Facing the Unknown: Behavioural Experiments for Intolerance of Uncertainty

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ABSTRACT

Facing the Unknown: Behavioural Experiments for Intolerance of Uncertainty

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Intolerance of uncertainty (IU) is a dispositional characteristic that arises from negative beliefs about uncertainty and its implications (Koerner & Dugas, 2006). IU is an important factor in both the development and maintenance of generalized anxiety disorder (GAD; APA, 2013). A cognitive-behavioural treatment (CBT) for GAD that targets IU and additional factors has shown robust efficacy across five randomized controlled trials. IU is a key cognitive mechanism in this treatment, as reductions in IU precede (Dugas & Ladouceur, 2000; Goldman, Dugas, Sexton, & Gervais, 2007) and mediate reductions in GAD symptoms (Donegan et al., 2010). Despite these encouraging results, approximately 20-30% of individuals do not achieve full GAD remission by posttreatment. Non-remitted individuals continue to endorse elevated IU. Moreover, established CBT protocols for GAD are often lengthy and complex, involving multiple therapeutic techniques. Thus, GAD treatment development and evaluation must consider parsimony and efficiency in addition to efficacy. To that end, we developed a novel, focused CBT protocol that targets IU exclusively via behavioural experiments. This cognitive-behavioural technique is an experiential method of testing idiosyncratic beliefs (here, beliefs about uncertainty). Participants with a primary diagnosis of GAD ($N = 7$) completed 12 sessions of this CBT protocol with a licensed clinical psychologist at a local Montreal hospital. Treatment consisted of three components: (1) psychoeducation and uncertainty awareness training; (2) behavioural experiments targeting beliefs about uncertainty, and (3) relapse prevention. Our results suggest that this CBT protocol produces substantial reductions in GAD symptomatology, IU, and general psychopathology by posttreatment. These changes were generally maintained across a 6-month follow-up period, with some deterioration in safety behaviours, general anxiety, and depression. The majority of participants (6/7) demonstrated moderate to high end-state functioning from posttreatment to 6-month follow-up. Additionally, we examined rapid, non-linear changes in IU, worry, and safety behaviours between treatment sessions. Results indicated that sudden gains in IU tended to occur first and that sudden gains occurring early in treatment were associated with improved long-term treatment outcomes. Overall, our findings suggest that the systematic

application of behavioural experiments alone may provide substantial reductions in GAD symptoms and IU.

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CONTRIBUTION OF AUTHORS

The following thesis comprises two manuscripts:

Study 1 (Chapter 2)

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Study 2 (Chapter 4)

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I conceptualized the program of research presented in this dissertation, as well as the two specific studies that compose the dissertation. I chose the research question, designed the studies, and determined the hypotheses and statistical plan. I wrote the initial draft of the cognitive-behavioural treatment protocol on which the dissertation was based, and subsequently revised this treatment protocol in collaboration with my supervisor, Dr. Michel Dugas. Isabelle Geninet translated this treatment protocol from English into French for the purposes of this study. I wrote the components of this dissertation, with recommendations from Dr. Michel Dugas on the drafts. I met regularly during this program of research with Dr. Michel Dugas, who provided consultation and recommendations on the development, implementation, interpretation, and writing of this dissertation as well as the studies therein. Dr. Michel Dugas and Céline Doucet primarily recruited and scheduled participants. Céline Doucet was responsible for screening potential participants to determine their study eligibility. Catherine Laurin, Pascale Harvey, Julie Turcotte, and Thu Van Dao conducted clinical assessments for the purposes of this study. Dr. Michel Dugas and I met with the assessors for team meetings to determine final study eligibility for each participant. Isabelle Geninet conducted the therapy sessions, and I provided clinical supervision with Dr. Michel Dugas. Céline Doucet, Isabelle Geninet, and I were primarily responsible for data collection and entry. Delphine DiTecco assisted with data entry for treatment integrity ratings and I provided training for this purpose. I was responsible for statistical analyses and interpretation of the results. My committee members, Drs. Adam Radomsky and Mark Ellenbogen, provided recommendations and approved my study design during my dissertation proposal meeting in August 2013.

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CHAPTER 1
GENERAL INTRODUCTION

Facing the Unknown: Behavioural Experiments for Intolerance of Uncertainty

Intolerance of uncertainty (IU) is a dispositional characteristic that results from a set of negative beliefs about uncertainty and its consequences (Koerner & Dugas, 2006). IU is a key factor in the development and maintenance of generalized anxiety disorder (GAD; APA, 2013), a common and debilitating illness characterized by excessive worry and anxiety. IU has also been increasingly implicated in other psychopathology, including obsessive-compulsive disorder (Holaway, Heimberg, & Coles, 2006), panic disorder (Boswell, Thompson-Hollands, Farchione, & Barlow, 2013), major depressive disorder, social anxiety disorder (Carleton et al., 2012), and eating disorders (Guido et al., 2012). Refining our therapeutic approach to IU may therefore significantly impact the treatment of GAD as well as other disorders. In GAD, IU has traditionally been targeted directly via behavioural exposure, as well as through indirect methods such as motivational interviewing for positive beliefs about worry, problem solving training, and imaginal exposure for worry. Together, these cognitive-behavioural techniques compose an established, efficacious CBT protocol for GAD (Dugas & Robichaud, 2007) commonly known as CBT-IU. This protocol produces significant reductions in GAD symptoms (e.g., Dugas & Ladouceur, 2000; Gosselin, Ladouceur, Morin, Dugas, & Baillargeon, 2006; Ladouceur et al., 2000a), with posttreatment remission rates ranging from 70% (Dugas et al., 2010) to 80% (van der Heiden, Muris, & van der Molen, 2012). Although these results are encouraging, this means that a substantial minority of individuals do not fully benefit from treatment. These non-remitted individuals continue to endorse high levels of IU at posttreatment (Donegan & Dugas, 2013). Given that reductions in IU precede (Dugas & Ladouceur, 2000; Goldman, Dugas, Sexton, & Gervais, 2007) and mediate reductions in GAD symptoms during treatment (Donegan et al., 2010), optimizing IU-based treatment may produce greater reductions in GAD symptoms. A related, but more modest, proposal is that GAD treatment may become more parsimonious, time-efficient, and cost-effective by refining our therapeutic approach to IU. The main goal of the current program of research was to develop and evaluate a novel treatment protocol that targets IU using a single therapeutic technique: behavioural experiments.

IU and GAD: A Brief History

Researchers at Laval University conceptualized IU partially in response to puzzling clinical observations of GAD: symptoms appeared resistant to the cognitive restructuring techniques traditionally used to treat anxiety, such as re-evaluating the probability and cost of feared outcomes. Clients with GAD reported that their worries persisted unless absolute certainty could be achieved. This inability to tolerate even minute quantities of uncertainty perpetuates anxiety and worry (Dugas & Robichaud, 2007; Hebert, Senn, & Dugas, in press).

The association between IU and GAD symptoms has been cemented via experimental and treatment-based research. IU is a predictor of non-clinical levels of worry (Khawaja & Chapman, 2007), and is the strongest predictor of excessive worry (Dugas, Schwartz, & Francis, 2004). IU is a specific vulnerability factor for GAD symptoms in clinical (Norton, Sexton, Walker, & Norton, 2005) and non-clinical samples (Sexton, Norton, Walker, & Norton, 2003). Although IU has now been associated with disorders other than GAD (e.g., Carleton et al., 2012), non-clinical investigations have found that it is more related to worry than to depressive symptoms (Dugas, Schwartz, & Francis, 2004), obsessions, or symptoms of panic (Dugas, Gosselin, & Ladouceur, 2001). Dugas, Marchand, and Ladouceur (2005) also found IU to be specific to GAD, unlike cognitive avoidance, negative problem orientation, and positive beliefs about worry. In clinical samples, greater GAD severity predicts greater IU scores (Dugas et al., 2007). Worry itself, the hallmark of GAD, also demonstrates a unique relationship to IU. Worry has stronger associations with IU than with perfectionism or need for control, independent of the influences of anxiety and depression (Buhr & Dugas, 2006). Experimental manipulations of IU also produce changes in worry: worry increases in participants who undergo an IU induction as compared to participants whose level of IU is decreased (Ladouceur, Gosselin, & Dugas, 2000b; Rosen & Knaüper, 2009). IU thus shares a robust relationship with GAD, despite its association with other disorders (Hebert et al., in press).

Clinical Conceptualization of IU in GAD

In the standard CBT-IU protocol (Dugas & Robichaud, 2007), treatment is guided by a cognitive-behavioural conceptualization of GAD in which worry takes centre stage (see *Figure 1*). In this model, the situation triggers a "What if…?" question in the mind of a person with GAD. This initial "What if…?" question begins the cycle of worry, leading to anxiety, demoralization, and exhaustion. In this model, IU is conceptualized as a background process that

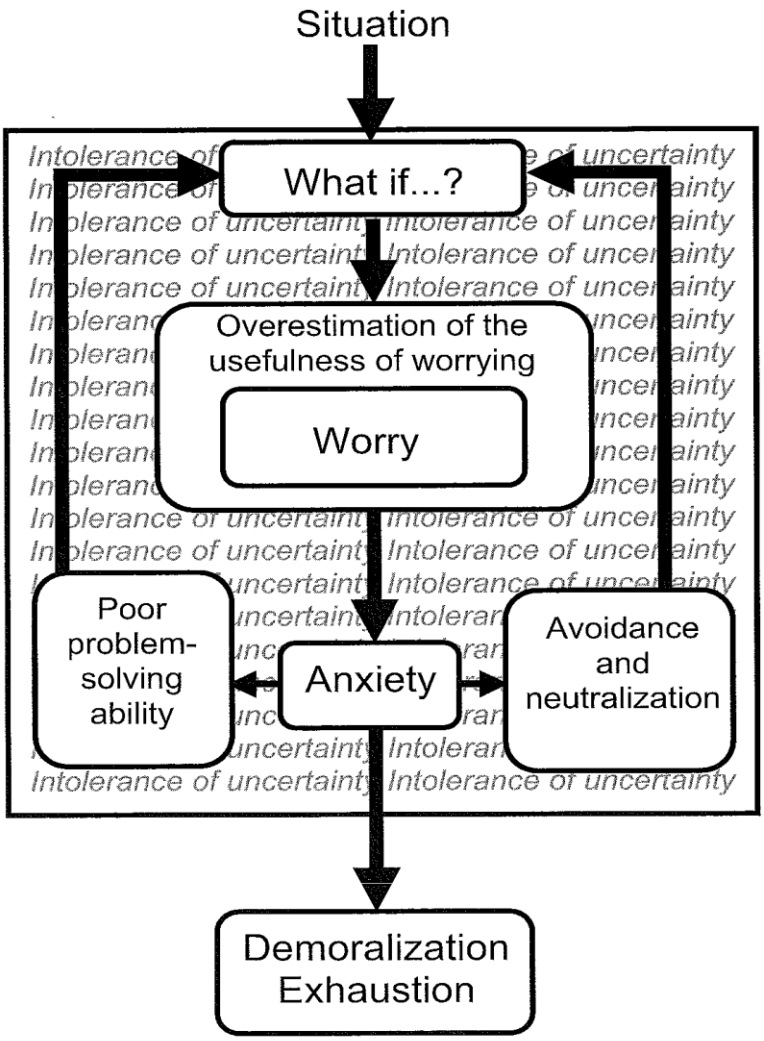

Figure 1. Standard cognitive-behavioural model of intolerance of uncertainty and generalized anxiety disorder symptoms (reprinted with permission from Dugas & Robichaud, 2007).

underpins "What if…?" questions, worry, and anxiety. IU also impacts this cycle indirectly through related cognitive mechanisms, including positive beliefs about worry, negative problem orientation, and cognitive avoidance. The individual's current emotional state as well as life events impact all aspects of this cycle, acknowledging the important role of personal history and life stressors on GAD symptoms (Mineka & Zinbarg, 2006; Moffit et al., 2007; Kendler, Hettema, Butera, Gardner, & Prescott, 2003).

This standard CBT conceptualization of IU and GAD has a number of strengths. First, it focuses on worry, widely considered to be the hallmark symptom of GAD. Given that GAD was regarded as a nebulous entity until the introduction of worry in the DSM-III-R (APA, 1987), this model highlights both the conceptual and historical importance of worry in GAD. Excessive and uncontrollable worry is recognizable as a specific diagnostic criterion of GAD, setting it apart from the criteria of other clinical disorders (Andrews et al., 2010). Second, IU is integrated into the majority of model components, highlighting its empirically-supported relationship with GAD symptoms. Third, the CBT-IU model integrates a number of additional cognitive mechanisms beyond IU that maintain GAD symptoms. For instance, cognitive avoidance has been highlighted in several additional models of worry and anxiety (e.g., Borkovec, Alcaine, & Behar, 2004; Newman & Llera, 2011). Finally, the model appears to be readily understood by both clinicians and clients and thus has significant clinical utility.

However, the standard CBT conceptualization of IU and GAD also has a number of drawbacks. First, the omission of uncertainty itself may make it more difficult for clients and clinicians to differentiate the state of uncertainty from its antecedents (i.e., situational triggers) and consequences (e.g., worry, anxiety). Second, the standard model does not specify *how* IU impacts the remaining components. Clients may benefit from a more detailed explanation as to how IU links the state of uncertainty and GAD symptoms. Third, behavioural symptoms of GAD do not appear in this model. Historically, GAD diagnostic criteria have not included behavioural symptoms – an anomaly amongst the anxiety disorders (Beesdo-Baum et al., 2012; Rapee, 1985). However, researchers and clinicians alike increasingly highlight the presence and importance of behavioural symptoms in GAD, such as reassurance seeking (Beesdo-Baum et al., 2012), procrastination (Stöber & Joorman, 2001), and refusal to delegate tasks to others (Dugas & Robichaud, 2007). Finally, the central placement of worry implies both temporal and theoretical precedence of worry over other GAD symptoms, such as anxiety and safety

behaviours. For these reasons, a new cognitive-behavioural conceptualization of IU and GAD symptoms was warranted.

Novel cognitive-behavioural conceptualization of IU. Our novel cognitive-behavioural treatment model has many familiar elements, but is centered upon IU rather than GAD symptoms (see *Figure 2*). IU is the most prominent feature of this model, highlighting its importance as a maintenance factor for GAD symptoms. This aligns with our overarching clinical goal of targeting IU more directly throughout therapy. The centrality of IU may also increase the model's applicability to other disorders – an important consideration given the increasing transdiagnostic appeal of IU (e.g., Carleton et al., 2012; McEvoy & Mahoney, 2012).

