Relationships Between Treatment Knowledge, Beliefs and Outcome Following Cognitive Behaviour Therapy for Panic Disorder and Agoraphobia

A thesis submitted in fulfilment of the requirements for the degree of Doctor of Philosophy in the School of Psychology at The University of Sydney

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Statement of Originality

I hereby declare that this submission is my own work and to the best of my knowledge it contains no materials previously published or written by another person, or substantial proportions of material which have been accepted for the award of any other degree or diploma at The University of Sydney or any other educational institution, except where due acknowledgement is made in the thesis. Any contribution made to the research by others, with whom I have worked at The University of Sydney or elsewhere, is explicitly acknowledged in this thesis. I also declare that the intellectual content of this thesis is the product of my own work, except to the extent that assistance from others in the project’s design and conception or in style, presentation, and linguistic expression is acknowledged.

Signed ..............................................

Dated ..............................................
Acknowledgments

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Abstract

The purpose of this thesis was to investigate relationships between treatment knowledge, beliefs and outcome in Panic Disorder and/or Agoraphobia (Panic-Ag). Research from the psychotherapy and medical literature indicates patients’ treatment knowledge and beliefs, specifically acceptance of the treatment rationale (ATR), expectancies of treatment outcome (ETO) and treatment self-efficacy (TSE), are associated with clinical outcomes for a range of disorders. However, methodological limitations surrounding measurement of these constructs have undermined conclusions and/or such relationships have not been investigated in the field of Panic-Ag.

Relationships between treatment knowledge, beliefs and outcome in Panic-Ag were examined using a 2 phase procedure. Phase 1 involved developing measures of treatment knowledge, ATR, ETO and TSE using patient and clinician samples. The psychometric properties of these measures were found to be satisfactory. Phase 2 investigated associations between treatment knowledge, beliefs and outcome following cognitive behaviour therapy (CBT) among 41 Panic-Ag participants. Measures were administered at pretreatment and 6-months posttreatment. It was hypothesised that treatment knowledge, ATR, ETO and TSE would be related to outcome, with associations mediated by belief in catastrophic cognitions. Of 4 Panic-Ag outcome measures (panic attack frequency, panic sensation severity, frequency of catastrophic cognitions and agoraphobic avoidance), results indicated improved treatment knowledge was significantly associated with frequency of catastrophic cognitions and agoraphobic avoidance. Posttreatment TSE was significantly associated with panic attack sensation severity, frequency of catastrophic cognitions and agoraphobic avoidance. Contrary to the hypothesis, ATR was not related to outcome.

Similar findings concerning TSE and ATR were obtained in an independent sample of 34 Panic-Ag participants. Exploratory analyses found that pretreatment beliefs including outcome expectancies were unrelated to outcome. Mediational analyses revealed relationships between TSE and outcome were partially mediated by belief in catastrophic cognitions while relationships between treatment knowledge and outcome were not. Results are discussed in light of previous research, methodological limitations, clinical implications and future research directions.
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Treatment Knowledge, Beliefs and Outcome in Panic Disorder and Agoraphobia: Is There a Relationship?

Introduction to Panic Disorder and Agoraphobia

Panic Disorder is a disabling anxiety disorder marked by recurrent unexpected episodes of brief, overwhelming physical symptoms consisting of heart palpitations, dizziness, shortness of breath, sweating, gastrointestinal distress, chills or hot flushes, numbness or tingling, feelings of unreality and a fear of dying or loss of control (American Psychiatric Association, 1994). In addition, fears of developing further attacks, worry over consequences (e.g., heart attack, going insane) and/or significant behavioural changes to prevent recurrence (e.g., avoidance of activities that trigger such sensations, restriction of travel, avoidance of being alone) emerges. Panic disorder often co-occurs with agoraphobia where specific situations are avoided or feared due to fear of embarrassment or absence of help should a panic attack occur (American Psychiatric Association, 1994).

The prevalence of panic disorder and/or agoraphobia (Panic-Ag) is relatively high with recent epidemiological studies in developed nations estimating 12-month prevalence rates between 1.6% and 2.8%, and lifetime rates of 2.1% to 5.1% (Andrews, Henderson, & Hall, 2001; Goodwin et al., 2005; Grant et al., 2006; Kessler et al., 2006). The condition is associated with comorbid poor physical health and health perceptions (Schmidt et al., 2003; Schmidt & Telch, 1997), elevated rates of depression (Brown & Barlow, 1992; Johnson & Lydiard, 1998), substance use (Cosci, Schruers, Abrams, & Griez, 2007; Marshall, 1997), and suicidal ideation and attempts (Boden, Fergusson, & Horwood, 2007; Goodwin & Roy-Byrne, 2006). Panic-Ag is also a costly disorder from a social and economic perspective (Batelaan...
et al., 2007; Edlund & Swann, 1987; Leon, Portera, & Weissman, 1995; Rees, Richards, & Smith, 1998; Salvador-Carulla, Segui, Fernandez-Cano, & Canet, 1995) with increased rates of medical service utilisation (Rees et al., 1998; Roberge et al., 2005; Weissman, 1990), absenteeism, unemployment and financial dependence (Edlund & Swann, 1987; Leon et al., 1995).

Current practice guidelines advocate the use of cognitive behaviour therapy (CBT) as the first line of treatment for Panic-Ag (American Psychiatric Association, 1998). Randomised controlled trials consistently demonstrate CBT to be effective for Panic-Ag (for reviews see Gould, Otto, & Pollack, 1995; Mitte, 2005; Westen & Morrison, 2001), with up to 87% of patients achieving panic-free status (defined as no panic attacks in the last month) at posttreatment (Landon & Barlow, 2004) and up to 81% remaining panic free at 1 to 2-year follow-up (Clark et al., 1994; Craske, Brown, & Barlow, 1991). However, the criterion of panic-free status is not an accurate and/or sensitive measure of true patient improvement since improvement rates markedly decrease when more conservative outcome criteria are applied. For example, a cross-sectional 2-year follow-up study by Brown and Barlow (1995) revealed that although 75% of patients were panic free, only 57% met criteria for high end state functioning (defined as no panic attacks in the last month and clinician severity rating of Panic-Ag as mild or below). When definitions of successful outcome include high end-state functioning and no further requirement for treatment, the percentage of patients classified as having successful outcomes reduces to 48%. Further, when even more stringent criteria of high end-state functioning at both 3-month and 2-year follow-up, no panic attacks in the past year and no further need for treatment are applied, successful outcome rates reduce to 21%. Similarly, a meta-analysis of CBT conducted by Westen and Morrison (2001) found the average Panic-
Ag patient remained at least mildly symptomatic at posttreatment and 35% sought additional treatment within 18 months of completing CBT. Thus although CBT is beneficial for the majority of Panic-Ag patients, there is further room for improvement. Therefore, the identification of processes and mechanisms associated with successful treatment outcome is crucial to improving the effectiveness of CBT.

CBT for Panic-Ag is based on Clark’s (1986) cognitive model which underscores the importance of catastrophic misinterpretations of harmless panic sensations in the maintenance of the disorder. CBT is assumed to be effective by assisting patients to identify, challenge and replace catastrophic cognitive interpretations with more realistic, less threatening ones. Considerable evidence has accumulated over the last two decades supporting the validity of the cognitive model and CBT produces a reliable decrease in both catastrophic cognitions and panic severity. However, it is unclear whether cognitive change is the cause or effect of treatment improvement. Furthermore, changes in catastrophic cognitions account for less than one third of the variance associated with symptom improvement (Hofmann et al., 2007) suggesting the influence of other contributing elements. This thesis aims to explore additional potential factors related to treatment knowledge and beliefs that may potentially influence the effectiveness of CBT.

**Treatment Compliance**

It is argued within the literature that the effectiveness of CBT is contingent on patient compliance with treatment recommendations (Burns & Spangler, 2000; Edelman & Chambless, 1993; Kazantzis, Deane, & Ronan, 2000; Kazantzis, Ronan, & Deane, 2001; Schmidt & Woolaway-Bickel, 2000; Westra & Dozois, 2006). However, treatment non-compliance is common (Sanderson & Bruce, 2007), with up
to 60% of patients failing to complete specific CBT directives as assigned (Helbig & Fehm, 2004). Patient treatment non-compliance therefore represents an important impediment to treatment outcome.

To ensure compliance, at least four conditions must be present. Patients must:

i) understand information presented

ii) be accepting of the treatment rationale

iii) believe treatment will be helpful, and

iv) have sufficient self-efficacy to implement therapeutic strategies.

A breakdown in any of these conditions may result in treatment non-compliance, while fulfilment of one condition does not guarantee fulfilment of others (Raynor, 1998). Although CBT treatment outcome studies often assess the extent to which therapists adhere to the content of treatment manuals (treatment integrity), the majority rarely include measures assessing patients’ knowledge and beliefs about treatment. Such studies implicitly assume that once patients are exposed to treatment information (the treatment rationale and application of techniques), they effectively understand information provided, accept it unquestioningly, believe it will be helpful and feel confident in applying it. However, as Primakoff, Epstein and Covi (1986) stated, “it is not sufficient to record what therapists ‘prescribe’ in order to know how much self-administered treatment the cognitive therapy patient actually ‘absorbs’” (p. 434).

As will be discussed below, research from the psychotherapy and medical literature has identified patients’ knowledge about treatment, and beliefs about the treatment rationale, helpfulness of treatment and self-efficacy to implement techniques, as fundamental factors that potentially interfere with the “psychological availability” of CBT, resulting in poorer treatment outcomes. However, these issues
have either not been rigorously investigated in the area of Panic-Ag or methodological limitations preclude firm conclusions being drawn. Hence, this thesis aims to examine relationships between patients’ treatment knowledge, beliefs and outcome following CBT for patients with Panic-Ag. Significant associations with such variables have important clinical implications that may enhance the effectiveness of CBT in reducing the burden of the disorder for both sufferers and society at large. Before examining the extant literature surrounding these issues, a discussion of the cognitive model and evidence in support of cognitive mediation of treatment effects will be reviewed.

The Cognitive Model of Panic-Ag

Clark (1986), in his seminal paper shaping current conceptualisations and treatments of Panic-Ag, advanced a cognitive model of panic highlighting catastrophic misinterpretations of bodily sensations as central to the aetiology and maintenance of Panic-Ag. This model proposes that Panic-Ag patients catastrophically interpret benign bodily sensations of anxiety, stress and arousal as evidence of imminent physical, mental or social danger; for example, heart palpitations may be misinterpreted as a sign of impending heart attack. A vicious cycle is set up such that catastrophic interpretations result in increased autonomic arousal causing anxiety symptoms to intensify, thereby reinforcing misinterpretations culminating in a panic attack. Associating specific situations (e.g., shopping centres, driving) with feared physical sensations lead patients to fear and/or avoid such situations and develop agoraphobia. Hence, according to the model, catastrophic cognitions about bodily sensations mediate panic attacks as well as associated
avoidance and disability. Treatment based on the cognitive model therefore consists of techniques focussed on reducing patients’ belief in catastrophic cognitions.

**Treatment Components of CBT for Panic-Ag**

There are three primary treatment components aimed at modifying catastrophic cognitions in Panic-Ag: psychoeducation, cognitive restructuring and exposure (in vivo and interoceptive) (Rayburn & Otto, 2003). Psychoeducation involves the provision of accurate information about the nature and physiology of anxiety and panic. Patients are informed of the *fight or flight response*, a physiological reaction enabling individuals to either fight or flee upon perceiving danger, and its relationship to panic attacks. Panic attacks are considered false alarms activated when individuals perceive physical or social threat (e.g., “I’m going to have a heart attack”, “I’m going to embarrass myself”) in the absence of real danger. Subsequently, patients are introduced to the concept of catastrophic misinterpretations of physical sensations and their role in perpetuating an anxious cycle ending in a panic attack. The impact of hyperventilation on the production of physical panic sensations is discussed and the medical reality of such intense physical sensations explained.

Cognitive restructuring involves assisting patients to identify underlying catastrophic misinterpretations of panic and teaching methods to challenge faulty threat appraisals. Patients typically overestimate the probability and/or cost of potential panic outcomes. Inflated probability and cost estimates are challenged either through examining evidence from past experiences or by conducting *behavioural experiments* which involve subjecting thoughts to reality testing. For example, if patients have the thought, “I am going to collapse” upon feeling dizzy
and lightheaded, this could be tested by standing without holding onto any supports while feeling panicky to determine whether or not they fall.

In vivo exposure involves confronting feared situations associated with panic such as shopping centres, lifts, public transport and driving. Interoceptive exposure involves deliberately inducing panic sensations (e.g., dizziness, shortness of breath, palpitations) through various exercises such as hyperventilating, breathing through a straw, running on the spot and spinning. A graded approach is used where patients construct a hierarchy of feared situations/sensations ranging from mildly to severely anxiety provoking and then systematically confront easier tasks before progressing to more difficult ones. In addition, exposure is accompanied with a gradual fading of avoidance, escape and safety seeking behaviours (i.e., reliance on people or items such as medication or mobile phone). In this way, exposure therapy, although often regarded as behavioural in nature, also has a strong cognitive focus; when used as a behavioural experiment, patients can test whether predicted catastrophes eventuate.

In essence, CBT adopts a psychoeducational approach (Blagys & Hilsenroth, 2002) where therapists present information and teach skills to help patients identify, test and replace cognitive misinterpretations and unhelpful behavioural responses with more realistic, less threatening ones. The process of change, however, requires patients to actively participate in therapy through learning, understanding and consistently applying skills.
Chapter 1 – Literature Review

Evaluation of the Cognitive Model of Panic-Ag

Clark (1986, 1996) delineated four predictions arising from the cognitive model:

1. “Panic patients will be more likely to interpret bodily sensations in a catastrophic fashion than individuals who do not experience panic attacks.
2. Procedures that activate catastrophic misinterpretations of bodily sensations will produce an increase in anxiety and panic in panic disorder patients.
3. Panic attacks can be prevented by reducing patients’ tendency to interpret bodily sensations in a catastrophic fashion.
4. Sustained improvement after the end of any treatment (whether psychological or pharmacological) will depend on cognitive change having occurred during the course of therapy” (Clark, 1996, p. 322).

These four predictions have received considerable research support over the last 20 years (Austin & Kiropoulos, 2008; Austin & Richards, 2006; Clark, 1993; Clark et al., 1994; Clark et al., 1997; Craske et al., 1991; Ehlers, Margraf, Roth, Taylor, & Birbaumer, 1988; Harvey, Richards, Dziadosz, & Swindell, 1993; McNally & Foa, 1987; Rapee, Mattick, & Murrell, 1986; Sanderson, Rapee, & Barlow, 1989; Schneider & Schulte, 2007; Telch, Silverman, & Schmidt, 1996; Westling & Öst, 1995) thus supporting the validity of the cognitive model for Panic-Ag. Evidence related to cognitive mediation of CBT’s treatment effects will now be reviewed.
Cognitive Mediation of CBT for Panic-Ag

To demonstrate that the effectiveness of CBT is cognitively mediated, three criteria must be satisfied (Hofmann et al., 2007; Oei, Llamas, & Devilly, 1999):

i) CBT must be demonstrated to be effective in reducing panic severity and catastrophic cognitions

ii) Effective CBT must produce greater change in catastrophic cognitions relative to other treatments (e.g., medication, relaxation)

iii) Change in catastrophic cognitions produces the observed treatment improvements.

Research investigating these criteria is reviewed below.

Criterion 1: Effective CBT Produces Changes in Catastrophic Cognitions

The majority of studies that have incorporated cognitive measures in their evaluation of the efficacy of CBT for Panic-Ag have found significant reductions in both catastrophic cognitions and panic severity following treatment. In their review of studies published between 1983 and 1996, Oei et al. (1999) found 15 of 16 studies demonstrated CBT to be effective in producing significant cognitive changes in the desired direction. Since this review, subsequent researchers have reported similar significant reductions in the frequency and strength of belief in catastrophic cognitions (Arntz, 2002; Bouchard et al., 2007; Casey, Oei, & Newcombe, 2005; Clark et al., 1999; Öst, Thulin, & Ramnerö, 2004; Poulton & Andrews, 1996; Richards & Alvarenga, 2002; Salkovskis, Hackmann, Wells, Gelder, & Clark, 2007; Wenzel, Sharp, Brown, Greenberg, & Beck, 2006). Percent reduction of catastrophic cognitions calculated from the data in their articles ranged from 17.4% (Bouchard et al., 1996) to 86.1% (Clark et al., 1999), with one study demonstrating 100%
reduction of primary catastrophic cognitions in five of seven patients treated with purely cognitive procedures (Salkovskis, Clark, & Hackmann, 1991). Hence, it would appear reasonable to conclude this aspect of cognitive mediation has been satisfied.

**Criterion 2: Effective CBT Leads to Greater Change in Catastrophic Cognitions Relative to Other Treatments**

CBT programs for Panic-Ag have been compared against pharmacotherapy, traditional exposure (in vivo and interoceptive) and relaxation therapies. Results differ according to type of therapy administered.

**CBT vs. Pharmacotherapy**

Two randomised controlled trials investigated cognitive change in CBT relative to pharmacotherapy and found results consistent with cognitive mediation of CBT. Clark et al. (1994) investigated 40 Panic-Ag patients and found CBT \( (n = 20) \) led to approximately twice as much improvement than imipramine \( (n = 20) \) on measures assessing catastrophic cognitions. At 3-month posttreatment, calculations from their data revealed CBT produced a 95% reduction in scores on the Body Sensations Interpretation Questionnaire (BSIQ, Clark et al., 1988) relative to 46% for imipramine. Similar results were shown for the Agoraphobic Cognitions Questionnaire (ACQ, Chambless, Caputo, Bright, & Gallagher, 1984) with CBT producing a 66% reduction in scores relative to 36% for imipramine. More recently, Hofmann et al. (2007) found catastrophic cognitions significantly mediated improvement in eight out of nine tests of mediation for CBT \( (n = 73) \) but in none of three tests of mediation for imipramine \( (n = 18) \). Hence, results from two
independent studies provide evidence of greater cognitive change for CBT than pharmacotherapy; however, different findings have been observed when comparing CBT to non-pharmacological therapies.

**CBT vs. Exposure Therapy**

Studies comparing CBT with exposure therapy (involving prolonged exposure in the absence of specifically challenging catastrophic cognitions) on degree of cognitive change revealed mixed findings. Of eight studies, five found equivalent rates of cognitive change (Arntz, 2002; Bouchard et al., 1996; Burke, Drummond, & Johnston, 1997; Michelson, Marchione, Greenwald, Testa, & Marchione, 1996; Öst et al., 2004); one study found CBT led to significantly greater levels of cognitive change than exposure therapy (Salkovskis et al., 2007), while another found CBT produced greater change than guided mastery (an exposure-based therapy solely focused on increasing patients’ sense of mastery in feared situations) on two of three cognitive measures, with equal change on the third (Hoffart, 1995b). In contrast, a further study (Williams & Falbo, 1996) found CBT produced significantly less cognitive change than guided mastery.

In explaining these discrepant results, sample sizes were low in the Salkovskis et al. (2007) study ($n = 8$ per group), hence their findings may have been affected by sampling error. Furthermore, the duration of exposure treatment was only 3.25 hours (in comparison to 20+ hours in traditional exposure treatments), suggesting longer exposure periods may be necessary to yield cognitive change observed in other studies.

The discordant findings may also be attributable to *researcher allegiance effects* (Luborsky et al., 1999), that is, better results are consistently obtained for
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treatments that researchers have an allegiance with. As the Clark and Salkovskis group have been responsible for the development of contemporary CBT programs based on the cognitive model and the Williams group developed the exposure-based treatment of guided mastery, the differential findings are consistent with researcher allegiance effects.

In summary, CBT and exposure therapies generally produce equivalent amounts of cognitive change. On this basis, both treatments are assumed to reduce anxiety through the same mechanism of changes to catastrophic misinterpretations of physical symptoms (e.g., Bouchard et al., 1996; Margraf, Barlow, Clark, & Telch, 1993). It is postulated that patients learn their feared bodily sensations are harmless through either direct experience (i.e., exposure) or verbal discourse (i.e., cognitive therapy plus reality testing with behavioural experiments). Such an interpretation suggests treatment effects for both CBT and exposure therapy may be cognitively mediated. Thus, to determine whether CBT produces greater cognitive change than other therapies, a treatment such as relaxation therapy, that does not focus on correcting harm-related interpretations of panic symptoms (either directly or indirectly), should provide a better test of cognitive mediation.

**CBT vs. Relaxation Therapy**

Of five comparative studies of CBT versus relaxation therapy, four did not yield significant differences between these interventions (Barlow, Craske, Cerney, & Klosko, 1989; Beck, Stanley, Baldwin, Edwin, & Averill, 1994; Carlbring, Ekselius, & Andersson, 2003; Öst & Westling, 1995). Only the study by Clark et al. (1994) found CBT led to significantly greater cognitive reduction relative to applied relaxation. With four of five studies showing non-significant differences, results
suggest that treatments not focussed on challenging catastrophic cognitions bring about equivalent levels of cognitive change. In explaining these counter-intuitive findings, Öst and Westling (1995) suggested reductions in panic severity generalised to improvement on cognitive measures; that is, cognitive change was a consequence or by-product of treatment improvement, similar to the way scores on self-report measures of depression and general anxiety also often decrease following successful treatment for Panic-Ag.

Results from a recent meta-analysis of CBT and relaxation therapy for Panic-Ag found the mean effect size of between-groups differences on cognitive measures was 0.48 (range = 0.10 to 1.08) indicating that overall, CBT produced greater cognitive change at posttreatment than relaxation therapy (Siev & Chambless, 2007). However, the only study finding a significant difference between CBT and relaxation in this meta-analysis was Clark et al.’s (1994) study suggesting researcher allegiance effects may have biased the outcome of this meta-analysis. Luborsky et al. (1999) found that correcting for researcher allegiance effects in meta-analyses of treatment comparison studies reduced observed differences between treatments to non-significant levels. Therefore, had Siev and Chambless (2007) assessed and controlled for allegiance effects in their meta-analysis, it is likely no significant difference would have occurred between the two treatments in the amount of cognitive change achieved.

In summary, in order to justify cognitive mediation of CBT’s effects, CBT should produce greater cognitive change relative to other therapies that do not target cognitions directly. However, the majority of studies comparing CBT with either exposure therapy or relaxation found these treatments did not differ significantly from CBT in cognitive changes produced. Some supportive evidence for cognitive
mediation is obtained from limited research comparing CBT with pharmacotherapy. Evidence in support of cognitive mediation across all three treatment comparisons (i.e., CBT vs. pharmacotherapy, exposure therapy and relaxation) emanates from the Clark and Salkovskis group (Clark et al., 1994; Salkovskis et al., 2007) who developed the cognitive model of Panic-LAg. Given their findings were partially replicated by only one of seven studies comparing CBT with exposure and none of four studies comparing CBT with relaxation therapy, one possible explanation is the influence of researcher allegiance effects in accounting for results obtained. Thus, the second criterion required for cognitive mediation receives only weak support.

**Criterion 3: Treatment Improvement is Produced by Change in Catastrophic Cognitions**

Although CBT has been shown to reduce panic severity and catastrophic cognitions, the possibility remains that observed changes in catastrophic cognitions are the result of improvements in panic severity (Öst & Westling, 1995) rather than its cause. Stronger support for cognitive processes underlying the effects of CBT can be demonstrated by showing that changes in catastrophic cognitions occurring during treatment are predictive of symptom improvement (Casey, Oei, & Newcombe, 2004), and that maintenance of treatment gains is dependent on degree of cognitive change (Clark, 1986).

