE LA SANTÉ

AVIS FAVORABLE :
Dre Chantal Lambert, scientifique non médecin, présidente
Mme Marie-France Thibaudeau, scientifique non médecin, vice-présidente
M. Guy Beauregard, personne spécialisée en éthique
Me Chantal Roy, juriste
Dr Marcel Boulanger, membre non affilié représentant la collectivité
Mme Henriette Bourassa, membre non affilié représentant la collectivité
Mme Isabelle Larouche, scientifique non médecin
Dre Jadranka Spahija, scientifique non médecin
Dr Axel Tosikyan, scientifique médecin
Dr Colin Verdant, scientifique médecin

M.F. Thibaudeau
Marie-France Thibaudeau

N.B. : Le Comité d'éthique de la recherche de l'HSCM poursuit ses activités en accord avec *Les bonnes pratiques cliniques (Santé Canada)* et tous les règlements applicables

[redacted]

Appendix E

THE DAILY SELF-MONITORING BOOKLET

Daily Self-Monitoring Booklet

Page 1: Percentage of Each Day Spent Worrying

No. Dossier: _____ Thérapeute: _____

Semaine du _____ au _____

1. Quelle a été la proportion de la journée pendant laquelle vous avez été inquiet-ète aujourd'hui? (cote en %)

0% 25% 50% 75% 100%

Date							
Jour							
Cote							

Page 2: Percentage of Each Day Spent Experiencing Somatic Anxiety

No. Dossier: _____ Thérapeute: _____

Semaine du _____ au _____

2. Quelle a été la proportion de la journée pendant laquelle vous avez été anxieux-se ou tendu-e aujourd'hui? (cote en %)

0% 25% 50% 75% 100%

Date							
Jour							
Cote							

Daily Self-Monitoring Booklet

Page 3: Percentage of Each Day Experiencing Feelings of Depression

No. Dossier: _____ Thérapeute: _____

Semaine du _____ au _____

3. Quelle a été la proportion de la journée pendant laquelle vous avez été triste ou déprimé-e aujourd'hui? (cote en %)

	0%	25%	50%	75%	100%
Date					
Jour					
Cote					

Page 4: Daily Record of the Type and Quantity of Psychoactive Medication

No. Dossier: _____ Thérapeute: _____

Semaine du _____ au _____

4. Si vous avez pris des médicaments aujourd'hui, indiquez la sorte et la quantité totale pour la journée.

	0%	25%	50%	75%	100%
Date					
Jour					
Rx					
Quan -tité					

Appendix F

THE WORRY AND ANXIETY QUESTIONNAIRE (WAQ)

QIA

QIA

No. Dossier _____ Date _____

1. Quels sont les sujets à propos desquels vous vous inquiétez le plus souvent?

a) _____ d) _____

b) _____ e) _____

c) _____ f) _____

Pour les numéros suivants, encerclez le chiffre correspondant (0 à 8).

2. Est-ce que vos inquiétudes vous semblent excessives ou exagérées?

Aucunement excessives			Modérément excessives				Complètement excessives
0	1	2	3	4	5	6	7	8

3. Durant les derniers six mois, combien de jours avez-vous été troublé-e par des inquiétudes excessives?

Jamais			1 jour sur 2				À tous les jours
0	1	2	3	4	5	6	7	8

4. Est-ce que vous avez de la difficulté à contrôler vos inquiétudes? Par exemple, lorsque vous commencez à vous inquiéter à propos de quelque chose, avez-vous de la difficulté à vous arrêter?

Aucune difficulté			Difficulté modérée				Difficulté extrême
0	1	2	3	4	5	6	7	8

5. Durant les derniers six mois, avez-vous souvent été troublé-e par une ou l'autre des sensations suivantes lorsque vous étiez inquiet-ète ou anxieux-se? Cotez chaque sensation en encerclant un chiffre (0 à 8).

a) Agité-e, surexcité-e ou avoir les nerfs à vif

Aucunement — Modérément — Très sévèrement

.....0..... .....1..... .....2..... .....3..... .....4..... .....5..... .....6..... .....7..... .....8.....

b) Facilement fatigué-e

Aucunement — Modérément — Très sévèrement

.....0..... .....1..... .....2..... .....3..... .....4..... .....5..... .....6..... .....7..... .....8.....

