Depressive Symptoms in the Cognitive-Behavioural Treatment of Generalized Anxiety Disorder

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Abstract

Depressive Symptoms in the Cognitive-Behavioural Treatment of Generalized Anxiety Disorder

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Generalized anxiety disorder (GAD) is characterized by anxiety, excessive and uncontrollable worry, and somatic symptoms such as muscle tension and difficulty concentrating (DSM-IV-TR, American Psychiatric Association, 2000). GAD is linked to symptoms of depression both theoretically and empirically, but there is currently no consensus as to how co-occurring depressive symptoms affect GAD treatment outcome. Dugas and colleagues have developed an efficacious cognitive-behavioural treatment (CBT) based on a model of GAD that centres upon intolerance of uncertainty. This CBT program has demonstrated consistent reductions in GAD symptom severity by posttreatment (e.g., Dugas et al., 2010); however, not all individuals achieve full remission of GAD for reasons that are currently unclear. The first goal of this study was to determine the relationship between depressive symptoms and short- and long-term GAD treatment outcome. The second goal was to determine the relationship between depressive symptoms and GAD treatment engagement. The results indicated that depressive symptoms at pretreatment were largely unrelated to posttreatment severity of GAD, worry, and somatic anxiety or to treatment engagement. Posttreatment depressive symptoms were not related to the severity of overall GAD symptoms, worry, and somatic anxiety at 18-month follow-up. The theoretical and clinical implications of these findings are discussed.

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Depressive Symptoms in the Cognitive-Behavioural Treatment of Generalized Anxiety Disorder

Numerous protocols have been developed for the treatment of generalized anxiety disorder (GAD; e.g., Borkovec & Costello, 1993; Dugas & Ladouceur, 2000; Wells & King, 2006), with most demonstrating good efficacy. However, some individuals do not achieve full remission of their disorder following treatment, for reasons that have not been conclusively established. One potential explanation is the presence of depressive symptoms, which often co-occur with GAD. Symptoms of depression are not directly targeted during treatments for GAD and may interfere with effective treatment implementation. The current study aims to clarify the impact of depressive symptoms on short- and long-term treatment outcome as well as treatment engagement in a cognitive-behavioural treatment for GAD.

GAD is one of the most common anxiety disorders and one that can have a profound effect on the lives of its sufferers in professional, personal, and social spheres (Wittchen, 2002). According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association, 2000), the main diagnostic feature of GAD is excessive anxiety and worry that occurs more days than not for at least six months and that is centred around a number of events or activities. This anxiety and worry causes significant distress or impairment in essential areas of functioning and is difficult to control. Additionally, in order to meet diagnostic criteria for GAD, an individual must experience three or more of the following somatic symptoms: restlessness, difficulty concentrating, irritability, fatigue, muscle tension, and sleep disturbance.

Approximately 5.1% of the population will meet GAD diagnostic criteria in their lifetime (Wittchen, Zhao, Kessler, & Eaton, 1994), with one-month prevalence rates as high as 7.9% in primary-care settings (Maier et al., 2000). In addition to its high prevalence, GAD results in significant impairments in numerous domains of functioning. GAD is associated with impairments in close relationships and social life (Massion, Warshaw, & Keller, 1993; Stein & Heimberg, 2004), career functioning (Greenberg et al., 1999; Wittchen, Carter, Pfister, Montgomery, & Kessler, 2000; Wittchen et al., 1994), and physical health (Hoffman, Dukes, & Wittchen, 2008; Wittchen et al., 2002). GAD is also associated with significant costs to the health care system, including increased visits to general practitioners and medical specialists such as cardiologists and gynaecologists (Greenberg et al., 1999). Considering the disorder's high prevalence rates, significant associated impairments, and economic burden, effective treatment for GAD is essential.

Dugas and Ladouceur (2000) have developed a cognitive-behavioural treatment (CBT) protocol based on a model of GAD that is centred upon intolerance of uncertainty (Dugas, Gagnon, Ladouceur, & Freeston, 1998). Intolerance of uncertainty is a dispositional characteristic that develops from a set of negative beliefs regarding uncertainty and its implications (Dugas & Robichaud, 2007). This CBT protocol has demonstrated efficacy in decreasing symptoms of GAD in numerous well-controlled studies (e.g., Dugas et al., 2003; Ladouceur, 2000). However, not all individuals treated for GAD achieve full remission of their disorder, the reasons for which remain unclear. As previously mentioned, one potential barrier to effective treatment for individuals with GAD may be symptoms of depression.

Defining Depression

What is commonly referred to as "depression" is increasingly recognized as a dimensional construct (Lewinsohn, Soloman, Seeley, & Zeiss, 2000) with a range of clinical presentations according to the number, duration, and severity of symptoms. Depressive disorders include both those traditionally recognized as "threshold" or "clinical" conditions (major depressive disorder and dysthymic disorder) as well as "subthreshold" or "subclinical" disorders (minor depressive disorder, recurrent brief depressive disorder, and subsyndromal symptomatic depression). These conditions differ in terms of the number, type, severity, and duration of depressive symptoms but are all associated with clinically significant distress and impairment. Common symptoms include low mood, anhedonia, appetite disturbance, fatigue, sleep disturbance, psychomotor retardation or agitation, and suicidal ideation.

Depression can be assessed in a number of ways, including: (1) diagnosis based on set criteria or (2) elevated depression scores on self-report measures. According to the DSM-IV-TR, a diagnosis of major depressive disorder (MDD) requires low mood or anhedonia and at least five additional symptoms of depression for at least two weeks. Dysthymic disorder (dysthymia) a requires low mood for at least two years with at least two additional symptoms. Minor depression has the same criteria as MDD, but with only two to four depressive symptoms (one of which must be low mood or anhedonia). Recurrent brief depression has the same criteria as MDD, but with symptom duration of less than two weeks (American Psychiatric Association, 2000). Subsyndromal symptomatic depression requires at least two symptoms of depression for at least two weeks, but with no low mood or anhedonia. Emerging research demonstrates that clinically significant distress and impairment can be present even when low mood or

anhedonia is absent, as is the case in subsyndromal symptomatic depression (Sadek & Bona, 2000).

Depression can also be measured by self-report questionnaires such as the *Beck Depression Inventory, 2nd Edition* and the *Center for Epidemiologic Studies Depression Scale* (Gotlib, Lewinsohn, & Seeley, 1995; Sadek & Bona, 2000). Clinically significant impairment and distress have been found in individuals with elevated scores on such self-report measures of depression even when criteria are not met for a threshold depressive disorder such as major depressive disorder (MDD) or dysthymia (Gotlib et al., 1995). There is therefore substantial evidence for conceptualizing depression as a dimensional syndrome, with clinically significant impairment and distress not limited to the highest end of the spectrum.

Depression and GAD

GAD and depression are strongly linked both empirically and theoretically. Empirical evidence is mainly derived from investigations of comorbidity, structural analyses, and phenotypic and genotypic similarities. The majority of this research has been conducted using MDD and dysthymia criteria as the definition of "depression".

There is a high rate of comorbidity between GAD and MDD/dysthymia even as compared to other comorbid conditions (Kendler, Gardner, Gatz, & Pederson, 2007; Sanderson, Di Nardo, Rapee, & Barlow, 1990). For instance, in a community-based epidemiological study, Kessler and colleagues (2005) found that the bivariate correlation between 12-month GAD and MDD was 62% – higher than the correlations between GAD and any other measured DSM-IV disorder. Further, both confirmatory and exploratory factor analyses suggest that GAD has more robust associations with MDD

and dysthymia than with other anxiety disorders (Brown, Chorpita, & Barlow,1998; Krueger, 1999; Vollebergh et al., 2001). GAD and MDD also have phenotypic and genotypic similarities. Somatic anxiety symptoms and depressive symptoms overlap significantly. In fact, the only somatic anxiety symptoms characteristic of GAD that do not overlap with DSM-IV criteria for MDD are irritability and muscle tension. Muscle tension appears to be the only somatic symptom uniquely related to worry, suggesting that the other somatic anxiety symptoms show little specificity to GAD (Joorman & Stöber, 1999). This symptom overlap may at least partially account for the disorders' high comorbidity and structural relationships (Menin, Heimberg, Fresco, & Ritter, 2008). Finally, GAD and MDD share genetic vulnerabilities that suggest pleiotropy (Gorwood, 2004).

Due to these similarities, GAD's status as an independent disorder has been questioned. However, GAD and unipolar depressive disorders can be reliably distinguished in terms of risk factors, clinical presentation, cognitive content, and cognitive biases. Again, most of this differentiation has been examined in terms of GAD and MDD. Environmental risk factors differ between GAD and MDD (Moffitt et al., 2007; Kendler et al, 2007; Kendler, Hettema, Butera, Gardner, & Prescott, 2003; Kessler, 2008). GAD and MDD also differ in terms of illness course (Fergusson, Horwood, & Boden, 2006), as well as some aspects of symptomatology.