Our new model begins with specific situational properties: ambiguity, novelty, and unpredictability. These three situational characteristics have an established relationship with anxiety (e.g., Lanzetta & Driscoll, 1966; Pervin, 1963) and have been theorized to induce uncertainty (Dugas & Robichaud, 2007; Krohne, 1989; 1993). We define uncertainty as the internal state of not knowing or being unsure (Hebert et al., in press). Although situations themselves are often colloquially referred to as "uncertain", it is critical clinically to distinguish between the internal experience of uncertainty and the situational characteristics that induce this state. For instance, clients may find that not all ambiguous, novel, or unpredictable situations induce uncertainty. This enhances therapeutic understanding and may later aid in decreasing IU. For individuals with high IU, we propose that the internal state of uncertainty will activate catastrophically negative beliefs about uncertainty. The specific catastrophic, negative belief about uncertainty activated will depend on the nature of the situation the individual finds him- or herself in. For instance, an individual who is uncertain while facing a new work task may have the thought "If I'm uncertain, I can't move forward with my task" whereas another individual may have the thought "If I'm uncertain, it means I am terrible at my job". These idiosyncratic, catastrophic beliefs about uncertainty have further emotional, cognitive, and behavioural consequences: namely, anxiety, worry, and safety behaviours. The exact nature of these emotional, cognitive, and behavioural sequelae will again depend on the specific situational characteristics and idiosyncratic beliefs about uncertainty that have been activated. For instance, the individual who feels uncertain in the face of a novel work task and has the belief that "If I'm uncertain, I can't move forward" may feel anxious; begin to worry about their future at the company, possible job loss, and later homelessness; and procrastinate on their task while

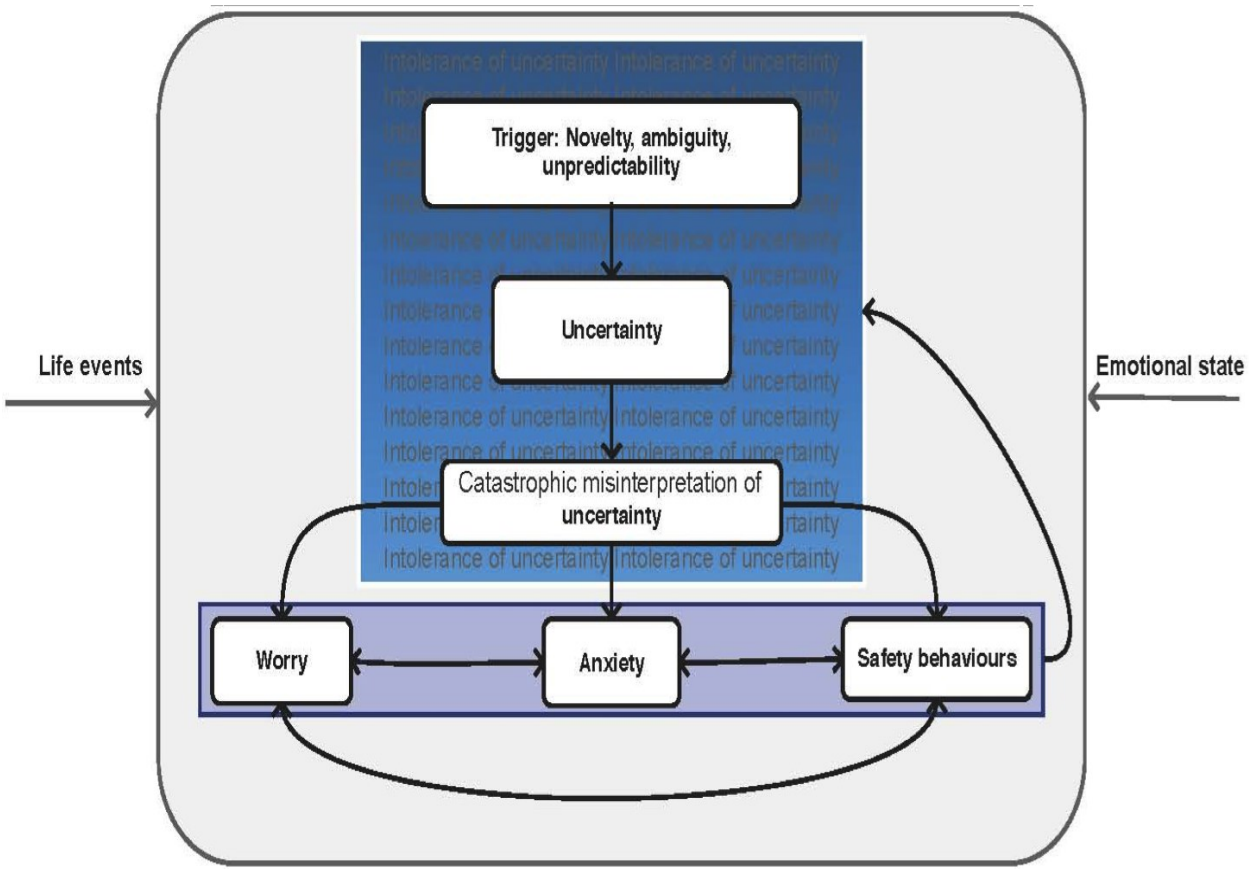

Figure 2. Novel cognitive-behavioural conceptualization of intolerance of uncertainty and generalized anxiety disorder symptoms.

researching the labour market in their field. Like our standard conceptualization of GAD and IU, an individual's current life events and emotional state impact all levels of this model.

In our new conceptual model, we propose that IU "runs in the background", impacting each model component individually. Those with high IU may be more likely to notice the situational characteristics relevant to our model: ambiguity, novelty, and unpredictability. For instance, Dugas, Hedayati, and colleagues (2005) found that individuals with high IU had better recall for uncertainty-related stimuli. Once a situational trigger is noticed, individuals with high IU may be more likely to experience the state of uncertainty. Based on the very definition of IU, individuals high in IU are hypothesized to make negative interpretations of uncertainty (Koerner & Dugas, 2006; Krohne, 1989). We have refined this to *catastrophically* negative beliefs about uncertainty, in order to distinguish from the near-universal preference for certainty present in the general population (e.g., Andreoni & Sprenger, 2012; Brim & Hoff, 1957; Schmidt, 1998; Tversky & Kahneman, 1986). Once catastrophically negative beliefs about uncertainty are activated, we propose that individuals with high IU experience worry and anxiety and engage in a variety of safety behaviours. This is consistent with previous definitions of IU as the negative cognitive, emotional, and behavioural patterns that develop in response to uncertainty-inducing stimuli (Freeston, Rheaume, Letarte, Dugas, & Ladouceur, 1994b). This is also largely consistent with previous theories that IU results in vigilant coping strategies (Krohne, 1989) as well as empirical findings that experimental increases in IU induce increases in worry (Ladouceur et al., 2000b). In addition, those with high IU display greater information seeking behaviours than those with low IU (Rosen & Knaüper, 2009) and require more certainty cues when responding to ambiguous tasks (Ladouceur, Talbot, & Dugas, 1997). More indirectly, GAD status has been associated with a variety of safety behaviours such as reassurance-seeking and situational avoidance (Beesdo-Baum et al., 2012) and worry has a unique relationship to procrastination (Stöber & Joorman, 2001). Although we will examine our novel conceptual model indirectly through the current program of research, future investigations must empirically evaluate the specific relationship between model components.

IU in GAD Treatment

IU has been traditionally targeted via behavioural exposure, a technique in which clients are asked to identify and enter into uncertainty-inducing situations. A largely habituation-based rationale is provided: repeatedly encountering and engaging with uncertainty-inducing triggers

will reduce anxiety and worry over time (Dugas & Robichaud, 2007). In combination with motivational interviewing for worry, problem-solving training, and imaginal exposure, behavioural exposure to uncertainty has been established as an efficacious treatment for GAD (e.g., Dugas & Ladouceur, 2000; Dugas et al., 2010; Ladouceur et al., 2000a). However, between 20-30% of individuals do not achieve full GAD remission by posttreatment (Dugas et al., 2010; van der Heiden, Muris, & van der Molen, 2012). One possible explanation for this is that IU has not been fully ameliorated: at posttreatment, non-remitted individuals continue to experience elevated IU (Donegan & Dugas, 2013). This is underscored by the pre-posttreatment effect sizes typically achieved for IU, which are smaller than pre-posttreatment effect sizes for GAD symptoms (e.g., Donegan & Dugas, 2013). Taken together, this suggests that IU could be targeted more effectively during treatment.

Behavioural experiments for IU. Behavioural experiments are a cognitive-behavioural technique that asks clients to identify idiosyncratic beliefs and test them as hypotheses via predetermined behaviours (Beck, Rush, Shaw, & Emery, 1979; Bennett-Levy et al., 2005). Behavioural experiments involve four main stages, conforming to the Lewin-Kolb experiential learning cycle (Kolb, 1984; Lewin, 1946): (1) planning, (2) experimentation, (3) observation, and (4) reflection. More specifically, behavioural experiments in our treatment protocol consisted of six steps: (1) identifying the problem; (2) identifying the belief about uncertainty to be tested; (3) identifying the prediction for the experiment, as well as any alternative possibilities; (4) planning the behavioural experiment, including when, where, how, and with whom it will be conducted; (5) conducting the experiment and recording the outcome; and (6) reflecting on what could be learned from the outcome of the behavioural experiment. Behavioural experiments were used to test either existing beliefs about uncertainty or new, alternative beliefs about uncertainty generated by the client.

Behavioural experiments have a strong basis in cognitive-behavioural theories. Cognitive mediation theories of psychopathology suggest that reductions in the cognitive mechanisms underlying anxiety symptoms will lead to reductions in the anxiety symptoms themselves (e.g., Beck, 1976; Beck et al., 1979). Behavioural experiments are consistent with cognitive mediation theory, as they target these underlying cognitive mechanisms. Behavioural experiments are hypothesized to reduce psychopathology symptoms via a combination of experiential learning and reflection (Kolb, 1984; Lewin, 1946), implicational and propositional information

processing (Teasdale, 1997; Teasdale & Barnard, 1993), and fear response extinction via violation of expectations (Bouton, 2004; McMillan & Lee, 2010). Due to the active, participatory nature of behavioural experiments, clients "learn by doing" as they design, carry out, and monitor the outcome of their experiments. The process of reflection following the behavioural experiment encourages the client to connect the behavioural experiment to the specific belief about uncertainty being tested and develop new beliefs as necessary. Initial experiential learning focuses on the existing landscape of uncertainty beliefs; in other words, clients conduct behavioural experiments to test what they already believe. The process of reflection encourages the generation of new beliefs, to then be evaluated in further experiential learning opportunities (Bennett-Levy et al., 2005; Gavetti & Levinthal, 2000). At the same time, behavioural experiments may impact both propositional and implicational systems of information processing, often expressed by clients as "intellectual" and "emotional" beliefs or the "head" and the "heart". Behavioural experiments may allow clients to not only evaluate current beliefs, but to generate alternative mental models via novel experiences (Bennett-Levy et al., 2005; Bouton, 2004; Teasdale, 1997; Teasdale & Barnard, 1993). This technique may violate outcome expectancies, thus facilitating fear response extinction via new inhibitory learning (Bouton, 2004). Behavioural experiments require clients to specify their predictions for the outcome of the experiment. Often, but not always, behavioural experiments provide disconfirmatory experiences that violate these expectations. This may promote extinction of fear (McMillan & Lee, 2010). Taken together, behavioural experiments represent a good theoretical fit with our new conceptual model, which centres on idiosyncratic beliefs about uncertainty. Because our conceptual framework places catastrophic beliefs about uncertainty "front-and-centre", we believe that our clinical technique must also do so. Behavioural experiments are a particularly appropriate vehicle for this, as experiential and behavioural change are theorized as perhaps the most powerful methods for changing cognition (e.g., Bandura, 1977; Jacobson et al., 1996; Waller, 2009).

Behavioural experiments for IU may have clinical advantages beyond traditional techniques. For example, a technique that focuses on beliefs about uncertainty rather than worry may be useful for GAD clients who focus excessively on the content of their worries during therapy sessions. This focus on beliefs about uncertainty may also be more effective than repeated exposure to uncertainty alone. Although a comparison of exposure and behavioural experiments in GAD has yet to be conducted, preliminary evidence suggests that, in general,

behavioural experiments may be more efficacious at reducing anxiety than exposure (McMillan & Lee, 2010; Salkovskis, Hackmann, Wells, Gelder, & Clark, 2006). Behavioural experiments can be conceptualized as cognitive change with a behavioural motor (Wells, 1997): in this case, individuals use pre-planned behaviours to induce a state of uncertainty and evaluate their uncertainty-relevant cognitions. This behaviourally-driven cognitive intervention may be particularly appropriate for clients with GAD, given that traditional cognitive techniques appear largely ineffective for GAD symptom reduction. For this reason, experiential learning may be especially important in GAD. As compared to thought records, behavioural experiments have been shown to reduce target cognitions more quickly and with greater generalization (McManus, van Doorn, & Yiend, 2012), and provide greater sensory information and higher emotional arousal (Bennett-Levy, 2003). Moreover, behavioural experiments appear more effective at changing target cognitions than exposure. If cognitive mediation is a mechanism of change in both behavioural experiments and exposure, then behavioural experiments may provide superior results (Raes, Koster, Loeys, & De Raedt, 2011).

Goals for Current Program of Research

The overarching goals for this program of research were to design a novel CBT protocol targeting IU and to evaluate this protocol within a GAD population. More specifically, we were interested in designing and evaluating a streamlined, parsimonious, and efficient protocol that would target IU via behavioural experiments. To our knowledge, there is no established GAD treatment that uses behavioural experiments exclusively, nor is there a behavioural experiment-driven treatment for IU. In fact, previously published clinical resources for behavioural experiments in GAD often appear aimed increasing feelings of certainty and attempting to resolve ambiguity (Butler & Rouf, 2005). Thus, a CBT protocol that exclusively targets IU using behavioural experiments represents a novel advancement in the field. We were concerned with not only determining if the protocol was efficacious, but also how change occurred. Thus, we conducted a preliminary evaluation of the CBT protocol's efficacy in reducing IU and GAD symptoms as well as an assessment of the temporal sequence of change across treatment sessions. We evaluated both linear and non-linear change over time in IU and GAD symptoms to gain a more comprehensive understanding of how the protocol exerts its effects.

CHAPTER 2

Challenging Uncertainty: Behavioural Experiments in the Treatment of Generalized Anxiety Disorder

Generalized anxiety disorder (GAD) is a debilitating illness characterized by chronic anxiety and excessive and uncontrollable worry (APA, 2013). Following its official recognition in the DSM-III (APA, 1980), clinicians noted that traditional cognitive restructuring approaches, such as re-evaluating the cost and probability of worries, were largely ineffective in reducing GAD symptoms (Hebert, Senn, & Dugas, in press). In response to this clinical observation, several efficacious treatments for GAD have been developed since the 1990s. These treatments have been largely based on cognitive-behavioural models of the disorder, including the cognitive avoidance model of worry (Borkovec, 1994), the metacognitive model (Wells, 1995), and the intolerance of uncertainty model (Dugas, Gagnon, Ladouceur, & Freeston, 1998). Treatment-based research in GAD has mainly focused on iterative improvements in efficacy. Given that several efficacious treatments have already been established, existing treatments should be refined (and new treatments designed) with a focus on parsimony and efficiency in addition to efficacy (Cougle, 2012). The present study focuses on the development and preliminary evaluation of a novel, three-component GAD treatment that utilizes behavioural experiments to target intolerance of uncertainty (IU).

Intolerance of Uncertainty in GAD Treatment

IU is a dispositional characteristic arising from a set of negative beliefs about uncertainty and its consequences (Dugas & Robichaud, 2007). IU has been conceptualized as a causal risk factor in the development of GAD symptoms, as well as a key maintenance factor of these symptoms. An established cognitive-behavioural treatment (CBT) for GAD targets IU directly via several sessions of behavioural exposure, as well as indirectly via re-evaluating the usefulness of worry, problem-solving training, and imaginal exposure for worry (Dugas & Robichaud, 2007). Although this treatment has demonstrated efficacy in four individual randomized controlled trials (Dugas et al., 2010; Gosselin, Ladouceur, Morin, Dugas, & Baillargeon, 2006; Ladouceur et al., 2000a; van der Heiden, Muris, van der Molen, 2012), approximately 20-30% of individuals do not achieve GAD remission by posttreatment. These symptomatic individuals continue to endorse elevated IU at post-treatment. In addition, pre-posttreatment effect sizes for IU are smaller than those for GAD symptoms (Donegan & Dugas,

2013). Thus, although the standard CBT-IU protocol significantly reduces GAD symptomatology and IU, there is room for improvement. Moreover, CBT protocols that are not IU-specific produce short- and long-term reductions in IU comparable to that of the CBT-IU protocol, despite not addressing this variable directly (van der Heiden et al, 2012). This suggests that IU could be more effectively targeted within treatment. Given that reductions in IU precede reductions in worry (Dugas & Ladouceur, 2000) and are an important mediator of GAD symptom reduction during treatment (Donegan et al., 2010), improving our ability to target IU is essential.

One method of optimizing IU-focused treatment may be to alter the techniques used to target it. In the standard CBT-IU protocol, IU is targeted directly through behavioural exposure. Although behavioural exposure is an established therapeutic technique, emerging evidence suggests that behavioural experiments may be more effective than exposure (McMillan & Lee, 2010; Salkovskis, Hackmann, Wells, Gelder, & Clark, 2006). Whereas behavioural exposure involves exposure to situations that evoke relevant symptoms and relies on a habituation-based paradigm of symptom reduction, behavioural experiments involve the identification and testing of idiosyncratic beliefs via predetermined behaviours or situations. Behavioural experiments may thus be particularly applicable to IU, given that IU is based on a *set of negative beliefs about uncertainty*. If individuals can identify and design experiments to test out their catastrophically negative beliefs about uncertainty, GAD symptoms are likely to decrease. This technique also allows individuals to identify and directly modify safety behaviours used to avoid uncertainty, such as refusing to delegate tasks, reassurance-seeking, and procrastination.

Behavioural experiments are an ideal fit for our new cognitive-behavioural conceptualization of IU (see *Figure 2*). We propose that catastrophically negative beliefs about uncertainty, once activated by situational triggers, lead to the cognitive, emotional, and behavioural symptoms of GAD (i.e., worry, anxiety, and safety behaviours). Given the inherent structure and flexibility of this cognitive-behavioural technique, our novel treatment protocol exclusively relies on behavioural experiments to target IU. To our knowledge, no existing treatment protocol for GAD has systematically and exclusively used behavioural experiments to target either GAD symptoms or underlying process variables. The current CBT protocol consisted of 3 modules: (1) psychoeducation and uncertainty awareness training, (2) testing beliefs about uncertainty via behavioural experiments, and (3) relapse prevention.