c) Difficulté à se concentrer ou blanc de mémoire

Aucunement — Modérément — Très sévèrement

.....0..... .....1..... .....2..... .....3..... .....4..... .....5..... .....6..... .....7..... .....8.....

d) Irritabilité

Aucunement — Modérément — Très sévèrement

.....0..... .....1..... .....2..... .....3..... .....4..... .....5..... .....6..... .....7..... .....8.....

e) Tensions musculaires

Aucunement — Modérément — Très sévèrement

.....0..... .....1..... .....2..... .....3..... .....4..... .....5..... .....6..... .....7..... .....8.....

f) Problèmes de sommeil (difficulté à tomber ou rester endormi-e ou sommeil agité et insatisfaisant)

Aucunement — Modérément — Très sévèrement

.....0..... .....1..... .....2..... .....3..... .....4..... .....5..... .....6..... .....7..... .....8.....

6. À quel point est-ce que l'anxiété ou l'inquiétude interfère avec votre vie, c'est-à-dire votre travail, activités sociales, famille, etc?

Aucunement — Modérément — Très sévèrement

.....0..... .....1..... .....2..... .....3..... .....4..... .....5..... .....6..... .....7..... .....8.....

Dugas, M. J., Freeston, M. H., Provencher, M. D., Lachance, S., Ladouceur, R., & Gosselin, P. (2001). Journal de Thérapie *Comportementale et Cognitive, 11(1), 31-36.*

Appendix G

THE PENN STATE WORRY QUESTIONNAIRE (PSWQ)

QIPS

No. Dossier _____ Date _____

Veuillez utiliser l'échelle ci-dessous pour exprimer jusqu'à quel point chacun des énoncés suivants correspond à vous. Encerclez le numéro (1 à 5) approprié.

	Pas du tout correspondant	Un peu correspondant	Assez correspondant	Très correspondant	Extrêmement correspondant
1. Si je n'ai pas assez de temps pour tout faire, je ne m'inquiète pas.	1	2	3	4	5
2. Mes inquiétudes me submergent.	1	2	3	4	5
3. Je n'ai pas tendance à m'inquiéter à propos des choses.	1	2	3	4	5
4. Plusieurs situations m'amènent à m'inquiéter.	1	2	3	4	5
5. Je sais que je ne devrais pas m'inquiéter mais je n'y peux rien.	1	2	3	4	5
6. Quand je suis sous pression, je m'inquiète beaucoup.	1	2	3	4	5
7. Je m'inquiète continuellement à propos de tout.	1	2	3	4	5
8. Il m'est facile de me débarrasser de pensées inquiétantes.	1	2	3	4	5

	Pas du tout correspondant	Un peu correspondant	Assez correspondant	Très correspondant	Extrêmement correspondant
9. Aussitôt que j'ai fini une tâche, je commence immédiatement à m'inquiéter au sujet de toutes les autres choses que j'ai encore à faire.	1	2	3	4	5
10. Je ne m'inquiète jamais.	1	2	3	4	5
11. Quand je n'ai plus rien à faire au sujet d'un tracas, je ne m'en inquiète plus.	1	2	3	4	5
12. J'ai été inquiet tout au long de ma vie.	1	2	3	4	5
13. Je remarque que je m'inquiète pour certains sujets.	1	2	3	4	5
14. Quand je commence à m'inquiéter, je ne peux pas m'arrêter.	1	2	3	4	5
15. Je m'inquiète tout le temps.	1	2	3	4	5
16. Je m'inquiète au sujet de mes projets jusqu'à ce qu'ils soient complétés.	1	2	3	4	5

Version originale: Meyer, T.J., Miller, M.L., Metzger, R.L., & Borkovec, T.D. (1990). *Behaviour Research and Therapy, 28*, 487-495.

Version française: Ladouceur, R., Freeston, M.H., Dumont, J. Letarte, H., Rhéaume, J. Thibodeau, N. & Gagnon, F. (1992). *Canadian Psychology/ Psychologie Canadienne, 33,* 240.