Additionally, specificity has been demonstrated in terms of both cognitive content and biases. For example, both anxiety and depression are associated with interpretive biases regarding the probability of the occurrence of negative events (e.g., Butler & Matthews, 1983). However, Clark and colleagues (1990) found that threat-related

cognitions relating to anticipated harm and danger were more prominent in anxious individuals, whereas negative cognitions related to failure, loss, low self-worth, and hopelessness were more prominent in depressed individuals. More specifically, individuals with GAD demonstrate attentional biases to threatening stimuli, a phenomenon not found in individuals with MDD (Mineka, Rafaeli, & Yovel, 2003). There is also evidence of differentiation between GAD and depression in terms of approach to certainty. Intolerance of uncertainty is associated with both GAD (Dugas, Buhr, & Ladouceur, 2004) and MDD (Gentes & Ruscio, 2011). Repetitive, negative cognitions may explain this relationship. Worry partially mediates the relationship between intolerance of uncertainty and anxiety, whereas rumination fully mediates the relationship between intolerance of uncertainty and depression (Yook, Kim, Suh, & Lee, 2010). When intolerant of uncertainty, worrying increases anxiety whereas ruminating increases depression. This may relate to hopelessness, a key distinguishing feature between anxiety and depression (Alloy, 1991; Alloy, Kelly, Mineka, & Clements, 1990). Individuals with MDD may react to uncertainty with hopelessness, converting an uncertain stimulus into a certain, negative one (Gentes & Ruscio, 2011). This coincides with research showing greater certainty in the occurrence of future negative events in individuals with MDD (Miranda & Mennin, 2007). Furthermore, certainty in an absence of future positive events is associated with depression but not GAD, with hopelessness mediating this relationship (Miranda, Fontes, & Marroquín, 2008). Thus, GAD and MDD share similarities in cognitive content and biases but can be successfully differentiated.

Theoretical models have been used to account for these similarities and differences between GAD and depressive disorders. Perhaps most widely known is the

tripartite model, which states that anxiety and depressive disorders share a common factor of negative affect. These broad disorder types are distinguished by positive affect and physiological hyper-arousal: according to the tripartite model, depressive disorders demonstrate comparatively low positive affect whereas anxiety disorders demonstrate comparatively high physiological arousal. This has been examined empirically in structural analyses of GAD and MDD with generally good support. GAD and MDD consistently share a strong association with a higher-order factor of negative affect when examining diagnostic data (Brown et al., 1998; Krueger, 1999; Vollebergh et al., 2001) as well as self-report symptom data (Brown et al., 1998). MDD demonstrates a relationship with low positive affect when examining both symptom clusters and diagnostic data (Brown et al., 1998, Watson et al., 1995). Finally, a recent conceptualization suggests that worry increases the baseline level of both negative affect and physiological arousal, while suppressing *further* increases in arousal when exposed to fear-evoking stimuli (Newman & Llera, 2011). This is consistent with the tripartite model as well as findings that anxiety symptoms in general are associated with higher levels of physiological arousal (Watson et al., 1995). It also clarifies earlier findings showing a relationship between GAD and suppression of autonomic reactivity (Borkovec, Alcaine, & Behar, 2004; Brown et al., 1998; Thayer, Friedman, & Borkovec, 1996).

Other theoretical models have been proposed to further elucidate the similarities and differences between unipolar depressive and anxiety disorders – and between GAD and MDD more specifically. An integrative hierarchical model (Mineka, Watson, & Clark, 1998) was put forth as an extension and amalgamation of the tripartite model and Barlow's hierarchical organization of anxiety disorders (Barlow, 1991; Zinbarg &

Barlow, 1996). This integrative hierarchical model posits that each disorder has both a common and a unique component – that is, a component it shares with other disorders and a component that is uniquely its own. For example, depression and GAD share the common component of negative affectivity. Depression's theorised specific components are disinterest and anhedonia (Mineka et al., 1998), as an absence of positive affect is also present in social anxiety disorder (Brown et al., 1998). This does not account for possible depression subtypes that present without anhedonia, such as subsyndromal symptomatic depression or depression presentations in certain ethnic groups. In terms of GAD's unique components, two elements that appear relatively specific to GAD are muscle tension and generalized worry. In sum, GAD and depressive disorders share substantial similarities but can be successfully differentiated both empirically and theoretically.

As previously stated, the co-occurrence of GAD and depression is common. It also comes with substantial impairment and distress. As compared to "pure" GAD, GAD with comorbid depression is marked by more severe GAD symptoms and increased impairments. Using data from the National Comorbidy Study and the Midlife Development in the United States Survey, Kessler and colleagues (1999) found that individuals with comorbid GAD and MDD show lower quality of life in terms of social functioning, physical health, and vitality. Unsurprisingly, individuals with comorbid GAD and MDD also had greater impairment in mental health than individuals with "pure" GAD or "pure" MDD. Risk of suicide is heightened in those with comorbid GAD and MDD as compared to pure GAD (Masi, Mucci, Favilla, & Millepiedi, 2001; Wittchen et al., 2002). Individuals with comorbid MDD and GAD also have earlier ages

of onset than those with "pure" GAD or "pure" MDD (Moffitt et al., 2007). Finally, although individuals with GAD already utilize a disproportionate level of health services (Greenberg et al., 1999), comorbidity appears to increase treatment seeking (Wittchen et al., 1994). In sum, comorbid depression increases the distress and impairment associated with GAD. For this reason, examining the impact of co-occurring depressive symptoms on GAD treatment outcome is essential.

Examining the Role of Depressive Symptoms in GAD Treatment

Despite the increased impairment associated with GAD and comorbid depression, there is a lack of consensus as to how symptoms of depression affect GAD treatment efficacy. In fact, there is a dearth of research examining the effects of total or specific comorbidities on GAD treatment; for example, in a large meta-analysis examining the effects of comorbidity on anxiety treatment outcomes only one of the included studies examined GAD treatment specifically (Olatunji, Cisler, & Tolin, 2010). In the extant literature, findings regarding the impact of depression on GAD treatment outcome are mixed.

In the available literature, several studies show that comorbid depression has a detrimental effect on short-term GAD treatment outcome. For example, van Balkom and colleagues (2008) found that comorbid depression (MDD and/or dysthymia) predicted lowered responsiveness to CBT for GAD. Similarly, Crits-Christoph and colleagues (2004) found that comorbid MDD was associated with poorer short-term treatment outcome in a supportive-expressive psychodynamic therapy for GAD (Crits-Christoph et al., 2004). Comorbid MDD has also been associated with decreased likelihood of spontaneous GAD remission and greater GAD chronicity when untreated (Bruce,

Machan, Dyck, & Keller, 2001; Bruce et al., 2005). The reverse has also been demonstrated: individuals with MDD and comorbid GAD show higher rates of attrition and increased latency of therapeutic medication effects (Brown, Schulberg, Madonia, Shear, & Houck, 1996). These findings are supplemented by numerous studies suggesting that comorbid depressive symptoms are associated with poorer short- and long-term treatment outcomes in other anxiety disorders (e.g., Emmanuel, Simmonds, & Tyrer, 1998; Lydiard & Brawman-Mintzer, 1998), including social phobia (Chambless, Tran, & Glass, 1997; Ledley et al., 2005; Marom, Gilboa-Schechtman, Aderka, Weizman, & Hermesh, 2009) and panic disorder (Lecrubier, 1998), suggesting these results may also be found in GAD treatment outcome.

However, several recent studies suggest that comorbid depressive disorders may not negatively impact treatment for GAD (Davis, Barlow, & Smith, 2010). In fact, in two recent investigations elevated depression scores at pre-treatment predicted improved short- and long-term CBT outcomes in adults (Newman, Przeworksi, Fisher, & Borkovec, 2010) and older adults (Wetherell et al., 2005) with GAD. Thus, given equivocal findings in the extant literature, our study aims to further elucidate the relationship between comorbid depression and GAD treatment outcome.

If elevated depression negatively impacts GAD treatment, it may do so in a number of ways. First, there is a robust relationship between treatment expectancy and CBT outcome for GAD (Barlow, Rapee, & Brown, 1992; Borkovec & Costello, 1993; Borkovec & Mathews, 1988; Newman & Fisher, 2010), which has been replicated in other forms of psychotherapeutic interventions for GAD (Crits-Christoph et al., 2004). Change in treatment expectancy has also been shown to partially mediate the relationship

between pretreatment and posttreatment severity of GAD (Newman & Fisher, 2010). Although this has not been studied directly, the presence of depressive symptoms may interfere with expectancies in the treatment of GAD, thus reducing symptom change by posttreatment. A significant symptom associated with depression is hopelessness, which mediates the relationship between depressive symptoms and reduced expectancy of positive future outcome (Miranda et al., 2008). Hopelessness and certainty in a lack of positive future events may extend to expectancies regarding treatment outcome. That is, individuals with depressive symptoms may have less positive treatment outcome expectancies. Given the robust relationship between treatment expectancy and outcome, this may be particularly problematic.

Second, although both depression and GAD are associated with negative problem orientation, depression has been linked with specific deficits in rational problem-solving style. Depressed individuals with high trait rumination generate poorer problem solutions than depressed individuals with low trait rumination and non-depressed individuals (Donaldson & Lam, 2004). Several studies have demonstrated that depression is associated with an avoidant problem-solving style, in which individuals seek to avoid thinking about or attempting to solve their problems (Becker-Weidman, Jacobs, Reinecke, Silva, & March, 2010; Reinecke, DuBois, & Schultz, 2001). This avoidant problem solving style predicts less improvement in depression severity over the course of treatment and greater suicidality (Becker-Weidman et al., 2010). Furthermore, this style of problem-solving could be particularly problematic in an active therapy context such as CBT: it may interfere with the individual's approach to treatment in general or the cognitive and behavioural exercises in particular. More specifically, in our CBT

treatment protocol for GAD an avoidant problem-solving style could be problematic in terms of between-session and in-session exercises such as monitoring of worry; accessing automatic thoughts, interpretations, and beliefs regarding uncertainty and willingness to challenge them; effective creation of anxiety-provoking imaginal exposure scenarios; and effective problem-solving training.