Goals and Hypotheses

The main goal of this study was to conduct a preliminary evaluation of the efficacy of a novel IU-specific CBT protocol for GAD. We assessed treatment efficacy based on changes in GAD symptoms, general psychopathology, and IU. We evaluated these changes using remission rates, effect sizes (Cohen's *d*), clinically significant change, and end-state functioning. First, we hypothesized that GAD symptoms, general psychopathology, and IU would significantly reduce from pre- to posttreatment, with at least moderate effect sizes. We predicted that at least 70% of participants would achieve GAD remission by posttreatment, consistent with the results of other IU-focused treatment studies (e.g., Dugas et al., 2010). Similarly, we predicted that the majority of participants would achieve clinically significant change on these measures. Second, we hypothesized that GAD symptoms, general psychopathology, and IU would remain stable from posttreatment to 6-month follow-up, with negligible to small effect sizes. We predicted that at least 70% of participants would be remitted across the follow-up period. We also predicted that the majority of participants would continue to have clinically significant change at 3- and 6-month follow-ups. Finally, we hypothesized that the majority of participants would have at least moderate end-state functioning from posttreatment to 6-month follow-up.

Method

Participants

Seven Francophone participants (71.43% female) with a primary diagnosis of GAD took part in the study. A primary diagnosis of GAD consisted of a score of 4 or greater on the Clinician's Severity Rating (CSR) from the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Di Nardo, Brown, & Barlow, 1994), with no other disorder having a higher score. At pretreatment, participants had an average GAD severity rating of 5.5 (SD = 0.82) and had experienced GAD symptoms for an average of 20.37 years (SD = 19.00). Participants had a mean age of 47.29 years (SD = 12.31) and all self-identified as White. The majority of the sample (57.10%) endorsed current antidepressant usage. The same percentage of participants denied use of anxiolytics and prior psychotherapy experience.

Procedures

Recruitment procedures. The study was approved by Human Research Ethics Committees of Concordia University and the Hôpital du Sacré-Cœur de Montréal. Participants were self-referred to our clinic via advertisements placed in a local newspaper (see *Appendix A*).

Interested individuals completed a telephone screening following informed consent (see *Appendix B*). Retained individuals then completed two clinical interviews: the ADIS-IV with a licensed clinical psychologist and the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) with a team psychiatrist. A team meeting was then held to discuss the clinical interview results and arrive at a final diagnosis. Participants were included in the study if the following criteria were met: (1) primary diagnosis of GAD; (2) a total score of 58 or greater on the Intolerance of Uncertainty Scale, indicating high IU (Koerner & Dugas, 2008); (3) 18 years of age or older; (4) French language fluency; (5) no current suicidal ideation; (6) no current or past history of bipolar depression or psychosis; (7) no current substance dependence or abuse; (8) no change in psychotropic medication dose or type for at least 12 weeks prior to the initial assessment; (9) willingness to maintain stable psychotropic medication for the 12-week treatment duration; and (10) not currently undergoing another psychological treatment. Individuals who did not meet the study's inclusion criteria were provided with alternative resources, including services at the Hôpital du Sacré-Cœur de Montréal or appropriate alternative referrals.

Assessment procedures. In addition to the pretreatment clinical interviews, participants completed a battery of self-report questionnaires (see *Measures*) using online software. These questionnaires assessed GAD symptoms, IU, and general psychopathology. During this clinic visit, participants also provided informed consent for treatment (see *Appendix C*). Participants completed these self-report questionnaires and the ADIS-IV again at mid-treatment, posttreatment, and at 3- and 6-month follow-ups. All assessments were conducted by one of two licensed clinical psychologists who did not conduct the treatment itself.

Treatment procedures. The CBT protocol for GAD was delivered over 12 weekly, 50-minute sessions by a licensed clinical psychologist. To ensure treatment integrity, the study authors (E.A.H. and M.J.D.) and the therapist conducted weekly clinical supervision meetings. Treatment consisted of three main components: (1) psychoeducation and uncertainty awareness training, (2) testing beliefs about uncertainty via behavioural experiments, and (3) relapse prevention training. The first component presented clients with information about CBT, GAD, and the role of uncertainty in their symptoms. Clients were asked to monitor uncertainty and their reactions to uncertainty in their daily lives. This component was delivered over two sessions. The second treatment component focused on identifying and testing clients'

idiosyncratic beliefs about uncertainty via behavioural experiments. Clients identified catastrophically negative interpretations of uncertainty via weekly monitoring and Socratic discussion. The therapist provided the rationale and steps for behavioural experiments, including planning and conducting the experiment, outcome monitoring, and reflection. The treatment manual included 60 possible behavioural experiments that could be selected from to target specific beliefs about uncertainty, including both negative and alternative beliefs. However, the therapist and participants were encouraged to generate personalized behavioural experiments based on each participant's idiosyncratic beliefs about uncertainty. Thus, the behavioural experiments differed across participants. This treatment component was delivered over 9 sessions and represented the bulk of therapy sessions. The final treatment component was composed of relapse prevention with a behavioural experiment focus. This included planning future behavioural experiments, applying a behavioural experiment framework to unexpected future events, and creating a plan of action to identify and manage possible increases in IU. The third component of treatment was delivered in one session. Participants were provided with a client manual of the treatment protocol to enhance their memory for the treatment procedures.

Clinician-Rated Measures

The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Di Nardo, Brown, & Barlow, 1994) is a semi-structured clinical interview that assesses anxiety, depressive, and other disorders. Clinicians rate the severity of each diagnosed condition on a 9-point Likert scale using the Clinician's Severity Rating (CSR). Scores range from 0 ("*absent or none*") to 8 ("*very severe*"), with a score of 4 representing clinically significant severity. The ADIS-IV demonstrates good inter-rater reliability for GAD diagnostic criteria ($\kappa = .67$), and dimensional ratings of GAD (clinical severity rating $r = .72$, excessive worry $r = .73$, uncontrollable worry $r = .78$; Brown, Di Nardo, Lehman, & Campbell, 2001).

The *Mini International Neuropsychiatric Interview, Version 5.0* (MINI; Sheehan et al., 1998) is a semi-structured clinical interview that assesses 17 DSM-IV-TR disorders, including anxiety and depressive disorders. In this study, the ADIS-IV CSR was used to rate the severity of each disorder diagnosed on the MINI. The MINI demonstrates excellent inter-rater reliability ($\kappa = .98$) and good test-retest reliability ($\kappa = .78$) for GAD over two days.

Self-Report Measures

The *Intolerance of Uncertainty Scale* (IUS; Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994b) assesses the tendency to endorse negative beliefs about uncertainty and its consequences. The IUS consists of 27 items assessed on a 5-point Likert scale, with higher scores representing greater IU. The IUS demonstrates criterion, convergent, and divergent validity and excellent internal consistency ($\alpha = .91$). The measure has a consistent two-factor structure. Factor 1 has been defined as "Uncertainty has negative self-referential and behavioural implications" (inhibitory IU) whereas Factor 2 has been defined as "Uncertainty is unfair and spoils everything" (prospective IU).

The *Worry and Anxiety Questionnaire* (WAQ; Dugas et al., 2001) assesses GAD diagnostic criteria including excessive and uncontrollable worry and somatic anxiety symptoms. Each item is rated on an 8-point Likert scale, with greater scores corresponding to greater self-rated symptoms of GAD. The French version of the WAQ has demonstrated adequate test-retest reliability over 64 days ($r = .83$ for those not meeting GAD diagnostic criteria; $r = .75$ for those meeting GAD diagnostic criteria).

The *Penn State Worry Questionnaire* (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) is a self-report questionnaire assessing the tendency to worry excessively and uncontrollably. The measure consists of 16 items rated on a 5-point Likert scale, with greater scores corresponding to greater worry. The French version of the PSWQ has demonstrated excellent test-retest reliability over four weeks ($r = .86$; Gosselin, Dugas, Ladouceur, & Freeston, 2001).

The *Generalized Anxiety Disorder – Safety Behaviours Questionnaire* (GAD-SBQ; Hebert & Dugas, 2013) is an 18-item questionnaire designed for use in this study (see *Appendix D*). This self-report measure assesses the tendency to use safety behaviours that have been clinically associated with GAD and anxiety. Safety behaviours include reassurance-seeking, over-preparation, avoidance of uncertainty-inducing situations, and refusal to delegate tasks to others. Participants rate each item on a 5-point Likert scale with responses ranging from 1 ("*not at all typical of me"*) to 5 ("*very typical of me*"). Numerical item responses are summed to create the total score, with greater total scores corresponding to greater use of safety behaviours over the previous one-month period.

The *Beck Anxiety Inventory* (BAI; Beck, Epstein, Brown, & Steer, 1988) is a 21-item self-report questionnaire that assesses cognitive, somatic, and affective anxiety symptoms over

the previous one-week period. The French translation of the BAI demonstrates convergent, divergent, and factorial validity, as well as adequate test-retest reliability (r = 0.63) and good internal consistency (α = .93; Freeston, Ladouceur, Thibodeau, Gagnon, & Rhéaume, 1994a).

The *Beck Depression Inventory, 2nd Edition* (BDI-II; Beck, Steer, & Brown, 1996) is a self-report questionnaire that assesses depressive symptoms such as sadness, worthlessness, and anhedonia. The measure is comprised of 21 items, each containing 4 statements that reflect differing levels of depressive symptoms. Respondents indicate which of the 4 statements best captures their experiences over the preceding two weeks. Higher scores indicate greater depressive symptoms. The BDI-II demonstrates evidence of content, discriminant, and factorial validity (Beck, Steer, & Brown, 1996).

Treatment Integrity

We assessed the extent to which the therapist adhered to the treatment protocol via audio recordings of treatment sessions. All treatment sessions were recorded. Two participants (28.57% of the sample) were randomly selected to have all 12 of their treatment sessions coded. One participant was randomly selected from the first half of the sample and the second participant was randomly selected from the second half of the sample, to control for the effects of therapist practice and therapist drift. A trained independent assessor coded each treatment session from the two participants for both structure and content, timed in accordance with the therapist treatment manual. Across participants, treatment integrity reached 98.31% for structure and 99.38% for content.

Results

We assessed treatment efficacy in terms of (1) remission rates, (2) effect size comparisons, (3) clinically significant change, and (4) end-state functioning. See *Table 1* for sample means and standard deviations at pretreatment, posttreatment, and 6-month follow-up. Missing data were present on self-report questionnaires at 6-month follow-up for 1 participant, due to a technical error during data collection. The data were imputed using the last available observation point, as this provided more conservative estimates of treatment outcome overall than did removing the participant from relevant analyses (see *Table 2*). Results from the completers-only sample are provided in a supplementary table (see *Table 3*).

GAD Remission

Table 1

Sample Means and Standard Deviations at Pretreatment, Posttreatment, and 6-Month Follow-up

Measure	Pretreatment		Posttreatment		6-month	
	M	*SD*	*M*	*SD*	*M*	*SD*
ADIS-IV	5.5	0.82	3.14	1.03	2.43	1.90
WAQ	38.36	5.25	24.36	9.06	25.43	6.42
IUS	85.71	14.71	55.00	15.55	54.29	12.30
PSWQ	61.57	6.00	50.00	8.25	51.14	6.15
GAD-SBQ	47.00	9.95	32.71	5.53	36.14	5.11
BAI	23.71	10.47	9.86	10.56	12.57	12.12
BDI-II	14.71	4.39	4.00	5.69	5.57	5.16

Note. ADIS = Clinical Severity Rating of the Anxiety Disorders Interview Schedule for DSM-IV; WAQ = Worry and Anxiety Questionnaire; IUS = Intolerance of Uncertainty Scale; PSWQ = Penn State Worry Questionnaire; GAD-SBQ = Generalized Anxiety Disorder-Safety Behaviour Questionnaire; BAI = Beck Anxiety Inventory; and BDI-II = Beck Depression Inventory, 2nd Edition.

Table 2

Effect Sizes (Cohen's d) for GAD Symptoms and IU

Comparison	Measure						
	ADIS-IV	WAQ	IUS	PSWQ	GAD-SBQ	BAI	BDI-II
Pre – Post	2.06	1.32	1.72	1.13	1.41	1.64	2.08
Pre – 3-month	1.94	1.07	1.14	0.89	1.34	1.25	0.53
Pre – 6-month	1.34	1.29	1.66	1.06	1.65	1.47	2.15
Post – 3-month	0.94	0.00	-0.15	-0.27	-0.60	-0.41	-0.50
Post – 6-month	0.37	-0.15	0.07	-0.18	-0.70	-0.56	-0.55

Note. "Pre – Post" = Cohen's *d* effect sizes between pretreatment and posttreatment; "Pre – 3-month" = Cohen's *d* effect sizes between pretreatment and 3-month follow-up; "Pre – 6-month" = Cohen's *d* effect sizes between pretreatment and 6-month follow-up; "Post – 3-month" = Cohen's *d* effect sizes between posttreatment and 3-month follow-up; "Post – 6-month" = Cohen's *d* effect sizes between posttreatment and 6-month follow-up; ADIS = Clinical Severity Rating of the Anxiety Disorders Interview Schedule for DSM-IV; WAQ = Worry and Anxiety Questionnaire; IUS = Intolerance of Uncertainty Scale; PSWQ = Penn State Worry Questionnaire; GAD-SBQ = Generalized Anxiety Disorder-Safety Behaviour Questionnaire; BAI = Beck Anxiety Inventory; and BDI-II = Beck Depression Inventory, 2nd Edition.

Table 3

Effect Sizes (Cohen's d) for GAD Symptoms and IU (Completers Sample)

Comparison	Measure						
	ADIS-IV	WAQ	IUS	PSWQ	GAD-SBQ	BAI	BDI-II
Pre – Post	2.06	1.32	1.72	1.13	1.41	1.64	2.08
Pre – 3-month	1.94	1.07	1.14	0.89	1.34	1.25	0.53
Pre – 6-month	1.34	2.07*	1.51*	1.82*	1.29*	1.92*	2.33*
Post – 3-month	0.94	0.00	-0.15	-0.27	-0.60	-0.41	-0.50
Post – 6-month	0.37	-0.18*	-0.11*	-0.10*	0.10*	-0.50*	-1.04*

Note. $N = 7$ unless otherwise noted. "Pre – Post" = Cohen's *d* effect sizes between pretreatment and posttreatment; "Pre – 3-month" = Cohen's *d* effect sizes between pretreatment and 3-month follow-up; "Pre – 6-month" = Cohen's *d* effect sizes between pretreatment and 6-month follow-up; "Post – 3-month" = Cohen's *d* effect sizes between posttreatment and 3-month follow-up; "Post – 6-month" = Cohen's *d* effect sizes between posttreatment and 6-month follow-up; ADIS = Clinical Severity Rating of the Anxiety Disorders Interview Schedule for DSM-IV; WAQ = Worry and Anxiety Questionnaire; IUS = Intolerance of Uncertainty Scale; PSWQ = Penn State Worry Questionnaire; GAD-SBQ = Generalized Anxiety Disorder-Safety Behaviour Questionnaire; BAI = Beck Anxiety Inventory; and BDI-II = Beck Depression Inventory, 2nd Edition.

* $n = 6$ for 6-month follow-up

A participant was considered to have achieved GAD remission if they had a score of less than 4 on the ADIS-IV's CSR. At posttreatment, six out of seven participants had remitted from GAD. This was relatively consistent across the follow-up period: at both 3- and 6-month follow-up, five participants maintained GAD remission on the ADIS-IV.

Mean Comparisons

We calculated effect sizes (Cohen's *d*) to assess the relative magnitude of change between assessment points (see *Table 1* for means and standard deviations). To evaluate the short- and long-term effects of treatment on participants' GAD symptoms and underlying cognitive variables, we examined the effect sizes between pre- and posttreatment and between pretreatment and 6-month follow-up. We also calculated effect sizes between pretreatment and 3-month follow-up and between posttreatment and the follow-up points (see *Table 2*). Paired samples t-tests were used to evaluate the statistical significance of key comparisons (i.e., pre- to posttreatment, pretreatment to 6-month follow-up, and posttreatment to 6-month follow-up).

Pretreatment to subsequent time points. Across all measures, the general pattern of results indicated substantial reductions from pre- to posttreatment. All measures demonstrated large, positive effect sizes from pre- to posttreatment. Paired samples t-tests showed that these comparisons were statistically significant across all measures. When examining the entire study period, all measures demonstrated large, positive effect sizes from pretreatment to 6-month follow-up. Paired samples t-tests demonstrated statistical significance for each comparison, with the exception of a trend toward statistical significance for the GAD-SBQ.

Posttreatment to follow-up. From posttreatment to 6-month follow-up, the general pattern of results indicated no statistically significant change across measures. Paired samples t-tests were non-significant for all comparisons. Effect sizes were of negligible size for the WAQ, PSWQ, and IUS. We found a small, positive effect size for the ADIS-IV, suggesting small, continued improvement in overall GAD symptomatology. The BAI, BDI-II, and GAD-SBQ demonstrated moderate, negative effect sizes over this time period.