Appendix H

THE STATE-TRAIT ANXIETY INVENTORY-TRAIT VERSION (STAI-T)

IASTA Trait

No. Dossier _____ Date _____

Vous trouverez ci-dessous des énoncés qui ont déjà été utilisés par des gens pour se décrire. Lisez chaque énoncé puis, en encerclant le numéro correspondant (1 à 4), indiquez comment vous vous sentez en général. Il n'y a pas de bonnes ou de mauvaises réponses. Ne vous attardez pas trop longtemps sur les énoncés et donnez la réponse qui semble le mieux décrire les sentiments que vous éprouvez en général.

	Presque Jamais	Quelquefois	Souvent	Presque Toujours
1. Je me sens bien.	1	2	3	4
2. Je me sens nerveux(se) et agité(e).	1	2	3	4
3. Je me sens content(e) de moi-même.	1	2	3	4
4. Je voudrais être aussi heureux(se) que les autres semblent l'être.	1	2	3	4
5. J'ai l'impression d'être un(e) raté(e).	1	2	3	4
6. Je me sens reposé(e).	1	2	3	4
7. Je suis d'un grand calme.	1	2	3	4
8. Je sens que les difficultés s'accumulent au point où je n'arrive pas à les surmonter.	1	2	3	4
9. Je m'en fais trop pour des choses qui n'en valent pas vraiment la peine.	1	2	3	4

	Presque Jamais	Quelquefois	Souvent	Presque Toujours
10. Je suis heureux(se).	1	2	3	4
11. J'ai des pensées troublantes.	1	2	3	4
12. Je manque de confiance en moi.	1	2	3	4
13. Je me sens en sécurité.	1	2	3	4
14. Prendre des décisions m'est facile.	1	2	3	4
15. Je sens que je ne suis pas à la hauteur de la situation.	1	2	3	4
16. Je suis satisfait(e).	1	2	3	4
17. Des idées sans importance me passent par la tête et me tracassent.	1	2	3	4
18. Je prends les désappointements tellement à coeur que je n'arrive pas à les chasser de mon esprit.	1	2	3	4
19. Je suis une personne qui a les nerfs solides.	1	2	3	4
20. Je deviens tendu(e) ou bouleversé(e) quand je songe à mes préoccupations et à mes intérêts récents.	1	2	3	4

Dévelopé par Charles D. Spielberger en collaboration avec R. L. Gorsuch, R. Lushene, P. R. Vagg, et G. A. Jacobs. Traduit et adapté par Janel G. Gauthier en collaboration avec Stéphane Bouchard

Appendix I

THE BECK DEPRESSION INVENTORY-II (BDI-II)

IDB-II

No. Dossier _____ Date _____

Ce questionnaire comporte 21 groupes d'énoncés. Veuillez lire avec soin chacun de ces groupes puis, dans chaque groupe, choisissez **l'énoncé** qui décrit le mieux comment vous vous êtes senti(e) **au cours des deux dernières semaines, incluant aujourd'hui.** Encerclez alors le chiffre placé devant l'énoncé que vous avez choisi. Si, dans un groupe d'énoncés, vous en trouvez plusieurs qui semblent décrire également bien ce que vous ressentez, choisissez celui qui a le chiffre le plus élevé et encerclez ce chiffre. Assurez-vous bien de ne choisir qu'**un seul** énoncé dans chaque groupe, y compris le groupe no. 16 (modifications dans les habitudes de sommeil) et le groupe no. 18 (modifications de l'appétit).

1.

0 Je ne me sens pas triste.
1 Je me sens très souvent triste.
2 Je suis tout le temps triste.
3 Je suis si triste ou si malheureux(se), que ce n'est pas supportable.

2.

0 Je ne suis pas découragé(e) face à mon avenir.
1 Je me sens plus découragé(e) qu'avant face à mon avenir.
2 Je ne m'attends pas à ce que les choses s'arrangent pour moi.
3 J'ai le sentiment que mon avenir est sans espoir et qu'il ne peut qu'empirer.

3.

0 Je n'ai pas le sentiment d'avoir échoué dans la vie, d'être un(e) raté(e).
1 J'ai échoué plus souvent que je n'aurais dû.
2 Quand je pense à mon passé, je constate un grand nombre d'échecs.
3 J'ai le sentiment d'avoir complètement raté ma vie.