Several studies have found that greater changes in catastrophic cognitions during CBT predicted better treatment outcome at posttreatment and up to 1-year follow-up (Casey, Newcombe, & Oei, 2005; Hoffart, 1998). Furthermore, stronger endorsement of catastrophic cognitions at posttreatment has been linked with poorer outcomes at posttreatment and 1-year follow-up (Clark et al., 1994; Clark et al.,
1999; Westling & Öst, 1995), thus providing additional evidence supporting cognitive mediation. However, the cross-sectional design of these studies still cannot exclude the possibility that changes in catastrophic cognitions occurred as a consequence rather than a cause of symptom improvement (Cho, Smits, Powers, & Telch, 2007; Westling & Öst, 1995). Research incorporating regular assessment of catastrophic cognitions during the treatment period is required to clarify directions of causality.

Bouchard and colleagues (2007) addressed the issue of causality in a study incorporating a 6-week pretreatment phase, an 18-week treatment phase and a 6-week posttreatment phase. These authors examined daily belief ratings of primary catastrophic cognitions and panic apprehension over this 30-week period in 12 Panic-Ag patients who responded positively to CBT. Using multivariate time series analysis, changes in catastrophic beliefs preceded changes in panic apprehension in only six patients (50%). The authors noted that despite remaining panic free, many patients still endorsed catastrophic cognitions at a low to moderate degree (Bouchard et al., 2007). Hence, effects of CBT are only partly mediated by catastrophic cognitions. Indeed, Hofmann et al. (2007) found catastrophic cognitions accounted for only 20% to 30% of the change in panic severity.

In summary, independent studies show CBT reduces catastrophic cognitions, and reduction in catastrophic cognitions during treatment is associated with decreased panic severity at posttreatment and follow-up, supporting cognitive mediation of treatment effects. However, similar rates of cognitive change also occur with non-cognitively focussed treatments and a study investigating causality found improvements in panic severity were not preceded by cognitive change in half of patients. Hence, it remains uncertain whether cognitive change is the cause or effect
of symptom relief. Furthermore, it is unclear whether catastrophic cognitions are the primary underlying mechanism contributing to treatment change or whether non-specific factors common to many treatments, such as patients’ knowledge and beliefs about treatment, are also involved in mediating the effectiveness of CBT.

**Role of Treatment Knowledge and Beliefs**

As stated, CBT requires patients to actively participate in treatment, however non-compliance with therapeutic instructions is common (Helbig & Fehm, 2004). In order to effectively comply with treatment instructions, patients must know what they are supposed to do (Jette, 1982). Patients cannot implement treatment advice that they do not understand or remember. Given Panic-Ag patients present to treatment with high rates of anxiety and depression, such mood and arousal states frequently interfere with cognitive processes (e.g., attention, concentration, memory) essential for learning and retaining information (Asmundson, Stein, Larson, & Walker, 1994; Barbee, 1993; Lucas, Telch, & Bigler, 1991). If it is assumed CBT is effective through transfer of specific therapeutic information and techniques, it follows that even highly effective CBT treatment programs will fail if patients are unable to recall or comprehend what is discussed during treatment (Schraa & Dirks, 1982).

Investigations into provider-patient communication in patients with medical disorders have repeatedly demonstrated that patients forget much of what they are told (Ley, 1988), and of information recalled, misunderstandings and misinterpretations are common. Hence, poor treatment knowledge promotes *unintentional* non-compliance, which in turn contributes to compromised treatment
outcomes. However, even despite knowing what to do, patients may deliberatively decide not to adhere to treatment recommendations. Such *intentional* non-compliance is often influenced by patients’ beliefs about treatment (Horne, 1999) which have been shown to adversely affect treatment outcome. Research from the wider psychotherapy and medical literature has identified patients’ beliefs concerning the treatment rationale, the helpfulness of treatment and their self-efficacy to apply the therapeutic techniques as being highly influential to treatment outcome. Before reviewing the literature on patients’ beliefs about treatment, patients’ knowledge of treatment and its impact on clinical outcomes will be discussed, commencing with a definition of treatment knowledge.

**Patient Knowledge of Treatment**

**Definition of Treatment Knowledge**

The Australian Concise Oxford Dictionary, Fourth Edition (2004) defines knowledge as “Awareness or familiarity gained by experience (of a person, fact or thing); a theoretical or practical understanding of a subject, language, etc” (p. 777). Numerous studies have investigated relationships between patient knowledge and various clinical outcomes, for example, treatment compliance, illness control, quality of life, with results ranging from no association (e.g., Blalock et al., 2000; Chan & Molassiotis, 1999; Coates & Boore, 1996; Ho et al., 2003; Ivens & Sabin, 2006; Lee, Wing, & Wong, 1992; Sands & Holman, 1985; Scherer & Bruce, 2001) to significant positive associations (e.g., Abramowitz, Franklin, Zoellner, & DiBernardo, 2002; Barth, Campbell, Allen, Jupp, & Chisholm, 1991; Croquelois & Bogousslavsky,
2006; Kallich, McDermott, Xu, Fayers, & Cella, 2006; Kim et al., 2007; Kronmüller et al., 2006; Miller et al., 2003; Ngamvitroj & Kang, 2007; Ni et al., 1999; Soriano, Rabe, & Vermeire, 2004; Surawy, 1989; Weiss et al., 2003). However, patient knowledge is a broad term encompassing knowledge of diagnosis, symptoms, pathophysiology, further investigations, risks associated with procedures, prognosis and treatment instructions/advice. Such differing definitions of patient knowledge may in part explain these discordant findings (Eraker, Kirsch, & Becker, 1984).

This thesis will confine its investigation of patient knowledge to the domain of treatment. For this thesis, treatment knowledge is defined as the patients’ awareness, familiarity or understanding of treatment information, instructions, recommendations or advice provided by treating clinicians considered necessary to control, cure, manage or prevent their presenting condition. Evidence of treatment knowledge, for example, would be provided if the doctor told a patient he/she needed to take one tablet in the morning with food every day to control hypertension and the patient was able to correctly remember and understand instructions (irrespective of actual behavioural compliance). In the CBT domain, treatment knowledge may include an understanding of the treatment rationale and ability to describe how to set up and complete a behavioural experiment in order to test catastrophic cognitions.

Accurate understanding of treatment may be considered to reflect a deeper degree of knowledge than merely having an awareness of treatment. Obtaining an estimate of the degree of knowledge a patient possesses is dependent upon the manner in which such knowledge was assessed.
Assessment of Treatment Knowledge

Treatment knowledge can be assessed through a range of methods including written tests, interviews and behavioural observation; however all methods require information to be stored in and retrieved from memory. The most common method for assessing patients’ treatment knowledge is to elicit their recall of information provided in relation to treatment. Ley (1988) distinguished between free recall, cued recall and probed recall. Free recall refers to patients freely reporting what the clinician has said. Cued recall is when cues are provided and patients are asked what was said in specified areas (e.g., “What was said about your medication?”). Probed recall is when patients are persistently questioned on a topic until they cannot recall any more information. Procedural differences influence the amount of information patients report. For example, probed recall with a cue elicits more information than cued recall without probing, which in turn elicits more information than free recall alone (Kortman, 1992; Tuckett, Boulton, & Olson, 1985).

Other methods of assessing treatment knowledge involve administration of written questionnaires. The format of questions can be open-ended short-answer questions (e.g., Jarvie, Espie, & Brodie, 1993b; Westra et al., 2004), true/false style (e.g., Rees, Abed, & Sheard, 2003; Westreich, Levine, Ginsburg, & Wilets, 1995) or multiple-choice (e.g., Baker, Uus, Bamford, & Marteau, 2004; Pande et al., 2000). Multiple-choice methods, however, assess recognition memory of treatment information rather than recall and may therefore yield a different estimate of the proportion of knowledge obtained as compared to recall methods.

Recall methods, although assessing the quantity of information patients retain, may not provide a true measure of patients’ knowledge of treatment instructions since recall of treatment does not guarantee understanding of instructions
or concepts conveyed. Patients often interpret information in a different way from how it was intended, as documented by Mazzullo, Lasagna and Griner (1974). These authors interviewed 67 medical patients on their understanding of common prescription labels and reported 52% thought a tablet for fluid retention actually caused, rather than reduced, fluid retention. As highlighted by Mazzullo et al. (1974), such misinterpretations may impact on a patient’s implementation of treatment (e.g., avoidance of taking the tablet upon noticing signs of oedema), resulting in unintentional non-compliance and poorer treatment outcome.

Patients’ understanding of treatment provides a more accurate evaluation of treatment knowledge. Several different approaches have been used, including self-report, use of expert judges and behavioural (or quasi-behavioural) observation (Ley, 1988). Examples of self-report items to assess understanding include, “How well do you understand how to take your medication?” (Heisler, Bouknight, Hayward, Smith, & Kerr, 2002) and “I did not understand today’s health information, because it was too complicated for me” (Lukoschek, Fazzari, & Marantz, 2003).

The use of expert judges to assess understanding involves using individuals highly knowledgeable about illness and treatment to interview patients about treatment recommendations. Any jargon or terms used by patients are queried to determine their interpretations of such words (Tuckett et al., 1985). For example, if a patient said they took a tablet for fluid retention, their interpretation of fluid retention would be carefully probed. Discrepancies between patients’ interpretation of what was told and what the doctor actually meant are taken as evidence of reduced treatment knowledge.

Finally, behavioural observation is perhaps the most reliable method for assessing patients’ treatment knowledge (Cleaveland & Denier, 1998). This method
involves asking patients to specify or demonstrate the behaviours required for treatment compliance. An example of behavioural observation to assess diabetes treatment knowledge would be, “Show me how you would sterilise your needle.”

In summary, assessment of treatment knowledge involves having patients recall what they were told, either orally or through questionnaires. Recall methods estimate the quantity of information retained, however, they do not ensure information recalled is correctly interpreted. Assessment of patients’ understanding of treatment information through interview or behavioural demonstrations can provide a more valid impression of patients’ treatment knowledge. Regardless of methods used, research from the medical literature clearly demonstrates patients’ recall and understanding of treatment is frequently flawed.

**Recall of Treatment Instructions**

Numerous researchers have found patients’ free recall of treatment instructions to be poor. Such investigations began with the work of Philip Ley and his colleagues. In his earliest report, Ley and Spelman (1965) interviewed 47 first-time attendees of a medical outpatient clinic shortly after consultation (0 – 80 minute delay). These patients recalled only 44% of treatment instructions provided. In later studies with 20 and 157 general practice patients, Ley and colleagues found patients recalled only 28% and 44% of treatment instructions/advice, respectively (Ley, Bradshaw, Eaves, & Walker, 1973; Ley et al., 1976). Similar rates of recall (44% – 52%) were found in rheumatology patients (Anderson, Dodman, Kopelman, & Fleming, 1979), although lower rates were observed after a longer delay period (> 1 week, 0% – 39%) (Joyce, Caple, Mason, Reynolds, & Mathews, 1969).
Several large-scale, multi-site studies found high proportions of patients were unable to recall specific treatment instructions when knowledge was assessed via free recall. Among 1,751 patients, Kravitz et al. (1993) found 68.4%, 54.9% and 39.6% of patients with hypertension, diabetes and heart disease failed to recall recommended self-care behaviours, respectively. More recently, Flock and Stange (2004) found, on average, 58% of 2,670 primary care patients were unable to recall health behaviour recommendations.

As stated, type of recall method influences the amount of information remembered. Kortman (1992) used both free and cued recall to assess what 28 patients with tendon injuries remembered about treatment and found more information was recalled in response to cues. For example, of five instructions provided, all 28 patients (100%) failed to recall at least one instruction without cue; however this percentage dropped to 64.3% (18 of 28 patients) with the addition of specific cues. Indeed, other studies found higher rates of recall using cued recall methods. Bertakis (1977) found patients attending a family practice clinic recalled 63.2% of treatment information shortly after their consultation. Among 32 Panic-Ag patients, Westra and colleagues found 68% of psychoeducation information was recalled immediately after presentation (Westra et al., 2004).

Even higher recall rates were found by Tucket et al. (1985) in 328 general practice patients using a probed recall method. Following patients’ consultation with the doctor, a third party judgement was made regarding key points of the consultation patients were expected to know. The authors found 97% and 95% of patients recalled all key points regarding treatment and prevention, respectively. Such high recall rates are inconsistent with findings from other investigators and likely reflect differing procedural methods concerning operationalisation of treatment
knowledge (key points vs. number of statements made), assessment of recall (probed recall vs. free recall) and sample characteristics (repeat attendees vs. first-time attendees with a new illness). Patients making a second or third visit tend to recall more information than patients presenting with a new illness (Ley, 1988).

In summary, patients are unable to spontaneously recall between one third and over one half of treatment information. However, providing a cue or probing can yield higher recall rates. Assessment of what patients recall does not necessarily imply they understand the information or comprehend it in the same manner as it was intended. Studies examining patient understanding of treatment indicate instructions and advice provided by doctors are often misinterpreted or not understood by patients.

**Understanding Treatment Instructions**

With regard to patients’ self-reported understanding of treatment, between 14% and 43% of patients reported that they did not understand what they were told about treatment (Ley, 1988; Lukoschek et al., 2003). Heisler et al. (2002) recently surveyed 2,000 patients receiving diabetes care, eliciting their understanding of treatment using a self-report scale from 0 to 100. On average, patients scored 76.3, indicating a substantial gap between perceived current and ideal understanding of treatment.

A disadvantage of relying on patient self-report to assess understanding is the risk that patients’ believe they understand information when in actuality they do not, thereby potentially overestimating treatment knowledge (Ley, 1988). For example, although patients in Anderson et al.’s (1979) study recalled 49% of treatment information, the authors noted that much of what patients recalled was incorrect. Of
what patients believed they were told, 39% was found to be either imagined to have been said or misinterpreted. Similarly, Tucket et al. (1985) found that while over 90% of patients recalled key points regarding treatment and prevention advice, discrepancies in the interpretation of what was said between doctor and patient was evident in 25% of cases.

Researchers who interviewed patients to assess understanding of treatment found high rates of treatment non-comprehension. Kerzman, Baron-Epel and Toren (2005) differentiated between patients’ reported knowledge and correct knowledge about medication therapy. Among 288 patients discharged from hospital, only 35 (12%) reported knowledge about required lifestyle changes, and 20 of those (57%) demonstrated incorrect knowledge. Therefore, correct knowledge of necessary lifestyle changes was demonstrated in only 15 of 288 patients (5.2%).

Lack of treatment understanding is considered a major contributor of patients’ non-compliance with treatment regimens, with reports revealing 32% to 58% of patients fail to accurately understand or comprehend medication schedules (Brody, 1980; Hulka, Cassel, Kupper, & Burdette, 1976; Parkin, Henney, Quirk, & Crooks, 1976) and up to 98% misunderstand instructions regarding timing of doses (see Ley, 1988, for a review). One further study reported 82% of medical patients discharged from hospital were either unaware, or had incomplete understanding, of treatment advice despite over half receiving a written copy (Ellis, Hopkin, Leitch, & Crofton, 1979). Such high rates of treatment non-comprehension are worrying as inadequate knowledge adversely affects treatment compliance and subsequently affects health outcomes.

Using behavioural observation, a more ecologically valid measure of patient treatment knowledge, Watkins and colleagues (Watkins, Williams, Martin, Hogan, &
Anderson, 1967) found similar results. Sixty diabetic patients were asked to demonstrate daily diabetic management routines. Over 76% of patients were classified as having unacceptable performance on urine testing and 93.3% were considered to perform inadequately in at least one of three diabetic management areas expected to directly affect diabetic control.

Thus, whether treatment understanding is assessed via self-report, interview or behavioural methods, patients frequently demonstrate insufficient knowledge of treatment and its requirements. Patients cannot adhere to advice they do not remember or understand. It follows that poor treatment knowledge promotes treatment non-compliance which in turn adversely affects the effectiveness of treatments and contributes to reduced clinical outcomes. Therefore, it is important to identify variables associated with patients’ knowledge (and related issues surrounding memory and learning) so strategies can be directed towards improving acquisition and retention of information among patients at risk of misunderstanding and/or forgetting treatment.

Factors Associated with Knowledge, Memory and Learning

Of studies investigating factors associated with patient knowledge, the majority assessed knowledge concerning a range of health issues (e.g., diagnosis, symptoms, prognosis, tests required); hence it is unclear whether such findings generalise to the more specific issues of treatment knowledge per se. The following section describes relationships between variables theoretically expected to share associations with patient knowledge and related constructs of learning and memory (i.e., cognitive abilities necessary for knowledge acquisition and retention).
Specifically, the role of age, education, intelligence, anxiety and psychotropic medication will be examined.

**Age, Education and Intelligence**

The relationship between age and knowledge shows an inconsistent association. Some studies reported a negative relationship between increased age and amount of information recalled (Anderson et al., 1979; Croquelois & Bogousslavsky, 2006; Ellis et al., 1979; Joyce et al., 1969; Kronmüller et al., 2006; Ley et al., 1976; McPherson, Smith, Powers, & Zuckerman, 2008; Surawy, 1989; Westra et al., 2004), while several studies reported no significant association (Brody, 1980; Flocke & Stange, 2004; Kayaniyil et al., 2009; Kerzman et al., 2005; Kortman, 1992; Lukoschek et al., 2003; Rees et al., 2003) and another found an age effect such that older patients knew more than their younger counterparts (Ley & Spelman, 1965).

Education level has often been associated with patients’ knowledge, with more educated patients recalling or understanding more information (Beeney, Dunn, & Welch, 1994; Bertakis, 1977; Ho et al., 2003; Kayaniyil et al., 2009; Kronmüller et al., 2006; Lukoschek et al., 2003; Miller et al., 2003; Reid et al., 1995; Scherer & Bruce, 2001; Westra et al., 2004; Yeh, Sung, Yorker, Sun, & Kuo, 2008) although others failed to replicate this association (Flocke & Stange, 2004; Kerzman et al., 2005; McPherson et al., 2008). Of the few studies examining the relationship between intelligence and patient knowledge, higher intelligence was related to greater patient knowledge (Beeney et al., 1994; Ley, 1988; Ley & Spelman, 1965; Reid et al., 1995).
Anxiety

Mixed finding have been observed for the relationship between patient anxiety and information recalled. Ley and Spelman (1965) reported a Yerkes-Dodson-type inverted U-shape relationship between anxiety and recall of medical information, where patients with moderate levels of anxiety remembered more than those with higher or lower anxiety. In contrast, a positive linear relationship between anxiety and recall was observed by Anderson et al. (1979), indicating patients with low anxiety recalled less information. The above patients were medical patients rather than patients with anxiety disorders, the latter of which often exhibit clinically higher levels of trait anxiety.

Among patients with anxiety disorders, attention and concentration (cognitive processes essential for memory and learning) are negatively affected (Barbee, 1993) and result in inefficiencies in the acquisition and retention of treatment knowledge. Adults with Panic-Ag scored significantly lower on measures of verbal learning and verbal and visual recall than non-anxious controls (Asmundson et al., 1994; Lucas et al., 1991), although another study failed to replicate this effect (Gladsjo et al., 1998). Such discrepant findings may be attributable to differences in neuropsychological tests, sample sizes and medication status. Medication status is important as some psychotropic medications, notably benzodiazepines, are associated with deficits in memory and learning as described below.

Psychotropic Medication

Pharmacological treatment guidelines for Panic-Ag have shifted away from using benzodiazepines and instead recommend antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs), as first-line pharmacological interventions
(American Psychiatric Association, 1998). Despite these changes in prescribing recommendations, Bruce et al. (2003) reported benzodiazepines are still the most commonly prescribed medication for Panic-Ag. The effects of benzodiazepines and antidepressants on cognitive functioning will now be discussed.

**Benzodiazepines**

Impairment in cognitive functioning associated with benzodiazepine use has been documented in numerous studies (see Barker, Jackson, Greenwood, & Crowe, 2003, for a review). Benzodiazepines may cause *anterograde amnesia*, that is, reduced memory for events occurring after administration of the drug (Ashton, 1995). Mintzer et al. (2001) postulated that benzodiazepines cause impairments in episodic memory (memory for personally experienced events), specifically at the level of encoding. Thus, memory is impaired for information presented to patients while affected by the drug, even when tested under drug-free conditions.

Of particular clinical importance, benzodiazepine use has been shown to affect patients’ learning and memory of information presented during CBT. Westra et al. (2004) compared 16 daily benzodiazepine using Panic-Ag patients with 16 age- and education-matched non-medicated Panic-Ag patients on memory of psychoeducation information regarding the development and treatment of Panic-Ag. Benzodiazepine users recalled significantly less information relative to their non-medicated counterparts (58% vs. 78%, *p* < .05); furthermore, benzodiazepine status was a stronger predictor of memory performance than education and age, accounting for 38% of variance. Thus, benzodiazepine use appears to adversely affect memory and recall of treatment knowledge in patients.
Antidepressants

Contrary to the case of benzodiazepines, minimal evidence suggests antidepressant use contributes to cognitive impairment. Antidepressants with the highest anticholinergic side-effect profile, namely tricyclics (Noble & Benfield, 1997), have been found to negatively impact memory performance after acute initial administration (Naudon, Hotte, & Jay, 2007), however tolerance to such effects tends to develop within 1 to 3 weeks of use (Danion, 1993). In contrast, SSRIs are less cognitively impairing than tricyclics (van Laar et al., 2002). One study reported a significant negative effect of paroxetine relative to placebo on a delayed recall word list task using healthy volunteers taking the drug for 2 weeks (Schmidt, Kruizinga, & Riedel, 2001); however, the authors noted the effect was weak (words recalled: paroxetine = 11.0, placebo = 11.6, p < .05), likely due to paroxetine’s anticholinergic effects and may be clinically relevant only for elderly individuals more sensitive to anticholinergic induced memory impairment and slower to develop tolerance to such effects (Danion, 1993).

In summary, although inconsistent findings exist, adults who are older, less educated, of lower intellect, highly anxious and/or benzodiazepine users during treatment appear at increased risk of experiencing difficulties in acquiring and/or retaining treatment information. From a clinical perspective, assuming a causal relationship between treatment knowledge and outcome exists, these factors could flag patients who would benefit from receiving additional interventions to enhance treatment knowledge.
Relationship Between Treatment Knowledge and Clinical Outcomes

Within the literature, clinical outcomes refer either to (a) treatment compliance, or (b) treatment outcome (symptomatic improvement). Interpretation of research examining relationships between patient knowledge and outcomes is hampered by inconsistencies in definitions and assessment of patient knowledge. Most studies do not purely focus on treatment knowledge but include other related aspects of knowledge (e.g., aetiology, prognosis, Ho et al., 2003; Scherer & Bruce, 2001; Watkins et al., 1967). Ley (1988) argued that specific aspects of knowledge should theoretically hold different relationships with outcome. For example, knowledge of the treatment regimen for hypertension is a prerequisite for medication compliance and blood pressure control. Knowledge of the illness may also affect medication compliance (and therefore blood pressure control) but it is not as necessary as knowing how many tablets to take. Studies incorporating other aspects of knowledge are therefore likely to underestimate the true relationship between treatment knowledge and clinical outcomes. Bearing these limitations in mind, research on this issue will now be reviewed. Studies examining the relationship with treatment compliance will be discussed first followed by studies comparing patient knowledge with treatment outcome.

Treatment Compliance

Ley (1988) reviewed 16 studies published between 1967 and 1986 examining the relationship between knowledge and compliance in patients with medical disorders. He categorised studies into those assessing patients’ understanding (14 studies) and those assessing memory (two studies). Correlations between
understanding and compliance ranged from .02 (German, Klein, McPhee, & Smith, 1982) to .73 (Parkin et al., 1976), with a mean of .36. Variations in patient disease characteristics, demographics and scope of patient knowledge assessed are likely to explain the discrepant findings. The relationship between memory and compliance was slightly lower with a mean correlation of .29.