Third, clinical observation suggests that depressive symptoms may interfere with imaginal exposure, an important component of the present study's treatment for GAD (Dugas & Robichaud, 2007). Clients with comorbid depression may include depressogenic elements into their imaginal exposure scenarios, thus increasing feelings associated with depression while inhibiting the elicitation of anxiety.

Fourth, the presence of depressive symptoms may decrease treatment motivation. For example, common symptoms of unipolar depression include a loss of interest in activities, fatigue, and behavioural inactivity. Such depressive symptoms may be particularly problematic in the context of CBT, which is an active treatment modality. Typical CBT protocols involve collaboration between the clinician and client as well as active involvement of the client in- and between- sessions in terms of both cognitive and behavioural exercises. Furthermore, the cognitive and behavioural exercises used in CBT paradigms can create short-term discomfort and distress in clients as they challenge and reassess their beliefs, interpretations, and behaviours; these exercises also require significant effort both in and outside of therapy sessions (Beck, 1995). In our CBT program designed to target GAD, clients are asked to engage in a number of potentially distressing exercises requiring substantial effort such as monitoring worry and imaginal exposure. Completing such effortful and anxiety-provoking exercises requires

considerable motivation, which may be lower in individuals with depressive symptoms. For these reasons, the presence of depressive symptoms may interfere with the effective implementation of CBT protocols in general and our CBT program for GAD in particular.

In sum, we must consider (1) research demonstrating the potentially problematic nature of depression in the treatment of GAD and anxiety disorders in general, (2) clinical observations regarding the treatment-interfering nature of depression, and (3) the potential mechanisms of interference proposed. Given these factors, this study posits the more conservative hypothesis that depressive symptoms will be associated with poorer treatment outcome on a number of short- and long-term indices.

Addressing Limitations of Previous Research

This study aims to address a number of limitations prevalent in the extant literature. Considering the broader treatment outcome literature, examining the potential impact of depressive symptoms on short- and long-term treatment outcomes is a valuable and necessary exercise. Despite the possibility that depressive symptoms interfere with treatment outcome in GAD, studies often exclude participants with comorbid depression (e.g., Borkovec, Abel, & Newman, 1995; Borkovec & Costello, 1993; Butler & Anastasiades, 1988; Durham, Allan, & Hackett, 1997) even when specifically examining the effects of Axis I comorbidity on GAD treatment outcome. Given the high rate of comorbidity between GAD and depressive disorders, the exclusion of participants with comorbid depression creates unrepresentative samples and results from such studies will lack complete information and generalizability.

A limitation present in previous research examining the role of comorbid depression in the treatment of GAD relates to the categorical assessment of depression (e.g., Provencher, Ladouceur, & Dugas, 2006). These studies have assessed depression dichotomously, meaning that depression is either present or absent. Typically, this is assessed as the presence or absence of MDD and dysthymia. This approach to assessing depression has several important disadvantages. One is that there may be few participants who meet the clinical threshold for depression, creating severely unbalanced groups and low power for statistical tests. Another disadvantage is that the presence of subthreshold depressive symptoms may have an impact on treatment outcome, even though these symptoms do not meet the clinical threshold for a depressive disorder, such as MDD. For example, patients with an anxiety disorder who also have subthreshold depressive symptoms display delayed latency for the onset of therapeutic effects, lower overall recovery rates, and higher relapse rates (Brown et al., 1996; Lecrubier, 1998; Maier, Gansicke, & Weiffenbach, 1997). In this sense, information with potential clinical importance may be lost. To address these issues, the present study assessed depression continuously. In other words, depression was viewed as a syndrome ranging in severity.

Another limitation in the available literature relates to the lack of long-term outcome assessment following treatment termination. To our knowledge, only one study examining GAD treatment has had a follow-up point beyond one year (Newman et al., 2010). Thus, the current study will examine long-term GAD treatment outcome at 18-months posttreatment in order to provide a more realistic, conservative, and clinically useful estimate of the long-term maintenance of treatment gains.

Goals and Hypotheses

This study examined the role of depressive symptoms in treatment outcome for a CBT protocol for GAD. The two main goals of this study were to determine if: (1) higher levels of depressive symptoms at pre- and posttreatment predict poorer short- and long-term treatment outcomes; and (2) higher pretreatment levels of depressive symptoms predict treatment non-engagement. Specifically, the study had four main hypotheses: (1) greater severity of depressive symptoms at pretreatment would predict greater severity of GAD symptoms at posttreatment; (2) greater severity of depressive symptoms at posttreatment would predict greater severity of GAD symptoms at 18-month follow-up; (3) greater severity of depressive symptoms at pretreatment would predict greater likelihood of drop-out during treatment; and (4) greater severity of depressive symptoms at pretreatment would predict fewer treatment sessions completed.

Method

Participants

The final sample consisted of 91 Francophone adults (females = 72) between the ages of 18 and 64 with a primary diagnosis of GAD. The sample was on average middle-aged (M = 43.28 years, SD = 12.07) with the following ethno-racial composition: 90.1% White (n = 82), 4.4% Native American (n = 4), 1.1% African-American (n = 1), and 2.2% "other" (n = 2). The majority of participants were married (42.9%, n = 39) or in conjugal relationships (33.0%, n = 30). The sample was also highly educated, with 63.8% of participants having completed a Bachelor's degree or higher. The majority of participants currently maintained full-time (51.6%, n = 47) or part-time (17.6%, n = 16) employment, with this being the primary source of income for most participants (72.5%, n = 66). At intake, the sample had had GAD for an average of 9.92 years (SD = 10.33). In

addition, the sample had an average GAD severity rating of 5.84 (*SD* = 0.73) on the Clinician's Severity Rating scale of the *Anxiety Disorders Interview Schedule for DSM-IV*, an average worry score of 63.02 (*SD* = 7.31) on the *Penn State Worry Questionnaire*, and an average somatic anxiety score of 21.09 (*SD* = 3.58) on the Somatic subscale of the *Worry and Anxiety Questionnaire*. The sample also had an average of 0.98 (*SD* = 0.82) non-depressive comorbid conditions at pretreatment. Finally, the sample's average pretreatment depression score was 16.78 (*SD* = 9.57) on the *Beck Depression Inventory, 2nd Edition*, which corresponds to "mild" depression (see *Measures* for all instrument properties).

Procedure

Participants were recruited from the Anxiety Disorders Clinic of the Hôpital du Sacré-Cœur de Montréal or from bi-annual advertisements placed in a local newspaper. Prior to enrolment in the treatment program, two independent assessors interviewed each potential participant using different structured diagnostic interviews in order to assess the reliability of the initial diagnoses. A team psychiatrist conducted the initial assessment using the *Mini International Neuropsychiatric Interview Version 5.0* (Sheehan et al., 1994), rating the severity of each diagnosed condition on a scale of 0-8 using the Clinician's Severity Rating scale (Di Nardo, Brown, & Barlow, 1994). If the participant met criteria for primary diagnosis of GAD (i.e., GAD severity was 4 or greater and was at least 1 point higher than other diagnosed conditions), a team psychologist then conducted a second diagnostic assessment using the *Anxiety Disorders Interview Schedule for DSM-IV* (Di Nardo, Brown, & Barlow, 1994). The severity of each diagnosed condition was again rated using the Clinician's Severity Rating scale. Following these assessments, a

final severity rating for each disorder was determined by consensus during a team meeting with the Principal Investigator, M. J. Dugas. Participants who received a primary diagnosis of GAD and who met the following additional inclusion criteria were accepted into the study: (1) no change in medication dose or type in 4 to 12 weeks before study entry (4 weeks for benzodiazepines, 12 weeks for antidepressants and hypnotics); (2) willingness to keep medication status stable while participating in the study (i.e., no changes in type of medication and no increase in dosage); (3) no use of herbal products known to have central nervous system effects 2 weeks before study entry; (4) no evidence of suicidal intent, based on clinical judgment; (5) no evidence of current substance abuse, current or past schizophrenia, bipolar disorder, or organic mental disorder; (6) no current participation in other clinical trials; and (7) no evidence of anxiety symptoms due to a general medical condition, based on clinical judgment (e.g., hyperthyroidism, anaemia, hypoglycemia). Participants who met all inclusion criteria then completed another standardized interview, cognitive tasks and pretreatment questionnaires (see *Measures*) prior to beginning treatment. The standardized interview and cognitive tasks were not part of the current study and are not reported below. The order of the study questionnaires was quasi-counterbalanced across participants.

The study used a within-subjects design, with measures administered at 8 assessment points: pre- and posttreatment as well as 3-, 6-, 9-, 12-, 15-, and 18-month follow-ups. Treatment consisted of an empirically-supported CBT protocol for GAD, administered over 14 weekly sessions utilizing a treatment manual initially developed in earlier studies and revised for the current one (Dugas & Ladouceur, 2000; Dugas et al., 2003; Ladouceur et al., 2000). The treatment components were: psychoeducation and

worry awareness training, reevaluation of the usefulness of worry, uncertainty recognition and behavioural exposure, problem-solving training, imaginal exposure, and relapse prevention. These components were designed to target specific processes in a cognitive model of GAD (Dugas & Robichaud, 2007), as well as simultaneously build tolerance and acceptance of uncertainty at each stage of treatment. As such, intolerance of uncertainty – the main component around which this cognitive model of GAD is based – was directly targeted throughout treatment. Four licensed psychologists experienced with CBT conducted treatment for all participants.