4.

0 J'éprouve toujours autant de plaisir qu'avant aux choses qui me plaisent.
1 Je n'éprouve pas autant de plaisir aux choses qu'avant.
2 J'éprouve très peu de plaisir aux choses qui me plaisaient habituellement.
3 Je n'éprouve aucun plaisir aux choses qui me plaisaient habituellement.

5.

0 Je ne me sens pas particulièrement coupable.
1 Je me sens coupable pour bien des choses que j'ai faites ou que j'aurais dû faire.
2 Je me sens coupable la plupart du temps.
3 Je me sens tout le temps coupable.

6.

0 Je n'ai pas le sentiment d'être puni(e).
1 Je sens que je pourrais être puni(e).
2 Je m'attends à être puni(e).
3 J'ai le sentiment d'être puni(e).

7.

0 Mes sentiments envers moi-même n'ont pas changé.
1 J'ai perdu confiance en moi.
2 Je suis déçu(e) par moi-même.
3 Je ne m'aime pas du tout.

8.

0 Je ne me blâme pas ou ne me critique pas plus que d'habitude.
1 Je suis plus critique envers moi-même que je ne l'étais.
2 Je me reproche tous mes défauts.
3 Je me reproche tous les malheurs qui arrivent.

9.

0 Je ne pense pas du tout à me suicider.
1 Il m'arrive de penser à me suicider, mais je ne le ferais pas.
2 J'aimerais me suicider.
3 Je me suiciderais si l'occasion se présentait.

10.

0 Je ne pleure pas plus qu'avant.
1 Je pleure plus qu'avant.
2 Je pleure pour la moindre petite chose.
3 Je voudrais pleurer mais je n'en suis pas capable.

11.

0 Je ne suis pas plus agité(e) ou plus tendu(e) que d'habitude.
1 Je me sens plus agité(e) ou plus tendu(e) que d'habitude.
2 Je suis si agité(e) ou tendu(e) que j'ai du mal à rester tranquille.
3 Je suis si agité(e) ou tendu(e) que je dois continuellement bouger ou faire quelque chose.

12.

0 Je n'ai pas perdu d'intérêt pour les gens ou pour les activités.
1 Je m'intéresse moins qu'avant aux gens et aux choses.
2 Je ne m'intéresse presque plus aux gens et aux choses.
3 J'ai du mal à m'intéresser à quoi que se soit.

13.

0 Je prends des décisions toujours aussi bien qu'avant.
1 Il m'est plus difficile que d'habitude de prendre des décisions.
2 J'ai beaucoup plus de mal qu'avant à prendre des décisions.
3 J'ai du mal à prendre n'importe quelle décision.

14.

0 Je pense être quelqu'un de valable.
1 Je ne crois pas avoir autant de valeur ni être aussi utile qu'avant.
2 Je me sens moins valable que les autres.
3 Je sens que je ne vaux absolument rien.

15.

0 J'ai toujours autant d'énergie qu'avant.
1 J'ai moins d'énergie qu'avant.
2 Je n'ai pas assez d'énergie pour pouvoir faire grand-chose.
3 J'ai trop peu d'énergie pour faire quoi que ce soit.

16.

0 Mes habitudes de sommeil n'ont pas changé.
1a Je dors un peu plus que d'habitude.
1b Je dors un peu moins que d'habitude.
2a Je dors beaucoup plus que d'habitude.
2b Je dors beaucoup moins que d'habitude.
3a Je dors presque toute la journée.
3b Je me réveille une ou deux heures plus tôt et je suis incapable de me rendormir.

17.

0 Je ne suis pas plus irritable que d'habitude.
1 Je suis plus irritable que d'habitude.
2 Je suis beaucoup plus irritable que d'habitude.
3 Je suis constamment irritable.

18.

0 Mon appétit n'a pas changé.
1a J'ai un peu moins d'appétit que d'habitude.
1b J'ai un peu plus d'appétit que d'habitude.
2a J'ai beaucoup moins d'appétit que d'habitude.
2b J'ai beaucoup plus d'appétit que d'habitude.
3a Je n'ai pas d'appétit du tout.
3b J'ai constamment envie de manger.