Since Ley’s (1988) review, significant positive associations between knowledge and compliance have been reported by other researchers in diabetes (Barth et al., 1991; Kravitz et al., 1993), hypertension (Kim et al., 2007), heart failure (Ni et al., 1999), asthma (Kolbe, Vamos, Fergusson, Elkind, & Garrett, 1996; Ngamvitroj & Kang, 2007), HIV (Miller et al., 2003; Weiss et al., 2003) and depression (Yeh et al., 2008). Knowledge was typically assessed via questionnaires (multiple-choice or true/false formats) although some studies interviewed patients (Kravitz et al., 1993; Miller et al., 2003). Where correlations were provided, the magnitude of coefficients were similar to the average reported by Ley (1988). For example, correlations of .38 and .33 (Barth et al., 1991; Ni et al., 1999, respectively). However, other researchers failed to find significant associations between knowledge and compliance (Ho et al., 2003; Lee et al., 1992; Sands & Holman, 1985; Scherer & Bruce, 2001) for reasons comparable to those cited above, namely differing disease populations, patient characteristics, illness severity and extent of knowledge examined.

Within the psychotherapy literature, significant positive correlations between patients’ treatment knowledge and compliance have also been reported. In a study involving 28 patients with Obsessive Compulsive Disorder (OCD), Abramowitz and colleagues (Abramowitz et al., 2002) found patients’ understanding of the treatment rationale for exposure/response prevention correlated .57 with clinician ratings of in-
session treatment compliance and .34 with homework compliance. More recently, among 61 depressed inpatients, Kronmüller et al. (2006) reported higher treatment knowledge for depression was associated with increased self-rated proactive problem-oriented coping ($r = .41, p < .001$). Thus, evidence to date suggests patient knowledge (specifically treatment knowledge) is related to compliance behaviour in psychotherapy, although findings are mixed for medical disorders.

**Treatment Outcome**

Within the medical literature, significant associations between patients’ treatment knowledge and outcome have been found, although other studies reported non-significant findings. The discrepant results appear due to differences in aspects of knowledge examined (treatment knowledge vs. illness knowledge), knowledge assessment procedures (questionnaires vs. interview) and psychometric properties of knowledge instruments, as illustrated in the fields of diabetes and asthma.

McPherson et al. (2008) recently interviewed 44 diabetic patients and found greater treatment knowledge was strongly associated with better glycemic control ($r = -.61, p < .001$, lower score reflects better glycemic control) and accounted for 40% of the variation in blood glucose levels. Using a diabetes knowledge questionnaire, Meadows et al. (1988) reported patients with good metabolic control ($n = 17$) had greater treatment knowledge than those with poor control ($n = 38$; 61.0 vs. 52.3, $p < .01$), and Surawy (1989) replicated this association using the same questionnaire in 25 patients, finding a correlation of -.55 between overall treatment knowledge and metabolic control.

However, using a different measure, the Diabetes Knowledge Questionnaire (Dunn et al., 1984), other researchers failed to find significant relationships (Chan &
Molassiotis, 1999; Coates & Boore, 1996; Dunn, Beeney, Hoskins, & Turtle, 1990). Ceiling effects were observed for the knowledge measure used in these three studies indicating a lack of sensitivity in differentiating between levels of patient knowledge (Beeney et al., 1994; Coates & Boore, 1996), thus reducing its clinical and theoretical usefulness.

In contrast, two early studies found negative relationships whereby patients with poor control had greater knowledge of the disease (Watkins et al., 1967; Williams, Martin, Hogan, Watkins, & Ellis, 1967). A possible explanation for this unexpected result is that patients with poor control may have had a greater number of associated problems requiring additional interventions, staff contact and diabetes education, resulting in their acquiring more knowledge, yet other factors contributed to poor metabolic control (Williams et al., 1967). When examining the relationship between treatment knowledge and outcome in diabetes, controlling for number of complications would be useful to clarify the association.

Within the asthma literature, Soriano et al. (2004) interviewed asthma patients regarding their treatment knowledge and found those with poor asthma control \( (n = 179) \) had significantly less knowledge than those with good control \( (n = 252) \) (77.4 vs. 93.6, \( p < .01 \)). However, other researchers using asthma knowledge questionnaires (Ho et al., 2003; Scherer & Bruce, 2001) failed to replicate this relationship with a range of outcome measures (asthma attack frequency, emergency department visits, hospitalisations). However, the reliability of the knowledge scale used by Ho et al. (2003) was low, thus undermining their conclusions. Furthermore, items from these asthma knowledge questionnaires incorporated non-treatment related knowledge (prevalence, aetiology, symptoms). For example, an item from the asthma knowledge questionnaire used by Scherer and Bruce (2001) was, “The
number of people with asthma in the United States is approximately ______.” Such
items provide a diluted measure of treatment knowledge, thereby weakening its
association with outcome.

In the psychotherapy domain, to date only Abramowitz and colleagues
(2002), in their study of 28 OCD patients described above, investigated the
relationship between treatment knowledge and outcome. Controlling for pretreatment
symptom severity, comprehension of the treatment rationale correlated -.65 with
treatment outcome ($p < .01$), indicating patients demonstrating greater understanding
of the rationale had less severe symptoms at posttreatment. Moreover,
comprehension of the treatment rationale was the strongest predictor of treatment
outcome (in comparison to in-session and homework compliance ratings).

In summary, studies from the medical and psychotherapy literature have
found patients’ knowledge of treatment is related to clinical outcomes, whereby
greater knowledge is associated with increased compliance and improved treatment
outcome. Although conflicting findings abound, studies confining their examination
to *treatment* knowledge (Abramowitz et al., 2002; Kronmüller et al., 2006; Meadows
et al., 1988; Surawy, 1989) tended to yield higher correlations with compliance and
outcome than those including other aspects of patient knowledge (Ho et al., 2003;
Scherer & Bruce, 2001; Watkins et al., 1967). However, as discussed below, the
majority of studies have substantive methodological problems regarding the
measurement of knowledge, rendering conclusions regarding the true relationship
unclear.
Methodological Limitations

Brown (1990) described the measurement of knowledge in the diabetes literature as being “seriously flawed” (p. 57) with few studies using measures with demonstrated validity and reliability. Such concerns are equally applicable to the assessment of knowledge within the wider range of medical and psychiatric disorders, with numerous studies utilising unvalidated measures of patient knowledge to examine relationships with treatment outcome in the fields of congestive heart failure (Ni et al., 1999), diabetes (Barth et al., 1991; Heisler et al., 2002), hypertension (Sands & Holman, 1985), HIV (Miller et al., 2003; Weiss et al., 2003), renal patients (Durose, Holdsworth, Watson, & Przygrodzka, 2004), patients on lithium therapy (Lee et al., 1992) and OCD (Abramowitz et al., 2002). Although it is possible that patient knowledge of treatment is not related to expected clinical outcomes, the lack of robust correlations may in part be due to the psychometrically unsound knowledge assessment instruments utilised in these studies.

Another important methodological problem previously alluded to concerns the content of knowledge measures; many scales combine items assessing treatment knowledge with other information regarding symptoms, aetiology and illness prevalence (Ho et al., 2003; Scherer & Bruce, 2001; Wigal et al., 1993) thereby diluting the measure of treatment knowledge. As argued by Ley (1988), these other aspects of knowledge theoretically hold weaker relationships with outcome as they are not essential for treatment compliance. Therefore, treatment knowledge needs to be separated from other aspects of illness information when investigating the relationship between treatment knowledge and outcome.

Providing an objective assessment of patients’ knowledge is also essential when examining the relationship between knowledge and outcome. The impressive
negative correlation of -.65 reported in OCD patients (Abramowitz et al., 2002) must be interpreted with caution. Not only were patients’ comprehension of treatment rationale rated on a single-item Likert-type scale (ranging from 0 = poor to 6 = outstanding) with no information provided on the validity or reliability of either the measure or its scoring system, but also such ratings were made by the patient’s therapist following the final treatment session. Hence, these were non-blind ratings potentially prone to therapist-bias. For example, improvements shown by patients over the course of treatment may have inadvertently influenced therapists to conclude such patients had greater understanding of the rationale than those showing less change, resulting in an inflated association between treatment knowledge and outcome. Despite these limitations, should such an association exist, the clinical implications for treatment delivery are significant.

On the basis of the studies reviewed above, deficits in patients’ treatment knowledge have been identified as a factor that can substantially hinder involvement in therapy. In clinical practice, both patients and clinicians may believe patients understand treatment and its rationale, when in actuality they do not. This phenomenon has been described as an “illusion of knowing” (Glenberg, Wilkinson, & Epstein, 1982); an individual’s belief that comprehension has been attained when in fact it has not. If ignored, such an illusion may represent a major obstacle to patients effectively learning and applying treatment techniques, which in turn could impair treatment outcome. To counteract this illusion, clinicians are recommended to regularly assess patients’ treatment knowledge and correct areas of confusion or misunderstanding.
Summary

Research data indicate that patients often forget and/or misunderstand much of what they have been told about treatment. Factors associated with poorer treatment knowledge include older age, less education, lower intelligence, greater anxiety and benzodiazepine use. While not specifically examined in relation to CBT for Panic-Ag, studies investigating relationships between patients’ treatment knowledge and clinical outcomes have produced conflicting and inconclusive results. Several studies suggest increased knowledge is associated with better treatment compliance and outcome, with a greater number failing to demonstrate such relationships. Variations in definitions and scope of knowledge, disorders, patient populations, severity levels and clinical outcome variables contribute to the inconsistent findings reported.

Conclusions regarding the true relationship between treatment knowledge and outcome have also been hampered by the use of knowledge measures with poor or unknown psychometric properties, often containing items assessing information extraneous to treatment, or that rely on subjective ratings of patient knowledge. Hence, one aim of this thesis is to extend the existing literature by developing a valid, reliable, sensitive and objective measure of patients’ knowledge of CBT for Panic-Ag to more accurately determine whether greater treatment knowledge is associated with improved outcome in this patient group. If such a relationship is present, clinicians are recommended to presume patients’ understanding of treatment is imperfect and apply steps to ensure adequate comprehension.

However, even with such knowledge enhancing strategies, compliance rates in medical disorders are estimated to increase from 50% to only 66% to 76% (Ley, 1986). Furthermore, the magnitude of the relationship between treatment knowledge
and outcome is only moderate at best. Therefore, treatment knowledge is a necessary but not sufficient factor for treatment compliance and successful outcome. Patients may choose not to comply with recommendations despite full comprehension of treatment information.

Horne (1999) asserted patients’ beliefs about treatment are the “hidden determinant of treatment outcome” (p. 491). Prior to commencing therapy, patients hold an abundance of pre-existing beliefs regarding a range of issues that impact on their willingness to engage in therapy. These beliefs are based on personal experiences as well as information from family and friends, the media and other health practitioners (Leventhal, Diefenbach, & Leventhal, 1992). Clinicians’ treatment recommendations must compete with these beliefs and if discrepant, such advice may be dismissed (Donovan, Blake, & Fleming, 1989), contributing to reduced treatment outcomes (Horne, 1999).

Even with sound understanding of treatment and its rationale, patients may not accept it as relevant to their problem, hold low expectations of its helpfulness and/or lack confidence in implementing treatment procedures dictated. Indeed, patients’ acceptance of the treatment rationale, expectancies of treatment outcome and self-efficacy to implement therapy have been identified as specific treatment beliefs critical to the success of any therapy program. Commencing with acceptance of the treatment rationale, the next sections will discuss the clinical importance of these beliefs and review their relationships with treatment outcome.
Acceptance of the Treatment Rationale

Definition

Acceptance of the treatment rationale is defined as the extent to which individuals believe or agree the rationale for treatment (including the aetiology of the problem and treatment procedures) is relevant and helpful to their problems (Addis & Carpenter, 2000; Fennell & Teasdale, 1987). Acceptance of the rationale has been assessed in numerous ways, for example asking patients, “To what extent does the treatment you are receiving match with your ideas of what helps people in psychotherapy?” (Addis & Jacobson, 2000). In addition, independent judges have rated patient responses to the rationale. For example, the response, “It’s like a mirror. Now I understand it, I can work on it,” reflects a high degree of acceptance (Fennell & Teasdale, 1987, p. 264). Treatment credibility, on the other hand, reflects patients’ perceptions of how logical treatment appears (Kazdin, 1979). For instance, an item from Borkovec and Nau’s (1972) treatment credibility scale is, “How logical does this treatment seem to you?”

Acceptance of the treatment rationale differs from rationale credibility as, although they are likely to be positively associated, it is conceivable that a rationale regarded as logical may simultaneously be poorly accepted. To illustrate this point, a doctor may provide a highly cogent explanation of how imbalances in serotonin level cause and maintain depression, thus requiring prescription of serotonin reuptake inhibitors. The patient, while perceiving the treatment rationale to be logical, may still reject it if it is incompatible with his/her ideas of what caused the problem and what is needed to achieve recovery. Instead, the patient may believe negative childhood experiences are at the root of his/her psychological distress, hence treatment should involve discussion of childhood issues. Items assessing acceptance
of the rationale typically have a more personal focus in contrast to credibility ratings (e.g., “Antidepressants are an effective treatment in general” vs. “Antidepressants are an effective treatment for me”) (Addis & Carpenter, 1999).

**Importance of the Treatment Rationale**

The treatment rationale, although widely differing in content, is considered to play a central role in all psychotherapies, including CBT. Frank (1982) referred to the treatment rationale as a conceptual scheme or “myth” that attempts to provide a persuasive explanation to account for and explain the patient’s distressing symptoms. The rationale also sets the stage for prescribing specific treatment procedures or “rituals” designed to promote recovery (Addis & Carpenter, 2000). Beck and colleagues ascribed considerable importance to the presentation of a convincing treatment rationale early in CBT to encourage treatment compliance (Beck, Rush, Shaw, & Emery, 1979). Indeed, withholding the rationale for treatment is associated with decreased acceptability of and willingness to use therapeutic instructions (Lee, Uhlemann, & Wikman, 1994) and reduced treatment change (Oliveau, Agras, Leitenberg, Moore, & Wright, 1969).

A credible treatment rationale has the potential to heighten positive expectations regarding benefits of therapy, with the latter being related to positive treatment outcomes (as discussed in the next section). Nau, Caputo and Borkovec (1974) empirically demonstrated that rationale credibility was related to expectancy of improvement across three studies using snake-fearful adults (study 1: \( N = 49 \); study 2: \( N = 18 \); study 3: \( N = 86 \)). Participants were presented with a rationale for one of several treatment procedures (including systematic desensitisation and placebo procedures). They were instructed to rate the credibility of treatment
rationales and role-play expected treatment effects during an approach test after imagining they received 5 weeks of that therapy. That is, their simulated response to treatment served as a behavioural measure of expectancy of treatment improvement. Credibility ratings for the total group correlated significantly with participants’ simulated treatment response for all three experiments ($r = .30 – .60$, $p < .02$).

Regarding treatment outcome however, perceived credibility of the treatment rationale holds an inconsistent relationship. Morrison and Shapiro (1987) found patients’ credibility ratings correlated significantly with overall clinical improvement for 40 patients receiving CBT or relationship-oriented therapy for depression or anxiety. However, using the same scale, other researchers failed to identify rationale credibility as a significant predictor of outcome in patients with Panic-Ag (Ramnerö & Öst, 2004) and Generalised Anxiety Disorder (Borkovec & Costello, 1993; Borkovec & Matthews, 1988). Differences in diagnoses, therapies and temporal administration of measures may account for the conflicting findings.

As noted, the mere presentation of a credible rationale does not guarantee all patients will accept it. While rationale credibility holds an inconsistent relationship with outcome, research suggests acceptance of the rationale is more strongly related to patients’ aetiological beliefs and clinical outcomes.

**Relationship Between Acceptance of the Treatment Rationale and Clinical Outcomes**

For the treatment rationale to be accepted it must be congruent with patients’ pre-existing beliefs and conceptualisations of their symptoms (Butler & Strupp, 1986). Highlighting this, in a sample of 51 non-clinical individuals, Addis and Carpenter (1999) found the more reasons offered for depression contrary to an
action-oriented treatment rationale, the less accepting patients were of that rationale \((r = -.31, p < .05)\). Negative reactions to the rationale are not uncommon, with 58% of patients voicing disagreements or doubts after being probed for their opinion (Addis & Carpenter, 2000). Theoretically, patients are unlikely to remain in treatment or benefit from psychotherapy if the treatment rationale is inconsistent with their beliefs about what should occur during therapy (Connor-Greene, 1993).

**Treatment Attrition**

Studies of different psychiatric disorders have linked poor acceptance of the treatment rationale with attrition. Davis and Addis (2002) examined predictors of dropout from an outpatient behavioural medicine program for 118 patients presenting with stress, insomnia or pain. Patients were classified as early dropouts \((n = 37)\), late dropouts \((n = 22)\) or treatment completers \((n = 59)\) according to number of sessions completed. Of predictor variables investigated (including physical and mental health functioning, self-efficacy expectations and treatment outcome expectations), agreement with the treatment rationale was the strongest predictor of treatment completion status, with early dropouts reporting significantly less acceptance of the rationale \((M = 3.93, SD = .83)\) than completers \((M = 4.55, SD = .52, p < .001)\). Consistent results were obtained by Hofmann and Suvak (2006) in the area of Social Phobia. These authors reported that dropouts \((n = 34)\) from a CBT group program endorsed the treatment rationale significantly less positively than treatment completers \((n = 99)\) despite not differing on demographic variables, Axis I or Axis II psychopathology.

In a similar vein, Elkin et al. (1999) found lower attrition when patients receive treatment congruent with their beliefs regarding the aetiology and treatment
of their problem. Prior to treatment, 82 patients with major depression completed a questionnaire assessing belief in different causes of depression (e.g., being very self-critical, biochemical problems) and the perceived helpfulness of different treatment approaches (e.g., learning more realistic attitudes about oneself and the world, medication) to ascertain treatment preference for psychotherapy or pharmacotherapy. Patients were randomly assigned to receive either psychotherapy (CBT or interpersonal therapy) or medication. Forty patients received their preferred treatment option (congruent group) and 42 received their non-preferred treatment (non-congruent group). Attrition rates were significantly lower in the congruent compared to non-congruent group (5% vs. 21%, \( p < .05 \)). Similarly, Foulks,Persons and Merkel (1986) found disagreement between patients’ aetiological beliefs and a biopsychosocial rationale for depression was associated with poorer therapy attendance and premature termination among 60 psychiatric outpatients.

These studies suggest patients are at increased risk of dropping out of treatment if the treatment rationale is poorly accepted, particularly if it is incompatible with patients’ pre-existing aetiological beliefs. Evidence also suggests patients’ acceptance of the treatment rationale is associated with outcome; however, surprisingly, no study has examined this in CBT for Panic-Ag.

**Treatment Outcome**

From a conceptual perspective, acceptance of the treatment rationale should promote stronger engagement with therapy, positive expectations of improvement and increased treatment compliance, which in turn should improve treatment outcomes. A pivotal study, in that it was the first to highlight the importance of patients’ acceptance of the treatment rationale in influencing outcome, and also set
the foundation for subsequent studies, was conducted by Fennell and Teasdale (1987). These authors attempted to understand factors contributing to individual differences in response to CBT for depression. In their study, 15 patients with depression were instructed to read a booklet concerning principles of cognitive therapy for depression following their first therapy session. Responses to the booklet (recorded at the beginning of Session 2) were rated by independent judges on the extent to which they accepted the treatment rationale. Consistent with the conceptual model, greater acceptance of the rationale was associated with greater compliance with treatment recommendations assigned in Session 2, and was highly predictive of treatment outcome, correlating significantly with independent observer ratings of depression at posttreatment ($r = -.76, p < .01$) and 6-month follow-up ($r = -.65, p < .02$). The same trend was observed at 1-year follow-up ($r = -.52, p < .1$) but this failed to reach significance. However, with a sample size of only 15, these high correlations may have reflected an early trend in the data which may not generalise to the larger population of depressed patients (Tversky & Kahneman, 1971). As sampling error increases with decreasing sample size, replication with a larger sample is clearly required.

Using a larger sample, Addis and Jacobson (1996) partially replicated the findings of Fennell and Teasdale (1987) in 98 depressed patients randomly allocated to receive cognitive therapy ($n = 48$) or behavioural activation ($n = 50$). Ratings of acceptance of the treatment rationale were combined with ratings of homework helpfulness to form a composite of “treatment helpfulness”. Controlling for pretreatment depression, the authors found Session 2 ratings of treatment helpfulness significantly predicted posttreatment outcome for patients receiving behavioural activation ($r = .47, p < .01$) but not cognitive therapy ($r = .17, p > .05$). As
acceptance ratings were combined with ratings of homework helpfulness, the true association between acceptance of the rationale and outcome remains unclear.

Strengthening the results of the previous two studies, Addis and Jacobson (2000) obtained similar findings using a purer measure of acceptance of the treatment rationale in a larger sample. In a sample of 150 patients undergoing CBT for depression, acceptance of the rationale assessed across the first three sessions correlated significantly with treatment outcome ($r = .35, p < .05$), and early- and mid-homework compliance ($r = .18, p < .05$; $r = .17, p < .05$, respectively). Interestingly, mediational analyses demonstrated acceptance of the treatment rationale had an independent direct relationship with clinical outcome rather than simply through encouraging increased homework compliance, arguing in favour of non-specific effects influencing outcome.

In summary, consistent findings from independent lines of research using different methodologies suggest that poor acceptance of the treatment rationale is associated with reduced therapeutic outcome in patients with depression. However, such a conclusion is weakened by methodological limitations inherent in studies and it remains unclear whether the same relationship exists for patients with Panic-Ag.

**Methodological Limitations**

An important methodological difficulty concerns the use of measures with unknown psychometric properties. Addis and Jacobson (1996, 2000) relied on single-item measures to assess acceptance of the treatment rationale. The reliability and validity of these scales were not reported, raising questions about their psychometric properties. Moreover, single-item scales are unreliable as they are associated with substantial random error, do not permit estimation of important
reliability information (e.g., internal consistency) and the magnitude of reliability is found to increase with the number of items (Loo, 2002; Nunnally, 1967). In addition, the validity of single-item scales is often poor, as it is difficult to depict complex constructs with a single item (Nunnally, 1967). As noted earlier, one study (Addis & Jacobson, 1996) combined rationale acceptance and homework helpfulness ratings, thereby potentially obscuring the true association between acceptance of the rationale and outcome.

A further problem involves the temporal assessment of acceptance of the rationale. The studies reviewed above assessed acceptance of the rationale within the first three sessions and compared it with treatment outcome. However, CBT typically involves numerous treatment components introduced to patients gradually over the course of therapy. For example, CBT for Panic Ag typically involves an introduction to the cognitive model and presentation of psychoeducation material during the first two sessions, with the rationale for cognitive therapy, behavioural experiments, in vivo exposure and interoceptive exposure unfolding over sessions three to six. Comparing treatment outcome with acceptance of the rationale before it has been fully described seems premature. Indeed, Addis and Jacobson (1996) observed differential relationships between acceptance of the rationale and outcome for patients allocated to behavioural activation or cognitive therapy. They speculated the relationship with behavioural activation was stronger as, unlike cognitive therapy, it utilised the same treatment approach consistently throughout treatment.

This thesis therefore aims to address these methodological weaknesses by developing a psychometrically sound measure comprising items focussed purely on acceptance of the treatment rationale for CBT in Panic-Ag. In this way, the relationship between acceptance of the rationale and outcome can be assessed more accurately. This
measure will be administered to patients both before and after CBT to determine whether pretreatment and posttreatment beliefs about the rationale are associated with treatment improvement.