Measures

Two structured diagnostic interviews were used prior to treatment to determine the presence and severity of GAD: the *Mini International Neuropsychiatric Interview* (MINI; Sheehan et al., 1998) and the *Anxiety Disorders Interview Schedule for DSM-IV* (ADIS-IV; Di Nardo, Brown, & Barlow, 1994). Three measures were used throughout treatment and follow-ups to measure GAD symptoms: overall GAD symptomatology was assessed using the Clinician's Severity Rating scale of the ADIS-IV; worry was assessed using the *Penn State Worry Questionnaire* (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990); and somatic anxiety was assessed using the somatic subscale of the *Worry and Anxiety Questionnaire* (WAQ; Dugas et al., 2001). Depressive symptoms such as self-dislike, loss of interest, and worthlessness were assessed using the *Beck Depression Inventory, 2nd Edition* (BDI-II; Beck, Steer, & Brown, 1996).

The *Mini International Neuropsychiatric Interview, Version 5.0* (MINI; Sheehan et al., 1998) is a semi-structured diagnostic interview that assesses 17 DSM-IV-TR Axis I disorders using 210 items. The MINI has demonstrated excellent inter-rater reliability for

GAD (κ = .98) and good test-retest reliability for GAD, measured over two days (κ = .78), using different interviewers for each assessment. The Clinician's Severity Rating used with the ADIS-IV (see below) was used in conjunction with the MINI in the current study to provide a severity rating for each diagnosed disorder on a 9-point Likert scale.

The *Anxiety Disorders Interview Schedule for DSM-IV* (ADIS-IV; Di Nardo, Brown, & Barlow, 1994) is a semi-structured interview that assesses anxiety disorders and screens for several other Axis I disorders, including mood disorders. The Clinician's Severity Rating provides severity ratings for each diagnosed disorder on a 9-point Likert scale, with scores ranging from 0 ("*absent or none*") to 8 ("*very severe*"). A severity rating of 4 (moderate) represents the threshold of clinical significance. This means that scores of 4 or above indicate the presence of a clinically significant disorder, whereas scores below 4 indicate sub-clinical levels of a disorder. The ADIS-IV has good inter-rater reliability for dimensional ratings of GAD (excessive worry, r = .73; uncontrollable worry, r = .78; clinical severity rating, r = .72) and for diagnostic reliability for GAD (κ =. 67; Brown, Di Nardo, Lehman, & Campbell, 2001). For the purposes of this study, the Clinician's Severity Rating on the ADIS-IV (heretofore referred to as "ADIS-IV") was used at pre- and posttreatment as well as across the six follow-ups to assess the severity of GAD. For the diagnostic assessment at posttreatment and across follow-ups, scores below 4.0 represented remission of GAD and scores of 4.0 or above represented non-remission of GAD.

The *Penn State Worry Questionnaire* (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) is a self-report questionnaire consisting of 16 items that measure the tendency to worry uncontrollably and excessively. Items are rated on a 5-point Likert

scale, with responses ranging from 1 ("*not at all typical of me*") to 5 ("*very typical of me*"). The French translation used in this study demonstrated good internal consistency (α = .81) in the current study and has shown excellent test-retest reliability over four weeks (*r* = .86; Gosselin, Dugas, Ladouceur, & Freeston, 2001). For the purposes of this study, the PSWQ was used at pre- and posttreatment as well as across follow-ups to assess the severity of worry continuously.

The *Worry and Anxiety Questionnaire* (WAQ; Dugas et al., 2001) is a self-report measure consisting of 11 items pertaining to GAD diagnostic criteria. The Somatic subscale of the WAQ (WAQ-Som) consists of six items assessing the somatic symptoms associated with GAD, which include restlessness, difficulty concentrating, irritability, muscle tension, sleep disturbance, and fatigue. The WAQ-Som was used in this study to complement the assessment of worry using the PSWQ. Each item is rated on a 5-point Likert scale, with responses ranging from 1 ("*not at all*") to 5 ("*very severe*"). The French version of the WAQ demonstrated adequate internal consistency (α = .74) in this study and has shown adequate test-retest reliability over 64 days (*r* = .75 for those who meet GAD diagnostic criteria; *r* = .83 for those who do not meet GAD diagnostic criteria; Dugas et al., 2001). For the purposes of this study, the WAQ-Som was used at pre- and posttreatment as well as across follow-ups to assess the severity of GAD somatic symptoms using a continuous approach.

The *Beck Depression Inventory, 2nd Edition* (BDI-II; Beck, Steer, & Brown, 1996) is a self-report questionnaire assessing depressive symptoms such as worthlessness, loss of interest, and self-dislike. It consists of 21 groups of statements, each containing four statements that reflect differing degrees of depressive symptoms.

For each group, respondents indicate which statement best describes them over the preceding two weeks. Scores on each item range from 0 to 3. This measure demonstrated good internal consistency ($\alpha = .87$) in the current study and shows evidence of content, factorial, and discriminant validity (Beck, Steer, & Brown, 1996). For the purposes of this study, the BDI-II was used at pre- and posttreatment to assess depressive symptoms continuously.

Statistical Analyses

When testing all hypotheses, two variables were controlled for: the initial severity of the relevant GAD outcome measure (i.e., at pretreatment in the case of *Hypotheses 1*, *3*, and *4* and at posttreatment in the case of *Hypothesis 2*) and the total number of non-depressive comorbid conditions (i.e., at pretreatment in the case of *Hypotheses 1*, *3*, and *4* and at posttreatment in the case of *Hypothesis 2*). The total number of non-depressive comorbid conditions was controlled for as overall comorbidity has been linked to poorer treatment outcomes and higher rates of relapse (e.g., Durham, Allan, & Hackett, 1997). *Hypotheses 1* and *2* were examined using hierarchical linear regression due to its appropriateness in terms of our data set and research questions. Although Hierarchical Linear Modelling (Raudenbush, Bryk, & Congdon, 2006) was an analytic option, given the number of available assessment points, it was not pursued in the present investigation largely due to the nature of our treatment program. Conceptually, given the nature of the inclusion and exclusion criteria as well as the process of treatment itself, we expected there to be less variability in how participants change over time as compared to the variability between participants at a particular time point (in this case, at posttreatment and 18-month follow-up). Our research group has found this to be the case when

examining data from previous randomized control trials, and thus regressions were used in the current investigation. *Hypothesis 3* was analyzed using hierarchical logistic regression, given its dichotomous outcome variable (i.e., drop-out status), whereas *Hypothesis 4* was analyzed using hierarchical linear regression.

Results

Prior to the main analyses, statistical assumptions were assessed. All relevant variables were found to be normally distributed and free of multicollinearity. Linearity and homoscedasticity requirements were met for the relationships between pretreatment BDI-II scores and the GAD outcome measures (i.e., ADIS-IV, PSWQ, and WAQ-Som) at posttreatment. Linearity assumptions were met for the relationships between posttreatment BDI-II scores and the GAD outcome measures at 18-month follow-up. However, the assumption of homoscedasticity was violated between posttreatment BDI-II scores and the GAD outcome measures at 18-month follow-up. Heteroscedasticity may be more common in clinical data, for several theoretical reasons (Grissom, 2000). No action was taken to transform these variables as this can result in further statistical and interpretive problems. Therefore, the resulting regression analyses must be interpreted with some caution.

Interrater Agreement on Diagnostic Status

We assessed interrater agreement on pretreatment GAD by comparing (1) primary diagnoses and (2) the severity of primary diagnoses on the MINI and ADIS-IV diagnostic interviews. Interrater agreement was met when the assessors agreed upon: (1) the primary diagnosis and (2) the severity of the primary diagnosis (i.e., a one-point difference or less between assessors on the Clinician's Severity Rating of the primary diagnosis). Interrater

agreement was assessed in two ways. First, interrater agreement was assessed for the total sample of individuals assessed for inclusion in the treatment study ($N = 147$). The percentage agreement for this sample was 74.15%. Second, interrater agreement was assessed for the total sample of individuals included in the current study ($N = 91$), all of whom met diagnostic criteria for primary GAD at intake. The percentage agreement for this sample was 81.32%.

Preliminary Analyses Regarding Treatment Outcome

Prior to testing our four main hypotheses, univariate *t*-tests were conducted to determine how GAD and depressive symptomatology changed as a result of GAD treatment and over the follow-up period. From pretreatment to posttreatment, scores on the BDI-II and all GAD measures (i.e., ADIS-IV, PSWQ, and WAQ-Som) decreased a statistically significant amount. The observed pattern of change from posttreatment to 18-month follow-up on these measures was somewhat similar. There was a statistically significant decrease on ADIS-IV scores from posttreatment to 18-month follow-up, $M = 0.383$, $t\ (46) = 1.861$, $p = .034$. There were also further decreases in PSWQ and WAQ-Som scores over this time period, but they did not reach statistical significance. Finally, BDI-II scores increased slightly from posttreatment to 18-month follow-up, although this was also not statistically significant, $M = -0.973$, $t\ (46) = -0.792$, $p = .217$.