Clinically Significant Change

We assessed clinically significant change on all study variables. For the ADIS-IV, CSR scores below 4 were considered to be clinically significant. On the PSWQ, BAI, and BDI-II, clinically significant change was calculated by determining if a participant's posttreatment score was closer to the functional group mean than to the dysfunctional group mean (Jacobson &

Truax, 1991). In this case, the dysfunctional group was defined as the GAD clinical population and the functional group was defined as the non-clinical population. If a participant's score fell below the cut score derived using this formula ($c = SD_0M_1 + SD_1M_0 / SD_0 + SD_1$), they were considered to be closer to the functional population than to the dysfunctional population. Due to the lack of non-clinical norms available for the GAD-SBQ and French non-clinical norms for the WAQ, we used an alternative formula ($a = M_1 – 2SD_1$; Jacobson & Truax, 1991). Thus, clinically significant change on the WAQ and GAD-SBQ was said to have occurred if a participant's score fell at least 2 standard deviations below the pretreatment sample mean. This provides a conservative estimate of clinically significant change.

Clinically significant change varied considerably across outcome measures (see *Table 4*). At posttreatment, six participants demonstrated clinically significant change on clinician-rated GAD symptoms (ADIS-IV) versus five participants in self-rated GAD symptoms (WAQ). Four participants experienced clinically significant change on the PSWQ, in contrast to only one on the GAD-SBQ. Three of seven participants experienced clinically significant change on the IUS. The majority of participants demonstrated clinically significant change on the BDI-II (6/7) and BAI (5/7).

Clinically significant change was generally maintained across the follow-up period. On the ADIS, clinically significant change decreased slightly across follow-up (5/7 participants at both time points). On the WAQ, clinically significant change decreased slightly at 3-month follow-up (4/7 participants) but returned to posttreatment levels (5/7 participants) by 6-month follow-up. Clinically significant change on the PSWQ increased to five participants by 3-month follow-up and remained unchanged at 6-month follow-up. Similarly, the rate of clinically significant change on the IUS increased by one participant at 3-month follow-up and was maintained at 6-month follow-up. Five and four participants demonstrated clinically significant change on the BDI-II and BAI, respectively, at both follow-up points. The rate of clinically significant change on the GAD-SBQ remained low across follow-up.

End-State Functioning

End-state functioning was calculated based on the number of measures on which a given participant experienced clinically significant change (see *Table 5*). This calculation was based on six key outcome variables: ADIS-IV, WAQ, IUS, PSWQ, BAI, and BDI-II. The GAD-SBQ was

Table 4

Clinically Significant Change Across Measures

Measure	Time Point		
	Post-Tx	3-month	6-month
ADIS-IV	6/7	5/7	5/7
WAQ	5/7	4/7	5/7
IUS	3/7	4/7	4/7
PSWQ	4/7	5/7	5/7
GAD-SBQ	1/7	1/7	0/7
BAI	5/7	4/7	4/7
BDI-II	6/7	5/7	5/7

Note. "Post-Tx" = number of participants who achieved clinically significant change at posttreatment; "3-month" = number of participants who achieved clinically significant change at 3-month follow-up; "6-month" = number of participants who achieved clinically significant change at 6-month follow-up; ADIS = Clinical Severity Rating of the Anxiety Disorders Interview Schedule for DSM-IV; WAQ = Worry and Anxiety Questionnaire; IUS = Intolerance of Uncertainty Scale; PSWQ = Penn State Worry Questionnaire; GAD-SBQ = Generalized Anxiety Disorder-Safety Behaviour Questionnaire; BAI = Beck Anxiety Inventory; and BDI-II = Beck Depression Inventory, 2nd Edition.

Table 5

End-State Functioning Across Participants

Participant	Time Point		
	Post-Tx	3-month	6-month
901	4/6	5/6	5/6
902	6/6	6/6	6/6
903	5/6	6/6	6/6
904	4/6	6/6	3/6
905	3/6	1/6	3/6
906	0/6	0/6	0/6
907	6/6	3/6	5/6

Note. End-state functioning was calculated as the total number of the following measures on which a participant achieved clinically significant change at a given time point: Anxiety Disorders Interview Schedule for DSM-IV, Worry and Anxiety Questionnaire, Penn State Worry Questionnaire, Intolerance of Uncertainty Scale, Beck Anxiety Inventory, and Beck Depression Inventory, 2nd Edition.

not included, as this measure has not yet been validated in clinical or non-clinical populations. Low end-state functioning was defined as clinically significant change on 0-2 measures. Moderate end-state functioning was defined as clinically significant change on 3-4 measures. Finally, high end-state functioning was defined as clinically significant change on 5-6 measures. These criteria are consistent with previous investigations involving CBT for GAD (e.g., Dugas & Ladouceur, 2000).

Using this formula, the majority of participants (6/7) displayed moderate to high end-state functioning at posttreatment. Three participants each experienced moderate and high end-state functioning, respectively. The majority of participants continued to experience moderate to high end-state functioning across the follow-up period. At 3-month follow-up, four participants displayed high end-state functioning and one participant had moderate end-state functioning. At 6-month follow-up, four participants displayed high end-state functioning and two had moderate end-state functioning. The remaining participants demonstrated low end-state functioning.

Discussion

The main goal of this study was to conduct a preliminary evaluation of a novel, IU-focused CBT protocol for GAD. The results supported our hypothesis that GAD symptoms would be significantly reduced by posttreatment: six of seven participants achieved GAD remission by posttreatment, as assessed by a structured clinical interview. Moreover, we found substantial pre- to posttreatment decreases in self-reported GAD symptomatology, general psychopathology, and IU. The magnitudes of these changes were large and statistically significant for all measures. This suggests that our IU-focused protocol does indeed target IU and produces meaningful change in GAD symptoms by posttreatment. Treatment gains were generally maintained across the 6-month follow-up period, with moderate deterioration in safety behaviours as well as general anxiety and depressive symptoms. However, these deteriorations were not statistically significant when evaluated in paired samples t-tests. Moreover, the relative magnitude of change for the overall study period (i.e., from pretreatment to 6-month follow-up) remained large and positive across all outcome measures. The majority of participants achieved clinically significant change on all measures by posttreatment, with the exception of the GAD-SBQ. Our findings regarding safety behaviours should be interpreted with some caution, given the unvalidated nature of the measure and the conservative cut-off used for clinically significant change (Jacobson & Truax, 1991). Our hypothesis that the majority of participants would

experience moderate to high end-state functioning from posttreatment to 6-month follow-up was also confirmed. Overall, our results suggest that this novel, streamlined CBT protocol produces meaningful change in key symptom dimensions and beliefs about uncertainty by posttreatment and that these changes are relatively maintained 6 months later.

Our pattern of results is largely consistent with previous investigations of a more complex IU-focused treatment for GAD (Dugas & Ladouceur, 2000; Dugas et al., 2010; Dugas et al., 2003; Ladouceur et al., 2000a; van der Heiden et al., 2012). These findings are particularly encouraging, given that the novel CBT protocol was delivered in fewer sessions, with fewer components, and only one major cognitive-behavioural intervention. Our findings suggest that we can significantly reduce GAD symptoms by exclusively targeting IU via behavioural experiments. Moreover, we achieved greater reductions in IU relative to previous investigations using the standard CBT-IU protocol. For instance, although large pre-posttreatment effect sizes for the IUS were found in previous studies (d = 0.93, Donegan & Dugas, 2013; d = 1.01, Dugas et al., 2010), these are smaller in comparison to our pre-posttreatment effect size (d = 1.72). It is also of note that this pre-posttreatment time period was not identical across studies: the current CBT protocol is two sessions shorter than the standard CBT protocol (12 vs. 14 sessions). Thus, we achieved a larger reduction in IU in fewer treatment sessions. However, despite these promising results we cannot draw any conclusions regarding the relative efficacy of our novel CBT protocol and the standard CBT-IU protocol based on our limited sample size and study design.

Our findings suggest that behavioural experiments targeting beliefs about uncertainty can produce meaningful changes in both IU and GAD symptoms. This is consistent with cognitive mediation theories of anxiety, which suggest that changing underlying beliefs will lead to changes in anxiety symptoms. Behavioural experiments target underlying beliefs to reduce problematic anxiety symptoms. In the current treatment, behavioural experiments were specifically designed to target the catastrophically negative beliefs about uncertainty that we propose lead to GAD symptoms (see *Figure 2*). The finding that IU-focused behavioural experiments decreased GAD symptoms thus indirectly supports our conceptual model.

This study had a number of strengths. First, the clinical case replication series design allowed us to conduct a small-scale evaluation of a novel protocol. This is an economical use of research and clinical resources, allowing us to determine if the treatment protocol warranted

further investigation in a randomized controlled trial. Second, we evaluated treatment outcome using a variety of methods, including remission rates, effect sizes, clinically significant change calculations, and mean comparisons. Third, we evaluated treatment outcome using a variety of clinician-administered and self-report measures for GAD symptomatology, general psychopathology, and IU. Fourth, we included a significant follow-up period in order to assess both short- and long-term treatment outcome. Fifth, we permitted comorbidity to enhance the representativeness of our sample. Sixth, our treatment protocol's focus on behavioural experiments reduces the need for future dismantling studies that would compare the relative contributions of multiple cognitive-behavioural techniques. This also resulted in a parsimonious and efficient treatment protocol, and may improve knowledge translation to clinicians (Cougle, 2012; Dimeff et al., 2009; Mansell, 2008; Shafran et al., 2009). This may be particularly important given the low rates of evidence-based psychological treatments in routine clinical settings (e.g., Goisman, Warsaw, & Keller, 1999; Stein et al., 2004). Our study also had several important limitations. First, our small sample size limited the number and type of statistical analyses that could be performed due to low power. Second, it is unclear how our results would generalize outside of our sample's geographic and demographic boundaries. Third, we did not employ a waitlist control condition. Thus, we cannot be certain that our results are not attributable to the effect of time. However, this is less of a concern for a preliminary investigation in a GAD sample, given that spontaneous remission in GAD is uncommon (Yonkers, Warshaw, Massion, & Keller, 1996). Fourth, the safety behaviours measure developed for this study has not yet been validated. Although it assesses a wide variety of safety behaviours that may exist in GAD (Dugas & Robichaud, 2007), the validity and reliability of this measure's results should be interpreted with caution.

Implications and Conclusions

IU has been conceptualized as a key factor in the development and maintenance of GAD symptoms. In this study, we found preliminary evidence suggesting that GAD symptoms can be reduced by directly and exclusively targeting IU via behavioural experiments. Behavioural experiments may reduce intolerance of uncertainty, and thus reduce GAD symptoms, in several ways. First, behavioural experiments may weaken catastrophically negative beliefs about uncertainty. A behavioural experiment may reveal disconfirmatory information about a previously held belief about uncertainty, thereby reducing IU. Second, behavioural experiments

may foster the creation of neutral or positive beliefs about uncertainty. These new neutral or positive beliefs about uncertainty may therefore also reduce IU. Behavioural experiments may be particularly valuable in selectively weakening or strengthening specific beliefs about uncertainty, as they may work on both implicational and propositional levels of cognition (Teasdale, 1997; Teasdale & Barnard, 1993) via experiential learning and reflection (Bennett-Levy et al., 2005; Kolb, 1984; Lewin, 1946). Future research should compare the relative performance of behavioural experiments, behavioural exposure, and other methods of targeting IU.

Overall, our study provides preliminary evidence that a novel CBT protocol targeting IU via behavioural experiments can produce significant reductions in GAD symptomatology. The remission rates and relative magnitude of changes on measures of GAD and general psychopathology were generally comparable to outcomes achieved via the standard CBT-IU protocol. We were also able to achieve greater reductions in IU than in previous investigations of the standard CBT-IU protocol. This suggests that our novel protocol does indeed target IU. Our pattern of findings is particularly encouraging as this novel CBT protocol appears to be more parsimonious and efficient than our standard CBT-IU protocol.

CHAPTER 3
BRIDGE

The current program of research focused on the development and preliminary evaluation of a novel CBT protocol that targets IU via behavioural experiments. In the first study, we examined short- and long-term treatment efficacy in terms of remission rates, effect sizes, clinically significant change, and end-state functioning. These analyses focused on change over time in GAD symptoms, general psychopathology, and IU. This provided us with information about how participants functioned in a variety of symptom domains at specific time points, such as posttreatment and across the 6-month follow-up period. We found that 85.71% of participants achieved GAD remission by posttreatment, which was maintained by 71.43% of participants across the 6-month follow-up period. The majority of participants (85.71%) had moderate to high end-state functioning at post-treatment and at 6-month follow-up. Effect sizes during the active treatment period as well as across the entire study period were large and positive for all measures of treatment outcome. Overall, our findings provided preliminary support for the efficacy of this treatment protocol.

The linear analytic strategy of our first study assumed that change in the outcome variables of interest occurs in a gradual fashion during therapy (Hayes, Laurenceau, Feldman, Strauss, & Cardaciotto, 2007). In our second study, we evaluated non-linear change during the treatment protocol's 12 therapy sessions using sudden gains. Sudden gains refer to rapid, large changes in a given variable between two therapy sessions (Tang & DeRubeis, 1999). We considered sudden gains in IU, worry, and safety behaviours and investigated their relative proportions, temporal sequence, and relationship to key measures of short- and long-term GAD treatment outcome.

CHAPTER 4

Sudden Gains in Generalized Anxiety Disorder: Changing Beliefs to Change Symptoms

Psychological treatments are improved by evaluating not only *if* a given treatment works, but also *how* that treatment works. Cognitive-behavioural mediation theories posit that changes in cognitive mechanisms precede changes in symptoms of emotional disorders, such as depression and anxiety. Specific modules or sessions within treatment may be particularly relevant to changes in these underlying cognitive mechanisms or symptoms. Sudden gains, or rapid changes that occur between two treatment sessions, provide key information about this temporal sequence of change on an individual, non-linear level (Present et al., 2008; Tang & DeRubeis, 1999). Examinations of sudden gains may also pinpoint key modules or sessions responsible for rapid change. Here, we consider sudden gains in intolerance of uncertainty (IU), excessive worry, and safety behaviours within a streamlined cognitive-behavioural treatment for generalized anxiety disorder (GAD) based on behavioural experiments.

Psychological treatments are most commonly evaluated in terms of pre-posttreatment changes in psychopathology symptoms. However, symptom change is not always a linear process (Hayes, Laurenceau, Feldman, Strauss, & Cardaciotto, 2007; Present et al., 2008). Sudden gains are common, occurring in 14.60-52.20% of treatments for anxiety and depression (Aderka, Nickerson, Bøe, & Hofmann, 2012). These sudden gains also account for a large proportion of total change across treatment (e.g., 64.68%, Norton, Klenck, & Barrera, 2010; 75%, Present et al., 2008; 105%, Stiles et al., 2003). Sudden gains do not appear to be random variations across treatment sessions. In fact, they have been associated with more positive treatment outcomes. For example, those who experience sudden gains during CBT for GAD experience greater pre-posttreatment reductions in excessive worry and self-rated GAD symptoms than those with no sudden gains (Deschênes & Dugas, 2013). Non-linear change that occurs early in treatment may be particularly relevant to short- and long-term outcomes. Several studies have found that the majority of sudden gains occur early in the treatment of depression (Kelly, Roberts, & Ciesla, 2005), panic disorder (Clerkin, Teachman, & Smith-Janick, 2008), and mixed emotional disorders (Stiles et al., 2003). Rapid early response in cognitive therapy accounts for 60-80% of total symptom reduction (Ilardi & Craighead, 1994). Moreover, individuals who experience sudden gains early in treatment have been shown to have higher posttreatment remission rates (Busch, Kanter, Landes, & Kohlenberg, 2006), as well as lower

posttreatment symptom severity and greater clinically significant change on measures of general psychopathology (Stiles et al., 2003) and depression (Busch et al., 2006; Kelly et al., 2005). Consistent with cognitive mediation theories, shifts in cognition may prompt these early sudden gains, precipitating further symptom change during treatment (Kelly et al., 2005; Tang & DeRubeis, 1999). Thus, we were particularly interested in determining the relevance of early sudden gains in the treatment of GAD.

Individuals with a primary diagnosis of GAD appear to experience sudden gains in both worry (20.34% of sample; Deschênes & Dugas, 2013) and general anxiety symptoms (16.8% of sample; Present et al., 2008). Sudden gains in GAD occur across of a variety of psychological treatments, including individual GAD supportive-expressive therapy (Present et al., 2008), individual GAD cognitive-behavioural therapy (CBT; Deschênes & Dugas, 2013) and transdiagnostic group CBT (Norton et al., 2010). However, the temporal sequence of sudden gains in underlying cognitive mechanisms, worry, and safety behaviours has not been examined.