**Clinical Implications**

Based on the above, clinicians are advised to encourage discussion of the treatment rationale regularly throughout treatment to elicit patient reactions contributing to its non-acceptance to minimise early termination and improve outcomes (Addis & Carpenter, 2000; Davis & Addis, 2002; Elkin et al., 1999; Iselin & Addis, 2003; Van Audenhove & Vertommen, 2000). Indeed, exploring patients’ beliefs about the treatment rationale improves compliance, the latter being related to outcome (Kazantzis et al., 2000). Worthington (1986) found exploring client attitudes about therapy recommendations significantly predicted subsequent treatment compliance among 61 counselling clients, while merely stressing its importance did not. Similarly, in 26 depressed patients undergoing cognitive therapy, Bryant, Simons and Thase (1999) found significantly greater treatment compliance when therapists elicited patients’ perceptions regarding treatment.

However, for patients who still fail to accept the rationale despite efforts to explore and address their reservations, matching treatment with their preferred alternative may be an option. Some studies in the field of depression found matching treatment preferences with treatment type contributed to improved patient outcome relative to receiving a randomised (Chilvers et al., 2001) or non-preferred treatment (Lin et al., 2005). However, other studies found no advantage for treatment matching relative to randomisation in patients with depression (e.g., Bedi et al., 2000; Elkin et
al., 1999). For patients with Panic-Ag, Bakker and colleagues (Bakker, Spinhoven, van Balkom, Vleugel, & van Dyck, 2000) compared patients treated with cognitive therapy through preference \( (n = 31) \) or randomisation \( (n = 35) \) and found no difference in outcome; however no studies have examined the impact of non-preferred therapy on outcome in Panic-Ag. Devine and Fernald (1973) specifically examined this issue among 32 snake phobics and found outcomes were reduced when phobic individuals were treated with therapies they strongly disliked \( (p < .01) \).

**Summary**

The treatment rationale serves a fundamental role in therapy through attempting to provide patients with an explanation of the cause of their problem and its solution. Patients vary in terms of their acceptance of the treatment rationale, which is often dependent upon its compatibility with pre-existing conceptualisations of their problem. Several studies have linked poor acceptance of the rationale with premature dropout and poorer outcome in CBT, suggesting clinicians should either discuss patients’ reservations about the rationale during therapy or offer preferred treatments. However, methodological problems surrounding the use of psychometrically unsound measures and timing of assessments cast doubt on the validity of this association, and it remains unclear whether the same relationship exists for patients with Panic-Ag. The present thesis aims to address the methodological problems inherent in the assessment of patients’ acceptance of the rationale such that its association with treatment improvement following CBT for Panic-Ag can then be examined.

Acceptance of the treatment rationale does not guarantee patients will believe treatment will be effective for them. Patients may strongly agree with the rationale
behind CBT yet not believe it will improve their symptoms. This latter treatment belief, often referred to as expectancies of treatment outcome, has frequently been linked to patient improvement in numerous clinical populations including Panic-Ag.

**Expectancies of Treatment Outcome**

**Definition**

Patients’ belief that therapy will be helpful and lead to improvement has been the focus of investigation for over 50 years (Arnkoff, Glass, & Shapiro, 2002). Also referred to as treatment outcome expectancy or expectation for therapeutic gain, patients’ beliefs about treatment outcome are prognostic beliefs that therapy will lead to change (Greenberg, Constantino, & Bruce, 2006). Therapy expectancies can be either positive (the belief that treatment will be helpful), negative (a lack of confidence that therapy will result in improvement) or ambivalent (conflicting feelings regarding the value of therapy) (Lipkin, 1954).

Treatment outcome expectancies have been measured in various ways, including, “How much do you really feel that therapy will help you to reduce your symptoms?” (Devilly & Borkovec, 2000), “How confident are you that this treatment will eliminate your fear of _____?” (Holt & Heimberg, 1990), and “How do you think you will feel at the end of treatment compared to how you feel now?” (Hansson & Berglund, 1987). Such expectancies must be distinguished from other types of expectancies. Recently, Dozois and Westra (2005) developed the Anxiety Change Expectancy Scale (ACES) to assess *agency expectancy*, a related yet different concept examining individuals’ beliefs that they are capable of change (e.g., “I feel
pessimistic that my anxiety problems could ever change for the better”), rather than focussing on expectancies concerning improvement from treatment as in the first three items listed above.

Treatment outcome expectancy should also be differentiated from similar concepts of treatment credibility and motivation. Kazdin (1979) defined treatment credibility as “how believable, convincing, and logical the treatment is” whereas treatment expectancy refers to “improvements that clients believe will be achieved on the basis of a particular treatment” (p. 82). Credibility involves logical and rational thought processes, while expectancy is related to emotional processes. What patients logically think is the case may differ from what is felt to be the case. For example, patients might believe their treatment is highly logical and credible, yet implicitly feel it will not be helpful for them (Devilly & Borkovec, 2000). Highlighting this distinction, Borkovec and Matthews (1988) found treatment expectancy and credibility ratings demonstrated different relationships with treatment outcome in 30 non-phobic anxiety patients. Expectancy ratings correlated with 13 of 10 outcome measures assessed at three time intervals (i.e., a total of 30 correlations), while ratings of credibility did not significantly correlate with any (i.e., 0 out of 30 such correlations).

Treatment motivation includes a desire to change, a commitment to attend appointments, and/or a willingness to participate in treatment and carry out homework assignments (Keijsers, Schaap, Hoogduin, Hoogsteyns, & de Kemp, 1999). However, as highlighted by Arknoff et al. (2002), although these concepts could be highly related to treatment expectancy ratings, patients can be motivated to engage in treatment but hold low expectations that therapy will be helpful.
Chapter 1 – Literature Review

**Historical Perspectives**

Research on expectancies of treatment outcomes dates back to a seminal article by Rosenthal and Frank (1956) highlighting the potential relevance of placebo effects in psychotherapy. These authors stated that “patients entering psychotherapy have various degrees of belief in its efficacy, and this may be an important factor in the results of therapy, but this has not been studied, to our knowledge” (p. 296). Frank, who went on to become a primary and influential proponent of treatment expectancies, asserted that positive expectations of outcome are more important than techniques specific to different psychotherapies (Frank, 1982). Furthermore, Kazdin and Wilcoxon (1976) argued for the importance of controlling for treatment outcome expectancies when determining if specific effects are responsible for improved outcomes.

The role of treatment outcome expectancies and the potential for researchers and clinicians alike to misattribute the cause of patient improvement to specific effects of therapy was highlighted in the classic work of Marcia, Rubin and Efran (1969). Forty-four snake and spider phobics received either systematic desensitisation, T-scope therapy (a bogus highly credible psychotherapy), or no treatment. Participants receiving T-scope therapy were told that images of feared stimuli would be presented tachistoscopically at subliminal levels, however they actually observed blank slides. Periodic shocks and false physiological feedback were also provided to enhance treatment credibility. Treatment expectancy for the T-scope condition was manipulated such that participants in the high-expectancy condition received the treatment, while low-expectancy participants were told a crucial aspect of treatment was missing so that no improvement could be expected. The results revealed no differences between the systematic desensitisation and high-
expectancy T-scope conditions on posttreatment measures. However, participants in these two conditions showed significantly more improvement than those receiving low-expectancy T-scope therapy or no treatment.

Weinberger and Eig (1999) classified patient outcome expectations as one of five factors responsible for outcome equivalence among the major classes of psychotherapy (the other factors being the therapeutic relationship, confronting problems, experience of mastery and attributions of improvement); however it was the only one not emphasised by any major school of psychotherapy. Often regarded as non-specific factors common to different therapies, treatment outcome expectancies have been conceptualised as “nuisance variables” to be excluded in order to investigate differences in improvement between specific brands of psychotherapy (Dozois & Westra, 2005). Perhaps as a result of this view, patient outcome expectancies have not been vigorously researched to date and have been referred to as the most “neglected” and “ignored” factor in psychotherapy research (Weinberger & Eig, 1999).

Highlighting the under-recognised role of outcome expectancies is the chronological trend of research investigating its relationship with treatment improvement in psychotherapy over the last five decades. Arnkoff et al. (2002) reviewed the psychotherapy literature and noted seven empirical studies were published between 1956 and 1963 (i.e., an average of one paper per year), while only eight were published during the 25-year period between 1965 and 1989 (i.e., an average of one paper approximately every three years), suggesting research interest in patient expectancies had waned. However, nine reports were published between 1990 and 2000 (i.e., an average of one paper every 1.2 years) and a growing collection of studies (Abouguendia, Joyce, Piper, & Ogrodniczuk, 2004; Davis &
Addis, 2002; Dozois & Westra, 2005; Goosens, Vlaeyen, Hidding, Kole-Snijders, & Evers, 2005; Greenberg et al., 2006; Joyce, Ogrodniczuk, Piper, & McCallum, 2003; Kenardy, McCafferty, & Rosa, 2003; Vogel, Hansen, Stiles, & Gotestam, 2006; Westra & Dozois, 2006; Westra, Dozois, & Marcus, 2007) have been published since Arnkoff et al.’s (2002) review indicating a renewed interest in the issue.

**Relationship Between Treatment Expectancies and Outcome**

Treatment outcome expectancies have frequently been associated with therapeutic improvement for a range of disorders, including borderline personality disorder (Antikainen, Koponen, Lehtonen, & Arstila, 1994), chronic pain (Goosens et al., 2005), complicated grief (Joyce et al., 2003), obesity (Bradley, Poser, & Johnson, 1980), social phobia (Chambless, Tran, & Glass, 1997; Safren, Heimberg, & Juster, 1997) and generalised anxiety disorder (Borkovec & Costello, 1993); however inconsistent findings are common (see Arnkoff et al., 2002; Noble, Douglas, & Newman, 2001, for reviews). Discrepant findings appear due to differing patient populations, symptom severity, treatment methods and outcome measures.

For Panic-Ag, the literature appears to suggest treatment outcome expectancies are generally predictive of improvement, irrespective of whether earlier or modern treatments and diagnostic criteria are applied. (Note that prior to the introduction of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III, American Psychiatric Association, 1980) Panic Disorder was termed *Agoraphobia with panic attacks*.)

Of five studies (Emmelkamp & Emmelkamp-Benner, 1975; Emmelkamp & Wessels, 1975; Mathews et al., 1976; Southworth & Kirsch, 1988; Stern & Marks, 1973) employing the more traditional habituation-based exposure paradigm (either in
vivo or imaginal) as the sole therapeutic component, three found significant findings (Emmelkamp & Emmelkamp-Benner, 1975; Mathews et al., 1976; Southworth & Kirsch, 1988). Mathews et al. (1976) examined 36 agoraphobic patients and found pretreatment expectancy ratings (defined as percent confidence they would eventually confront each of 15 feared situations) predicted a composite measure of treatment outcome (including behavioural avoidance tests and patient- and clinician ratings of severity). Likewise, Emmelkamp and Emmelkamp-Benner (1975) found scores on a pretreatment 3-item expectancy scale (where patients rated how much they expected to gain from treatment) significantly correlated with posttreatment patient and independent-observer ratings of phobic anxiety and avoidance in 29 agoraphobic patients.

Southworth and Kirsch (1988) manipulated treatment expectancies and found it impacted positively on outcome. Twenty agoraphobic patients were assigned to either a high or low expectancy group and were given 10 in vivo exposure sessions over 2 to 3 weeks. Patients in the high expectancy condition were informed they were receiving a treatment with demonstrated efficacy in reducing fear and avoidance for agoraphobia. In contrast, patients in the low expectancy group were told the 10 exposure sessions were conducted to collect a reliable baseline anxiety measure after which they would receive treatment. Patients in the high expectancy group showed greater and more rapid behavioural improvement than patients in the low expectancy group, although no differences between groups were observed on self-report measures of anxiety.

Two studies failed to obtain significant results, however they suffered from low sample sizes, reflecting inadequate power to detect relationships. For example, Emmelkamp and Wessels (1975) found a correlation of .38 with posttreatment
outcome (phobic anxiety and phobic avoidance) in a sample of 19 patients. Another report consisting of 16 patients also failed to find a significant relationship (Stern & Marks, 1973).

The introduction of the cognitive model (Clark, 1986) saw a fundamental shift in the theoretical understanding and treatment of Panic-Ag, with modern approaches incorporating cognitive elements designed to disconfirm feared catastrophes (Rayburn & Otto, 2003). Despite the wealth of evidence supporting the cognitive model, of two studies investigating contemporary CBT programs for Panic-Ag and treatment expectancies, both found positive associations. Clark et al. (1999) examined expectations of improvement as a predictor of treatment response to CBT for 43 patients with Panic-Ag. Controlling for pretreatment severity, expectancy ratings obtained at the end of the first session correlated significantly ($r = -.50, p < .01$) with posttreatment scores on a panic-anxiety composite (including patient self-report and assessor ratings, lower score reflects better outcome). Interestingly, none of the other predictor variables examined (e.g., depression, general anxiety, episode duration, treatment suitability) significantly correlated with outcome. Kenardy et al. (2003) found pretreatment outcome expectancy ratings of Internet-delivered CBT for Panic-Ag predicted posttreatment fear of bodily sensations ($r = -.59, p < .05$) and catastrophic cognitions ($r = -.64, p < .05$) but was not significantly associated with three other self-report outcome measures among 36 university students at risk of developing the disorder.

Hence, for more than 30 years, studies conducted by independent researchers found therapeutic outcome in Panic-Ag is predicted by positive expectations regarding the helpfulness of treatment. However, methodological problems exist
similar to those discussed in reference to treatment knowledge and acceptance of the treatment rationale, as described below.

**Methodological Limitations**

A serious methodological problem concerns the use of expectancy measures that combine items assessing treatment credibility with expectancy (Chambless et al., 1997; Goosens et al., 2005; Safren et al., 1997). The impact of treatment credibility needs to be disentangled from expectancy because treatment expectancy ratings tend to correlate more frequently and strongly with outcome than treatment credibility (Borkovec & Costello, 1993; Borkovec & Matthews, 1988; Devilly & Borkovec, 2000; Kenardy et al., 2003). As highlighted by Arnkoff et al. (2002), such combined measures no longer provide a “pure” assessment of treatment expectancy. Hence, scales including credibility items provide a diluted measure of outcome expectancy and its true relationship with outcome may be stronger than previously reported.

Another methodological weakness prevalent in the Panic-Ag and wider psychotherapy literature is the reliance on expectancy scales with minimal (Abouguendia et al., 2004) or no reported psychometric data (Clark et al., 1999; Emmelkamp & Emmelkamp-Benner, 1975; Hansson & Berglund, 1987; Joyce et al., 2003; Kenardy et al., 2003; Mathews et al., 1976; Stern & Marks, 1973). Several expectancy measures have been published, such as the Credibility/Expectancy Questionnaire (CEQ, Borkovec & Nau, 1972) and the Reaction to Treatment Questionnaire (RTQ, Holt & Heimberg, 1990). The 4-item CEQ contains three items assessing credibility of the treatment rationale and only one item assessing expectancy of success. The 13-item RTQ contains the same four items from the CEQ. These scales not only have the disadvantage of combining credibility and
expectancy ratings but their psychometric properties have not been established. However, a revised 6-item version of the CEQ (including an additional two expectancy items) demonstrated satisfactory internal consistency, test-retest reliability and some evidence of validity (Devilly & Borkovec, 2000) although it has not been used in studies investigating expectancy-outcome relationships in Panic-Ag. Other studies relied on single-item scales to assess outcome expectancy (Clark et al., 1999; Hansson & Berglund, 1987; Vogel et al., 2006), which, as previously discussed, are unreliable. Given the significance ascribed to patient expectancies of improvement and the potential theoretical and clinical implications stemming from its association with treatment outcome, the development of a valid and reliable measure of treatment expectancy is highly desirable.

Clinical Implications

Bearing the above limitations in mind, it has been asserted that “believing that one will feel better is enough to make one feel better” (Kirsch, 1990, p. 104). Despite its clinical appeal, such a conclusion is premature as causality cannot be inferred from correlational research. As articulated by Chambless et al. (1997), poor treatment expectancies could either cause patients to improve less or they may simply reflect patients’ accurate prediction that treatment would not be effective for them. However, Hansson and Berglund (1987) conducted a study using path analysis which suggested a causal association. Alternatively, an unknown third variable could be influencing both expectancies of improvement and treatment outcome.

If there is a causal relationship, the exact mechanisms through which treatment expectancies mediate change remains largely unknown. A number of researchers have suggested that positive expectancies promote greater engagement in
treatment and increased compliance with difficult CBT techniques such as exposure, thereby enhancing clinical outcomes (Arnkoff et al., 2002; Chambless et al., 1997). Such a hypothesis has not been tested and awaits further empirical investigation.

A further issue requiring clarification is whether treatment expectancies assessed *prior* to treatment are related to outcome. Most studies assessed patients’ expectancies either during the first two treatment sessions (Borkovec & Costello, 1993; Borkovec, Newman, Pincus, & Lytle, 2002; Chambless et al., 1997; Clark et al., 1999; Safren et al., 1997; Vogel et al., 2006) or prior to treatment but after the treatment rationale was presented (Emmelkamp & Emmelkamp-Benner, 1975; Emmelkamp & Wessels, 1975; Kenardy et al., 2003; Stern & Marks, 1973). However, patients are likely to have expectations regarding the helpfulness of treatment even before being introduced to it (Leventhal et al., 1992). It remains to be determined whether *pretreatment* expectancies are related to treatment improvement.

If pretreatment expectancies of improvement predict outcome, patients with negative pretreatment expectancies should receive preparatory counselling to discuss and restructure such beliefs. A similar approach was used by Westra and Dozois (2006) who found providing three sessions of motivational interviewing to patients with anxiety disorders prior to undergoing CBT increased their expectancies for anxiety control, treatment compliance and the number of treatment responders relative to patients who did not receive pretreatment interventions. Conversely, if pretreatment outcome expectancies were unrelated to clinical improvement it suggests clinicians have a window of opportunity early in treatment (e.g., during Session 1) to instil confidence in the helpfulness of treatment in order to positively influence outcomes.
Summary

Expectancies of treatment outcome refer to the extent patients believe treatment will lead to symptom improvement. This non-specific factor common to all treatments has frequently been associated with psychotherapy outcomes for a variety of disorders for over 50 years. Most studies examining associations between treatment expectancies and outcome for Panic-Ag showed significant relationships irrespective of whether treatment involved graded exposure only or more modern CBT procedures. However, methodological limitations regarding the measurement of treatment outcome expectancies (scales combining treatment credibility and expectancy ratings, poor psychometric data) weaken the veracity of such conclusions. Moreover, it remains to be determined whether patients’ pretreatment outcome expectancies are related to therapeutic improvement which, if significant, would signify the need to provide pretreatment counselling for patients with poor outcome expectations. This thesis aims to address these issues by developing a psychometrically sound measure consisting of items purely assessing treatment expectancies where it will be administered to patients prior to commencing CBT for Panic-Ag to assess its relationship with treatment improvement.

Thus far, poor treatment knowledge, acceptance of the rationale and expectancies of outcome have been associated with reduced clinical improvement, presumably in part through its impact on treatment compliance. A further issue known to interfere with compliance and treatment outcome concerns patients’ confidence or self-efficacy to carry out therapy instructions.
**Treatment Self-Efficacy**

Compliance with CBT procedures is estimated at approximately 50% (Detweiler & Whisman, 1999). Interestingly, of reasons offered for difficulty completing treatment directives, patients doubting their ability to complete therapy tasks was most commonly mentioned, being expressed by 57% of patients who experienced problems with CBT task completion (Helbig & Fehm, 2004). Hence, patients’ confidence or self-efficacy to implement prescribed CBT techniques is likely to play an important role in influencing treatment compliance and, in turn, outcome.

**Self-Efficacy**

First formally introduced by Bandura (1977), *self-efficacy* is defined as “the conviction that one can successfully execute the behaviour required to produce the outcome” (p. 193). This concept represents an individual’s belief in being able to effectively perform a particular behaviour. Self-efficacy beliefs are situation specific and intercorrelations between different domains tend to be low (O'Leary, 1992). Furthermore, self-efficacy is not static and may fluctuate significantly in relation to numerous factors. To illustrate, a student’s self-efficacy for completing mathematical equations can be quite different from his/her self-efficacy for driving, the latter of which can be influenced by factors such as the weather, driving terrain, or time of day.

According to self-efficacy theory, individuals’ belief in their effectiveness can influence whether or not they try to cope with difficult situations, as well as how much effort they use and how long they persevere when encountering obstacles (Bandura, 2001). Strong self-efficacy promotes greater effort and is intertwined with
behaviour. Successes tend to raise self-efficacy and encourage further success, whereas failures, especially those occurring early on, tend to reduce self-efficacy, which in turn hinders success (Bandura, 1977). Personal efficacy beliefs are essential for implementing behavioural changes required for anxiety reduction (Bandura, 1988). Patients are unlikely to be motivated to engage in or persist with treatment advice if they do not believe their actions will produce positive effects (Bandura, 2004).

**Definitional Issues**

Within the literature, self-efficacy has been assessed in different domains (managing symptoms, completing daily activities, overcoming barriers); however this thesis will confine its investigation to self-efficacy for implementing treatment directives (herein referred to as *treatment self-efficacy*). This construct has also been termed *adherence self-efficacy* (Barclay et al., 2007; Johnson et al., 2006; Nilsson Schönnesson, Diamond, Ross, Williams, & Bratt, 2006). Example items assessing treatment self-efficacy for diabetes self-management and hormone replacement therapy include, “How sure are you that you can manage your diabetes the way your health care team want you to, almost all the time?” (Iannotti et al., 2006) and “How confident are you in your ability to continue taking hormone therapy?” (Nagia, 1999), respectively.

Self-efficacy beliefs need to be distinguished from outcome expectancies (Bandura, 1977). Whereas outcome expectancies reflect the belief that specific behaviours, if implemented, will lead to predicted outcomes, self-efficacy beliefs denote individuals’ expectations that they possess skills required to successfully carry out those behaviours. Differentiating treatment self-efficacy from outcome
beliefs is important in CBT as patients may believe CBT will be helpful in reducing symptoms (high treatment outcome expectancies), yet simultaneously doubt their ability to carry out therapeutic techniques (low treatment self-efficacy). If self-efficacy for Panic-Ag treatment recommendations is poor, patients may not attempt to carry out such procedures, thereby promoting intentional non-compliance, ultimately compromising therapy outcomes.

It is also important to distinguish treatment self-efficacy from panic self-efficacy which, unlike treatment self-efficacy, has been extensively investigated within the Panic-Ag literature. While treatment self-efficacy is exclusively concerned with confidence in implementing treatment, panic self-efficacy concerns individuals’ confidence in coping with panic symptoms (sensations, cognitions and feared situations) irrespective of treatment (Casey, Oei, Newcombe, & Kenardy, 2004; Williams, 1990). Examples of panic self-efficacy items include, “How confident are you in controlling a panic attack when feeling short of breath?” (Gauthier, Bouchard, Côté, Laberge, & French, 1993) and “How confident are you that could drive 6 kilometres on a crowded freeway when unaccompanied?” (Kinney & Williams, 1988). If a patient’s treatment recommendation was to drive unaccompanied for 6 kilometres on a crowded freeway, the latter item would also be assessing treatment self-efficacy.

Numerous researchers have ascribed great importance to panic self-efficacy in theoretical models explaining the aetiology and maintenance of Panic-Ag (Barlow, 1988; Casey, Oei, Newcombe et al., 2004; Rachman, Craske, Tallman, & Solyom, 1986; Richards, Richardson, & Pier, 2002; Sanderson et al., 1989; Telch et al., 1996), and changes in panic self-efficacy have been shown to mediate the effectiveness of CBT for Panic-Ag (Hoffart, 1995a; Williams, Kinney, & Falbo, 1989; Zane &
Williams, 1993). Indeed, several studies have demonstrated that panic self-efficacy predicted treatment outcome to a similar (if not greater) degree as catastrophic cognitions (Borden, Clum, & Salmon, 1991; Bouchard et al., 2007; Casey, Newcombe et al., 2005; Casey, Oei et al., 2005; Reilly, Gill, Dattilio, & McCormick, 2005). Thus, panic self-efficacy and catastrophic cognitions have both been emphasised as central to mediating the effectiveness of CBT and may each need to be targeted to maximise treatment outcome.