Hypothesis 1

To test the hypothesis that greater severity of pretreatment depressive symptoms would predict greater severity of GAD symptoms at posttreatment, three hierarchical linear regressions were conducted using posttreatment ADIS-IV, PSWQ, and WAQ-Som as dependent variables while using pretreatment BDI-II scores as the main independent

variable. Only those participants who had completed all pre- and posttreatment measures (n = 78) were included in these analyses. All drop-outs (n = 13) were excluded. In each analysis, pretreatment severity of GAD symptoms on the relevant measure was controlled for in Step 1 and the number of non-depressive comorbid conditions at pretreatment was controlled for in Step 2.

Results showed that pretreatment depressive symptom severity (BDI-II scores) predicted posttreatment GAD severity (ADIS-IV scores) at a statistically significant level (see *Table 1*). However, pretreatment depression severity did not significantly predict posttreatment worry severity (PSWQ scores; see *Table 2*) or somatic anxiety (WAQ-Som scores; see *Table 3*). Pretreatment depression severity accounted for 7% of the variance in posttreatment GAD severity over and above the control variables, F (3, 74) = 5.026, p = .003. Pretreatment depression severity had a positive relationship with posttreatment GAD severity: as the severity of pretreatment depressive symptoms increased, posttreatment GAD severity increased. This finding remained virtually unchanged when participants with MDD or dysthymia, as defined by scores of 4 or greater on the Clinician's Severity Rating scale (n = 2), were excluded from the analysis.

Hypothesis 2

To test the hypothesis that greater severity of posttreatment depressive symptoms would predict greater severity of GAD symptoms at 18-month follow-up, three hierarchical linear regressions were conducted using the ADIS-IV, PSWQ, and WAQ-Som at 18-month follow-up as dependent variables while using posttreatment BDI-II scores as the main independent variable. Only those participants who had completed all follow-up assessment points (n = 47) were included in these analyses. All drop-outs (n =

13) as well as participants who had not fully completed the follow-up assessments (but who, nevertheless, had not dropped out of the study: $n = 31$) were not included. In each analysis, posttreatment severity of GAD symptoms on the relevant measure was controlled for in Step 1 and the number of non-depressive comorbid conditions at posttreatment was controlled for in Step 2.

Results showed that posttreatment depression severity (BDI-II scores) did not statistically significantly predict 18-month follow-up scores on any of the GAD outcome measures (see *Tables 4–6*). To determine if the pretreatment level of depression would better predict GAD symptom severity at 18-month follow-up, hierarchical regression analyses were conducted using pretreatment BDI-II scores as the main independent variable and the same control variables. Similar findings were produced: pretreatment depression severity did not account for a statistically significant proportion of variance in any of the GAD outcome measures at 18-month follow-up.

Hypothesis 3

To test the hypothesis that greater severity of pretreatment depressive symptoms would predict drop-out status (drop-out versus treatment completer), a logistic regression analysis was conducted using the full sample ($N = 91$). Pretreatment ADIS-IV scores were controlled for in Step 1 and the number of non-depressive comorbid conditions at pretreatment was controlled for in Step 2. Results showed that pretreatment BDI-II scores accounted for a non-statistically significant proportion of variance in attrition (4.2%; see *Table 7*). Moreover, the adjusted odds ratio showed that for every one-point increase on the pretreatment BDI-II, the likelihood of attrition increased by 1.050 times ($p = .154$). In

other words, pretreatment depressive symptoms had a negligible impact on the likelihood of drop-out that is not statistically or clinically significant.

Hypothesis 4

To test the hypothesis that greater severity of pretreatment depressive symptoms would predict treatment attendance, a hierarchical linear regression analysis was conducted using pretreatment BDI-II scores as the main independent variable and the total number of treatment sessions completed as the dependent variable. The full sample ($N = 91$) was included in the analysis. Pretreatment ADIS-IV scores were controlled for in Step 1 and the number of non-depressive comorbid conditions at pretreatment was controlled for in Step 2. The average number of treatment sessions completed was 12.55 ($SD = 3.643$). Results showed that pretreatment BDI-II scores accounted for a negligible amount of variance in treatment attendance that was not statistically significant (0.8%; see *Table 8*). The results remained virtually unchanged when only those participants who had completed at least one session of treatment ($n = 85$) were included in the analysis.

Discussion

The objectives of this study were twofold: to determine (1) if higher levels of depressive symptoms predicted poorer short- and long-term outcomes in an efficacious CBT program for GAD, and (2) if higher levels of pretreatment depressive symptoms predicted treatment non-engagement. Overall, our findings suggest that depressive symptoms do not substantially interfere with short or long-term treatment outcomes or with treatment engagement in this CBT program. Furthermore, our GAD treatment was successful at reducing both GAD and depressive symptomatology, despite not

specifically targeting depression. These gains were generally maintained over the follow-up period.

Predicting GAD at Posttreatment

Of our four hypotheses, only one (*Hypothesis 1*) was partially supported statistically: greater pretreatment severity of depressive symptoms predicted greater posttreatment GAD severity as measured by the ADIS-IV, at a statistically significant level. In this analysis, pretreatment depressive symptom severity accounted for 7% of the variance in posttreatment ADIS-IV scores after controlling for pretreatment ADIS-IV scores and the total number of pretreatment non-depressive comorbid conditions. Although this finding is statistically significant, its clinical significance is dubious: the effect size is approximately half that of the effect sizes found in studies showing a statistically significant effect of depression on GAD treatment outcome (e.g., 12%, Crits-Christoph et al., 2004) and social phobia treatment outcome (e.g., 15%, Marom et al., 2009). Further, pre-treatment severity of depressive symptoms did not predict the posttreatment severity of worry (PSWQ) or somatic anxiety (WAQ-Som) at a statistically significant level. Overall, these results align with others in the extant literature finding little to no relation between depression severity and poorer short-term GAD treatment outcome (e.g., Newman et al., 2010; Wetherell et al., 2005).

Although the severity of depression at pretreatment was mild in our sample (M_{pre} = 16.368, SD_{pre} = 9.770), it is unlikely that this negatively affected its predictive power. Our sample's pretreatment BDI scores are comparable to that of other studies investigating depressive symptoms and GAD (e.g., Butler, Fennell, Robson, & Gelder, 1991: M_{BDI} = 20.0, SD_{BDI} = 9.3; Hopko et al., 2000: M_{BDI} = 17.8, SD_{BDI} = 6.7; Wetherell

et al., 2010: M_{BDI} = 16.066, SD_{BDI} = 6.83). Moreover, even subthreshold levels of depression at pretreatment have been shown to impact treatment outcomes in a variety of anxiety disorders (Rivas-Vasquez, Saffa-Biller, Ruiz, Blais, & Rivas-Vasquez, 2004). In sum, this suggests that the relatively low level of pretreatment depression in our sample is not responsible for the lack of findings.

The finding that pretreatment depression severity predicts posttreatment GAD severity but not worry or somatic anxiety has several potential explanations, despite its dubious clinical significance. It suggests a differential relationship between depressive symptoms and GAD severity, worry, and somatic anxiety in the context of GAD treatment. This discrepant association between depressive symptoms and the measured GAD symptom dimensions could be due to: (1) instrument properties, (2) differential relationships among the constructs themselves, or (3) differential impact of depression on GAD treatment components. Each will be considered in turn.

First, measurement modality may have affected the results of *Hypothesis 1*. The ADIS-IV is a clinician-rated questionnaire, whereas the PSWQ and WAQ-Som are self-report questionnaires. Previous research suggests low (Beck, Stanley, & Zebb, 1995) to moderate (Hopko et al., 2000) associations between self- and clinician-rated measures of worry and anxiety. In our sample, the correlation between pretreatment ADIS-IV and PSWQ scores was moderate (r = .578, p = .010), as was the correlation between pretreatment ADIS-IV and WAQ-Som scores (r = .583, p = .010). Although simple correlations do not account for shared variances between these variables, they suggest that the measures may have sufficient differences to account for differential relationships with depression scores.

Due to shared method variance, a stronger predictive relationship would be anticipated between the BDI-II and both the PSWQ and WAQ-Som, as these are all self-report measures. Our findings stand in contrast to this, as statistically significant predictive relationships were not found between depression and worry or between depression and somatic anxiety. Examining the GAD outcome measures more closely, it is apparent that worry and somatic anxiety are captured in a very different way by their respective measures than is GAD severity by the ADIS-IV. The ADIS-IV assesses the presence of dysfunctional symptoms associated with GAD, impairment, distress, and the number of life domains affected. The PSWQ assesses the presence and degree of excessive worry, but does not directly assess associated impairment, distress, or the number of life domains affected. Similarly, the WAQ-SOM assesses self-rated severity of somatic anxiety symptoms, but does not directly measure impairment, distress or the number of life domains affected. The ADIS-IV is a closer approximation of Wakefield's (1992a) assertion that disorders are characterized by harmful dysfunction. Harmful dysfunction assumes that the disorder or condition (a) causes harm or deprivation of some benefit as judged by the values of the overarching culture and (b) causes an inability of a mental mechanism to perform its natural function. While distress, impairment, and the number of life domains affected are not sufficient measures of harmful dysfunction (Wakefield, 1992a; 1992b), the inclusion of these elements provides a closer approximation harmful dysfunction than their exclusion. Therefore, the ADIS-IV provides a more nuanced and valid assessment of the harmful dysfunction of GAD symptomatology than do the PSWQ and WAQ-Som. The BDI-II may have greater

predictive power of ADIS-IV scores relative to PSWQ and WAQ-Som scores due to this measurement difference.