Our research group has previously evaluated sudden gains in our standard CBT protocol for GAD (Deschênes & Dugas, 2013), which targets IU, positive beliefs about worry, cognitive avoidance, and negative problem orientation. In the present study, we turn our attention to sudden gains within a novel, streamlined CBT protocol exclusively targeting IU. Evaluating sudden gains within this streamlined CBT protocol offered several advantages. First, our novel CBT protocol solely targeted IU, rather than the additional cognitive mechanisms targeted by our standard CBT protocol (i.e., positive beliefs about worry, cognitive avoidance, and negative problem orientation). Thus, changes in GAD symptoms are more likely to be associated with changes in IU. Second, our novel CBT protocol employed one major treatment component (i.e., behavioural experiments targeting beliefs about uncertainty) versus the standard protocol's four major components. This allowed us to more accurately pinpoint key therapeutic interventions without the need for future dismantling studies. This increases treatment efficiency and parsimony, both of which may enhance later knowledge translation to clinicians (Cougle, 2012; Mansell, 2008). This also extends the extant literature, as few studies isolate the "active ingredients" of therapy (Longmore & Worrell, 2007). Moreover, behavioural experiments are designed to directly target idiosyncratic beliefs, and may therefore be more likely to produce sudden gains in these beliefs than exposure-based interventions (e.g., Raes, Koster, Loeys, & De Raedt, 2011) commonly used in GAD treatments. Third, we included weekly assessments of IU

and GAD-related safety behaviours in addition to worry. This allowed us to determine the temporal sequence of change among a key cognitive mechanism (i.e., IU), the hallmark feature of GAD (i.e., excessive and uncontrollable worry), and behavioural symptoms associated with GAD. Because our treatment protocol solely targets IU, we expected sudden gains in IU to be more common than sudden gains in worry or safety behaviours. Finally, given the potential importance of early sudden gains, we used the modified sudden gain criterion outlined by Kelly and colleagues (2005) to evaluate sudden gains that occur early or late in treatment.

The main objectives of this study were to investigate the relative proportion, sequence, and relationship to treatment efficacy of sudden gains in a streamlined CBT protocol for GAD. Specifically, we hypothesized that (1) more sudden gains would occur in IU than in worry or safety behaviours; (2) sudden gains in IU would precede sudden gains in worry or safety behaviours, and (3) early sudden gains would be associated with more positive short- and long-term treatment outcome.

Method

Participants

The sample consisted of seven Francophone participants (five women) with a primary diagnosis of GAD. A primary diagnosis of GAD consisted of a score of 4 or greater on the Clinician's Severity Rating of the Anxiety Disorders Interview Schedule for DSM-IV (ADIS), with no other clinical diagnosis having a greater score. All participants self-identified as White or of European descent, and had a mean age of 47.29 (SD = 12.31). The sample's mean GAD severity rating was 5.5 (SD = 0.82) with an average GAD duration of 20.37 years (SD = 19.00). The majority of the sample (57.10%) had no prior therapy experience and denied use of anxiolytics. The same percentage of participants endorsed current use of antidepressants.

Measures of Sudden Gains

The *Intolerance of Uncertainty Scale – Past Week* (IUS-PW; Dugas, 2008) is a 12-item questionnaire that assesses the tendency to view uncertainty and its consequences as negative over a one-week period (see *Appendix E*). The questionnaire was adapted from the Intolerance of Uncertainty Scale (IUS; Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994b), which has demonstrated strong validity and test-retest reliability. Items on the IUS-PW are rated on a 5-point Likert scale, with higher scores representing elevated IU.

The 3-Item *Penn State Worry Questionnaire – Past Week* (PSWQ-PW; Stöber & Bittencourt, 1998) is a brief questionnaire that assesses the tendency to worry excessively and uncontrollably over a one-week period. The 3-item version was adapted from the Penn State Worry Questionnaire – Past Week (Stöber & Bittencourt, 1998) and an abbreviated version of the Penn State Worry Questionnaire (Berle et al., 2011). Items on the PSWQ-PW are rated on a 5-point Likert scale, with higher scores representing greater worry. The PSWQ-PW has demonstrated strong validity and reliability for weekly assessments of excessive worry (Stöber & Bittencourt, 1998).

The *Generalized Anxiety Disorder-Safety Behaviours Questionnaire – Past Week* (GAD-SBQ-PW; Hebert & Dugas, 2013) is an 18-item questionnaire measuring the tendency to use a variety of safety behaviours associated with generalized anxiety disorder during the previous week (see *Appendix F*). Safety behaviours include avoidance of uncertainty-inducing situations, over-preparation, reassurance-seeking, and refusal to delegate tasks. This measure was adapted for use in this study from the *Generalized Anxiety Disorder – Safety Behaviours Questionnaire*, which assesses safety behaviours over the previous one-month period. Each item is rated on a 5-point Likert scale. These item scores are summed to create the total score, with higher scores corresponding to greater past-week use of safety behaviours.

Treatment Outcome Measures

The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Di Nardo, Brown, & Barlow, 1994) is a semi-structured clinical interview assessing anxiety, depression, psychosis, and related disorders. The Clinician's Severity Rating (CSR) provides a numerical rating for the severity of each diagnosed condition on a 9-point Likert scale. CSR scores range from 0 ("*absent or none*") to 8 ("*very severe*"), with scores of 4 or greater considered clinically significant. The ADIS-IV has demonstrated good inter-rater reliability for dimensional ratings of GAD (clinical severity rating $r = .72$, excessive worry $r = .73$, uncontrollable worry $r = .78$) as well as GAD diagnostic criteria ($\kappa = .67$; Brown, Di Nardo, Lehman, & Campbell, 2001).

The *Worry and Anxiety Questionnaire* (WAQ; Dugas et al., 2001) evaluates self-reported GAD diagnostic criteria. Participants rate each item on an 8-point Likert scale, with greater scores representing greater GAD symptoms. The French translation of the WAQ demonstrates adequate test-retest reliability over 64 days ($r = .83$ for non-GAD; $r = .75$ for GAD).

The *Penn State Worry Questionnaire* (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item measure assessing the self-reported tendency to worry excessively and uncontrollably. Items are rated on a 5-point Likert scale, with greater scores representing greater worry. The French translation of the PSWQ has excellent test-retest reliability over a four-week period (r = .86; Gosselin, Dugas, Ladouceur, & Freeston, 2001).

The *Intolerance of Uncertainty Scale* (IUS; Freeston et al., 1994b) is a 27-item measure of the tendency to view uncertainty and its consequences in a negative manner. Each item is rated on a 5-point Likert scale, with greater scores corresponding to greater IU. The questionnaire demonstrates strong test-retest reliability and a stable two-factor structure (Sexton & Dugas, 2009).

The *Beck Anxiety Inventory* (BAI; Beck, Epstein, Brown, & Steer, 1988; French translation: Freeston, Ladouceur, Thibodeau, Gagnon, & Rhéaume, 1994a) is a 21-item self-report measure assessing cognitive, somatic, and affective anxiety symptoms over a one-week period. The French version of the BAI has good internal consistency (α = .93) and adequate test-retest reliability (r = 0.63). It has also demonstrated convergent, divergent, and factorial validity.

The *Beck Depression Inventory, 2nd Edition* (BDI-II; Beck, Steer, & Brown, 1996) is a self-report measure of depressive symptoms such as sadness, worthlessness, and anhedonia. The measure has 21 items with 4 statements each. These statements reflect varying levels of depressive symptoms and respondents indicate which statement best captures their experiences from the previous two weeks. Higher scores represent greater depressive symptom severity. The BDI-II has shown discriminant, content, and factorial validity (Beck, Steer, & Brown, 1996).

Criteria for Sudden Gains

Sudden gains on the IUS-PW, PSWQ-PW, and GAD-SBQ-PW were assessed using the criteria outlined by Deschênes and Dugas (2013), adapted from Tang and DeRubeis (1999). The criteria for a sudden gain focus on both the absolute and relative magnitudes of the gain. A sudden gain was identified if the following three criteria were met. *Criterion 1* requires that absolute magnitude of the gain be large. Consistent with previous studies in anxiety disorders (e.g., Clerkin et al., 2008; Deschênes & Dugas, 2013), we assessed absolute magnitude using the Reliable Change Index (RCI; Jacobson & Truax, 1991). We calculated the RCI of each respective measure by dividing the mean pre- to posttreatment change score across participants by the standard error of the difference score. In order to meet this first criterion, the minimum

decrease required between two sessions was 2.63 for the IUS-PW, 3.87 for the PSWQ-PW, and 1.02 for the GAD-SBQ-PW, respectively. *Criterion 2* requires that the relative magnitude of the gain be large. Consistent with Deschênes and Dugas, we defined this as a 25% or greater reduction in scores from the pre-gain session. *Criterion 3* requires that the gain be large relative to fluctuations preceding and following the gain. We required that the gain be at least 1.5 SD relative to each individual's mean score across all treatment sessions, as advised by Kelly and colleagues (2005). This modification of the original criterion defined by Tang and DeRubeis accounts for individual variability and allows sudden gains to be identified early in treatment. A sudden gain reversal constituted a loss of 50% or more of the original gain at any subsequent session (Tang & DeRubeis, 1999).

Procedure

Concordia University and the Hôpital du Sacré-Cœur de Montréal provided ethical approval for the study. Participants were recruited via advertisements placed in a local newspaper (see *Appendix A*). Interested individuals contacted our clinic and underwent telephone screening after providing informed consent (see *Appendix B*). After initial screening for the presence of GAD and high IU, retained individuals completed two diagnostic interviews. A licensed clinical psychologist conducted the ADIS and a team psychiatrist conducted the MINI. The final diagnosis was confirmed at a team meeting following a discussion of the diagnostic interview results. Participants were included in the study if they met the following criteria: (1) primary diagnosis of GAD with a minimum severity of 4 on the ADIS; (2) elevated intolerance of uncertainty, defined as an IUS score of 58 or greater (Koerner & Dugas, 2008); (3) 18 years of age or over; (4) fluent in French; (5) no current suicidal ideation; (6) no current or past history of psychosis or bipolar depression; (7) no current substance abuse or dependence; (8) no change in psychoactive mediation dose or type 12 weeks prior to the initial assessment; (9) willingness to maintain stable dose and type of psychoactive medication during the treatment phase of the study; and (10) no concurrent psychological treatment for anxiety or depression. Participants provided informed consent for treatment following their enrollment in the study (see *Appendix C*). Potential participants who did not meet these inclusion criteria were provided with alternative services at the Hôpital du Sacré-Cœur de Montréal or referred to an appropriate treatment source.

Treatment consisted of a novel cognitive-behavioural treatment (CBT) targeting intolerance of uncertainty exclusively via behavioural experiments. Treatment was composed of

three modules: (1) psychoeducation and uncertainty awareness training, (2) testing beliefs about uncertainty through behavioural experiments, and (3) relapse prevention. Each participant completed 12 weekly, 50-minute therapy sessions with a licensed clinical psychologist trained in this protocol. The study authors (E.A.H. and M.J.D.) conducted weekly clinical supervision to ensure treatment integrity. Participants completed the PSWQ-PW, IUS-PW, and GAD-SBQ-PW prior to each therapy session. Participants completed all treatment outcome measures (i.e., ADIS, PSWQ, WAQ, BAI, BDI-II) at pre-, mid-, and posttreatment and at 3- and 6-month follow-up. For the purposes of this study, we have focused on pretreatment, posttreatment, and 6-month follow-up scores on these measures.

Results

Total scores for the IUS-PW, PSWQ-PW, and GAD-SBQ-PW were calculated for each participant at each treatment session. Missing items for past-week measures were handled with mean substitution, given that they composed only 1.19% of the data (Deschênes & Dugas, 2013; Tabachnick & Fidell, 2012). Missing treatment outcome data were present for one participant at 6-month follow-up. Data were imputed using the last available assessment point, as this provided more conservative estimates of treatment outcome than did removal of the participant.

Sudden Gains Across Measures

Across all measures (IUS-PW, PSWQ-PW, and GAD-SBQ-PW), a total of 21 sudden gains were identified. Six participants (85.71%) experienced at least one sudden gain over the course of treatment. Of these six participants, all experienced two or more sudden gains across all measures.

IUS-PW. Over the course of treatment, six participants experienced at least one sudden gain on the IUS-PW. Ten sudden gains in total were identified on the IUS-PW, with two participants experiencing two sudden gains and one participant experiencing three. Five of the ten sudden gains found in our sample were reversed (50%). An equivalent number of IUS-PW sudden gains occurred during the first and second halves of treatment. Two sudden gains occurred at session 5, two occurred at session 7, and one occurred at sessions 3, 4, 6, 8, 10, and 11, respectively. The average sudden gain magnitude (M = 13.70) on the IUS was substantially larger than the average pre-posttreatment change (M = 9.00). In other words, the average sudden gain magnitude accounted for 152.22% of the average pre-posttreatment change on this measure.

PSWQ-PW. Five participants experienced at least one sudden gain on the PSWQ-PW during treatment. Six sudden gains were identified on the PSWQ-PW, with one participant experiencing two sudden gains. Two of the six sudden gains on this measure (33.33%) were reversed at some point in treatment. The majority (66.67%) of PSWQ-PW sudden gains occurred within the first half of treatment. Two sudden gains occurred at Session 6 and one occurred at sessions 2, 4, 8, and 11, respectively. The average sudden gain magnitude accounted for 186.67% of the pre-posttreatment change in PSWQ-PW scores.

GAD-SBQ-PW. Three participants experienced at least one sudden gain on the GAD-SBQ-PW during treatment. A total of five sudden gains on the GAD-SBQ-PW were identified, with two participants experiencing two sudden gains. Two of the five sudden gains found in our sample were reversed (40%). The majority (60%) of GAD-SBQ-PW sudden gains occurred within the first half of treatment. One sudden gain occurred at sessions 3, 5, 6, 8, and 10, respectively. The average magnitude of sudden gains on this measure accounted for 119.32% of pre-posttreatment score changes.

Temporal Sequence of Sudden Gains

Of the 21 sudden gains that occurred in our sample across three measures (PSWQ-PW, IUS-PW, and GAD-SBQ-PW), the majority occurred in conjunction with at least one other sudden gain (see *Table 6* for sequence of sudden gains by participants). Eight (8) sudden gains occurred on one measure in isolation at a given time point, whereas 13 sudden gains occurred at the same time as at least one other gain within the same participant. Across the three measures, six participants experienced at least two sudden gains; three participants experienced at least three sudden gains; two participants experienced at least five sudden gains; and one participant experienced seven sudden gains.

First sudden gains. We examined first sudden gains, as these initial rapid changes are the first link in a chain of nonlinear changes. Which sudden gains came first? The majority of first sudden gains involved the IUS-PW (44.44%), followed by the PSWQ-PW (33.33%), and the GAD-SBQ-PW (22.22%). Like overall sudden gains in our sample, the majority of these first sudden gains occurred in conjunction with a sudden gain on another measure. This distinction made important differences in the likelihood of a subsequent sudden gain. If the first sudden gain occurred in combination with a sudden gain on another measure (6/9 first sudden gains), the likelihood of it leading to a second sudden gain was 66.66%. The likelihood of it leading to a

Table 6

Sequence of Sudden Gains Across Participants.

Participant	First Gain	Second Gain	Third Gain	Fourth Gain
901	PSWQ-PW	IUS-PW	GAD-SBQ-PW	IUS-PW, GAD-SBQ-PW
902	IUS-PW, GAD-SBW-PW	IUS-PW, PSWQ-PW GAD-SBW-PW	IUS-PW, PSWQ-PW	---
903	IUS-PW	PSWQ-PW	---	---
904	---	---	---	---
905	GAD-SBQ-PW	IUS-PW	---	---
906	IUS-PW, PSWQ-PW	---	---	---
907	IUS-PW, PSWQ-PW	IUS-PW	---	---

Note. PSWQ-PW = Penn State Worry Questionnaire – Past Week; IUS-PW = Intolerance of Uncertainty Scale – Past Week; and GAD-SBQ-PW = Generalized Anxiety Disorder Safety Behaviours Questionnaire – Past Week.

third sudden gain was 33.33%. First sudden gains that occurred in combination with another sudden gain were most likely to lead to second sudden gains on the IUS-PW (50%). They were equally likely to lead to second gains on the PSWQ-PW (25%) and the GAD-SBQ-PW (25%). In contrast, first sudden gains that occurred in isolation (3/9 first sudden gains) all lead to second sudden gains. The likelihood of a third sudden gain was 33.33%, as was the likelihood of a fourth sudden gain. These isolated first sudden gains were more likely to lead to second sudden gains on the IUS-PW (66.66%) than the PSWQ-PW (33.33%) or the GAD-SBQ-PW (0%).