It is also important to focus on treatment self-efficacy, as research from the medical literature identified this construct to be highly influential in contributing to clinical outcomes. However, few studies in the wider psychotherapy literature, and none in the Panic-Ag domain, have investigated treatment self-efficacy. Therefore, the present thesis will examine the role of treatment self-efficacy on outcome for Panic-Ag.

**Relationships Between Treatment Self-Efficacy, Compliance and Outcome**

Theoretically, treatment self-efficacy should hold a positive linear relationship with treatment outcome through promoting increased treatment compliance. Indeed, as will be reviewed below, the majority of studies from the medical and psychotherapy literature investigating relationships between treatment self-efficacy and clinical outcomes found significant positive associations across differing age groups, patient demographics, illness types and methodologies. The magnitude of effects varied as a function of illness type, outcome measures used (self-report vs. biological markers; stronger effects for self-report) and timing of assessments (baseline vs. concurrent).
Medical Illness

Patients’ confidence to perform necessary self-care behaviours correlated significantly and positively with adherence to such behaviours in the field of asthma (Ngamvitroj & Kang, 2007; Zebracki & Drotar, 2004) and diabetes (Hurley & Shea, 1992; Iannotti et al., 2006; Williams & Bond, 2002). For example, Williams and Bond (2002) found patients’ confidence to perform diabetic self-care behaviours (e.g., blood glucose testing, dietary restrictions) correlated between .39 and .61 ($p < .01$) with compliance behaviours in 94 patients. In regards to outcome, confidence adhering to treatment also correlated significantly with metabolic control in diabetic patients (Gerber et al., 2006; Grossman, Brink, & Hauser, 1987; Iannotti et al., 2006), although correlations ($r = -.21 – -.33$) tended to be lower than those obtained for compliance.

Among HIV-positive patients, confidence in being able to adhere to antiretroviral therapy (ART) has consistently been associated with treatment compliance in cross-sectional (Barclay et al., 2007; Johnson et al., 2006, 2007; Pinheiro, de-Carvalho-Leite, Drachler, & Silveira, 2002) and longitudinal (Godin, Coté, Naccache, Lambert, & Trottier, 2005; Johnson et al., 2007; Nilsson Schönnesson et al., 2006; Spire et al., 2002) studies. In addition, confidence in adhering to ART correlated significantly with immunologic functioning and viral load biomarkers in two large samples ($N > 260$) (Johnson et al., 2007).

A strong relationship between treatment self-efficacy and self-reported outcome was observed for patients taking hormone replacement therapy (Nagia, 1999). In this study of 50 women, confidence in continuing to take hormone therapy (assessed on a single-item scale) correlated .56 with quality of life and 31% of the
variance in quality of life was accounted for by hormone self-efficacy. However, problems associated with single-item measures may have inflated this relationship. One study failed to find significant results, however methodological problems may have contributed to this. Among 72 hypertensive patients, treatment self-efficacy scores were higher for patients with controlled blood pressure than for those with uncontrolled blood pressure (Ogedegbe, Mancuso, Allegrante, & Charlson, 2003), however the use of a 3-point Likert scale may have reduced variability in self-efficacy scores preventing this difference from achieving statistical significance. In summary, however, treatment self-efficacy was associated with patients’ treatment compliance and health outcomes across many different illnesses.

**Psychological Disorders**

Although aspects of self-efficacy have been widely investigated in the psychotherapy literature, treatment self-efficacy itself appears only to have been investigated for insomnia (Bélanger, Morin, Bastien, & Ladouceur, 2005; Bouchard, Bastien, & Morin, 2003) and pain (Heapy et al., 2005). In a study of 36 patients, Bouchard et al. (2003) found self-efficacy in performing requirements of a CBT program for insomnia was significantly associated with adherence behaviour scores over a 7-week program ($r = .17 – .67$).

Similar findings were found by Bélanger et al. (2005) who assessed self-efficacy for adhering to recommendations to reduce sleeping tablets across 10 weeks of a benzodiazepine taper program for 47 patients. These authors reported that patients compliant with recommendations to reduce sleeping tablets reported significantly higher confidence in being able to do so than their less compliant counterparts at several time periods during the program. For example, at week 10,
self-efficacy expressed out of 100 for compliant patients was 95.1 in comparison to 74.3 for non-compliant patients ($p < .01$). In addition, patients who were medication free at posttreatment (successful outcome) had significantly higher self-efficacy ratings than those who were not.

Interestingly, baseline self-efficacy ratings are not as predictive of patient compliance or outcome as are concurrent measurements described in the two studies reviewed above. In the Bélanger et al. (2005) study, baseline self-efficacy did not differ between compliant and non-compliant patients or those with and without successful outcomes. Lack of significant associations with compliance and outcome were also reported by Heapy et al. (2005) in a sample of 78 patients undergoing CBT for chronic pain. Given self-efficacy is not an immutable character trait but responsive to personal experiences (e.g., therapy experiences), this lack of baseline association is not surprising and suggests optimism when treating patients with low self-efficacy.

**Summary**

Patients’ confidence or self-efficacy for implementing treatment recommendations is positively related to treatment compliance and outcome for a range of disorders. However, baseline self-efficacy ratings appear less predictive than concurrent ratings. To date, no study has examined this issue in the field of CBT for Panic-Ag.

The present thesis will examine the relationship between treatment self-efficacy and outcome in Panic-Ag. Should a significant relationship exist, the resultant clinical implications include regular monitoring of patients’ self-efficacy for implementing CBT recommendations coupled with exploring and addressing reasons
for low self-efficacy. As self-efficacy is domain specific, a multi-item measure specifically assessing confidence in implementing CBT techniques for Panic-Ag firstly needs to be developed.

**The Present Study**

The effectiveness of CBT is linked to patients’ treatment compliance. To comply, patients must understand or have knowledge of treatment, accept the treatment rationale, perceive therapy as helpful and believe they have sufficient self-efficacy to execute treatment recommendations. A failure in any of these requirements can result in treatment non-compliance and interfere with therapeutic outcomes. Hence, patients’ knowledge and beliefs about treatment play an important role in influencing patient outcomes. With the exception of treatment outcome expectancies, treatment knowledge and beliefs and the impact of pretreatment expectancies on outcome have not been adequately examined in Panic-Ag.

**Aims and Hypotheses**

This thesis therefore aims to address a number of methodological limitations in examining associations between treatment knowledge, acceptance of the treatment rationale, pretreatment outcome expectancies and treatment self-efficacy on outcome following CBT for Panic-Ag. If significant relationships emerge, they highlight important clinical implications for treatment delivery, contributing to improved treatment outcomes, leading to reduced patient suffering and improved quality of life, thus lessening the burden of the disorder on the individual, the health care
system and society at large. The research undertaken in this thesis involved several stages.

**Stage 1: Development of Treatment Knowledge Measures**

Measures specifically assessing treatment knowledge of CBT for Panic-Ag were developed. Due to uncertainty regarding best methods for assessing treatment knowledge, a multiple-choice and structured interview measure was constructed. Psychometric properties of the treatment knowledge measures were evaluated. Reliability and validity estimates were obtained from patient and clinician samples.

It was expected that:

1. Scores on the multiple-choice knowledge measure would correlate positively with knowledge interview scores.
2. Treated patients would demonstrate greater treatment knowledge than untreated patients.
3. Clinicians with experience in CBT would score higher on the multiple-choice knowledge measure than entry-level intern clinical psychologists.
4. Treatment knowledge scores would significantly increase with the provision of treatment.
5. Treatment knowledge scores would be positively correlated with intelligence and years of education and negatively correlated with age.
Stage 2: Development of Treatment Beliefs Measures

Measures assessing patients’ treatment beliefs about CBT for Panic-Ag were developed; specifically, beliefs assessing acceptance of the treatment rationale, expectancies of treatment outcome and treatment self-efficacy. Psychometric properties of the treatment belief measures were assessed. Reliability and validity information was obtained from patient samples.

It was expected that:

1. Acceptance of the treatment rationale would be negatively associated with endorsement of aetiological beliefs inconsistent with a CBT rationale.
2. Acceptance of the treatment rationale and treatment outcome expectancies would be negatively associated with stronger belief in non-CBT treatments.
3. Acceptance of the treatment rationale would be positively correlated with treatment outcome expectancies.
4. Treatment outcome expectancies would correlate positively with published measures assessing this construct.
5. Treatment self-efficacy and outcome expectancies would be negatively correlated with stronger endorsement of factors perceived to interfere with treatment.
6. Treatment self-efficacy would be negatively correlated with level of self-deprecation.
7. Treated patients would demonstrate greater acceptance of the treatment rationale and treatment self-efficacy than untreated patients.
8. Patients’ acceptance of the treatment rationale and treatment self-efficacy would increase significantly with the provision of treatment.
Stage 3: Examination of Relationships Between Treatment Knowledge, Beliefs and Outcome

In this final stage, relationships between treatment knowledge, beliefs and outcome following CBT for Panic-Ag were examined. Relative contributions of these variables to treatment outcome and whether such relationships were mediated by belief in catastrophic cognitions was determined.

The following hypotheses were advanced:

1. Improving treatment knowledge will be associated with reduced Panic-Ag severity.

2. Greater posttreatment acceptance of the treatment rationale will be associated with reduced Panic-Ag severity.

3. Stronger treatment self-efficacy at posttreatment will be associated with reduced Panic-Ag severity.

4. Recovered participants will show greater treatment knowledge, pretreatment outcome expectancies, acceptance of the treatment rationale and treatment self-efficacy than non-recovered participants.

5. Relationships between treatment knowledge and outcome will be mediated by belief in catastrophic cognitions.

6. Relationships between treatment beliefs and outcome will be mediated by belief in catastrophic cognitions.

In addition, this thesis attempted to examine associations between pretreatment outcome expectancies, acceptance of the rationale and treatment self-efficacy on outcome. Finally, the impact of benzodiazepine use on treatment knowledge acquisition was explored.
Chapter 2

DEVELOPMENT OF THE MULTIPLE-CHOICE PANIC-AG TREATMENT KNOWLEDGE QUESTIONNAIRE (MC-PTKQ) ........................................................................................................................................74

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Development of the Multiple-Choice Panic-Ag Treatment Knowledge Questionnaire (MC-PTKQ)

This study describes the initial development of a measure assessing CBT knowledge for Panic-Ag. Knowledge measures for medical disorders have burgeoned in recent years (Dunn et al., 1984; Edworthy, Devins, & Watson, 1995; Hill, Bird, Hopkins, Lawton, & Wright, 1991; Jarvie, Espie, & Brodie, 1993a; John et al., 2009; Lubrano et al., 1998; Meadows et al., 1988; Pande et al., 2000; Rees et al., 2003; Wigal et al., 1993). However, to date there have been no valid and reliable instruments constructed for the assessment of CBT principles in general or tailored specifically to Panic-Ag. This thesis follows the guidelines on scale development set out by DeVellis (1991), additionally guided by research in the medical literature (Dunn et al., 1984; Edworthy et al., 1995; Lubrano et al., 1998; Pande et al., 2000; Rees et al., 2003) which suggest that several phases are involved in the construction of a treatment knowledge measure.

Development of the Treatment Knowledge Measure

Although formats may vary, the process of developing a knowledge measure typically involves three phases: (i) development of an item pool, (ii) expert review of items and initial item refinement, and (iii) analysis of items and final reduction of item pool. These phases are described below.

Phase 1 – Development of item pool: The realm of knowledge to be assessed is identified from either experts in the field or published texts related to the disorder and its treatment. Major domains of knowledge are defined and items written, usually in multiple-choice or true/false format, assessing specific aspects of
knowledge relevant to content domains. These items constitute the initial draft of the knowledge questionnaire.

**Phase 2 – Expert review of items and initial item refinement:** The draft knowledge questionnaire is then reviewed by experts in the field to obtain feedback on relevance, comprehensibility and accuracy of items. The draft questionnaire is also piloted on lay persons to obtain comments characteristic of the target population. Items are added, omitted or reworded on the basis of feedback obtained and this refined set of items comprises the revised questionnaire. Guidelines regarding the number of expert reviewers required are lacking, however reports from the literature range from two or three (Edworthy et al., 1995; Rees et al., 2003) to 14 (Jarvie et al., 1993a).

**Phase 3 – Analysis of items and final reduction of item pool:** Items from the revised questionnaire are analysed to determine their index of difficulty, index of discrimination and reliability. Index of difficulty refers to the proportion of respondents answering the item correctly (Murphy & Davidshofer, 1994). The item’s difficulty index, or *p value*, is calculated using the formula:

\[ p \text{ value for item } i = \frac{\text{Number of correct responses for item } i}{\text{Total number of responses for item } i} \]

A *p* value of .0 indicates no respondent answered the item correctly (difficult item), while a *p* value of 1.0 indicates all respondents answered the item correctly (easy item). Extreme *p* values minimise variability of scores and do not contribute to measurement of individual differences. Maximum variability within test scores is obtained when all *p* values cluster around .50 (Murphy & Davidshofer, 1994).
However, selection of appropriate item difficulties varies according to the purpose of the measure (Anastasi, 1988). If the measure is intended to determine pre-existing knowledge of material to be taught, item difficulty ratings are expected to be low. In such instances, difficult items should not be removed as they highlight what remains to be learnt. Similarly, if the purpose of the measure is to establish whether a respondent has adequately learnt the information, the difficulty indices should be high, around .80 or .90. In these circumstances, even very easy items (including those passed by 100% of respondents) are retained (Anastasi, 1988).

Item discrimination is defined as the degree to which an item is able to differentiate between respondents who do well and those who do poorly on the measure. The item discrimination index, or $D$, is calculated by constructing extreme groups (quartiles or thirds) and subtracting the percentage of respondents answering correctly in the lowest group from the percentage answering correctly in the highest group (Murphy & Davidshofer, 1994). Theoretically, $D$ values range from -100 through 0 to +100. Negative $D$ values indicate a higher proportion of respondents from the lower group answering the item correctly in comparison to respondents from the upper group. Positive $D$ values indicate a higher proportion of respondents in the upper group answering the item correctly relative to respondents from the lower group. A zero $D$ value indicates the proportion of respondents answering the item correctly from the upper group was equivalent to the proportion responding correctly from the lower group. A zero $D$ value therefore signifies the item lacks discriminating power. A mean $D$ value of +50 across an entire measure is associated with the highest level of item discrimination (Anastasi, 1988).

Reliability of items is commonly measured by Cronbach’s alpha, an index of internal consistency. Results from homogeneous groups are expected to reach
coefficient values between .8 and .9 (Cronbach, 1970). Less consistent items can be identified by producing an alpha score following the exclusion of individual items (alpha if item deleted). If the alpha score of a scale increases after removal of a particular item it indicates that inclusion of this item reduces the overall reliability of the scale and is therefore a poor item.

Following a comprehensive analysis of items, those demonstrating unsatisfactory indices of difficulty, discrimination and reliability are excluded. Remaining items comprise the final version of the questionnaire. However, before developing a knowledge measure, selection of a suitable assessment format is an important requirement.

Selection of Knowledge Questionnaire Format

Three different formats have been identified in the literature: multiple-choice, true/false and open-ended short answer formats (Beeney et al., 1994; Dunn et al., 1984; Edworthy et al., 1995; Hill et al., 1991; Jarvie et al., 1993a; Lubrano et al., 1998; Meadows et al., 1988; Pande et al., 2000; Rees et al., 2003; Wigal et al., 1993). The true/false format, although simple for participants to complete, was rejected for this thesis on grounds that participants have a 50% chance of guessing correctly, which in turn can undermine interpretation of item difficulty indices (Dunn et al., 1984). Some researchers (Beeney et al., 1994; Dunn et al., 1984) rejected open-ended short-answer formats because a high proportion of their participants were from non-English speaking backgrounds (NESB). Short-answer formats have been criticised for discriminating against NESB individuals, penalising individuals who experience difficulty expressing themselves orally, and regarded as “an assessment of verbal ability,” reducing the validity of the knowledge measure (Dunn et al., 1984, p. 38).
However, it was unclear whether a test of recognition memory (multiple-choice format) versus recall (open-ended short answer format) would be a more ecologically valid assessment of knowledge in the field of CBT. It could be argued that a person who is able to recognise correct responses from a list of alternatives demonstrates knowledge of that issue. However, during real life situations, individuals are often required to draw upon information from memory in the absence of prompts or cues. Multiple-choice measures capitalise on recognition memory which may serve to remind some participants of knowledge that would otherwise be inaccessible without prompts.

Open-ended short answer questions may provide a more sensitive measure of patient knowledge than other formats as they have the capacity to assess a wider range of knowledge sophistication. Responses to open-ended questions can determine whether individuals possess partial knowledge of a topic, in contrast to multiple-choice items that are typically scored either wholly correct or incorrect. Indeed ceiling effects have been observed on a well validated multiple-choice test of diabetes knowledge (Dunn et al., 1984) possibly indicating a lack of sensitivity (Beeney et al., 1994; Coates & Boore, 1996). In contrast, responses to open-ended short answer questions can be scored on a continuum ranging from completely incorrect, partially correct, mostly correct through to completely correct.

While possibly providing a more sensitive assessment of knowledge, short-answer formats are not without their disadvantages. In addition to potentially prejudicing less verbally skilled individuals, scoring open-ended responses can be time consuming, open to interpretation and unreliable across different raters in comparison to other formats where correct responses are identified objectively.
Formal discussions regarding format choice were held with clinical psychologists from the Department of Medical Psychology, Westmead Hospital and the School of Psychology, University of Sydney. Acknowledging the pros and cons of each approach, feedback from clinical psychologists and academics recommended using both a multiple-choice questionnaire and an open-ended short-answer interview. In this way, the multiple-choice questionnaire allowed objective assessment of treatment knowledge free from subjective scoring interpretations or therapist bias, while assessing knowledge via interview enabled participants’ responses to be queried and/or probed, minimising problems associated with reduced verbal fluency and incorrect use of terms. Incorporating two measures of patient knowledge also enabled assessment of convergent validity. Hence, for purposes of this thesis, a decision was made to incorporate both multiple-choice questionnaire and open-ended interview formats.

The remainder of this chapter focuses on the development of the multiple-choice treatment knowledge questionnaire. The development of the open-ended short answer structured knowledge interview and the psychometric properties of both knowledge measures are described in chapter 3.

**Method**

Construction of the multiple-choice knowledge questionnaire proceeded through the three previously described phases of questionnaire development: (i) development of the item pool, (ii) expert review of items and initial item refinement, and (iii) analysis of items and final reduction of the item pool.
Phase 1: Development of the Item Pool

Items were generated from material covered in contemporary CBT programs for Panic-Ag with empirical efficacy (Andrews et al., 2003; Craske & Barlow, 2001; Hawton, Salkovskis, Kirk, & Clark, 1989) and scientific literature relevant to the disorder (Clark, 1986, 1996; Rapee, 1997; Rayburn & Otto, 2003; Salkovskis, Clark, & Gelder, 1996; Schmidt et al., 2000; Uren, Szabo, & Lovibond, 2004). Four interrelating domains were repeatedly discussed in the literature:

1. *Psychoeducation*: consisting of the stress-diathesis model, fight/flight response, role of hyperventilation and medically accurate explanations of panic symptoms
2. *Cognitive Therapy*: consisting of the cognitive model of panic, identification of causal thoughts and methods for challenging the probability and cost of feared panic outcomes
3. *Avoidance*: consisting of the nature and function of avoidance and safety seeking behaviours
4. *Exposure Therapy*: consisting of behavioural experiments, graded in vivo exposure and interoceptive exposure

These four domains encompassed over 150 individual facets of knowledge relevant to treatment of Panic-Ag. As research has not investigated which specific aspects of treatment knowledge are essential to clinical improvement, a comprehensive measure of treatment knowledge was sought. However, lengthy measures have been known to promote undesirable factors such as fatigue, boredom and intimidation that may serve to reduce the validity (Dunn et al., 1984). To achieve a compromise between comprehensiveness of knowledge and questionnaire length, items were constructed with a stem question comprising five response alternatives. A “Don’t know” response was also included to minimise guessing, reduce performance
anxiety and improve compliance. While three or four response alternatives is often traditional for multiple-choice questionnaires (Dunn et al., 1984; Edworthy et al., 1995; Lubrano et al., 1998; Pande et al., 2000; Wigal et al., 1993), many items in the present questionnaire contained an “All of the above” and/or “None of the above” option within the response alternatives to maximise the scope of knowledge able to be assessed within one question whilst keeping questionnaire length to a minimum (see Figure 2.1). Similar formats have been applied by other researchers (Hill et al., 1991; Wigal et al., 1993).

A preliminary set of 39 multiple-choice questions was constructed (see Appendix A) consisting of 15 psychoeducation items (items 1-15), 11 cognitive therapy items (items 16-22, 24-27), six avoidance items (items 28-33) and seven exposure therapy items (items 23, 34-39). Example questions from each content domain are presented in Figure 2.1.
**Psychoeducation Items**

**In individuals without a history of such problems, panic attacks are likely to cause**

a. Heart disease or heart attacks  
b. Stroke  
c. Insanity (e.g., Schizophrenia)  
d. All of the above  
e. None of the above  
f. Don’t know

**Which of the following reactions does NOT occur during the fight or flight response?**

a. The face may go pale as blood is diverted away from parts of the body that do not immediately require nutrition.  
b. Heart rate and blood pressure increases so that oxygen and nutrients can be transported quickly to where they are needed.  
c. Breathing speeds up to increase the amount of oxygen available to the muscles.  
d. Muscles relax to help you stay calm and perform at your best.  
e. Sweating increases to cool the body to prevent it from overheating during strenuous physical activity.  
f. Don’t know
Figure 2.1 (Continued). Examples of multiple-choice questions for each knowledge domain.

Cognitive Therapy Items

According to the CBT approach, which of the following statements are correct?

If you are having difficulty identifying why you are feeling panicky…

a. It means there are simply no thoughts there and a medical explanation is required to explain the cause of your panic attacks and anxiety.
b. It is likely that the panic sensations or situation have become associated with danger (probably because of past experiences), so that you now automatically respond with fear without consciously having thoughts about the sensations.
c. You can repeatedly ask yourself what would be so bad if the worst thing happened until you get to the core of the problem (Downward Arrow Technique).
d. You can observe your own behaviour when you are anxious to look for clues that would help to explain the underlying thought.
e. b, c and df. Don’t know

According to the CBT approach, “Overestimating the probability” refers to:

a. Thinking about something that has happened and making it out to be much worse than it is in reality.
b. Thinking that something is more likely to happen than it is in reality.
c. Exaggerating the importance or significance of an event.
d. All of the above
e. b and cf. Don’t know
**Figure 2.1 (Continued).** Examples of multiple-choice questions for each knowledge domain.