To test the possibility that the ADIS-IV provides a significantly different picture of GAD symptomatology than the other two measures, the same hierarchical regression analysis was conducted using another measure of overall GAD severity: the full scale of the WAQ. The WAQ, like the ADIS-IV, assesses DSM-IV criteria for overall GAD symptomatology and associated impairment. However, unlike the ADIS-IV, the WAQ does not directly measure associated distress or the number of life domains affected. Thus, I hypothesized it would function as a poorer approximation of harmful dysfunction (Wakefield, 1992a; 1992b) than the ADIS-IV. A hierarchical regression was conducted using pretreatment BDI-II scores and control variables to predict posttreatment scores on the full scale of the WAQ. The results of this analysis supported my assertion: pretreatment BDI-II scores did not predict posttreatment WAQ scores while controlling for pretreatment WAQ scores and the total number of non-depressive comorbid conditions at pretreatment. In sum, it is a strong possibility that the discrepant statistical findings regarding *Hypothesis 1* are due to measurement modality.

Second, the equivocal statistical relationship between pretreatment depression severity and posttreatment GAD measures could be due to the constructs themselves. That is, overall depression severity may relate more to overall GAD severity than to worry or somatic anxiety. Distinct elements of depression may relate differently to worry and somatic anxiety in the context of GAD treatment. For example, depressive rumination is a form of negative, repetitive thought implicated in intolerance of uncertainty (Yook, Kim, Suh, & Lee, 2010) and deficits in problem-solving (Donaldson,

& Lam, 2004). Rumination may better predict changes in worry across treatment as it is a highly related but separate construct (Goring & Papageorgiou, 2008). Similarly, somatic-vegetative symptoms of depression overlap significantly with somatic anxiety symptoms (Menin et al., 2008). Although it is beyond the purview of the current study, the predictive power of rumination, somatic-vegetative depressive symptoms, hopelessness, and cognitive-affective depressive symptoms could be examined specifically in future research.

A third potential explanation for the *Hypothesis 1* findings is that depressive symptom severity differentially impacts the components of GAD treatment. If this is true, it could attenuate the effect of depression severity on overall GAD treatment outcome. Given depression's observed relationship with difficulties in problem-solving (Becker-Weidman et al., 2010; Reinecke et al., 2001) and imaginal exposure (Dugas & Robichaud, 2007), these are the most likely treatment components that could be affected. A second possibility is that particular symptom dimensions of depression could differentially impact GAD treatment outcome. For example, the BDI-II appears to have a two-factor structure, organizing depressive symptoms into cognitive-affective and somatic-vegetative dimensions (Dozois, Dobson, & Ahnberg, 1998). It is possible that either factor could impact overall GAD treatment outcome or specific components. Neither of these possibilities could be investigated in our study due to lack of measurement of specific treatment components and issues of sample size, respectively. Future studies should examine the effects of depression on specific treatment components as well as the impact of specific depressive symptom dimensions on GAD treatment outcome.

Overall, our current findings suggest that severity of depressive symptoms has minimal clinical impact on short-term GAD treatment outcome. However, the depression scores in our sample are lower than those of studies investigating GAD with depressive symptoms exclusively meeting DSM-IV criteria for MDD, such as Lawrence, Liverant, Rosellini, & Brown (2009): $M = 26.89$, $SD = 9.58$. This suggests our findings regarding short-term treatment outcome may only be representative of those with GAD and mild or subthreshold depressive symptoms. Further research is necessary to determine how moderate and severe comorbid depression may impact short- and long-term GAD treatment outcome.

Predicting GAD at 18-Month Follow-Up

Our second hypothesis (*Hypothesis 2*) was not supported: the posttreatment severity of depressive symptoms did not predict the severity of overall GAD symptomatology, worry, or somatic anxiety at 18-month follow-up. This result coincides with the findings of several studies that found no effect of depressive symptoms on long-term GAD treatment outcome (Newman et al., 2010; Wetherell et al., 2005). Although we had 6 available assessment points, ranging from 3 months posttreatment to 18-months, this follow-up point was chosen because it is the most conservative in examining maintenance of treatment gains and therefore may be most clinically useful. A similar pattern of findings emerged when the same analyses used to test *Hypothesis 2* were re-run using GAD outcome at all other available assessment points (3, 6, 9, 12, and 15 months posttreatment). Posttreatment depressive symptoms did not significantly predict ADIS-IV or PSWQ scores at any of the follow-up points, but accounted for a small amount of variance in WAQ-Som scores at 3-, 6-, and 9-month follow-ups. The finding that

depression is largely unrelated to GAD treatment outcome is supported by a number of studies in the available literature (Newman et al., 2010; Wetherell et al., 2005), although in contrast to our findings, these studies found that the presence of comorbid depression predicted improved outcomes. Only one study in the extant literature has examined a follow-up point beyond one year posttreatment (Newman et al., 2010); thus, the current study adds important information to existing research.

There are several potential explanations for the lack of support for *Hypothesis 2*. First, the sample's depressive symptom severity was "minimal" by posttreatment (M_{post} = 8.234, SD_{post} = 8.374). This truncated range of scores may have decreased the predictive power of the BDI-II (Pagano, 2004). Although not explicitly targeted during treatment, depressive symptoms decreased a statistically significant amount from pre- to posttreatment. This suggests that at least some of the factors maintaining these depressive symptoms were alleviated, possibly due to symptom overlap, overlap with the maintaining factors of GAD, or the transfer of cognitive and behavioural techniques. If depressive symptoms and their maintaining factors were successfully reduced to the point of minimal impact on participants' functioning, their power in predicting GAD severity 18 months later would likely have been subsequently lessened.

Second, the lack of support for *Hypothesis 2* could also be partially explained by additional statistical factors, such as issues with power and heteroscedasticity. Statistical power for this set of analyses may have been affected by the low sample size available at posttreatment (n = 47) combined with the number of predictors used. However, our data was within the suggested guidelines for adequate power. This set of analyses may also

have been affected by the heteroscedastic relationship between posttreatment depressive symptom severity and the GAD outcome measures at 18-month follow-up.

Third, it is possible that there is simply less to predict over the course of follow-up as compared to the treatment period. One would expect, and indeed our data show, that there is greater change in GAD symptoms during the treatment period than across follow-ups. Impairment is highest at pretreatment and treatment actively targets these symptoms and their maintaining factors, leading to greater change during treatment than in the months subsequent to treatment. Furthermore, the follow-up period is associated with increased error due to the longer time period examined. This increased error combined with less change in GAD scores could be responsible for a significant decrease in the predictive power of depressive symptoms during the follow-up period.

Predicting GAD Treatment Engagement

Our final major finding was that pretreatment severity of depressive symptoms did not predict treatment engagement, either in terms of attrition (*Hypothesis 3*) or treatment attendance (*Hypothesis 4*). Higher levels of pretreatment depressive symptoms did not significantly increase the likelihood of drop-out and did not significantly decrease the total number of treatment sessions completed. In other words, a participant's odds of dropping out of the treatment program and the total number of treatment sessions they completed were not affected by how depressed they were at pretreatment. This stands in contrast to research suggesting that individuals with comorbid MDD and GAD have higher attrition rates (e.g., Brown et al., 1996), although there is a paucity of literature specifically regarding the relationship between GAD, comorbidity, and the number of treatment sessions completed.

The possibility that depression severity has no relation to GAD treatment engagement is supported by the fact that comparable results were achieved when examining treatment engagement in two ways (i.e., number of sessions completed and drop-out status). This is complemented by our findings regarding short- and long-term GAD treatment outcome using measures of GAD symptom severity (*Hypotheses 1* and *2*). Additionally, treatment motivation as measured by the Nijmegen Motivation List 2 (Keijsers, Schaap, Hoogduin, Hoogsteyns, & de Kemp, 1999) was equivalent in individuals with the highest and lowest depression levels in our sample, as determined by a median split. Depression severity at pretreatment also did not predict treatment motivation in a hierarchical regression analysis when controlling for pretreatment GAD severity and the total number of comorbid conditions. In other words, depression severity had no relation to motivation to complete CBT for GAD. Perhaps this equivalency in treatment motivation partially accounts for the lack of relation between depression severity and treatment engagement.

It is again of note that depression levels were considered "mild" in our sample at pretreatment. It is possible that these findings only represent the relationship between mild depressive symptoms and treatment engagement, given the relatively low severity of depressive symptoms in our sample at intake. Similar analyses would need to be conducted in samples with moderate to severe depressive symptoms in order to fully elucidate this relationship.

Conclusions

Implications of findings. The severity of pretreatment depressive symptoms had little relation to short-term GAD treatment outcome: it was not predictive of the

posttreatment severity of worry and somatic anxiety, but accounted for a statistically significant amount of variance in posttreatment GAD severity. At this juncture the clinical significance of this finding appears to be negligible when examining GAD treatment in the aggregate. It remains unclear if this finding would be more pronounced if treatment components were examined separately or if the impact of different depressive symptom dimensions were examined. As it currently stands, our short-term treatment outcome finding has several clinical implications.