Sequence of sudden gains across measures. If the first sudden gain involved the IUS-PW (4/9 first sudden gains), there was a 75% probability of a second sudden gain and a 25% probability of a third. When a subsequent sudden gain occurred, the likelihood that it would involve the IUS-PW or the PSWQ-PW was 42.86% for both measures, respectively. The likelihood of a subsequent sudden gain on the GAD-SBQ-PW was 14.28%. Thus, initial sudden gains on the IUS-PW tended to lead to subsequent gains on the IUS-PW or PSWQ-PW.

If the first sudden gain involved the PSWQ-PW (3/9 first sudden gains), the probability of a second sudden gain was 66.67%, the probability of a third sudden gain was 33.33%, and the probability of a fourth sudden gain was 33.33%. When a subsequent sudden gain occurred, it was most likely to involve the IUS-PW (60.00%). The likelihood of a subsequent sudden gain on the GAD-SBQ-PW was 40.00%. Thus, initial sudden gains on the PSWQ-PW tended to lead to subsequent gains on the IUS-PW or GAD-SBQ-PW.

If the first sudden gain involved the GAD-SBQ-PW (2/9 first sudden gains), the probability of a second sudden gain was 100% and the probability of a third sudden gain was 50.00%. In other words, first sudden gains involving the GAD-SBQ-PW always lead to at least one subsequent sudden gain. When a subsequent sudden gain occurred, it was most likely to involve the IUS-PW (50%). The likelihood of subsequent sudden gains on the PSWQ-PW and GAD-SBQ-PW were 33.33% and 16.67%, respectively. Thus, first sudden gains involving the GAD-SBQ-PW were most likely to lead to subsequent sudden gains on the IUS-PW.

Sudden Gains and Treatment Outcome

We did not compare sudden gainers to non-sudden gainers, as only one participant did not experience a sudden gain over the course of treatment. Due to the importance of early sudden gains to treatment outcome, we created two groups: (1) those who experienced at least one sudden gain in the first six sessions of treatment (Early Sudden Gainers, or ESG) and (2) those

who did not experience a sudden gain in the first half of treatment (No Early Gains, or NEG). At pretreatment, the ESG group (5 participants) did not significantly differ from the NEG group (2 participants) on the severity of clinician- or self-rated GAD symptoms, worry, IU, general anxiety symptoms. The groups also did not significantly differ on age, sex, educational attainment, GAD duration, number of comorbid conditions, medication use, or previous treatment experience. However, the groups did significantly differ in pretreatment depression scores, with the NEG endorsing greater depressive symptoms on the BDI-II, $F = .006$, $t = 3.243$, $p = .023$. Due to our small sample size, we were not able to statistically control for this pretreatment difference. See *Table 7* for pretreatment, posttreatment, and 6-month follow-up scores on treatment outcome variables.

Short-term treatment outcome. First, we evaluated group differences at post-treatment. GAD remission was calculated as scores below 4 on the ADIS. At posttreatment, the ESG had a 100% remission rate whereas the NEG group had a 50% remission rate. We also assessed end-state functioning, calculating the total number of treatment outcome measures on which a participant achieved clinically significant change (i.e., ADIS, WAQ, PSWQ, IUS, BAI, and BDI-II). Low end-state functioning was defined as clinically significant change on 0-2 measures; moderate represented 3-4 measures; and high represented 5-6 measures. On average, the ESG group had moderate end-state functioning ($M = 4.8$, $SD = 1.30$) whereas the NEG group had low end-state functioning ($M = 2.00$, $SD = 2.83$). This difference was not statistically significant, as assessed by an independent samples *t*-test. The ESG and NEG groups significantly differed on post-treatment WAQ scores, with the ESG group having statistically significantly lower scores, $F = .000$, $t = 2.818$, $p = .037$. No significant group differences were found on the ADIS, PSWQ, BAI, or BDI-II. However, the ESG group had numerically lower scores than the NEG group on each of these measures.

Long-term treatment outcome. We then examined group differences at the last remaining follow-up point: 6-months post-treatment. Across all measures, the ESG group had numerically lower scores than the NEG group. At this time point, the ESG group had a 100% remission rate whereas the NEG group had a 0% remission rate – a statistically significant difference ($\chi = 7.00$, $p = .008$; likelihood ratio = 8.38, $p = .004$). On average, the ESG group had high end-state functioning ($M = 5.00$, $SD = 1.25$) whereas the NEG group had low end-state functioning ($M = 1.50$, $SD = 2.12$) – a statistically significant difference ($F = 1.250$, $t = 2.887$,

Table 7

Means and Standard Deviations for Early Sudden Gains and No Early Gains Groups

Measure and Time	ESG Group		NEG Group	
	M	*SD*	*M*	*SD*
ADIS				
Pretreatment	5.80	.57	4.75	1.06
Posttreatment	3.00	.61	3.50	2.12
6-month follow-up	1.60	1.52	4.50	.71
WAQ				
Pretreatment	38.00	5.43	39.25	6.72
Posttreatment	20.20	6.21	34.75	6.01
6-month follow-up	23.20	3.95	31.00	9.90
PSWQ				
Pretreatment	63.80	5.63	56.00	1.41
Posttreatment	48.40	9.29	54.00	4.24
6-month follow-up	48.4	4.39	58.00	4.24
BAI				
Pretreatment	20.00	10.17	33.00	1.41
Posttreatment	6.60	6.99	18.00	16.97
6-month follow-up	9.8	9.78	19.50	19.09
BDI-II				
Pretreatment	12.60	2.70	20.00	2.83
Posttreatment	1.2	1.30	11.00	7.07
6-month follow-up	2.8	2.49	12.5	.71

Note. ESG = Early sudden gains group; NEG = No early gains group; ADIS = Clinical Severity Rating of the Anxiety Disorders Interview Schedule for DSM-IV; WAQ = Worry and Anxiety Questionnaire for DSM-5; PSWQ = Penn State Worry Questionnaire; BAI = Beck Anxiety Inventory; and BDI-II = Beck Depression Inventory, 2nd Edition.

p = .034). The ESG and NEG groups differed significantly on PSWQ (F = .024, t = 2.630, p = .047) and BDI-II scores (F = 1.218, t = 5.154, p = .004). There was a trend toward significance on ADIS scores (F = 4.311, t = 2.489, p = .055), and no significant differences on WAQ or BAI scores.

Discussion

The main goals of this study were to examine the relative proportion and sequence of sudden gains in IU, worry, and safety behaviours as well as the relationship between early sudden gains and treatment outcome. We found that sudden gains were the rule rather than the exception for participants who completed a novel, 12-week CBT protocol targeting IU. More specifically, 85.71% of participants experienced a sudden gain in IU, 71.43% in worry, and 42.86% in safety behaviours. This is consistent with our expectation that the greatest number of sudden gains would occur in IU, given that our treatment solely targets this cognitive mechanism. Interestingly, our sample demonstrated a higher proportion of sudden gains than in previous investigations of generalized anxiety disorder (20.34%, Deschênes & Dugas, 2013; 34.50%, Present et al., 2008) or in a recent meta-analysis of sudden gains in anxiety and depressive disorder treatments (14.60-52.20%, Aderka et al., 2012). The reversal rate for sudden gains in our study ranged from 33.33-50.00% across 3 measures, largely consistent with previous literature in GAD (54.00%, Deschênes & Dugas, 2013; 40.00%, Present et al., 2008) as well as anxiety and depression (9.10-85.70%, Aderka et al., 2012). It is possible that the novel CBT protocol used in the current study produces more sudden gains than other psychological treatments for GAD, although this cannot be conclusively established from our study design.

To the best of our knowledge, the current study is the first investigation of the sequence of sudden gains in GAD symptoms and a cognitive mechanism (in this case, IU). We found that sudden gains were more likely to occur first in IU, rather than in worry or safety behaviours. These initial sudden gains in IU were most likely to lead to further sudden gains in IU and worry. This finding is relatively consistent with investigations of linear change demonstrating that reductions in IU precede reductions in excessive worry (Dugas & Ladouceur, 2000). Thus, there is some evidence that rapid, non-linear changes in IU and GAD symptoms follow a sequence similar to linear changes in these same variables. The pattern of results is consistent with the temporal aspects of cognitive mediation theories, which suggest that changes in beliefs precede changes in symptoms. In this case, rapid changes in beliefs about uncertainty were more likely to

occur first than were changes in excessive worry and safety behaviours. Our findings also provide indirect support for the cognitive-behavioural model of IU on which this novel treatment protocol is based, which highlights the temporal precedence and clinical significance of catastrophically negative beliefs about uncertainty.

Early sudden gains had a unique relationship to treatment outcome in our sample, particularly long-term outcome. Remission rates at posttreatment and 6-month follow-up for early sudden gainers was 100%, as compared to those who did not experience sudden gains in the first six sessions (50% and 0% at posttreatment and 6-month follow-up, respectively). Early sudden gainers had higher average end-state functioning at both posttreatment and 6-month follow-up, with statistically significant between-group differences at 6-month follow-up. Early sudden gainers exhibited lower scores on all outcome measures at posttreatment and 6-month follow-up as compared to those with no early gains. Several of these differences were statistically significant at 6-month follow-up (i.e., excessive worry, depressive symptoms), with one approaching statistical significance (i.e., clinician-rated GAD symptoms). Only one comparison was statistically significant at posttreatment (i.e., self-rated GAD symptoms). These between-group comparisons are limited by our small sample size and restricted statistical power. However, our findings suggest that early sudden gains may be especially relevant to long-term outcome in CBT for GAD. This is consistent with the positive treatment outcomes associated with early sudden gains in depression (Busch et al., 2006; Kelley et al., 2005) and general psychopathology (Stiles et al., 2003).

This study had several strengths. First, we assessed sudden gains in both GAD symptoms and a cognitive mechanism underlying GAD symptoms (i.e., IU). This allowed us to investigate the relative proportions of sudden gains amongst these variables as well as their temporal sequence. Second, we used multiple measures to assess past-week GAD symptoms. This provided information about the relative importance and sequence of sudden gains in excessive worry and safety behaviours. Additionally, this is the first study to our knowledge that has evaluated sudden gains in GAD-related safety behaviours. Third, we used multiple measures of treatment outcome, including clinician- and self-rated instruments. All but one of these treatment outcome measures did not overlap with measures used to assess sudden gains. Fourth, we assessed both short- and long-term treatment outcomes. Fifth, our treatment protocol focused on a single therapeutic strategy (i.e., behavioural experiments). This reduces the need for a future

dismantling study that would compare the relative impact of multiple cognitive-behavioural techniques.

This investigation also had important limitations. First, our small sample size decreased our statistical power and thus limited the statistical analyses that could be performed. Thus, it is unclear if the non-significant group comparisons at posttreatment are truly due to a lack of effect or alternatively due to limited power. Similarly, we could not statistically control for pretreatment group differences in depressive symptoms. Second, our sample consisted entirely of self-identified White Francophone participants. It is therefore unclear if our results will generalize to non-White and/or Anglophone populations. However, similar samples have been used in several previous investigations of CBT for GAD (e.g., Bélanger, Morin, Langlois, & Ladouceur, 2004; Primiano et al., 2014; Théberge-Lapointe, Marchand, Langlois, Gosselin, & Watts, 2015). Our Francophone sample also provides diversity within the sudden gains literature, which has primarily focused on Anglophones. Third, a waitlist or active control condition would have allowed us to assess spontaneous sudden gains (in the case of a waitlist control condition) or directly compare sudden gains across treatment modalities.

Implications and Conclusions

Early sudden gains may improve long-term GAD treatment outcome in a number of ways. Rapid changes in beliefs and/or GAD symptoms in the first half of treatment may produce greater feelings of self-efficacy (Bandura, 1977; Deschênes & Dugas, 2013; Kelly et al., 2005). This increased self-efficacy may become particularly relevant after active treatment has finished, as a participant must apply the skills learned in therapy without the support of his or her therapist. The novel treatment protocol used in this study may also partially explain the relationship between early sudden gains and treatment outcome. Early sudden gains in the context of the current study's CBT protocol may signal more effective use of behavioural experiments, the proposed "active ingredient" of this therapy. Early sudden gains might indicate greater client understanding of the behavioural experiment technique, the identification of more personally relevant beliefs about uncertainty, or the early development of alternative beliefs about uncertainty. These changes may foster continued application of therapy skills, leading to greater reductions in GAD symptoms or increased resistance to relapse.

This CBT protocol's emphasis on repeated behavioural experiments targeting beliefs about uncertainty may produce a greater proportion of sudden gains in IU and GAD symptoms

than previous studies. However, this possibility should be evaluated in a larger clinical trial. To establish the relative impact on cognitive versus behaviourally focused interventions in GAD, future investigations should compare sudden gains using behavioural experiments to sudden gains using habituation-based behavioural techniques (e.g., situational or imaginal exposure). Based on our findings regarding the precedence of sudden gains in beliefs over sudden gains in GAD symptoms, we also suggest that future sudden gains investigations incorporate measures of relevant cognitive mechanisms in addition to symptom measures.

CHAPTER 5
GENERAL DISCUSSION

IU is a dispositional characteristic that results from negative beliefs about uncertainty and the consequences of uncertainty (Dugas & Robichaud, 2007). In our novel cognitive-behavioural conceptualization of IU, we proposed that catastrophically negative beliefs about uncertainty are activated when situational characteristics provoke feelings of uncertainty. These catastrophic, negative beliefs about uncertainty then result in the worry, anxiety, and safety behaviours characteristic of GAD (see *Figure 2*). The current program of research involved the development and evaluation of a novel CBT protocol based on this conceptual model. The protocol employed behavioural experiments to target idiosyncratic beliefs about uncertainty. A total of seven participants completed the 12-week CBT protocol, which included (1) psychoeducation and uncertainty awareness training, (2) repeated behavioural experiments to test idiosyncratic beliefs about uncertainty, and (3) relapse prevention. Overall, our results demonstrated that the treatment produced substantial reductions in IU and GAD symptoms by posttreatment that were relatively maintained across a 6-month follow-up period. Treatment also reduced general psychopathology symptoms over the study period, specifically depression and general anxiety. In addition, we found that sudden gains in IU tended to occur first rather than sudden gains in worry and safety behaviours. Improved long-term treatment outcome was associated with sudden gains that occurred early in treatment. This pattern of findings provides preliminary support for the efficacy of this parsimonious CBT protocol, as well as the utility of behavioural experiments for IU. We will discuss the theoretical and clinical implications of these results.

Novel Conceptualization of IU

To maximize clinician understanding of, adherence to, and interest in CBT protocols, treatments must clearly articulate the specified theory of change. This should include detailed information about therapeutic concepts, their interrelationships, and how to best target these variables (David, 2004). The CBT protocol developed and evaluated for the current program of research was based on a novel cognitive-behavioural conceptualization of IU. This conceptualization is in keeping with traditional British models of psychopathology, including those for panic disorder (Clark, 1986), obsessive-compulsive disorder (Salkovskis, 1999), and hypochondriasis (Warwick & Salkvoskis, 1990). In each of these models, the individual misinterprets internal or external triggers, leading to a variety of emotional, physiological,

cognitive, and/or behavioural sequelae. Our CBT model of IU highlighted catastrophically negative beliefs about uncertainty as the motor that drives the symptoms of GAD. We proposed that specific situational characteristics – namely, ambiguity, novelty, and unpredictability – generate feelings of uncertainty. When this state of uncertainty is misinterpreted in a catastrophically negative manner, worry, anxiety, and safety behaviours occur. Overall, the findings from the current program of research indirectly supported this novel model of IU. The majority of therapy sessions within our protocol focused on catastrophically negative beliefs about uncertainty. Our largely positive short- and long-term treatment outcomes reinforce the centrality of these beliefs about uncertainty within GAD. Similarly, the temporal precedence of IU-related sudden gains emphasizes the proposed sequence of our conceptual model. However, this must be confirmed in future investigations, as the sequence of change within therapy may differ from the theoretical sequence of pathology prior to intervention. Moreover, the temporal sequence of change during treatment may differ when examined outside of sudden gains.

The equality of worry, anxiety, and safety behaviours within our model was also indirectly supported. Although many GAD treatment techniques – such as worry scheduling (Borkovec, Alcaine, & Behar, 2004) and imaginal exposure (Dugas & Robichaud, 2007) – address worry content directly, our model and treatment protocol did not highlight worry content per se. Our efficacy-related results suggest that it may not be necessary for cognitive-behavioural conceptualizations of GAD to focus on worry in order to produce successful therapeutic outcomes. This may have clinical utility for clients who have been unsuccessfully treated with worry-based interventions. Moreover, this may enhance the possible transdiagnostic applicability of our model and treatment for IU, given that the clinical emphasis remains on beliefs about uncertainty rather than disorder-specific symptoms. The conceptual addition of safety behaviours was also indirectly supported by our results, as this symptom dimension was endorsed by our clinical participants and decreased as a result of treatment. Future research should consider this often overlooked aspect of GAD.