**Avoidance Items**

**According to the CBT approach, which of the following is a safety seeking behaviour?**

a. Slowing your breathing to prevent a panic attack from developing  
b. Carrying (but not actually taking) anti-anxiety medication with you when you enter an anxiety provoking situation  
c. Carrying a paper bag  
d. All of the above  
e. None of the above  
f. Don’t know

**According to the CBT approach, which of the following statements about safety seeking behaviours is false?**

Using a safety seeking behaviour when you are in an anxiety provoking situation…

a. Is a sensible approach to overcoming anxiety as it can help prevent the terrible thing from happening (e.g., heart attack, fainting, embarrassing self in public).  
b. Is a form of avoidance.  
c. Stops you from testing out your thoughts about the dangerousness of panic.  
d. Keeps your fears alive.  
e. Is a problem because you will still believe that something bad would have happened if you had not used the safety seeking behaviour.  
f. Don’t know
Exposure Therapy Items

According to the CBT approach, which of the following statements involving facing your fears is false?

a. Behavioural experiments that involve facing your fears can cause intense anxiety and even panic attacks.
b. Facing your fears is not recommended for the treatment of Panic Disorder or Agoraphobia as anxiety levels can become so severe as to cause serious physical harm (e.g., heart attack, stroke, fainting) or mental harm (e.g., insanity).
c. The more often you confront your fear, the less your anxiety will rise and the faster your anxiety will fall.
d. Confronting a feared situation is an excellent method for testing out whether your thoughts about the dangerousness of panic attacks are correct.
e. By facing your fears you learn that the thing you feared did not happen (or was not that bad). This increases your confidence about facing your fear in the future.
f. Don’t know

According to the CBT approach, which of the following statements about deliberately bringing on the feared panic sensations (interoceptive exposure) is correct?

The aim of deliberately bringing on panic sensations is to…

a. Change your thoughts about the dangerousness of panic sensations.
b. Learn that panic sensations are unpleasant but harmless.
c. Help you become less anxious when you experience such sensations (e.g., dizziness, heart palpitations) as part of your every day life.
d. Break the association between your fear response and the feared sensation.
e. All of the above
f. Don’t know
Phase 2: Expert Review of Items and Initial Item Refinement

The 39-item draft multiple-choice questionnaire was reviewed by experts in the field for relevance, comprehensibility and accuracy of information. Following this, items were refined on the basis of feedback.

Participants and Procedure

The 39-item draft multiple-choice knowledge questionnaire was distributed to 27 clinical academics and clinical psychologists experienced in the provision of CBT specific to Panic-Ag and in general. These expert reviewers were recruited from three sources:

1. Clinical academics from the School of Psychology, University of Sydney (n = 3)
2. Clinical Psychologists working in tertiary referral anxiety clinics in Western Sydney (n = 5)
3. Clinical Psychologists working within the Department of Medical Psychology, Westmead Hospital (n = 19)

Brief demographic information comprising years of clinical experience and self-rated level of expertise with CBT for Panic-Ag from 1 (very low) to 5 (very high) was collected. Reviewers were requested to make three ratings regarding:

i. relevance of each question to cognitive behavioural treatment of Panic-Ag from 0 (not at all relevant) to 2 (very relevant)

ii. comprehensibility or wording of each question and respective answers from 0 (difficult for patients to understand) to 2 (easy for patients to understand) and to make changes to wording of items as they saw fit

iii. agreement with answer provided (Yes or No)
Reviewers were also asked to indicate whether any components of CBT were omitted or over-represented and extra space for each question was provided for additional comments (see Appendix A).

Thirteen of the 27 reviewers returned the questionnaire (48%). Three reviewers (100%) from the University of Sydney and five reviewers (100%) from tertiary referral anxiety clinics returned the questionnaire. Only five questionnaires (26%) were received from clinical psychologist reviewers in the Department of Medical Psychology, Westmead Hospital. This low return rate may in part be due to these reviewers working primarily with other disorders (depression, post-traumatic stress disorder, eating disorders, child and adolescent issues) which may have lowered their Panic-Ag expertise ratings, leading to reluctance in providing feedback. Khawaja and Oei (1992) reported a similar and lower return rate (37.5%) among psychologist reviewers. The 13 reviewers returning questionnaires had an average of 9.8 years clinical experience ($SD = 7.1$ years). Self-rated level of expertise with CBT for Panic-Ag was high ($M = 4.3, SD = 0.9$).

**Analysis of Feedback From Expert Reviewers**

Table 2.1 displays means and standard deviations for relevance and comprehensibility ratings and percentage agreement for each of the 39 items of the draft knowledge questionnaire. Overall, the draft measure appeared to assess knowledge relevant to CBT for Panic-Ag with relevance ratings ranging between 1.45 and 2.00 ($M = 1.92, SD = 0.13$). Comprehensibility ratings ranged between 0.92 and 2.00 ($M = 1.76, SD = 0.23$) indicating reviewers felt most items were relatively easy for patients to understand. Overall inter-rater agreement with answers was high at 100% with the exception of four items (78% – 92% agreement).
## Table 2.1 Ratings of Relevance, Comprehensibility and Agreement Across Expert Reviewers (N = 13) for the 39-Item Draft Multiple-Choice Knowledge Questionnaire

<table>
<thead>
<tr>
<th>Item no.</th>
<th>Relevance ( M ) (SD)</th>
<th>Comprehensibility ( M ) (SD)</th>
<th>Agreement (% Agree)</th>
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<td><strong>Total</strong></td>
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<td><strong>1.76 (0.23)</strong></td>
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</table>
Chapter 2 – Development of the Multiple-Choice Knowledge Questionnaire

**Initial Item Refinement**

Length of questionnaire was a recurring topic raised by reviewers. While shorter measures may be less taxing for participants to complete, a reduction in number of items generally reduces reliability (DeVellis, 1991). As a trade-off, attempts were made to reduce the number of items by approximately 25% (from 39 to 29 items), thereby substantially shortening the questionnaire yet conserving reliability.

Although all items were considered relevant, items with mean relevance ratings below 1.80 were excluded (items 4, 8, 9, 17). Items 1-3 related to the stress-diathesis model of panic. These items were omitted because, although considered part of the psychoeducation component of Panic-Ag, knowledge contained in these items did not assist in challenging catastrophic misinterpretations of panic symptoms, the core feature of treatment for Panic-Ag (Clark et al., 1997; Craske & Barlow, 2006). Items 10 and 11 both discussed symptoms of hyperventilation and were combined into one item. Similarly, items 17 and 18 related to the cognitive model of panic and items 29 and 30 to specific examples of avoidance. These four items were respectively combined into two single items, resulting in a refined set of 29 items.

While reviewers indicated all areas of CBT for Panic-Ag were adequately covered, specific details were added as requested (e.g., the notion that “the fight/flight response is a mechanism that does not need to be controlled or stopped and it will go away on it is own” was added to a question concerning the fight/flight response). Of the 29 items, those with comprehensibility ratings less than 2.0 were rephrased where possible. Items with agreement ratings less than 100% were reworded until consensus with reviewers’ comments was achieved.
The refined 29-item multiple-choice knowledge questionnaire was piloted on a small group of individuals consisting of three patients with a history of Panic-Ag, three lay persons and four intern clinical psychologists to assess time taken to complete the measure and identify aspects requiring further refinement. The questionnaire took approximately 20-25 minutes to complete and further adjustments to wording of items were made in light of their advice. The revised questionnaire, referred to as the Multiple-Choice Panic Disorder-Agoraphobia Treatment Knowledge Questionnaire (MC-PTKQ, see Appendix B), consisted of eight psychoeducation items (items 1, 3, 6, 10, 12, 14, 25, 26), nine cognitive therapy items (items 2, 4, 7, 16, 17, 19, 21, 22, 23), five avoidance items (items 5, 11, 15, 24, 27) and seven exposure therapy items (items 8, 9, 13, 18, 20, 28, 29).

The readability of the MC-PTKQ was assessed using the Flesch Reading Ease Index (Flesch, 1948). Readability scores are calculated according to average number of (a) syllables per word and (b) words per sentence. Reading ease scores range from 0 to 100 where higher scores reflect greater reading ease. Documents rated above 70 are regarded as fairly easy to read while scores of 50 and below are considered difficult. As the MC-PTKQ assessed knowledge of CBT principles, items typically contained multi-syllabic technical terms (e.g., hyperventilation, behavioural experiments, overestimating the probability) inflating the average number of syllables per word thereby reducing the total readability score. Accordingly, the obtained reading ease score ranged between 30 and 50 (rated as difficult). However, when based on average sentence length in words, the text was considered easy to read (reading ease score: 80 – 90).

Scores for the 29-item MC-PTKQ ranged from 0 to 40. Eighteen items were scored one point for a correct response. The remaining 11 items assessed multiple
aspects of knowledge (often containing an “All of the above” and/or “None of the above” response option). For such items, two points were awarded if all correct responses were selected and one point was awarded if only one of the correct responses was selected. Incorrect responses, unanswered questions or “Don’t know” responses were scored zero.

**Phase 3: Analysis of Items and Final Reduction of the Item Pool**

The next phase involved determining indices of item difficulty, discrimination and reliability enabling identification and elimination of poorly performing items to maximise validity and reliability of the questionnaire. The questionnaire was administered to a sample of CBT waitlisted patients with Panic-Ag for the purpose of identifying and deleting easy items. Items easy for most patients prior to receiving treatment contribute to a ceiling effect and thus reduce the measure’s ability to detect changes after treatment. A difficulty index, or $p$ value, exceeding .75 is deemed a poor discriminator (Hill et al., 1991; Lubrano et al., 1998; Pande et al., 2000). However, it was considered appropriate to retain difficult items as the provision of treatment allows scores on such items to increase. Furthermore, it was unclear whether items with low $p$ values reflected patients’ confusion with wording (indicating validity problems), or whether knowledge contained within such items was associated with successful treatment outcomes. However, items lacking ability to discriminate between high and low scoring participants should be removed. A discrimination index, or $D$ value, of less than 20 is deemed unacceptably low (Dunn et al., 1984). Similarly, items with low reliability coefficients should be discarded.
Participants

Participants were recruited from the Sydney West Area Health Service (SWAHS) Anxiety Treatment and Research Unit, Cumberland Hospital between December 2005 and September 2007. The SWAHS Anxiety Treatment and Research Unit is a tertiary referral anxiety clinic in Western Sydney typically serving patients from lower socioeconomic backgrounds. The clinic offers cognitive behavioural therapy for patients with a primary anxiety disorder.

The sample was drawn from a population of 83 consecutive first-time attendees seeking treatment for Panic-Ag. Inclusion criteria consisted of diagnosis of Panic Disorder and/or Agoraphobia as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV, American Psychiatric Association, 1994) as assessed through the Anxiety Disorders Interview Schedule for DSM-IV, Adult Version (ADIS-IV, Brown, DiNardo, & Barlow, 1994), fluency in written and spoken English, and aged between 18 and 70 years. Active psychosis, substance abuse and developmental delay were exclusion criteria.

A flow-chart of participant recruitment is presented in Figure 2.2. From this population, 79 (95.2%) met the study’s inclusion criteria (four patients were excluded: > 70 years = 1, non-fluent in English = 2, active psychosis = 1), and of these 79, 65 were able to attend the pretreatment research assessment representing a response rate of 82.3% of eligible participants. These 65 participants, referred to as Sample A, comprised 18 males (27.7%) and 47 females (72.3%), with a mean age of 37.9 years (SD = 12.6, range = 18 – 63). There was no significant age difference between males (M = 42.67, SD = 11.57) and females (M = 36.04, SD = 12.67), t(63) = 1.93, p > .05. Remaining demographic characteristics for Sample A participants are displayed in Table 2.2.
Figure 2.2. Recruitment of Sample A and Sample B participants.

Panic-Ag patients waiting for treatment from the SWAHS Anxiety Treatment & Research Unit recruited between December 2005 and September 2007

\[ N = 83 \]

1 = Exceeded age range

1 = Active psychosis

2 = Non-fluent in written or spoken English

Panic-Ag patients eligible to complete the pretreatment research appointment

\[ N = 79 \]

4 = Unable to attend treatment due to moving out of area or work commitments

10 = Unable to be assessed before treatment commenced.

Moved to Sample C

Sample A – Pre Treatment
Participants completing pretreatment research assessment

\[ N = 65 \]

3 = Did not commence treatment

14 = Dropped out within 3 sessions

Treated participants eligible for 6-month follow-up assessment

\[ N = 48 \]

3 = Unable to be contacted

1 = Withdrew from study

2 = Deceased

1 = Failed to attend 3 scheduled appointments

Sample B – Post Treatment
Participants completing 6-month follow-up assessment

\[ N = 41 \]
Chapter 2 – Development of the Multiple-Choice Knowledge Questionnaire

Of the 14 eligible participants unable to attend the pretreatment research appointment, four declined treatment due to work commitments or moving out of the area. These four were excluded from the study altogether. The remaining 10 participants could not attend the research assessment due to work (n = 5), study (n = 1), personal commitments (n = 2) or illness (n = 2). These 10 had a mean age of 38.9 years (SD = 10.9); there was nine females (90%) and one male (10%). They did not differ significantly from Sample A participants in age, t(73) = -.24, p > .05, or gender, χ²(1, N = 75) = 1.43, p > .05. These 10 participants were excluded from this aspect of the study, however they completed treatment, were assessed at 6-month follow-up and their data was used in subsequent aspects of the study described in chapters 3 and 4.

Following the pretreatment research assessment, participants were offered treatment for Panic-Ag. Of the 65 Sample A participants offered treatment, three (4.6%) did not commence and 14 (21.5%) dropped out within three sessions giving a final sample of 48 treatment completers. This sample represents a response rate of 60.7% of the total sample of eligible participants, and 73.8% of those offered treatment.

In respect to follow-up data, 41 (85.4%) of the 48 Sample A treatment completers were able to attend 6-month follow-up assessments. This subset of Sample A participants is referred to as Sample B. Of the seven treated participants unable to attend follow-up assessments, two died (one diabetic patient suffered a myocardial infarction, another died from suspected drug overdose), three could not be contacted, one withdrew from the study and one failed to attend three scheduled appointments. These seven participants did not differ significantly from Sample B on
pretreatment demographic or diagnostic characteristics. Sample B represents 51.9%
of the total eligible population, and 63.1% of those initially offered treatment.

Table 2.2 Pretreatment Demographic Characteristics for Samples A and B

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<tr>
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<th>Sample A</th>
<th>Sample B</th>
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<td><strong>N = 41</strong></td>
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<td>Marital status</td>
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<td>Divorced/Separated</td>
<td>9 (13.8%)</td>
<td>5 (12.2%)</td>
</tr>
<tr>
<td>Education in years (M \pm SD)</td>
<td>12.7 ± 2.8</td>
<td>13.0 ± 2.8</td>
</tr>
<tr>
<td>range = 7 – 19</td>
<td>range = 9 – 19</td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>23 (35.4%)</td>
<td>19 (46.3%)</td>
</tr>
<tr>
<td>Country of origin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>49 (75.4%)</td>
<td>30 (73.2%)</td>
</tr>
<tr>
<td>Other</td>
<td>16 (24.6%)</td>
<td>11 (26.8%)</td>
</tr>
<tr>
<td>Length of time in Australia in years(^a)</td>
<td>31.2 ± 15.6</td>
<td>34.2 ± 12.1</td>
</tr>
<tr>
<td>range = 1.5 – 57</td>
<td>range = 10 – 57</td>
<td></td>
</tr>
<tr>
<td>Duration of anxiety disorder in years (M \pm SD)</td>
<td>8.9 ± 11.2</td>
<td>6.8 ± 7.7</td>
</tr>
<tr>
<td>range = 0.25 – 50</td>
<td>range = 0.25 – 30</td>
<td></td>
</tr>
<tr>
<td>Previous treatments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medication</td>
<td>47 (72.3%)</td>
<td>29 (70.7%)</td>
</tr>
<tr>
<td>CBT</td>
<td>18 (27.7%)</td>
<td>11 (26.8%)</td>
</tr>
<tr>
<td>Counselling</td>
<td>31 (47.7%)</td>
<td>16 (39.0%)</td>
</tr>
<tr>
<td>Self-help books</td>
<td>31 (47.7%)</td>
<td>21 (51.2%)</td>
</tr>
<tr>
<td>Medication</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No medication</td>
<td>25 (38.5%)</td>
<td>17 (41.5%)</td>
</tr>
<tr>
<td>ADs only</td>
<td>12 (18.5%)</td>
<td>7 (17.1%)</td>
</tr>
<tr>
<td>BZs only</td>
<td>12 (18.5%)</td>
<td>8 (19.5%)</td>
</tr>
<tr>
<td>ADs &amp; BZs</td>
<td>15 (23.1%)</td>
<td>8 (19.5%)</td>
</tr>
<tr>
<td>Other</td>
<td>1 (1.5%)</td>
<td>1 (2.4%)</td>
</tr>
<tr>
<td>Primary diagnosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Panic Disorder without Agoraphobia</td>
<td>4 (6.2%)</td>
<td>3 (7.3%)</td>
</tr>
<tr>
<td>Panic Disorder with Agoraphobia</td>
<td>58 (89.2%)</td>
<td>36 (87.8%)</td>
</tr>
<tr>
<td>Agoraphobia without Panic Disorder</td>
<td>3 (4.6%)</td>
<td>2 (4.9%)</td>
</tr>
</tbody>
</table>
Chapter 2 – Development of the Multiple-Choice Knowledge Questionnaire

Table 2.2 (continued) Pretreatment Demographic Characteristics for Samples A and B

<table>
<thead>
<tr>
<th></th>
<th>Sample A</th>
<th>Sample B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 65</td>
<td>N = 41</td>
</tr>
<tr>
<td>Comorbidity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social phobia</td>
<td>22 (33.8%)</td>
<td>14 (34.1%)</td>
</tr>
<tr>
<td>GAD</td>
<td>34 (52.3%)</td>
<td>21 (51.2%)</td>
</tr>
<tr>
<td>OCD</td>
<td>5 (7.7%)</td>
<td>3 (7.3%)</td>
</tr>
<tr>
<td>Specific phobia</td>
<td>14 (21.5%)</td>
<td>7 (17.1%)</td>
</tr>
<tr>
<td>PTSD</td>
<td>6 (9.2%)</td>
<td>3 (7.3%)</td>
</tr>
<tr>
<td>Any comorbid anxiety disorder</td>
<td>49 (75.4%)</td>
<td>29 (70.7%)</td>
</tr>
<tr>
<td>Depressive disorder</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major depression</td>
<td>30 (46.2%)</td>
<td>19 (46.3%)</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>15 (23.1%)</td>
<td>6 (14.6%)</td>
</tr>
<tr>
<td>Any depressive disorder</td>
<td>36 (55.4%)</td>
<td>21 (51.2%)</td>
</tr>
<tr>
<td>No. comorbid diagnoses (M ± SD)</td>
<td>2.1 ± 1.5</td>
<td>2.0 ± 1.5</td>
</tr>
<tr>
<td>Range = 0 – 7</td>
<td>range = 0 – 5</td>
<td></td>
</tr>
</tbody>
</table>

Note. ADs = Antidepressants; BZs = Benzodiazepines; GAD = Generalised Anxiety Disorder; OCD = Obsessive Compulsive Disorder; PTSD = Post-traumatic Stress Disorder.

*Based on participants born outside Australia.

Sample B participants consisted of 11 males (26.8%) and 30 females (73.2%), with a total sample mean age of 37.8 years (SD = 11.6, range = 20 – 63). Again, no significant age differences existed between males (M = 40.00, SD = 9.06) and females (M = 37.03, SD = 12.06), t(39) = 0.72, p > .05. Remaining demographic characteristics of Sample B are displayed in Table 2.2. Comparisons between Sample B and the combined group of treatment non-starters and dropouts (n = 17) revealed Sample B participants were significantly more likely to be married, 61.0% vs. 23.5%, χ²(1, N = 58) = 6.74, p < .01, employed, 46.3% vs. 17.6%, χ²(1, N = 58) = 4.20, p < .05, and less likely to have received previous counselling, 39.0% vs. 76.5%, χ²(1, N = 58) = 6.74, p < .01. These differences suggest Sample B participants were a higher functioning sample relative to treatment dropouts and non-starters. However, no
significant differences existed on other demographic variables (age, sex, education, country of origin, duration of anxiety disorder, medication, other previous treatments), intelligence (assessed by Matrix Reasoning and the Wechsler Test of Adult Reading), comorbidity or self-report measures (described below) assessing frequency and severity of anxiety and depressive symptoms ($p > .05$).

**Measures**

*Personal Details Questionnaire.* A 12-item demographic questionnaire was administered to collect information on age, gender, ethnicity, length of time in Australia, marital status, education, employment, duration of anxiety disorder, previous anxiety treatments and psychotropic medication.

*Anxiety Disorders Interview Schedule for DSM-IV, Adult Version* (ADIS-IV, Brown et al., 1994). The ADIS-IV is a structured clinician-administered psychiatric interview assessing current and lifetime episodes of anxiety, mood, somatoform and substance use disorders based on DSM-IV criteria (American Psychiatric Association, 1994). Severity of symptoms is rated on dimensional scales from 0 to 8. Each diagnosis is assigned a clinical severity rating from 0 (*none*) to 8 (*very severe*) based on functional interference and distress associated with the disorder. Clinical severity ratings of four or more are of diagnostic significance, ratings of 1 to 3 are regarded as subclinical and a rating of 0 is assigned when no features of the disorder are present.

The ADIS-IV has demonstrated good to excellent reliability for the majority of anxiety and depressive disorders, with the exception of Dysthymia ($\kappa = .22 - .31$). Test-retest reliability of anxiety disorders ranged from .57 to .86 and reliability of Major Depression was also good ($\kappa = .59 - .67$). Inter-rater reliability of key
syndrome across anxiety and depressive disorders was generally sound \((r = .36 - .86, \text{mean } r = .69)\) (Brown, Di Nardo, Lehman, & Campbell, 2001). Evidence of convergent and discriminant validity has also been established with self-report measures of Panic-Ag, Social Phobia, GAD, OCD and depression loading onto the respective latent diagnostic factors (e.g., the checking subscale of an OCD questionnaire loaded .94 on the OCD latent factor) without cross-loading on latent factors of non-corresponding diagnoses (Brown, Chorpita, & Barlow, 1998).

*Number of Panic Attacks Assessed on the ADIS-IV (nPA-ADIS).* Interviewers assessed the number of DSM-IV defined panic attacks experienced in the previous month using the Panic Disorder module of the ADIS-IV (Brown et al., 1994).

*Panic Attack Sensation Severity Assessed on the ADIS-IV (PASS-ADIS).* Participants were interviewed regarding the severity of 14 physical and mental sensations experienced during an unexpected panic attack using the Panic Disorder module of the ADIS-IV (Brown et al., 1994). Items were rated on a 9-point visual analogue scale ranging from 0 (*none*) to 8 (*very severe*). Scores on this measure range from 0 to 112 with higher scores reflecting more severe symptoms. While the psychometric properties of this subscale have not been published, internal consistency estimates (Cronbach’s alpha) based on participants in the present study were sound, both at pretreatment and posttreatment \((\alpha = .81 - .92)\).

*Agoraphobic Cognitions Questionnaire – Frequency* (ACQ-Frequency, Chambless et al., 1984). The ACQ is a 14-item self-report questionnaire assessing frequency of thoughts concerning catastrophic consequences of anxiety (e.g., “I am going to pass out”, “I am going crazy”) when feeling anxious. Items are rated on a 5-point scale from 1 (*never*) to 5 (*always*). Total scores range from 14 to 70. It has sound reliability (internal consistency: \(\alpha = .80\), test-retest reliability: \(r = .79 - .86\))
and showed evidence of convergent \((r = .21 - .67, p < .01)\) and discriminant validity \((r = -.08 - -.14, p > .05)\) when correlated with theoretically related and unrelated measures, respectively. The ACQ is sensitive to treatment effects and clearly discriminates between normal and agoraphobic samples (Chambless et al., 1984). For the purpose of this thesis, the ACQ is subsequently referred to as ACQ-Frequency to differentiate it from the ACQ-Belief scale described below.