First, it adds to the emerging literature demonstrating that depressive symptoms do not negatively impact short-term outcomes in CBT for GAD (Newman et al., 2010; Wetherell et al., 2005). However, our findings may only be generalizable to minimal and mild depressive symptoms. Second, depression severity appears to decrease over the course of GAD treatment even when not directly targeted. GAD and depression may have similar maintaining factors that decrease during manualized treatment for GAD. Third, GAD treatment outcome should be assessed using continuous and categorical methods. This provides a more complete picture of symptom change and treatment outcome. Finally, our findings do not suggest that our CBT protocol targeting GAD must be fundamentally changed or adjusted according to the level of depressive symptoms. It is possible that specific components of treatment are particularly susceptible to the influence of depressive symptoms, such as problem-solving training and imaginal exposure. However, further studies are necessary to determine the impact of depression on specific components of CBT protocols for GAD.

The finding that the severity of posttreatment depressive symptoms was not predictive of GAD symptoms at 18-month follow-up also has several implications. First,

it provides evidence of no effect of mild depressive symptoms on GAD outcome at a conservative, long-term assessment point. This adds further credence to the growing body of literature suggesting that comorbid depression does not predict poorer long-term GAD treatment outcome (Newman et al., 2010; Wetherell et al., 2005). Second, it suggests that individuals with residual depressive symptoms at posttreatment do not need to be offered a greater number of booster sessions across follow-ups than individuals without residual depressive symptoms in order to maintain their treatment gains. Third, it implies that it is not imperative to assess depressive symptoms at posttreatment, although this may only apply to samples with mild to minimal levels of depression.

Finally, the finding that the severity of pretreatment depressive symptoms does not predict treatment engagement in terms of either treatment attendance (i.e., total number of treatment sessions completed) or attrition also has several implications. First, it implies that individuals with concurrent, mild depressive symptoms do not need additional intervention or monitoring to improve treatment engagement or attendance. Second, it suggests that greater pretreatment severity of depressive symptoms does not interfere with motivation to complete GAD treatment, which was found to be true in a follow-up analysis.

Limitations and directions for future research. Although this study was designed with the limitations of previous research in mind, the current study has a number of limitations of its own. First, our sample was likely not representative of those with "pure" GAD, "pure" depressive disorders, or comorbid GAD/depression in the general population, due to both the nature of recruitment procedures and certain inclusion and exclusion criteria. For example, the exclusion of current suicidal ideation may be

problematic due to its frequency in GAD, clinically depressed, and comorbid GAD/depression populations (Sareen et al., 2005). Second, few participants in our sample met the clinical threshold for MDD or dysthymia (i.e., only 2 participants met diagnostic criteria). This may have had a detrimental impact on our observed effect sizes, despite the fact that subthreshold depressive symptoms have been shown to impact treatment outcome (Rivas-Vasquez et al., 2004). Further, this low level of depression may negatively impact the generalizability of our findings. Future research must examine the impact of moderate and severe depressive symptoms on GAD treatment outcome. Third, due to ongoing data collection substantially fewer participants had completed the 18-month follow-up as compared to those who had completed the treatment program itself. Although our samples were within acceptable power limits for the analyses conducted, this may have affected the outcome of analyses examining GAD outcome at 18-month follow-up. Fourth, we did not have a sufficient number of treatment completers to examine GAD outcome dichotomously either at posttreatment or at 18-month follow-up. Although assessing GAD outcome in a continuous manner has significant benefits, assessing GAD remission status would also be informative. One advantage of a categorical approach is that it sorts participants into subgroups according to the severity of their symptoms, thus allowing us to bluntly examine treatment outcome in terms of remission and relapse. Another advantage of categorically assessing outcome is its consistency with the most widely used current classification systems – notably, the DSM-IV and ICD-10 – and the extant literature. Thus, future investigations should assess GAD outcome both continuously and categorically to maximize the informative value of findings. Fifth, the current study included self-report and clinician-rated GAD outcome

measure, both of which may be subject to expectancy biases. Future studies should include behavioural or implicit measures if possible, as these are relatively free of such biases. Finally, dimensions of depressive symptoms (e.g., cognitive-affective or somatic-vegetative) may differentially impact treatment outcome, a possibility that was not investigated in the present study. Similarly, depression may differentially affect components of CBT protocols, such as imaginal exposure and problem-solving training. Examining this differential impact would more effectively guide clinicians in monitoring and targeting problematic symptoms of depression during GAD treatment.

Table 1

Pretreatment BDI-II Scores Predicting Posttreatment ADIS-IV Scores

	ΔR^2	*B*	SE_B	*t*-ratio	*df*	*p*
Step 1						
ADIS-IV pre-tx	.099	0.542	0.188	2.885	76	.005
Step 2						
ADIS-IV pre-tx		0.537	0.198	2.708		
No. comorbid cons	.000	0.015	0.174	0.087	75	.931
Step 3						
ADIS-IV pre-tx		0.350	0.205	1.701		
No. comorbid cons		0.041	0.169	0.243		
BDI-II pre-tx	.070	0.036	0.015	2.506	74	.014

Note. "ADIS-IV pre-tx" is the pretreatment severity of overall GAD symptomatology as measured by the *Anxiety Disorders Interview Schedule for DSM-IV*, "No. comorbid cons" is the total number of non-depressive comorbid conditions at pretreatment, and "BDI-II pre-tx" is the pretreatment severity of depressive symptoms as measured by the *Beck Depression Inventory, 2nd Edition*.

Table 2

Pretreatment BDI-II Scores Predicting Posttreatment PSWQ Scores

	ΔR^2	*B*	SE_B	*t*-ratio	*df*	*p*
Step 1						
PSWQ pre-tx	.165	0.553	0.143	3.871	76	.000
Step 2						
PSWQ pre-tx		0.544	0.144	3.766		
No. comorbid cons	.003	0.715	1.341	0.533	75	.595
Step 3						
PSWQ pre-tx		0.477	0.150	3.175		
No. comorbid cons		0.676	1.331	0.508		
BDI-II pre-tx	.024	0.172	0.117	1.475	74	.145

Note. "PSWQ pre-tx" is the pretreatment severity of worry as measured by the *Penn State Worry Questionnaire*, "No. comorbid cons" is the total number of non-depressive comorbid conditions at pretreatment, and "BDI-II pre-tx" is the pretreatment severity of depressive symptoms as measured by the *Beck Depression Inventory, 2nd Edition*.

Table 3

Pretreatment BDI-II Scores Predicting Posttreatment WAQ-Som Scores

	ΔR^2	*B*	SE_B	*t*-ratio	*df*	*p*
Step 1						
WAQ-Som pre-tx	.307	0.738	0.127	5.803	76	.000
Step 2						
WAQ-Som pre-tx		0.716	0.125	5.710		
No. comorbid cons	.034	1.061	0.536	1.979	75	.051
Step 3						
WAQ-Som pre-tx		0.696	0.136	5.103		
No. comorbid cons		1.057	0.539	1.959		
BDI-II pre-tx	.001	0.018	0.049	0.375	74	.709

Note. "WAQ-Som pre-tx" is the pretreatment severity of somatic anxiety as measured by the Somatic subscale of the *Worry and Anxiety Questionnaire*, "No. comorbid cons" is the total number of non-depressive comorbid conditions at pretreatment, and "BDI-II pre-tx" is the pretreatment severity of depressive symptoms as measured by the *Beck Depression Inventory, 2nd Edition*.

Table 4

Posttreatment BDI-II Scores Predicting ADIS-IV Scores at 18-Month Follow-Up

	ΔR^2	*B*	SE_B	*t*-ratio	*df*	*p*
Step 1						
ADIS-IV post-tx	.332	0.840	0.178	4.726	45	.000
Step 2						
ADIS-IV post-tx		0.775	0.180	4.300		
No. comorbid cons	.033	0.386	0.256	1.505	44	.139
Step 3						
ADIS-IV post-tx		0.702	0.241	2.911		
No. comorbid cons		0.379	0.259	1.462		
BDI-II post-tx	.003	0.014	0.031	0.465	43	.644

Note. "ADIS-IV post-tx" is the posttreatment severity of overall GAD symptomatology as measured by the *Anxiety Disorders Interview Schedule for DSM-IV*, "No. comorbid cons" is the total number of non-depressive comorbid conditions at posttreatment, and "BDI-II post-tx" is the posttreatment severity of depressive symptoms as measured by the *Beck Depression Inventory, 2nd Edition*.

Table 5

Posttreatment BDI-II Scores Predicting PSWQ Scores at 18-Month Follow-Up

	ΔR^2	*B*	SE_B	*t*-ratio	*df*	*p*
Step 1						
PSWQ post-tx	.513	0.899	0.131	6.884	45	.000
Step 2						
PSWQ post-tx		0.841	0.127	6.603		
No. comorbid cons	.052	3.507	1.530	2.292	44	.027
Step 3						
PSWQ post-tx		0.793	0.146	5.424		
No. comorbid cons		3.379	1.550	2.179		
BDI-II post-tx	.005	0.108	0.158	0.686	43	.497

Note. "PSWQ post-tx" is the posttreatment severity of worry as measured by the *Penn State Worry Questionnaire*, "No. comorbid cons" is the total number of non-depressive comorbid conditions at posttreatment, and "BDI-II post-tx" is the posttreatment severity of depressive symptoms as measured by the *Beck Depression Inventory, 2nd Edition*.