Behavioural Experiments for IU

Techniques that activate the sensory, experiential systems of anxiety in addition to verbal representations may be more successful than treatments that only impact one of these aspects. The development of new treatments may be most fruitful when both components are incorporated (McManus, Grey, & Shafran, 2008). In other words, clinicians should focus on

"doing therapy" rather than "talking therapy" (Waller, 2009). Behavioural experiments may represent the ideal technique for addressing both cognitions and behaviours in an experiential manner. Behavioural experiments, which have been identified as an effective therapeutic technique in the treatment of anxiety, are present in several CBT protocols for anxiety disorders (Woody & Ollendick, 2006). To our knowledge, the current program of research was the first to evaluate the use of systematic behavioural experiments as a stand-alone treatment for GAD.

We found that behavioural experiments targeting beliefs about uncertainty were efficacious in reducing IU, GAD symptoms, and general psychopathology. This suggests that behavioural experiments have both specific and generalized effects. On the one hand, this technique can effectively target highly specialized constructs: our results demonstrate that we were indeed able to target IU successfully. On the other hand, targeting beliefs about uncertainty via behavioural experiments reduced GAD symptoms as well as more peripheral symptoms of general psychopathology. This is consistent with cognitive mediation theories of emotion and psychopathology (e.g., Beck, 1976; Beck et al., 1979), which suggest that changes in underlying beliefs precede changes in symptomatology. Our behavioural experiment-based treatment targeted underlying beliefs about uncertainty throughout the 12-week therapy period, and produced substantial changes in GAD symptoms at posttreatment and 6-month follow-up. Our findings regarding sudden gains also provide some support for cognitive mediation, as sudden gains involving IU were most likely to occur first. Cognitive mediation theory suggests that symptoms may not need to be targeted directly in order for treatment to be successful. Indeed, our behavioural experiments did not need to target GAD symptoms in order to reduce them. This provides a novel focus within the GAD literature, as previously published behavioural experiments for GAD have often emphasized worry content even when targeting IU (Butler & Rouf, 2005).

The current program of research also demonstrated that behavioural experiments can be used in isolation as the sole means of targeting IU. This stands in contrast to the traditional method of treating IU, in which a combination of cognitive-behavioural techniques has been used (Dugas & Robichaud, 2007). The inherent flexibility of behavioural experiments allowed participants to test idiosyncratic beliefs about uncertainty rather than a narrow, prescribed range of cognitions. This encouraged creativity and individualization within a structured framework. Thus, behavioural experiments represent a parsimonious, flexible, and direct method of targeting

beliefs about uncertainty that may provide comparable reductions in IU. In fact, we found preliminary evidence to suggest that the current behavioural experiment-based protocol may produce larger changes in IU than our standard CBT-IU protocol. However, this finding should be interpreted with caution given the small sample size and lack of direct comparison between treatment protocols.

Behavioural experiments produced sudden gains in IU, suggesting that beliefs about uncertainty can undergo rapid between-session changes in addition to gradual change over time. Behavioural experiments may promote sudden gains in beliefs about uncertainty via the experiential learning and reflection inherent in the technique (Bennett-Levy et al., 2005; Kolb, 1984; Lewin, 1946). Participants were encouraged to treat their beliefs about uncertainty as hypotheses to be tested. Participants then monitored and actively reflected on the relationship between the experiment's outcome and their beliefs about uncertainty. Behavioural experiments may have also fostered sudden gains in IU via activation of both the implicational and propositional information processing systems (Teasdale, 1997; Teasdale & Barnard, 1993). This participatory style of learning, active reflection, and activation of both implicational and propositional information processing may all promote rapid between-session changes in IU as well as GAD symptoms. Sudden gains that occur early in a behavioural experiment-based therapy may be more relevant to long-term GAD treatment outcome than to short-term outcomes. Early "successes" in behavioural experiments may promote sudden gains. "Successes" might include the targeting of more relevant cognitions, experiments that generate greater information, or enhanced client understanding of the rationale or procedural steps of a behavioural experiment. These early successes could create a "positive spiral" (Kelly, Roberts, & Ciesla, 2005) that enhances perceived self-efficacy. When sudden gains occur early in therapy, they may allow clients to capitalize on their gains and have greater opportunities to practice newly learned skills, facilitated by the therapist. If the client has greater mastery of the cognitive-behavioural technique, this may provide a "safety net" for participants once therapy has ended, promoting long-term maintenance of gains and preventing relapse.

Behavioural experiments for IU may have reduced symptoms of GAD and general psychopathology in several ways. Behavioural experiments employ principles of experiential learning and reflection (Kolb, 1984; Lewin, 1946) to promote change. This perspective suggests that participants gained information by engaging in experiential exercises and later used active

reflection to process this information in the context of their pre-existing belief structures. The information gained in early behavioural experiments possibly disconfirmed some or all aspects of participants' pre-existing negative beliefs about uncertainty. In modern learning theory, the violation of negative expectations (Bouton, 2004; McMillan & Lee, 2010) is theorized to lead to reduced anxiety and fear responses. The disconfirmatory experiences fostered by behavioural experiment may have modified participants' existing beliefs about uncertainty (Salkovskis, Hackmann, Wells, Gelder, & Clark, 2006) or may have generated new beliefs or mental representations (Bouton, 2004; Gavetti & Levinthal, 2000; Pearce & Hall, 1980). Similarly, behavioural experiments may have generated alternative mental representations by engaging both the propositional and implicational information processing systems (Bennett-Levy et al., 2005; Teasdale, 1997; Teasdale & Barnard, 1993). These changes in beliefs about uncertainty then, according to cognitive mediation theory, may have caused cascading changes in GAD symptomatology.

Implications for GAD Treatment Practices

Despite the introduction of several empirically-supported treatments since the 1990s, GAD remains the least successfully treated of all anxiety disorders (Gould, Safren, O'Neill Washington, & Otto, 2004). For instance, GAD relapse rates of 50% are not uncommon (Holaway, Rodebaugh, & Heimberg, 2006). Without treatment, the clinical picture is disheartening: GAD has a chronic course (Lydiard, 2000) with few spontaneous remissions (Yonkers, Warshaw, Massion, & Keller, 1996). Thus, improvements in GAD treatment remain critical. In addition to concerns of efficacy, there have been calls for greater parsimony, efficiency, and cost-effectiveness in treatment protocols (Cougle, 2012; Mansell, 2008; McManus, Van Doorn, & Yiend, 2012). Available treatments for GAD are based on several theoretical perspectives and incorporate a variety of cognitive-behavioural techniques (Behar, DiMarco, Hekler, Mohlman, & Staples, 2009), making clinical decision-making difficult. This may be particularly problematic in routine clinical settings, given the reduction in supervised training in CBT for GAD between 1993-2003 (Woody, Weisz, & McLean, 2005).

The current program of research provides preliminary evidence for the efficacy of a novel cognitive-behavioural treatment for GAD. Equally, this treatment demonstrates the potential power of parsimony and efficiency. Empirically supported treatments for GAD have typically been complex, involving multiple cognitive-behavioural techniques (e.g., Borkovec, Newman,

Pincus, & Lytle, 2002; Dugas & Robichaud, 2007; Mennin, 2004; Wells, 2006) and taking upwards of 14 sessions to administer in a research context (e.g., Newman, Przeworski, Fisher, & Borkovec, 2010; van der Heiden et al., 2012). The current treatment protocol requires clinicians to master only one major therapeutic strategy – thus reducing an important barrier to effective knowledge translation (Cougle, 2012; Dimeff et al., 2009; Mansell, 2008). This may be particularly important for clinicians with less specialized clinical training in psychological treatments, such as nurses and social workers (Bright, Baker, Neimeyer, 1999). Fewer training hours are likely required for less complex interventions (Dimeff et al., 2009; Nadort et al., 2009; Rollinson et al., 2007). Moreover, clients may benefit from the repeated practice of a single therapeutic technique, promoting the maintenance of therapeutic gains (Cougle, 2012). Because the current treatment protocol required only 12 sessions to administer, it may reduce some of the direct and indirect costs of GAD treatment such as therapist fees, transportation costs to and from therapy appointments, and missed time at work (Cougle, 2012; McManus et al., 2012).

The current treatment protocol also has advantages that enhance its clinical utility. The flexibility of this treatment protocol allows for customization based on individual client needs. Although the protocol offers 60 behavioural experiments to select from based on the target cognition, clients and clinicians are encouraged to generate alternative behavioural experiments to ensure feasibility and relevance. In other words, participants were taught the skills necessary to design and carry out successful behavioural experiments rather than simply led through predetermined exercises. This improves the ecological validity of the treatment protocol, as it allows for individualization based on case formulation (Persons, 2006). Moreover, this may enhance treatment dissemination efforts, as perceived restriction of clinical creativity and innovation may be a barrier to the implementation of empirically-supported treatments in routine clinical practice (Gunter & Whittal, 2010).

Future Directions

The current program of research built upon previous treatment studies for GAD (e.g., Dugas & Ladouceur, 2000), and attempted to address increasing calls for treatment parsimony and efficiency (Cougle, 2012). The program of research had a number of strengths. First, conducting a small-scale evaluation of a novel treatment protocol conserves research and clinical resources while ensuring that only promising avenues are pursued. Second, we used a variety of measures and methods to evaluate treatment outcome. We used both clinician-rated and self-

report measures and assessed the statistical and clinical significance of change using several methods. Third, we investigated sudden gains in terms of both GAD symptoms and IU. Fourth, we assessed outcomes at a variety of time points to determine the short and long-term effects of therapy. This provides information about immediate outcomes as well as the durability of treatment effects. Fifth, our protocol design reduced the need for a future dismantling study, as it focused on one major therapeutic technique. This is a considerable advantage within the CBT literature, as the majority of efficacy studies concern multicomponent CBT protocols (Westbrook, Kennerly, & Kirk, 2005) and do not identify the most critical intervention strategies (Longmore & Worrell, 2007). Using one major therapeutic technique increases treatment efficiency, but may also aid dissemination efforts given that clinician training would focus on a narrow range of cognitive-behavioural skills (Cougle, 2012; Mansell, 2008). In addition to these strengths, our studies also had several weaknesses. First, our small sample size reduced our statistical power and thus restricted our selection of statistical analyses. Second, our sample was restricted to White, Francophone participants. However, the inclusion of French-speaking participants contributes diversity to the GAD and anxiety literatures. Third, we did not include a waitlist or active control condition. Thus, we were not able to statistically control for the effects of time or common therapy factors. Finally, our measure of safety behaviours in GAD has not yet been empirically validated. Thus, these findings must be interpreted with caution.

Combining the strengths and limitations of the current program of research, there are several interesting avenues that could be pursued in future investigations. First, future research should extend our findings to address the question, “Does this treatment work?” This would include a randomized controlled trial with a larger sample size and a waitlist control condition. This will provide greater statistical power and permit analyses to be conducted in multilevel modeling – providing a more nuanced account of treatment outcome. This would also allow the results to be generalized beyond the current studies’ participants, therapist, hospital site, and geographical location. Following this, comparison to an active control condition may be warranted. Future replication studies should consider measures of short- and long-term cost effectiveness, quality of life indices, as well as additional variables known to be associated with GAD (e.g., neuroticism). The transdiagnostic applicability of this novel model and treatment for IU may warrant investigation, given the calls for the provision of CBT based on cognitive

mechanisms rather than diagnostic categories (Mansell, 2008). Our protocol's flexibility, structure, and focus on IU may be advantageous in this context.

Second, future investigations should extend our findings to address the question, "How does this therapy work?" via process-based research. Although we found interesting evidence concerning the temporal sequence of change within this treatment protocol, future research should confirm and extend these results. Future sudden gains studies should consider the inclusion of general anxiety and somatic anxiety symptoms in addition to the variables assessed in the current program of research. Daily monitoring could be considered as an alternative to weekly self-monitoring of symptoms to allow for time series analyses.

Third, experimental research should directly compare behavioural experiments and exposure for IU within a GAD population. This would provide key information regarding the relative efficacy and acceptability of these two techniques, as has been examined in panic disorder (Salkovskis et al., 2007). Based on our study design, it is also currently unclear if behavioural experiments that focus on IU have greater clinical utility than ones focusing on worry. However, behavioural experiments that focus on underlying cognitive mechanisms may avoid the potential pitfalls of those focused on worry. For example, behavioural experiments targeting worry may focus on disproving worry content while inadvertently fostering the need for certainty (Butler & Rouf, 2005). However, this should be evaluated empirically.

Finally, future research should address key measures of safety behaviours and IU. The safety behaviour measure introduced in this study should be empirically validated. Clinical observations (Dugas & Robichaud, 2007) as well as empirical investigations (e.g., Beesdo-Baum et al., 2012) implicate a number of safety behaviours in GAD. Psychometric validation of a GAD-specific safety behaviours measure would improve our understanding of this phenomenon as well as its relationship to successful treatment. Similarly, future investigations should consider including a more general measure of beliefs about uncertainty in order to quantitatively assess treatment-induced changes in positive and neutral beliefs about uncertainty in addition to negative ones.

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Appendix A

Advertisement for Participant Recruitment

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 nouveau traitement psychologique.

Si vous avez entre 18 ou plus 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

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

Appendix B

Information and Consent for Assessment

HÔPITAL DU SACRÉ-CŒUR DE MONTRÉAL
HSCM *Doués pour la vie*

UNIVERSITÉ Concordia UNIVERSITY

AFFILIÉ À Université de Montréal

FORMULAIRE D'INFORMATION ET DE CONSENTEMENT TÉLÉPHONIQUE[1] (Évaluation de l'admissibilité)

Titre de l'étude : Une nouvelle psychothérapie pour le trouble d'anxiété généralisée: Les expériences comportementales pour l'intolérance à l'incertitude

Chercheur principal : Michel Dugas, Ph.D.
Chercheur, 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-chercheur: Elizabeth Hebert, M.A., candidate au Ph.D. (psychologie), Département de psychologie, Université Concordia

Collaborateurs : Pierre Savard, MD, Ph.D.; Julie Turcotte, MD, M.Sc.; Thu Van Dao, MD; Éric Bugeaud, MD; Psychiatres, Clinique des troubles anxieux, Hôpital du Sacré-Cœur de Montréal

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INFORMATION

A. BUT DE L'ÉTUDE

Le but de cette étude est de déterminer si une nouvelle thérapie cognitivo-comportementale visant principalement l'intolérance à l'incertitude peut s'avérer efficace pour des adultes souffrant du trouble d'anxiété généralisée (TAG). La thérapie cognitivo-comportementale est une forme de psychothérapie qui vise à vous aider à comprendre et à changer les comportements et pensées qui contribuent à vos difficultés.

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.

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[1] *Le genre masculin, employé pour alléger le texte, désigne autant les femmes que les hommes.*

B. PROCÉDURE

Dans un premier temps, vous participerez à une entrevue d'évaluation téléphonique (durée 1 h 30) avec un psychologue de l'équipe.

S'il semble que vous rencontrez les critères de sélection de l'étude, vous serez référé à la Clinique des troubles anxieux de l'Hôpital du Sacré-Cœur de Montréal, où vous serez évalué à nouveau par un psychiatre de notre équipe. Cette évaluation se déroule en personne et est d'une durée d'une heure environ. Au début de cette rencontre, vous signerez le présent formulaire de consentement. Après cette évaluation, les membres de l'équipe de recherche (psychologues, psychiatres et chercheur principal) se réuniront pour discuter et s'assurer que 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 dans l'étude, vous aurez à signer un autre formulaire de consentement concernant la suite de l'étude. Si vous ne rencontrez pas les critères requis pour participer à l'étude, une liste de ressources sera mise à votre disposition.

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, ces entrevues ont déjà été utilisées 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. Compensation

Vous ne recevrez aucune compensation financière pour votre participation à cette étude.

2. Confidentialité

Tous les renseignements recueillis à votre sujet demeureront strictement confidentiels, dans les limites prévues par la loi, et vous ne serez identifié 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.

5. Personnes à contacter

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 (1) la Direction Générale de l'Hôpital du Sacré-Coeur, au (514) 338-2222, poste 3581; ou (2) le Conseiller en Éthique de la Recherche de l'Université Concordia, au (514) 848-2424, poste 7481 ou à ethics@alcor.concordia.ca.