**Agoraphobic Cognitions Questionnaire – Belief (ACQ-Belief).** Belief in catastrophic cognitions was assessed using a modified version of the ACQ. Ratings of ACQ items were modified to assess belief in catastrophic cognitions across a 5-point Likert scale from 0 (do not believe at all), 1 (slightly believe), 2 (somewhat believe), 3 (mostly believe) to 4 (completely believe). Scores range from 0 to 56, with higher scores reflecting stronger beliefs. Cronbach’s alpha for this scale based on participants in the present study was sound \((\alpha = .87)\) and test-retest reliability (1-2 week re-test interval) was very high \((r = .95 - .96)\). Similar modifications to the ACQ have been used by Salkovskis et al. (2007) to assess belief in catastrophic cognitions.

**Mobility Inventory for Agoraphobia (MI, Chambless, Caputo, Jasin, Gracely, & Williams, 1985).** The MI is a 26-item self-report questionnaire assessing frequency of avoidance of common agoraphobic situations (e.g., supermarkets, enclosed spaces, trains). Items are rated on a 5-point scale from 1 (never avoid) to 5 (always avoid). Each item is rated twice according to when experienced (a) alone and (b) accompanied by a trusted companion. Scores for the alone subscale range from 26 to 130, while the accompanied subscale ranges from 25 to 125 (the “staying at home alone” item is not applicable for this subscale). Both subscales are highly internally consistent \((\alpha > .91)\) and correlate significantly with each other \((r = .67)\) as
well as independent measures of agoraphobia ($r = .44 – .66, p < .001$) and trait anxiety ($r = .25 – .38, p < .01$). The measure is highly sensitive to treatment effects and can successfully distinguish agoraphobic patients from socially phobic and normal control samples.

*Body Sensations Questionnaire* (BSQ, Chambless et al., 1984). The BSQ is a 17-item self-report questionnaire assessing fear of panic sensations. Items are rated on a 5-point scale from 1 (*not at all frightening*) to 5 (*extremely frightening*) with total scores ranging from 17 to 85. The BSQ has demonstrated high internal consistency (alpha = .87 – .88), moderate pretreatment stability (test-retest reliability = .66 – .67, retest interval = 6-31 days), and evidence of convergent ($r = .17 – .67, p < .05$) and discriminant validity ($r = -.19 – .11, p > .05$). BSQ scores decreased significantly with treatment and were able to successfully distinguish between agoraphobic patients and normal controls.

*Panic Belief Inventory* (PBI, Greenberg, 1989; Wenzel et al., 2006). The PBI is a self-report questionnaire consisting of 35 statements assessing strength of beliefs regarding unrealistic ideas about panic and its consequences. Factor analysis identified four subscales: *anticipatory anxiety* (e.g., “I must be able to reach my ‘support system’ at all times or a catastrophe could happen”), *physical catastrophes* (e.g., “A panic attack can give me a heart attack”), *emotional catastrophes* (e.g., “A panic attack can drive me insane”), and *self-deprecation* (e.g., “Having panic attacks means I’m weak, defective, or inferior”). Items are rated on a 6-point Likert scale from 1 (*totally disagree*) to 6 (*totally agree*). Analysis of the psychometric properties of the PBI revealed good internal consistency of the subscales ($\alpha = .82 – .91$) and of the measure as a whole ($\alpha = .95$). Significant moderate correlations were observed between the PBI and measures of anxiety demonstrating evidence of convergent
validity. Evidence of discriminant validity was achieved with a lack of association between the PBI and measures of depressive cognitions and suicidal ideation. The PBI has also been shown to be sensitive to treatment gains with scores across the four subscales decreasing significantly from pretreatment to posttreatment.

*Beck Depression Inventory – II* (BDI-II, Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report questionnaire assessing severity of depressive symptoms. Items are reflective of DSM-IV criteria for major depressive episode. Each symptom is rated on a 4-point severity scale from 0 to 3 such that total scores range from 0 to 63. The BDI-II has demonstrated high levels of internal consistency ($\alpha > .90$) and test-retest reliability ($r = .93$). It correlated well ($r > .50$) with self-report and clinician ratings of depression in clinical and non-clinical samples and was more strongly associated with measures of depression than anxiety (see Steer & Beck, 2000, for a review), attesting to its sound convergent and discriminant validity.

*Work and Social Adjustment Scale* (WSAS, Mundt, Marks, Shear, & Greist, 2002). The WSAS is a brief 5-item self-report measure assessing functional impairment resulting from a specified disorder across five domains of functioning (work, home management, social leisure activities, private leisure activities, maintaining close relationships). Items are rated on a 9-point visual analogue scale from 0 (*not at all impaired*) to 8 (*very severely impaired*). Total scores range from 0 to 40. The WSAS demonstrated good reliability with internal consistency estimates (Cronbach’s alpha) ranging between .79 to .94, pretreatment test-retest reliability of .73 and inter-rater reliability (patient vs. clinician ratings) of .81 to .86. WSAS scores correlated significantly with measures of symptom severity in patients with depression ($r = .63 – .77, p < .001$) and OCD ($r = .45 – .69, p < .001$). Furthermore, patients reporting higher clinical improvement ratings scored significantly lower on
the WSAS than those indicating little or no improvement. These results provide strong evidence of the measure’s convergent validity.

Matrix Reasoning subtest of the Wechsler Adult Intelligence Scale – Third Edition (WAIS-III, Wechsler, 1997). Matrix Reasoning is an untimed non-verbal measure of intelligence consisting of 26 items of increasing difficulty. Each item comprises a matrix of coloured geometric shapes in which one part of the pattern is omitted. Participants must identify the missing part that best completes the pattern from five choices. Raw scores range from 0 to 26. The psychometric properties of Matrix Reasoning are very sound (The Psychological Corporation, 1997). Reliability of the measure was high with split-half internal consistency coefficients ranging from .87 to .94 (M = .90) across different age groups and test-retest reliability (mean retest interval = 35 days) ranging from $r = .75 – .81$. Age-scaled scores correlated highly with full-scale IQ ($r = .75$), performance IQ ($r = .79$) and a similar independent non-verbal intelligence test (Raven’s Standardised Progressive Matrices, $r = .81$), demonstrating strong construct validity.

Wechsler Test of Adult Reading (WTAR, The Psychological Corporation, 2001). The WTAR is a verbal intelligence test originally designed to estimate premorbid IQ. It requires the reading and pronunciation of 50 irregularly spelled words of increasing difficulty (e.g., know, ogre, hyperbole) without requiring word comprehension. The words do not follow standard grapheme-to-phoneme translation and generally require prior knowledge of the words for correct pronunciation. Raw scores range from 0 to 50, however scores are adjusted according to age, sex, and level of education. The WTAR demonstrated excellent internal consistency ($r = .87 – .97$) and temporal stability (test-retest reliability, $r = .92 – .94$, mean retest interval = 35 days). It correlated highly with other measures of reading recognition ($r = .73 –$
.90) and verbal and full-scale IQ ($r = .75, .73$, respectively), providing evidence of good construct validity.

**Multiple-Choice Panic Disorder-Agoraphobia Treatment Knowledge Questionnaire (MC-PTKQ).** Constructed by the author for this thesis, the MC-PTKQ contains 29 items assessing knowledge of CBT for Panic-Ag. Scores on the MC-PTKQ range from 0 to 40. All information required by participants to correctly answer the questionnaire was provided by the therapists in the treatment program as detailed below.

**Procedure**

This phase of the study examined item refinement and formed part of the broader research question assessing relationships between treatment knowledge, beliefs and outcome. Ethics approval for all aspects of the study was provided from the Sydney West Area Health Service (SWAHS) Human Research Ethics Committee [HREC2005/5/4.12(2083)].

As part of the routine clinical procedures used in the clinic, participants completed an initial clinical diagnostic assessment using the ADIS-IV and a battery of self-report psychosocial measures consisting of the ACQ-Frequency, MI, PBI, BSQ, WSAS and BDI-II. These self-report measures were completed by participants in their own home after administration of the ADIS-IV. The ADIS-IV was administered by one of three clinical psychologists with between 8 and 15 years clinical experience. In addition, 16 clinical masters or doctoral students completing an internship at the SWAHS Anxiety Treatment and Research Unit between November 2005 and November 2007 also conducted ADIS-IV assessments following training in the administration of the instrument. Symptoms and ratings of
interns were discussed in weekly supervision meetings in order to ensure diagnoses were accurate and justified.

Prior to treatment, participants were invited to attend a research assessment where the purpose of the study was explained and informed consent was obtained. Participants were subsequently administered a battery of measures comprising the WTAR, Matrix Reasoning, MC-PTKQ (described above) in addition to an open-ended short-answer structured interview assessing knowledge of CBT for Panic-Ag, and questionnaires assessing beliefs about Panic-Ag and its treatment for research purposes (descriptions of these latter measures are provided in chapters 3 and 4, respectively).

Treated participants (Sample B) attending the 6-month follow-up (for brevity, referred to as posttreatment) research assessment were readministered the ADIS-IV, followed by the same pretreatment measures assessing treatment knowledge and beliefs. Pre- and posttreatment research assessments each lasted approximately 2 hours after which participants were thanked for their time and effort. A 6-month posttreatment assessment was selected to provide a more representative measure of treatment outcome for two reasons. Firstly, “honeymoon effects” observed immediately post treatment (e.g., rapid reduction of panic attack frequency) may not be sustained over time. Secondly, agoraphobic avoidance continues to decrease following treatment with ongoing application of CBT skills.

In both the pre- and posttreatment assessment procedures, previous feedback from expert reviewers suggested participants might feel threatened about answering questions related to treatment knowledge. Furthermore, it was highlighted that some participants lacking familiarity with multiple-choice measures may respond to questions impulsively without reading through all answer options. To minimise such
performance anxiety and impulsivity, the following instructions were provided and read aloud to participants immediately prior to completion of the knowledge measure.

Below are some questions exploring what people know about Cognitive Behaviour Therapy (CBT) for Panic Disorder and Agoraphobia. Some of the questions are quite difficult and you are not expected to know all of the answers. It does not matter if you do not know any of the answers or if you know them all. This is not a test or exam, we are just interested in what you currently know about CBT for Panic Disorder and Agoraphobia. The information obtained will be treated in the strictest confidence and used only for research.

**Important Instructions**

- Read each question carefully before answering
- Make sure you read **all** the options before making your selection
- Circle the letter of the answer that you think is most correct
- Circle only one answer per question
- If you think you do not know the answer to a question, circle ‘Don’t know’ rather than simply guess
- Do not spend too long on any question

Following explanation and clarification of instructions, participants were asked to complete the questionnaire alone in a quiet office unaided by textbooks or material relevant to treatment of Panic-Ag.

**Treatment**

Treatment was based on the cognitive model of Panic-Ag (Clark, 1986) and focussed on correcting catastrophic misinterpretations of bodily sensations through a
range of cognitive and behavioural techniques. Therapy was conducted in a group-based format and involved eight sessions over 8 consecutive weeks. Each session ran for 2½ hours with the exception of Session 5, which consisted of a 4-hour in vivo exposure session. A total of eight groups were run comprising between six and 10 patients per group. Treatment was administered by one of three registered clinical psychologists experienced in CBT for anxiety disorders, in combination with two intern clinical psychologists in their second or third year of a masters or doctoral degree in clinical psychology (16 interns in total). Interns were supervised weekly to ensure therapist competence. Of the eight treatment groups, six were run by the author, and the other two clinical psychologists each conducted one group. Treatment was manualised and all clinicians utilised the same manual.

The treatment manual was based on procedures outlined in a number of published texts on Panic-Ag (Andrews et al., 2003; Barlow, 2001; Clark, 1986, 1996, 1999; Hackmann, 2004; Hawton et al., 1989; Salkovskis et al., 1996). Session 1 introduced patients to the role of catastrophic cognitions in maintaining anxiety and panic attacks and provided psychoeducation surrounding the nature of anxiety and the role of the fight/flight response. Session 2 examined the impact of hyperventilation on the production of panic sensations and accurate medical information was provided to challenge patients’ catastrophic misinterpretations. Session 3 focussed on identifying and challenging catastrophic cognitions through examining the realistic probability and cost of feared outcomes and behavioural experiments. Session 4 discussed graded exposure to feared situations and sensations. In-session interoceptive exposure was conducted (e.g., hyperventilation, breathing through a straw, spinning). Session 5 involved a 4 hr exposure session where patients tested their catastrophic cognitions in vivo (e.g., in shopping centres,
trains, lifts), initially accompanied or prompted by the therapist and then independently. Safety seeking behaviours were gradually withdrawn to reinforce the lack of feared consequences. Sessions 6 and 7 involved revision of concepts, further behavioural experiments, cognitive challenging and planning of exposure goals. Relapse prevention strategies were discussed in Session 8. Patients were given weekly homework assignments corresponding to session content which was reviewed at the beginning of the following session.

**Statistical Analyses**

SPSS for Windows Version 15.0 (SPSS Inc., 2006) was used to conduct statistical analyses. Initial indices of item difficulty, discrimination and reliability were based on Sample A participants. Item difficulty indices, or $p$ values, were calculated by dividing the number of participants responding correctly to the item by the total number of participants. Item discrimination indices were obtained by firstly constructing extreme groups consisting of participants scoring in the upper and lower 33.3% of the questionnaire. The use of thirds over quartiles provided a more stringent evaluation of item discrimination. Scores on the knowledge measure ranged from 0 to 39 (out of a possible 40). The lower group consisted of 22 participants scoring 0-14, while the upper group comprised 22 participants scoring 23-39. A choice-distribution table was generated for each item to determine percentage of participants answering the item correctly in the upper and lower groups. The discrimination index, or $D$ value, was calculated by subtracting the percentage of participants in the lower group responding correctly to the item from the percentage scoring correctly in the upper group. Reliability of individual items was determined using Cronbach’s alpha and calculating the alpha coefficient when the item was
excluded from the total scale (alpha if item deleted). An increase in alpha of .01 was selected as a conservative cut-off to identify unreliable items.

**Results**

Table 2.3 displays indices of item difficulty and discrimination and reliability coefficients for the 29 items of the MC-PTKQ. Item difficulty indices ranged between .08 to .72, with an average of .38. As participants consisted of patients due to commence treatment, all item difficulty indices fell within the appropriate range of .00 – .75 (Anastasi, 1988).

Eleven items (items 2, 8, 11, 12, 17, 18, 21, 23, 25, 26, 27) had $p$ values below .30 indicating they were difficult (Meadows et al., 1988). To determine whether these items became easier with provision of treatment, pre- and posttreatment item difficulty indices were calculated and compared among Sample B participants. As can be seen from Table 2.4, scores increased significantly for all items following treatment with the exception of item 17, indicating items became easier. Item 17 assessed participants’ ability to understand the concept of overestimating the probability of potential events and to differentiate it from overestimating the cost of such events (see Appendix B). This suggests that participants confused these concepts and treatment was unhelpful in clarifying their meaning. Posttreatment difficulty indices exceeded .30 for most items, with the exceptions of items 17 and 18. However, these difficult items were retained so the contribution of such knowledge to treatment outcome could be examined.

Item discrimination indices, or $D$ values, for Sample A ranged between 9 and 86, with an average $D$ value of 50.41 (see Table 2.3) indicating most items were able to discriminate well between high and low scoring participants. Only three items
Table 2.3 *Index of Difficulty (p), Index of Discrimination (D), and Alpha Coefficients for the 29-Item Multiple-Choice Panic Disorder-Agoraphobia Treatment Knowledge Questionnaire (MC-PTKQ) for Sample A (N = 65)*

<table>
<thead>
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<th>Item no.</th>
<th>Item difficulty index</th>
<th>Upper group</th>
<th>Lower group</th>
<th>Item discrimination index</th>
<th>Percentage passing</th>
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<td>.52</td>
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<td>27</td>
<td>55</td>
<td>.909</td>
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<td>82</td>
<td>32</td>
<td>50</td>
<td>.909</td>
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<tr>
<td><strong>Total</strong></td>
<td><strong>.38</strong></td>
<td><strong>63.72</strong></td>
<td><strong>13.31</strong></td>
<td><strong>50.41</strong></td>
<td><strong>.911</strong></td>
<td></td>
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</table>
Table 2.4 Pre- and Posttreatment Item Difficulty Indices (p) for MC-PTKQ Items With Pretreatment Indices of \( p < .30 \) for Sample B (\( N = 41 \))

<table>
<thead>
<tr>
<th>Item No.</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>( p )</td>
<td>( p )</td>
</tr>
<tr>
<td>2</td>
<td>.37</td>
<td>.85***</td>
</tr>
<tr>
<td>8</td>
<td>.12</td>
<td>.51***</td>
</tr>
<tr>
<td>11</td>
<td>.24</td>
<td>.66***</td>
</tr>
<tr>
<td>12</td>
<td>.24</td>
<td>.51**</td>
</tr>
<tr>
<td>17</td>
<td>.27</td>
<td>.27</td>
</tr>
<tr>
<td>18</td>
<td>.10</td>
<td>.29*</td>
</tr>
<tr>
<td>21</td>
<td>.22</td>
<td>.49***</td>
</tr>
<tr>
<td>23</td>
<td>.22</td>
<td>.49***</td>
</tr>
<tr>
<td>25</td>
<td>.29</td>
<td>.61***</td>
</tr>
<tr>
<td>26</td>
<td>.27</td>
<td>.78***</td>
</tr>
<tr>
<td>27</td>
<td>.27</td>
<td>.51***</td>
</tr>
</tbody>
</table>

*Note.* \( *p < .05. \) **\( p < .01. \) ***\( p < .001. \)

(items 7, 8, 17) had unacceptably low \( D \) values (< 20), however these three items also had \( p \) values below .35, indicating a floor effect made it difficult to discriminate between participants at pretreatment. As scores on item 8 increased following treatment it was retained. Posttreatment \( D \) values (based on Sample B) for items 7 (upper group = 39, lower group = 8) and 17 (upper group = 46, lower group = 15) were each 31, indicating that these items were able to discriminate between high and low scoring participants following treatment. Hence, items 7 and 17 were also retained. Internal consistency of the 29-item knowledge questionnaire was high (\( \alpha = .911 \)). The alpha coefficient did not substantially increase following deletion of any item (alpha if item deleted = .903 – .912), indicating all 29 items were internally consistent (see Table 2.3).
Discussion

This study aimed to develop a multiple-choice measure assessing patients’ knowledge of CBT for Panic-Ag, (Multiple-Choice Panic Disorder-Agoraphobia Treatment Knowledge Questionnaire, MC-PTKQ). Thirty-nine items were generated and reviewed for relevance, comprehensibility and accuracy. Reviewers’ feedback resulted in reducing the questionnaire by 10 items to a set of 29 items. Results of item difficulty, discrimination and reliability analyses on these 29 items indicated that all items were sound, hence all items were retained.

The difficulty, discrimination and reliability ratings obtained in this study must be interpreted with caution. Firstly, over one quarter of participants reported having previously received CBT. However, prior exposure to CBT varies considerably according to factors such as nature of presenting problem (Panic-Ag, depression, general anxiety), therapy intensity (3 sessions vs. 12 sessions) as well as therapist orientation and experience. These variables could not be ascertained retrospectively yet may have influenced item difficulty, discrimination and reliability ratings to some extent.

Secondly, as Sample B comprised only half the number of eligible participants, the posttreatment item difficulty indices obtained in this study may not generalise to the larger population of Panic-Ag patients. Sample B participants were more likely to be married, employed and less likely to have previously received counselling than treatment dropouts and non-starters thereby comprising a higher functioning sample. Consequently, a more representative sample of Panic-Ag patients may score substantially lower on the MC-PTKQ after treatment than Sample B.
Nevertheless, this study provides the first attempt to develop a measure assessing knowledge of CBT for Panic-Ag using a multiple-choice format. However, multiple-choice measures are not without their shortcomings. The next chapter attempts to address such disadvantages through the development of a parallel knowledge interview where the psychometric properties of both knowledge measures will also be examined.
# Chapter 3

**Development of the Knowledge Interview**

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<th>Section</th>
<th>Page</th>
</tr>
</thead>
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<td>Scoring</td>
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<tr>
<td>Analysis of Items and Final Reduction of Item Pool</td>
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<td>EVALUATION OF THE PSYCHOMETRIC PROPERTIES OF THE KNOWLEDGE MEASURES</td>
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<td>Measures</td>
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<td>Construct Validity</td>
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<tr>
<td>Discussion</td>
<td>138</td>
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</table>
Development of the Interview of Panic-Ag Treatment Knowledge (Int-PTK)

This chapter describes the development of a structured knowledge interview designed specifically to confirm findings from the MC-PTKQ using a more sensitive format as discussed earlier. As noted, interview methods permit a more sensitive assessment of knowledge than multiple-choice and other written measures. In addition, they not only maximise the potential to distinguish between partial and full knowledge of concepts but also reduce the impact of correct guesses on total knowledge scores. For these reasons, an open-ended short-answer structured interview was developed as a supplementary assessment of CBT knowledge for Panic-Ag.

Construction of the multiple-choice knowledge questionnaire (MC-PTKQ) described in chapter 2 laid the foundations for the development of the Interview of Panic Disorder-Agoraphobia Treatment Knowledge (Int-PTK). That is, of the three phases involved in the development of a knowledge measure, phases one (development of the item pool) and two (expert review of items and initial item refinement) had already been conducted. Interview questions were therefore identical in content to the 29 multiple-choice questionnaire items but restructured into 24 questions to accommodate the open-ended short-answer format.

The Int-PTK (see Appendix C) comprised 24 questions across domains of psychoeducation (questions 1a, 1b, 2a, 2b, 2c), cognitive therapy (questions 3a, 3b, 3c, 4, 5a, 5b, 5c, 5d), role of avoidance (questions 6a, 6b, 6c, 6d) and exposure therapy (questions 7a, 7b, 7c, 7d, 8a, 8b, 8c). Examples of interview questions for each domain appear in Figure 3.1. The interview was tightly scripted. The
interviewer, an experienced clinical psychologist, read each question aloud to participants irrespective of their experience with CBT for Panic-Ag.

**Figure 3.1.** Example questions from the Int-PTK for each knowledge domain.

<table>
<thead>
<tr>
<th>Psychoeducation</th>
</tr>
</thead>
<tbody>
<tr>
<td>▪ What is the Fight or Flight Response and what is its purpose?</td>
</tr>
<tr>
<td>▪ Name 5 symptoms that can be caused by hyperventilating.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cognitive Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>▪ According to the CBT approach, why are panic attacks/panic sensations so frightening?</td>
</tr>
<tr>
<td>▪ According to the CBT approach, if you are overestimating the probability, how could you go about decreasing your probability estimates?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Role of Avoidance</th>
</tr>
</thead>
<tbody>
<tr>
<td>▪ According to the CBT approach, how does avoidance maintain fear and anxiety?</td>
</tr>
<tr>
<td>▪ According to the CBT approach, what is the problem with using safety seeking behaviours?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exposure Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>▪ According to the CBT approach, if your behavioural experiment was too hard, describe 3 ways to make it easier for yourself.</td>
</tr>
<tr>
<td>▪ According to the CBT approach, what is the purpose of deliberately bringing on panic sensations (e.g., dizziness, heart palpitations, lightheadedness, shortness of breath)?</td>
</tr>
</tbody>
</table>

**Administration**

To ensure a thorough assessment of knowledge had been obtained, participants were advised they would be probed exhaustively for each question until they indicated they had no more to report. As the interview required participants to discuss a range of (often unfamiliar) CBT concepts in front of the interviewer, the potential to feel anxious, intimidated and/or threatened was also acknowledged. As such, the following instructions were provided prior to commencing the interview.
This section involves a brief interview about what you currently know about cognitive behaviour therapy for panic disorder and agoraphobia. When assessing people’s knowledge, their first response will often capture, say, only 70% of what they know about the topic. If I want to get the remaining 30% I have to probe further by asking “Is there anything else you would like to add?” until they say “no”. I will use this same procedure with you for every question, regardless of your response.