Table 6

Posttreatment BDI-II Scores Predicting WAQ-Som Scores at 18-Month Follow-Up

	ΔR^2	*B*	SE_B	*t*-ratio	*df*	*p*
Step 1						
WAQ-Som post-tx	.386	0.741	0.139	5.317	45	.000
Step 2						
WAQ-Som post-tx		0.617	0.151	4.092		
No. comorbid cons	.046	1.566	0.828	1.890	44	.065
Step 3						
WAQ-Som post-tx		0.554	0.165	3.362		
No. comorbid cons		1.557	0.829	1.878		
BDI-II post-tx	.012	0.071	0.075	0.956	43	.345

Note. "WAQ-Som post-tx" is the posttreatment severity of somatic anxiety as measured by the Somatic subscale of the *Worry and Anxiety Questionnaire*, "No. comorbid cons" is the total number of non-depressive comorbid conditions at posttreatment, and "BDI-II post-tx" is the posttreatment severity of depressive symptoms as measured by the *Beck Depression Inventory, 2nd Edition*.

Table 7

Pretreatment BDI-II Scores Predicting Drop-Out Status at Posttreatment

	Negelkerke ΔR^2	*B*	SE_B	Wald	Exp(*B*)	*p*
Step 1						
ADIS-IV pre-tx	.000	-0.016	0.426	0.001	0.984	.970
Step 2						
ADIS-IV pre-tx		-0.032	0.446	0.005	0.969	.943
No. comorbid cons	.000	0.047	0.397	0.014	1.048	.906
Step 3						
ADIS-IV pre-tx		-0.283	0.492	0.331	0.753	.565
No. comorbid cons		0.074	0.394	0.035	1.076	.852
BDI-II pre-tx	.042	0.049	0.034	2.035	1.050	.154

Note. "ADIS-IV pre-tx" is the pretreatment severity of overall GAD symptomatology as measured by the *Anxiety Disorders Interview Schedule for DSM-IV*, "No. comorbid cons" is the total number of non-depressive comorbid conditions at pretreatment, and "BDI-II pre-tx" is the pretreatment severity of depressive symptoms as measured by the *Beck Depression Inventory, 2nd Edition*.

Table 8

Pretreatment BDI-II Scores Predicting Number of Treatment Sessions Completed

	ΔR^2	B	SE_B	t-ratio	df	p
Step 1						
ADIS-IV pre-tx	.003	0.260	0.528	0.492	89	.624
Step 2						
ADIS-IV pre-tx		0.304	0.557	0.546		
No. comorbid cons	.001	-0.130	0.498	-0.262	88	.794
Step 3						
ADIS-IV pre-tx		0.463	0.588	0.787		
No. comorbid cons		-0.136	0.499	-0.272		
BDI-II pre-tx	.008	-0.037	0.043	-0.854	87	.395

Note. "ADIS-IV pre-tx" is the pretreatment severity of overall GAD symptomatology as measured by the *Anxiety Disorders Interview Schedule for DSM-IV*, "No. comorbid cons" is the total number of non-depressive comorbid conditions at pretreatment, and "BDI-II pre-tx" is the pretreatment severity of depressive symptoms as measured by the *Beck Depression Inventory, 2nd Edition*.

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Appendix A
RECRUITMENT ADVERTISEMENT

Êtes-vous une personne inquiète?

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

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

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

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

UNIVERSITÉ Concordia UNIVERSITY

www.concordia.ca

Appendix B
CONSENT FORMS FOR STUDY PARTICIPATION

HÔPITAL DU SACRÉ-COEUR
DE MONTRÉAL

HSCM

UNIVERSITÉ Concordia UNIVERSITY

Formulaire d'information et de consentement téléphonique

(1e partie : Évaluation de l'admissibilité)

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

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

INFORMATION

A. BUT DE L'ÉTUDE

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

B. PROCÉDURES

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

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

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

C. RISQUES ET BÉNÉFICES

1. Risques, effets secondaires et désagréments

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

2. Bénéfices et avantages

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

D. CONDITIONS DE PARTICIPATION

1. Versement d'une indemnité

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

2. Confidentialité

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

3. Indemnisation en cas de préjudice

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

4. Participation volontaire et retrait de l'étude

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

CONSENTEMENT

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

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

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

OUI_____ NON_____

NOM DU PARTICIPANT : _____ DATE : _____

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

SIGNATURE _____ DATE _____

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

HÔPITAL DU SACRÉ-COEUR
DE MONTRÉAL

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FORMULAIRE D'INFORMATION ET DE CONSENTEMENT

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

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

Chercheur régulier, Centre de recherche, HSCM

Psychologue, Clinique des troubles anxieux, HSCM

Professeur titulaire, Département de psychologie, Université Concordia

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

Courriel : Michel.Dugas@concordia.ca

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

Professeur adjoint, Département de psychologie, Université Concordia

Tél : 514-848-2424 (poste 2202)

Natalie Phillips, Ph.D. (psychologie)

Professeur agrégé, Département de psychologie, Université Concordia

Tél : 514-848-2424 (poste 2218)

William Bukowski, Ph.D. (psychologie)

Professeur titulaire, Département de psychologie, Université Concordia

Tél : 514-848-2424 (poste 2184)

Julie Turcotte, M.D. (psychiatrie)

Professeur adjoint, Département de psychiatrie,

Faculté de Médecine, Université de Montréal

Psychiatre, Clinique des troubles anxieux, HSCM

Tél : 514-338-4201

Pierre Savard, M.D., Ph.D. (microbiologie et immunologie)

Professeur adjoint, Département de psychiatrie,

Faculté de Médecine, Université de Montréal

Psychiatre, Clinique des troubles anxieux, HSCM

Tél : 514-338-4201

Adrienne Gaudet, M.D. (psychiatrie)

Professeur adjoint, Département de psychiatrie,

Faculté de Médecine, Université de Montréal

Psychiatre, Clinique des troubles anxieux, HSCM

Tél : 514-338-4201

Organisme

de subvention : Instituts de recherche en santé du Canada

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

Ottawa, Ontario, K1A 0W9

INFORMATION

1. Nature et objectif de l'étude

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

plus importants dans leur façon de traiter l'information répondent différemment aux interventions psychologiques.

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

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

2. Déroulement de l'étude et méthodes utilisées

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

Premier volet : Évaluation pré-thérapie

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

comprendre certaines situations. Chacune des trois tâches prend environ 20 minutes à compléter. Vous répondrez ensuite à des questionnaires qui ont pour but d'évaluer votre état général. Cela vous prendra environ 20 minutes pour répondre aux questionnaires. La durée totale de cette rencontre (directives, tâches informatiques, pause et questionnaires) sera d'environ une heure et demie.

Deuxième volet : Thérapie cognitivo-comportementale

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

Troisième volet : Évaluation post-thérapie

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

3. Risques, effets secondaires et désagréments

Évaluations

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

Psychothérapie

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

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

4. Bénéfices et avantages

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

5. Versement d'une indemnité

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

6. Confidentialité

Tous les renseignements recueillis à votre sujet au cours de l'étude demeureront strictement confidentiels, dans les limites prévues par la loi, et vous ne serez identifié(e) que par un code. Les rencontres avec les psychologues seront enregistrées sur cassettes audio afin de nous permettre d'évaluer la qualité des interventions offertes par celles-ci (les cassettes seront aussi identifiées par un code). Immédiatement après l'étude, toutes les cassettes seront détruites. Aucune publication ou communication scientifique résultant de cette étude ne renfermera quoi que ce soit qui puisse permettre de vous identifier.

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

7. Indemnisation en cas de préjudice

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

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

8. Participation volontaire et retrait de l'étude

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

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

9. Personnes à contacter

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

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

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

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

HÔPITAL DU SACRÉ-COEUR
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CONSENTEMENT

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

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

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

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

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

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

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

Appendix C
ETHICS APPROVAL FORMS

Concordia
UNIVERSITY

CERTIFICATION OF ETHICAL ACCEPTABILITY FOR RESEARCH INVOLVING HUMAN SUBJECTS

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

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

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

31/05/2007

HSCM

APPROBATION D'UN PROJET DE RECHERCHE

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

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

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

PROVENANCE DES FONDS: Instituts de recherche en santé du Canada

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

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

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

LES CONSÉQUENCES ÉTHIQUES:

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

FORMULAIRE DE CONSENTEMENT:

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

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

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

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

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

M.F. Thibaudeau

Marie-France Thibaudeau

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

Cette approbation est valable pour une période d'un an seulement. Une demande de renouvellement doit être faite après cette période.

Appendix D

THE WORRY AND ANXIETY QUESTIONNAIRE (WAQ)

QIA

No. Dossier _____ Date _____

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

a) _____ d) _____

b) _____ e) _____

c) _____ f) _____

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

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

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

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

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

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

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

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

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

Aucunement Modérément Très sévèrement

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

b) Facilement fatigué-e

Aucunement Modérément Très sévèrement

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

c) Difficulté à se concentrer ou blanc de mémoire

Aucunement Modérément Très sévèrement

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

d) Irritabilité

Aucunement Modérément Très sévèrement

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

e) Tensions musculaires

Aucunement Modérément Très sévèrement

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

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

Aucunement Modérément Très sévèrement

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

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

Aucunement Modérément Très sévèrement

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

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

Appendix E

THE PENN STATE WORRY QUESTIONNAIRE (PSWQ)

QIPS

No. Dossier _____ Date _____

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

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

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

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

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

Appendix F

THE BECK DEPRESSION INVENTORY, 2nd EDITION (BDI-II)

IDB-II

No. Dossier _____ Date _____

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

1.

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

2.

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

3.

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

4.

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

5.

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

6.

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

7.

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

8.

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

9.

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

10.

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

11.

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

12.

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

13.

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

14.

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

15.

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

16.

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

17.

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

18.

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

19.

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

20.

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

21.

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