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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 si je suis invité à l'évaluation qui se déroulera en personne à la Clinique des troubles anxieux, j'y signerai le présent formulaire de consentement.
- 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 révèleront 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 PSYCHIATRE DE L'ÉQUIPE :

OUI____ NON____

Nom du participant	Date (consentement verbal)	Heure (consentement verbal)

Nom de la personne qui obtient le consentement verbal	Signature	Date

Signature du participant (si évaluation en personne)	Date (consentement écrit)

Signature du chercheur responsable	Date

Appendix C

Information and Consent for Treatment

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FORMULAIRE D'INFORMATION ET DE CONSENTEMENT [2]

Titre de l'étude: **Une nouvelle psychothérapie pour le trouble d'anxiété généralisée: Les expériences comportementales pour l'intolérance à l'incertitude**

Chercheur:

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

- Elizabeth Hebert, M.A., candidate au Ph.D. (psychologie), Département de psychologie, Université Concordia

Collaborateurs :

- Pierre Savard, MD, Ph.D.; Julie Turcotte, MD, M.Sc.; Thu Van Dao, MD; Éric Bugeaud, MD; Psychiatres, Clinique des troubles anxieux, Hôpital du Sacré-Cœur de Montréal

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INFORMATION

Préambule :

Nous sollicitons votre participation à un projet de recherche. Cependant, avant d'accepter de participer à ce projet et de signer ce formulaire d'information et de consentement, veuillez prendre le temps de lire, de comprendre et de considérer attentivement les renseignements qui suivent.

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[2] *Le genre masculin, employé pour alléger le texte, désigne autant les femmes que les hommes.*

Ce formulaire peut contenir des mots que vous ne comprenez pas. Nous vous invitons à poser toutes les questions que vous jugerez utiles au chercheur responsable du projet ou aux autres membres du personnel affecté au projet de recherche et à leur demander de vous expliquer tout mot ou renseignement qui n'est pas clair.

Une participation simultanée à plusieurs études pourrait vous être préjudiciable. Si vous participez déjà à d'autres études, veuillez en informer le chercheur.

1. Nature et objectif de l'étude

Le trouble d'anxiété généralisée (TAG) se caractérise par la présence excessive et chronique d'inquiétudes et d'anxiété. Notre équipe de recherche a précédemment développé et validé un protocole de traitement cognitivo-comportemental pour les personnes atteintes du TAG. L'efficacité de notre traitement a maintenant été évaluée dans cinq essais randomisés. En général, les données indiquent que le traitement mène à la rémission du TAG chez 75% des personnes atteintes et que les gains thérapeutiques se maintiennent pour au moins deux ans suite à l'intervention. Alors que ces résultats sont encourageants, il n'en demeure pas moins que 25% des personnes présentent une faible réponse au traitement. De plus, ce protocole est passablement complexe puisqu'il compte 6 composantes administrées sur 14 à 16 rencontres. En d'autres mots, malgré sa relative efficacité, l'utilité clinique de notre traitement demeure un point d'interrogation.

L'étude proposée vise à évaluer de façon préliminaire l'acceptabilité et l'efficacité d'un nouveau protocole de traitement pour le TAG. Ce nouveau protocole est moins complexe que son prédécesseur. Il cible uniquement le facteur principal du traitement précédent; à savoir l'intolérance à l'incertitude qui est une caractéristique importante chez les personnes qui s'inquiètent de façon excessive. De plus, le nouveau protocole prévoit qu'une seule intervention (les expériences comportementales) sera utilisée pour cibler l'intolérance à l'incertitude sur un maximum de 10 rencontres de psychothérapie. Ainsi, cette étude permettra de faire une évaluation préliminaire d'une nouvelle forme de thérapie cognitivo-comportementale plus simple; celle-ci sera potentiellement plus facile à enseigner aux thérapeutes, plus facile à suivre et moins contraignante pour les personnes atteintes du TAG.

Dix (10) adultes avec un diagnostic principal de trouble d'anxiété généralisée participeront à cette étude qui se déroulera dans les locaux de la Clinique des troubles anxieux de l'Hôpital du Sacré-Cœur de Montréal. Les participants seront recrutés à cette Clinique et par le biais d'annonces placées dans les journaux comme *La Presse,* par exemple.

2. Déroulement de l'étude et méthodes utilisées

Si vous acceptez de participer à cette étude, vous devrez signer ce formulaire d'information et de consentement.

L'étude se divise en trois volet : (1) évaluation pré-thérapie; (2) thérapie cognitivo-comportementale administrée en 10 rencontres hebdomadaires; (3) évaluation après 5 rencontres de thérapie, une semaine après la fin de la thérapie et deux évaluations de suivi (3 et 6 mois après la fin de la thérapie).

Premier volet : Évaluation pré-thérapie

Suite à l'évaluation de vos symptômes d'anxiété – entrevues téléphoniques et entrevue avec un psychiatre 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 une heure avec un psychologue de notre équipe. Au cours de cette rencontre vous répondrez à des questionnaires portant sur vos symptômes d'anxiété et votre état général.

Deuxième volet : thérapie cognitivo-comportementale (TCC)

En participant à cette étude, vous recevrez une psychothérapie pour le TAG. Cette thérapie, de type cognitivo-comportementale, vise à vous aider à comprendre et à changer les comportements et pensées qui contribuent à vos difficultés. La durée de cette thérapie est d'environ trois mois (10 rencontres hebdomadaires d'une durée de 50 minutes) et elle vous sera administrée par un des psychologues de notre équipe. Entre les rencontres, vous aurez des lectures à faire et des exercices à compléter.

Troisième volet : Évaluation après 5 rencontres de thérapie, à la fin de la thérapie et 2 évaluations de suivi

Afin d'évaluer les effets de la psychothérapie à court et à long terme, vous serez évalué à quatre reprises : après 5 rencontres de thérapie ainsi qu'une semaine, 3 mois et 6 mois après la fin de la thérapie. Ces rencontres d'évaluation comprennent une entrevue diagnostique et des questionnaires.

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 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 prescrit par votre médecin ou 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 pour le TAG. 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 6 mois après la fin de la psychothérapie. Parallèlement, cette étude permettra de savoir si une thérapie plus simple, ciblant uniquement l'intolérance à l'incertitude, s'avère efficace pour diminuer les symptômes du TAG. Ainsi, cette étude contribuera à l'avancement des connaissances dans le domaine.

5. Compensation

Vous ne recevrez aucune compensation financière pour votre participation à cette étude.

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 que par un code. Les rencontres avec les psychologues seront enregistrées (audio seulement) afin de nous permettre d'évaluer la qualité des interventions offertes par ceux-ci (les fichiers audio seront aussi identifiés par un code). Immédiatement après la publication de cette étude, tous ces fichiers seront détruits. 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 de Montréal 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 leur responsabilité civile et professionnelle.

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 connaitre 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.

Le chercheur responsable, le comité d'éthique de la recherche de l'Hôpital du Sacré-Cœur de Montréal ou l'organisme subventionnaire (IRSC) peuvent mettre fin à votre participation, sans votre consentement, si de nouvelles découvertes ou informations indiquent que votre participation au projet n'est plus dans votre intérêt, si vous ne respectez pas les consignes du projet de recherche ou s'il existe des raisons administratives d'abandonner le projet.

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 :

- Département de psychologie, Université Concordia : (514) 848-2424, poste 2215
- Clinique des troubles anxieux, Hôpital du Sacré-Cœur de Montréal : (514) 338-4201

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 (1) la Direction Générale de l'Hôpital du Sacré-Cœur de Montréal, au (514) 338-2222, poste 3581; ou (2) le Conseiller en Éthique de la Recherche de l'Université Concordia, au (514) 848-2424, poste 7481 ou à « ethics@alcor.concordia.ca ».

10. Surveillance des aspects éthiques du projet

Les comités d'éthique de la recherche de l'Hôpital du Sacré-Cœur de Montréal et de l'Université Concordia ont approuvé ce projet de recherche et en assurent le suivi. De plus, ils approuveront au préalable toute révision et toute modification apportée au formulaire d'information et de consentement et au protocole de recherche.

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CONSENTEMENT

Titre de l'étude : Une nouvelle psychothérapie pour le trouble d'anxiété généralisée: Les expériences comportementales pour l'intolérance à l'incertitude

La nature de cette étude, les procédés utilisés, 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 participant	Signature	Date

Signature de la personne qui a obtenu le consentement si différent du chercheur responsable du projet de recherche.

J'ai expliqué au participant les termes du présent formulaire d'information et de consentement et j'ai répondu aux questions qu'il m'a posées.

Nom de la personne qui obtient le consentement	Signature	Date

Signature et engagement du chercheur responsable du projet

Je certifie qu'on a expliqué au participant les termes du présent formulaire d'information et de consentement, que l'on a répondu aux questions qu'il avait à cet égard et qu'on lui a clairement indiqué qu'il demeure libre de mettre un terme à sa participation, et ce, sans préjudice.

Je m'engage, avec l'équipe de recherche, à respecter ce qui a été convenu au formulaire d'information et de consentement et à en remettre une copie signée au participant.

Nom du chercheur responsable du projet de recherche	Signature	Date

Appendix D

Generalized Anxiety Disorder-Safety Behaviours Questionnaire

QCS-TAG

No de participant: _____ Date: _____

Les énoncés suivants présentent des comportements sécurisants rapportés par des personnes ayant un trouble d'anxiété généralisée. Veuillez indiquer jusqu'à quel point chacun de ces comportements est typique de vous **au cours du dernier mois** en encerclant le chiffre approprié (1 à 5). Il n'y a pas de bonne ou mauvaise réponse. **Si votre réponse est 3 ou plus,** veuillez svp inscrire un exemple personnel de ce comportement que vous avez eu au cours du dernier mois.

	Pas du tout typique de moi	Peu typique de moi	Assez typique de moi	Très typique de moi	Extrêmement typique de moi
1. Je fais constamment autre chose pour éviter de commencer ce que je devrais faire. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
2. J'évite fréquemment des situations dont l'issue pourrait être négative *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
3. Je demande souvent aux autres (conjoint, amis, famille, collègue) de me rassurer. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
4. À répétition, je refais ou change des choses à des tâches que j'ai déjà terminées. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
5. Je cherche beaucoup d'information afin de me sentir rassuré. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
6. J'évite de m'engager totalement dans un emploi, un projet ou une relation parce que je ne suis par certain de comment ça va se passer. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5

7. Je prends plus de temps que nécessaire (ou que la plupart des gens) pour préparer les choses. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
8. Je vérifie constamment pour m'assurer que j'ai bien terminé une tâche ou un projet. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
9. Je cherche constamment de la réassurance à cause de mes inquiétudes. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
10. J'observe constamment mon corps et mes sensations physiques à la recherche d'un «signe » de maladie. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
11. Je vérifie constamment pour m'assurer que j'ai bien fait une tâche ou un projet correctement. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
12. Je dis toujours aux autres quoi faire parce que je ne suis pas certain de comment les choses vont se passer. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
13. Je remets souvent une prise de décision ou un comportement à cause de mes inquiétudes. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
14. J'évite constamment des situations dans lesquelles je me sens incertain. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
15. Je recherche souvent des gens					

en particulier, des objets ou des routines dans le but de me sentir plus « certain ». *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
16. J'essaie de prendre le contrôle des choses en faisant tout moi-même *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
17. Je prends beaucoup de temps et d'effort pour me préparer à des situations pour lesquelles un résultat négatif peut arriver *Exemple personnel (si 3 ou +) :*	1	2	3	4	5
18. Je vérifie à répétition si j'ai bien verrouillé la porte ou si j'ai bien éteint la cuisinière. *Exemple personnel (si 3 ou +) :*	1	2	3	4	5

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Appendix E: Intolerance of Uncertainty Scale – Past Week

ÉII 1.1

ÉII-DS

No. Dossier _____ Rencontre _____ 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 **au cours de la dernière semaine.**

	Pas du tout correspondant	**Un peu correspondant**	**Assez correspondant**	**Très correspondant**	**Tout à fait correspondant**
1. L'incertitude m'empêchait de prendre position.	1	2	3	4	5
2. L'incertitude me rendait la vie intolérable.	1	2	3	4	5
3. L'incertitude me rendait mal à l'aise, anxieux(se) ou stressé(e).	1	2	3	4	5
4. Les imprévus me dérangeaient énormément.	1	2	3	4	5
5. Ça me frustrait de ne pas avoir toute l'information dont j'avais besoin.	1	2	3	4	5
6. L'incertitude m'empêchait de profiter pleinement de la vie.	1	2	3	4	5
7. Lorsque c'était le temps d'agir, l'incertitude me paralysait.	1	2	3	4	5
8. Lorsque j'étais incertain(e), je ne pouvais pas bien fonctionner.	1	2	3	4	5
9. L'incertitude me rendait vulnérable, malheureux(se) ou triste.	1	2	3	4	5
10. Le moindre doute pouvait m'empêcher d'agir.	1	2	3	4	5
11. L'incertitude m'empêchait de bien dormir.	1	2	3	4	5
12. Les ambiguïtés de la vie me stressaient.	1	2	3	4	5

Appendix F: Generalized Anxiety Disorder-Safety Behaviours Questionnaire – Past Week

QCS-TAG-DS

No de participant: _____ Rencontre : _____ Date: _____

Les énoncés suivants présentent des comportements sécurisants rapportés par des personnes ayant un trouble d'anxiété généralisée. Veuillez indiquer jusqu'à quel point chacun de ces comportements est typique de vous **au cours de la dernière semaine** en encerclant le chiffre approprié (1 à 5). Il n'y a pas de bonne ou mauvaise réponse.

	Pas du tout typique de moi	Peu typique de moi	Assez typique de moi	Très typique de moi	Extrêmement typique de moi
1. Je fais constamment autre chose pour éviter de commencer ce que je devrais faire. Ex : Regarder la télévision pour retarder le moment de commencer une tâche.	1	2	3	4	5
2. J'évite fréquemment des situations dont l'issue pourrait être négative Ex : Éviter d'aller manger dans un nouveau restaurant.	1	2	3	4	5
3. Je demande souvent aux autres (conjoint, amis, famille, collègue) de me rassurer. Ex : Demander à un ami de me rassurer à propos d'une décision que j'ai prise au travail.	1	2	3	4	5
4. À répétition, je refais ou change des choses à des tâches que j'ai déjà terminées. Ex : Reprendre un projet que j'ai déjà fini et y apporter des changements.	1	2	3	4	5
5. Je cherche beaucoup d'information afin de me sentir rassuré. Ex : Vérifier les rapports de circulation afin de savoir s'il y a eu un accident sur la route qu'emprunte mon conjoint.	1	2	3	4	5

6. J'évite de m'engager totalement dans un emploi, un projet ou une relation parce que je ne suis par certain de comment ça va se passer. Ex : Refuser de m'impliquer dans un projet ambitieux au travail.	1	2	3	4	5
7. Je prends plus de temps que nécessaire (ou que la plupart des gens) pour préparer les choses. Ex : Passer des heures à réviser de l'information pour une courte réunion.	1	2	3	4	5
8. Je vérifie constamment pour m'assurer que j'ai bien terminé une tâche ou un projet. Ex : Vérifier mon courriel à répétition pour m'assurer que j'ai bien envoyé un courriel important.	1	2	3	4	5
9. Je cherche constamment de la réassurance à cause de mes inquiétudes. Ex : Demander à mon conjoint de me rassurer à propos de mes inquiétudes face à notre relation.	1	2	3	4	5
10. J'observe constamment mon corps et mes sensations physiques à la recherche d'un «signe » de maladie. Ex : Vérifier mon pouls pour voir si j'ai un problème cardiaque.	1	2	3	4	5
11. Je vérifie constamment pour m'assurer que j'ai bien fait une tâche ou un projet correctement. Ex : Vérifier un courriel plusieurs fois pour m'assurer que j'y ai joint le bon document.	1	2	3	4	5
12. Je dis toujours aux autres quoi faire parce que je ne suis pas certain de comment les choses vont se passer. Ex : Dire à mon conjoint comment il/elle devrait réagir à un problème.	1	2	3	4	5

13. Je remets souvent une prise de décision ou un comportement à cause de mes inquiétudes. Ex : Retarder un appel téléphonique à cause de mes inquiétudes.	1	2	3	4	5
14. J'évite constamment des situations dans lesquelles je me sens incertain. Ex : Éviter des fêtes où des gens que je ne connais pas sont présents.	1	2	3	4	5
15. Je recherche souvent des gens en particulier, des objets ou des routines dans le but de me sentir plus « certain ». Ex : Me rendre seulement à une fête si un tel ami s'y rend avec moi.	1	2	3	4	5
16. J'essaie de prendre le contrôle des choses en faisant tout moi-même Ex : Faire un projet « de groupe » seul en refusant de déléguer des tâches aux membres du groupe.	1	2	3	4	5
17. Je prends beaucoup de temps et d'effort pour me préparer à des situations pour lesquelles un résultat négatif peut arriver Ex : Avant un rendez-vous chez le médecin, passer des heures à faire des recherches sur internet à propos de symptômes.	1	2	3	4	5
18. Je vérifie à répétition si j'ai bien verrouillé la porte ou si j'ai bien éteint la cuisinière. Ex : Vérifier la porte plusieurs fois pour être certain qu'elle est bien verrouillée avant de quitter la maison.	1	2	3	4	5

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