19.

0 Je parviens à me concentrer toujours aussi bien qu'avant.
1 Je ne parviens pas à me concentrer aussi bien que d'habitude.
2 J'ai du mal à me concentrer longtemps sur quoi que ce soit.
3 Je me trouve incapable de me concentrer sur quoi que ce soit.

20.

0 Je ne suis pas plus fatigué(e) que d'habitude.
1 Je me fatigue plus facilement que d'habitude.
2 Je suis trop fatigué(e) pour faire un grand nombre de choses que je faisais avant.
3 Je suis trop fatigué(e) pour faire la plupart des choses que je faisais avant.

21.

0 Je n'ai pas noté de changement récent dans mon intérêt pour le sexe.
1 Le sexe m'intéresse moins qu'avant.
2 Le sexe m'intéresse beaucoup moins maintenant.
3 J'ai perdu tout intérêt pour le sexe.

Appendix J

THE INTOLERANCE OF UNCERTAINTY SCALE (IUS)

ÉII

No. Dossier _____ Date _____

Voici une série d'énoncés qui représentent comment les gens peuvent réagir à l'incertitude dans la vie. Veuillez encercler le numéro (1 à 5) approprié pour exprimer jusqu'à quel point chacun des énoncés suivants correspond à vous.

	Pas du tout correspondant	Un peu correspondant	Assez correspondant	Très correspondant	Tout à fait correspondant
1. L'incertitude m'empêche de prendre position.	1	2	3	4	5
2. Être incertain(e) veut dire qu'on est une personne désorganisée.	1	2	3	4	5
3. L'incertitude rend la vie intolérable.	1	2	3	4	5
4. C'est injuste de ne pas avoir de garanties dans la vie.	1	2	3	4	5
5. Je ne peux pas avoir l'esprit tranquille tant que je ne sais pas ce qui va arriver le lendemain.	1	2	3	4	5
6. L'incertitude me rend mal à l'aise, anxieux(se) ou stressé(e).	1	2	3	4	5
7. Les imprévus me dérangent énormément.	1	2	3	4	5
8. Ça me frustre de ne pas avoir toute l'information dont j'ai besoin.	1	2	3	4	5
9. L'incertitude m'empêche de profiter pleinement de la vie.	1	2	3	4	5
10. On devrait tout prévenir pour éviter les surprises.	1	2	3	4	5
11. Un léger imprévu peut tout gâcher, même la meilleure des planifications.	1	2	3	4	5

	Pas du tout correspondant	Un peu correspondant	Assez correspondant	Très correspondant	Tout à fait correspondant
12. Lorsque c'est le temps d'agir, l'incertitude me paralyse.	1	2	3	4	5
13. Être incertain(e) veut dire que je ne suis pas à la hauteur.	1	2	3	4	5
14. Lorsque je suis incertain(e), je ne peux pas aller de l'avant.	1	2	3	4	5
15. Lorsque je suis incertain(e), je ne peux pas bien fonctionner.	1	2	3	4	5
16. Contrairement à moi, les autres semblent toujours savoir où ils vont dans la vie.	1	2	3	4	5
17. L'incertitude me rend vulnérable, malheureux(se) ou triste.	1	2	3	4	5
18. Je veux toujours savoir ce que l'avenir me réserve.	1	2	3	4	5
19. Je déteste être pris(e) au dépourvu.	1	2	3	4	5
20. Le moindre doute peut m'empêcher d'agir.	1	2	3	4	5
21. Je devrais être capable de tout organiser à l'avance.	1	2	3	4	5
22. Être incertain(e), ça veut dire que je manque de confiance.	1	2	3	4	5
23. Je trouve injuste que d'autres personnes semblent certaines face à leur avenir.	1	2	3	4	5
24. L'incertitude m'empêche de bien dormir.	1	2	3	4	5
25. Je dois me retirer de toute situation incertaine.	1	2	3	4	5

	Pas du tout correspondant	Un peu correspondant	Assez correspondant	Très correspondant	Tout à fait correspondant
26. Les ambiguïtés de la vie me stressent.	1	2	3	4	5
27. Je ne tolère pas d'être indécis(e) au sujet de mon avenir.	1	2	3	4	5