Sometimes people feel threatened when being asked these types of questions. As you have not started treatment you are not expected to know the answers to these questions. However, whatever you say is useful and interesting for the purpose of this research and your responses are completely confidential.

In addition, less confident participants have been noted to prefer to say nothing or “I don’t know” rather than risk responding incorrectly and appearing foolish. In such cases, prompts were used to encourage responding (e.g., “Just have a go”). Care was taken to query vague or unclear responses to ensure participants were not being disadvantaged by reduced verbal abilities (e.g., “Tell me more about that,” “Give me an example of what you mean”). Similarly, ambiguous terms or phrases used by participants were queried to determine their exact understanding of concepts. For example, the term negative thoughts, when queried, implied both catastrophic misinterpretations of physical sensations and negative (non-panic) memories of the past by different patients. Administration of the Int-PTK lasted approximately 15 to 25 min, with less knowledgeable participants typically taking less time. Interviews were audiotaped and transcribed to assist scoring.
Scoring

Correct responses to questions consisted of either core CBT concepts (e.g., purpose of the fight or flight response, role of catastrophic cognitions in the maintenance of panic) or identification of a specific number of points (e.g., three symptoms of the fight or flight response and their function, five symptoms that can be caused by hyperventilating). Ten treated and 10 untreated participant interview transcripts were analysed and responses classified across a 5-point continuum as operationalised in Figure 3.2 below.

**Figure 3.2.** Operationalisation of scoring for Int-PTK items.

<table>
<thead>
<tr>
<th>Score</th>
<th>Knowledge rating</th>
<th>Necessary criteria</th>
</tr>
</thead>
</table>
| 4     | Excellent        | ▪ All necessary information provided  
|       |                  | ▪ Excellent understanding of core components  
|       |                  | ▪ Required number of points |
| 3     | Very Good        | ▪ Majority of information provided  
|       |                  | ▪ Sound understanding of core components  
|       |                  | ▪ One point overlapping or similar to another |
| 2     | Good             | ▪ Moderate understanding of core components  
|       |                  | ▪ Missing one concept or 1-2 points |
| 1     | Minimal          | ▪ Minimal information provided  
|       |                  | ▪ Vague reference to core concept or elaboration that indicates misunderstanding  
|       |                  | ▪ Missing 3 or more points |
| 0     | Poor             | ▪ Wholly incorrect information provided  
|       |                  | ▪ Poor understanding of core concept or “Don’t know” response  
|       |                  | ▪ Missing all points |

Scores across the 24 items therefore ranged from 0 to 96. Examples of 0 to 4 point responses are displayed in Figure 3.3.
**Figure 3.3.** Examples of 0 to 4-point responses.

**What is the fight or flight response and what is its purpose?**

<table>
<thead>
<tr>
<th>Score</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>The fight or flight response is a term used when people are put into a threatening situation, whether that be a real threat or a perceived threat, and it’s when your body activates the fight or flight response, which is basically whether you’re going to fight the danger that’s ahead of your or flee it, run away from it. Even just a distressing thought can trigger it off. Its purpose is to protect you from danger, so it’s a natural response.</td>
</tr>
<tr>
<td>3</td>
<td>Fight or flight response is if you’re put into a dangerous situation as you see it, you fight aggressively to survive or you flee the situation for the same reason, to survive…I guess it’s a trigger for you to survive.</td>
</tr>
<tr>
<td>2</td>
<td>It’s when you think you’re in a dangerous situation, it brings on fear and palpitations, sweating and lightheadedness, that kind of thing and it’s…to alert you to danger…To kind of get you out of danger when you’re in a dangerous situation.</td>
</tr>
<tr>
<td>1</td>
<td>Either run away or you fight it…To overcome the panic and know that nothing’s going to happen, you just have to fight it and not give in…your body’s responding to the panic and your body’s either going to run away and keep going until it ends.</td>
</tr>
<tr>
<td>0</td>
<td>Something about fighting the symptoms, trying to just fight them and get through it, and then the flight is sort of when you’ve gotten over it.</td>
</tr>
</tbody>
</table>

**According to the CBT approach, why are panic attacks/panic sensations so frightening?**

<table>
<thead>
<tr>
<th>Score</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>It’s because of what you think about them. If you think that the physical symptoms are because of a heart attack or a stroke or something, that’s quite frightening…just from your thoughts about them.</td>
</tr>
<tr>
<td>3</td>
<td>Well they’re usually frightening from the point of view, it’s more your thoughts. So your thoughts are usually quite negative more than anything else.</td>
</tr>
<tr>
<td>2</td>
<td>They’re frightening because you don’t understand what’s going on…you say “Is this going to be forever, what’s wrong with me?”</td>
</tr>
<tr>
<td>1</td>
<td>You feel like you don’t have control of the feelings and the situation and they feel like they’re just gonna keep on getting worse.</td>
</tr>
<tr>
<td>0</td>
<td>Because of the symptoms….Because the symptoms are not pleasant so you don’t want them.</td>
</tr>
</tbody>
</table>

To assess inter-rater reliability of the scoring system, an independent rater was initially trained in the scoring system through reviewing five pretreatment and
five posttreatment Int-P TK transcripts. Following review of these 10 transcripts, the 
reviewer independently scored an additional five transcripts blind to participants’ 
treatment status (i.e., pretreatment vs. posttreatment) and symptom severity. Ratings 
to individual items were compared and discrepancies discussed until consensus was 
achieved. Finally, blind ratings were made to an additional random sample of 20 Int-
PTK transcripts. Intraclass correlations (ICC) across the 20 transcripts ranged 
between .65 and .96, \( p < .001 \), (mean ICC = .84) indicating satisfactory inter-rater 
reliability.

**Analysis of Items and Final Reduction of Item Pool**

The next phase in the development of the Int-P TK involved analysing items 
to remove those that were too easy, poor discriminators or unreliable. Although all 
items in the multiple-choice version were deemed appropriate, differences in the 
administration and phrasing of questions in the knowledge interview may have 
affected difficulty, discrimination and reliability indices.

Participants, measures and assessment procedures used to determine such 
indices were identical to those for the multiple-choice version described in chapter 2. 
The Int-P TK was administered prior to the MC-PTKQ to prevent information from 
the latter measure influencing participants’ responses. Item difficulty, discrimination 
and reliability indices were based on Sample A participants. Scores for interview 
items were collapsed into two categories to calculate item difficulty and 
discrimination indices. Responses scored 0 and 1 were coded as incorrect, while 
scores of 2, 3 and 4 were coded as correct. As can be seen from Table 3.1, item 
difficulty indices (\( p \) values) ranged from .05 to .69 indicating all items fell within the
appropriate range (.00 – .75). The mean difficulty rating was .31 indicating that on average, interview items were quite difficult for patients to answer at pretreatment.

Table 3.1 Index of Difficulty (p), Index of Discrimination (D), and Alpha Coefficients for the 24-Item Interview of Panic Disorder-Agoraphobia Treatment Knowledge (IntPTK) for Sample A (N = 65)

<table>
<thead>
<tr>
<th>Item no.</th>
<th>p</th>
<th>Item difficulty index</th>
<th>Item discrimination index</th>
<th>Alpha if item deleted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Upper group</td>
<td>Lower group</td>
<td>D</td>
</tr>
<tr>
<td>1a</td>
<td>.42</td>
<td></td>
<td>73</td>
<td>14</td>
</tr>
<tr>
<td>1b</td>
<td>.25</td>
<td></td>
<td>55</td>
<td>0</td>
</tr>
<tr>
<td>2a</td>
<td>.49</td>
<td></td>
<td>86</td>
<td>18</td>
</tr>
<tr>
<td>2b</td>
<td>.37</td>
<td></td>
<td>68</td>
<td>9</td>
</tr>
<tr>
<td>2c</td>
<td>.69</td>
<td></td>
<td>91</td>
<td>41</td>
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<tr>
<td>3a</td>
<td>.55</td>
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<td>0</td>
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<tr>
<td>5a</td>
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<td>5d</td>
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<td>0</td>
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<tr>
<td>6a</td>
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</tr>
<tr>
<td>6b</td>
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<td>14</td>
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<tr>
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<tr>
<td>Total</td>
<td>.31</td>
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<td>57.0</td>
<td>9.5</td>
</tr>
</tbody>
</table>
Thirteen of the 24 items (54%) had \( p \) values below .30, suggesting participants found the interview more difficult than the multiple-choice version (c.f. 38% of items with \( p \) values < .30). Examination of difficulty indices for pre- and posttreatment data confirmed the provision of treatment improved performance; \( p \) values increased significantly with treatment for all such items and exceeded .30 (see Table 3.2).

<table>
<thead>
<tr>
<th>Question</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1b</td>
<td>.29</td>
<td>.63*</td>
</tr>
<tr>
<td>3b</td>
<td>.29</td>
<td>.76*</td>
</tr>
<tr>
<td>4</td>
<td>.10</td>
<td>.49*</td>
</tr>
<tr>
<td>5c</td>
<td>.17</td>
<td>.51*</td>
</tr>
<tr>
<td>5d</td>
<td>.07</td>
<td>.42*</td>
</tr>
<tr>
<td>6a</td>
<td>.24</td>
<td>.73*</td>
</tr>
<tr>
<td>6d</td>
<td>.12</td>
<td>.73*</td>
</tr>
<tr>
<td>7a</td>
<td>.24</td>
<td>.83*</td>
</tr>
<tr>
<td>7b</td>
<td>.20</td>
<td>.93*</td>
</tr>
<tr>
<td>7c</td>
<td>.20</td>
<td>.90*</td>
</tr>
<tr>
<td>8a</td>
<td>.24</td>
<td>.90*</td>
</tr>
<tr>
<td>8b</td>
<td>.24</td>
<td>.93*</td>
</tr>
<tr>
<td>8c</td>
<td>.12</td>
<td>.49*</td>
</tr>
</tbody>
</table>

*Note. *\( p < .001.\)

Sample A participants scoring in the upper and lower thirds of the Int-PTK were used to construct extreme groups for the evaluation of item discrimination. Scores ranged from 0 to 58 (out of a possible 96). The lower third \( (n = 22) \) scored 0-16 and the upper third \( (n = 22) \) scored 28-58. Item discrimination indices, or \( D \)
values, ranged between 14 and 86, with a mean of 47.5 (see Table 3.1) indicating most items were able to adequately discriminate between high and low scoring participants. Only two items (items 4 and 5d) had $D$ values below 20, however these items also had very low pretreatment $p$ values (.06 and .05, respectively), indicating a floor effect made item discrimination difficult. Posttreatment discrimination values were calculated on items 4 and 5d using Sample B data. $D$ values of 54 and 85 were obtained for items 4 (upper group = 77, lower group = 23) and 5d (upper group = 85, lower group = 0), respectively, indicating both items discriminated well between high and low scoring participants after treatment. Internal consistency of the Int-LPTK was high ($\alpha = .914$). The alpha coefficient did not substantially increase following deletion of any item (alpha if item deleted = .907 – .916), indicating all 24 items were internally consistent. On the basis of these indices, all items were retained.

**Summary**

This section described the development of a structured interview assessing knowledge of CBT for Panic-Ag (the Interview of Panic Disorder-Agoraphobia Treatment Knowledge, Int-PTK). The Int-PTK was constructed to obtain a more sensitive assessment of knowledge than the MC-PTKQ. Items for the Int-PTK borrowed heavily from the development of the MC-PTKQ described in chapter 2. As found for the MC-PTKQ, item difficulty, discrimination and reliability ratings were sound hence all items were retained.

Several limitations of the Int-PTK must be acknowledged. Firstly, as described in chapter 2, over one quarter of participants reported prior treatment with CBT which may have influenced indices of difficulty, discrimination and reliability. Secondly, treated participants’ were interviewed immediately following
administration of the ADIS-IV. Hence the interviewer was not blind to participants’ diagnostic status (or the research question) which may have biased the interview in some way. However, interviews were scored from decoded transcripts, thus raters were blind to participants’ treatment status (pretreatment vs. posttreatment) and symptom severity. Moreover, intraclass correlations revealed the inter-rater reliability of the scoring was sound.

Evaluation of the Psychometric Properties of the Knowledge Measures

This section describes the reliability and validity of the MCLPTKQ and Int-PTK. Reliability data was collected using a pretreatment and posttreatment sample. Convergent and discriminant validity was assessed by comparing total knowledge scores with variables theoretically related and unrelated to patient knowledge, respectively. The known-group method (Murphy & Davidshofer, 1994) was used to test the ability of the MCLPTKQ to discriminate between independent samples of pretreatment and posttreatment patients as well as between clinical psychologists and intern clinical psychologists to further support the construct validity of the measures. Finally, the measures’ sensitivity to change was assessed as a final marker of validity.
Method

Participants

A summary of patient and clinician samples used to evaluate the psychometric properties of the knowledge measures is displayed in Table 3.3.

Patient Samples

All patient participants were recruited from the SWAHS Anxiety Treatment and Research Unit, Cumberland Hospital. Three patient samples were used, hereafter referred to as Samples A, B and C. Samples A and B were identical to those described in chapter 2. Sample C was recruited from two groups of treated Panic-Ag patients assessed 6 to 12 months posttreatment (again, for brevity referred to as posttreatment): (a) an independent group of 40 consecutive patients who completed treatment prior to December 2005 and, as described in chapter 2, (b) 10 patients receiving treatment from the clinic between December 2005 and September 2007 who were unavailable for the pretreatment research assessment. Inclusion criteria consisted of primary pretreatment DSM-IV diagnosis of Panic Disorder and/or Agoraphobia, fluency in written and spoken English and aged 18 to 70 years. Exclusion criteria involved current substance abuse, active psychosis and developmental delay.
### Table 3.3 Summary Characteristics of Participant Samples Used to Evaluate the Psychometric Properties of the Knowledge Measures

<table>
<thead>
<tr>
<th>Sample Description</th>
<th>Purpose</th>
<th>N</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample A - Pretreatment Panic-Ag patients</td>
<td>Reliability and validity testing</td>
<td>65</td>
<td>Patients from the SWAHS Anxiety Treatment &amp; Research Unit on the waiting list for treatment</td>
</tr>
</tbody>
</table>
| Sample B - Panic-Ag patients who completed treatment | Validity testing  
Sensitivity to change | 41 | Subset of Sample A participants who completed treatment and were re-assessed 6-12 months posttreatment |
| Sample C - Panic-Ag patients who completed treatment | Reliability and validity testing  
Replication analyses  
Known-group validity | 35 | Independent sample of patients from the SWAHS Anxiety Treatment & Research Unit assessed 6-12 months posttreatment |
| Clinical psychologists | Known-group validity | 16 | University of Sydney (n = 3)  
Department of Medical Psychology, Westmead Hospital (n = 13) |
| Second and third year interns | Known-group validity | 28 | Intern clinical psychologists enrolled in their second or third year of a post-graduate degree in clinical psychology who completed a field placement at the SWAHS Anxiety Treatment & Research Unit |
| Entry-level interns | Known-group validity | 20 | Intern clinical psychologists enrolled in the Doctorate of Clinical Psychology degree from the University of Sydney assessed at the commencement of their first year |
A flow-chart of Sample C recruitment is depicted in Figure 3.4. One patient was excluded due to English non-fluency. Of the remaining 49 patients, four (8.2%) declined participation, five (10.2%) were unable to be contacted, another five (10.2%) failed to attend three scheduled assessment appointments, leaving 35 patients available for follow-up assessment which constituted Sample C. Sample C represents a response rate of 71.4% of the total sample of eligible posttreatment participants.

*Figure 3.4. Recruitment of Sample C participants.*
Demographic characteristics of Samples A, B and C are displayed in Table 3.4. For ease of comparison, data from Table 2.2 is repeated here.

Table 3.4 Pretreatment Demographic Characteristics for Samples A, B and C

<table>
<thead>
<tr>
<th></th>
<th>Sample A</th>
<th>Sample B</th>
<th>Sample C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) ($M \pm SD$)</td>
<td>37.9 ± 12.6</td>
<td>37.8 ± 11.6</td>
<td>37.7 ± 11.4</td>
</tr>
<tr>
<td></td>
<td>range = 18-63</td>
<td>range = 20-63</td>
<td>range = 21-68</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>18 (27.7%)</td>
<td>11 (26.8%)</td>
<td>7 (20.0%)</td>
</tr>
<tr>
<td>Females</td>
<td>47 (72.3%)</td>
<td>30 (73.2%)</td>
<td>28 (80.0%)</td>
</tr>
<tr>
<td>Country of origin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>49 (75.4%)</td>
<td>30 (73.2%)</td>
<td>28 (80.0%)</td>
</tr>
<tr>
<td>Other</td>
<td>16 (24.6%)</td>
<td>11 (26.8%)</td>
<td>7 (20.0%)</td>
</tr>
<tr>
<td>Length of time in</td>
<td>31.2 ± 15.6</td>
<td>34.2 ± 12.1</td>
<td>18.1 ± 10.3</td>
</tr>
<tr>
<td>Australia (years) a</td>
<td>range = 1.5 – 57</td>
<td>range = 10 – 57</td>
<td>range = 6 – 32</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never married</td>
<td>25 (38.5%)</td>
<td>11 (26.8%)</td>
<td>9 (25.7%)</td>
</tr>
<tr>
<td>Married/de facto</td>
<td>31 (47.7%)</td>
<td>25 (61.0%)</td>
<td>23 (65.7%)</td>
</tr>
<tr>
<td>Divorced/Separated</td>
<td>9 (13.8%)</td>
<td>5 (12.2%)</td>
<td>3 (8.6%)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>12.7 ± 2.8</td>
<td>13.0 ± 2.8</td>
<td>13.5 ± 2.6</td>
</tr>
<tr>
<td>($M \pm SD$)</td>
<td>range = 7 – 19</td>
<td>range = 9 – 19</td>
<td>range = 10 – 19</td>
</tr>
<tr>
<td>Employed</td>
<td>23 (35.4%)</td>
<td>19 (46.3%)</td>
<td>22 (62.9%)</td>
</tr>
<tr>
<td>Duration of anxiety</td>
<td>8.9 ± 11.2</td>
<td>6.8 ± 7.7</td>
<td>6.3 ± 6.9</td>
</tr>
<tr>
<td>disorder (years)</td>
<td>range = .25 – 50</td>
<td>range = .25 – 30</td>
<td>range = .5 – 30</td>
</tr>
<tr>
<td>($M \pm SD$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous treatments b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medication</td>
<td>47 (72.3%)</td>
<td>29 (70.7%)</td>
<td>19 (54.3%)</td>
</tr>
<tr>
<td>CBT</td>
<td>18 (27.7%)</td>
<td>11 (26.8%)</td>
<td>35 (100%)</td>
</tr>
<tr>
<td>Counselling</td>
<td>31 (47.7%)</td>
<td>16 (39.0%)</td>
<td>17 (48.6%)</td>
</tr>
<tr>
<td>Self-help books</td>
<td>31 (47.7%)</td>
<td>21 (51.2%)</td>
<td>16 (45.7%)</td>
</tr>
<tr>
<td>Medication status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No medication</td>
<td>25 (38.5%)</td>
<td>17 (41.5%)</td>
<td>18 (51.4%)</td>
</tr>
<tr>
<td>ADs only</td>
<td>12 (18.5%)</td>
<td>7 (17.1%)</td>
<td>11 (31.4%)</td>
</tr>
<tr>
<td>BZs only</td>
<td>12 (18.5%)</td>
<td>8 (19.5%)</td>
<td>1 (2.9%)</td>
</tr>
<tr>
<td>ADs &amp; BZs</td>
<td>15 (23.1%)</td>
<td>8 (19.5%)</td>
<td>5 (14.3%)</td>
</tr>
<tr>
<td>Other</td>
<td>1 (1.5%)</td>
<td>1 (2.4%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Number of medications</td>
<td>1.0 ± 0.9</td>
<td>1.0 ± 0.9</td>
<td>0.6 ± 0.8</td>
</tr>
<tr>
<td>($M \pm SD$)</td>
<td>Range = 0 – 3</td>
<td>Range = 0 – 3</td>
<td>Range = 0 – 2</td>
</tr>
</tbody>
</table>
Table 3.4 (continued) Pretreatment Demographic Characteristics Across Samples A, B and C

<table>
<thead>
<tr>
<th></th>
<th>Sample A</th>
<th>Sample B</th>
<th>Sample C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 65</td>
<td>N = 41</td>
<td>N = 35</td>
</tr>
<tr>
<td><strong>Primary diagnosis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td>4 (6.2%)</td>
<td>3 (7.3%)</td>
<td>4 (11.4%)</td>
</tr>
<tr>
<td>PD-Ag</td>
<td>58 (89.2%)</td>
<td>36 (87.8%)</td>
<td>27 (77.1%)</td>
</tr>
<tr>
<td>Ag</td>
<td>3 (4.6%)</td>
<td>2 (4.9%)</td>
<td>4 (11.4%)</td>
</tr>
<tr>
<td><strong>Comorbidity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social phobia</td>
<td>22 (33.8%)</td>
<td>14 (34.1%)</td>
<td>7 (20.0%)</td>
</tr>
<tr>
<td>GAD</td>
<td>34 (52.3%)</td>
<td>21 (51.2%)</td>
<td>14 (40.0%)</td>
</tr>
<tr>
<td>OCD</td>
<td>5 (7.7%)</td>
<td>3 (7.3%)</td>
<td>2 (5.7%)</td>
</tr>
<tr>
<td>Specific phobia</td>
<td>14 (21.5%)</td>
<td>7 (17.1%)</td>
<td>7 (20.0%)</td>
</tr>
<tr>
<td>PTSD</td>
<td>6 (9.2%)</td>
<td>3 (7.3%)</td>
<td>3 (8.6%)</td>
</tr>
<tr>
<td>Any comorbid anxiety disorder</td>
<td>49 (75.4%)</td>
<td>29 (70.7%)</td>
<td>20 (57.1%)</td>
</tr>
<tr>
<td><strong>Comorbid depressive disorder</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major depression</td>
<td>30 (46.2%)</td>
<td>19 (46.3%)</td>
<td>10 (28.6%)</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>15 (23.1%)</td>
<td>6 (14.6%)</td>
<td>5 (14.3%)</td>
</tr>
<tr>
<td>Any depressive disorder</td>
<td>36 (55.4%)</td>
<td>21 (51.2%)</td>
<td>14 (40.0%)</td>
</tr>
<tr>
<td><strong>No. comorbid diagnoses</strong></td>
<td>2.1 ± 1.5</td>
<td>2.0 ± 1.5</td>
<td>1.5 ± 1.6</td>
</tr>
<tr>
<td>(M ± SD)</td>
<td>range = 0 – 7</td>
<td>range = 0 – 5</td>
<td>range = 0 – 6</td>
</tr>
</tbody>
</table>

*Note. ADs = Antidepressants; BZs = Benzodiazepines; GAD = Generalised Anxiety Disorder; OCD = Obsessive Compulsive Disorder; PTSD = Post-traumatic Stress Disorder; PD = Panic Disorder without Agoraphobia; PD-Ag = Panic Disorder with Agoraphobia; Ag = Agoraphobia without Panic Disorder.

aData based on participants born outside Australia.

bData for Sample C collected at 6-12 months posttreatment and represents treatments received either before or after treatment from the Anxiety Treatment and Research Unit.

Sample C differed significantly from Sample A on several pretreatment variables: Sample C participants were more likely to be employed (62.9% vs. 35.4%), $\chi^2(1, N = 100) = 6.94, p < .01$, used fewer total psychotropic medications (0.6 vs. 1.0), $t(98) = 2.24, p < .05$, were less likely to be using benzodiazepines (17.1% vs. 41.5%), $\chi^2(1, N = 100) = 6.12, p < .05$, obtained higher age-scaled scores on matrix reasoning ($M = 12.17, SD = 2.23$ vs. $M = 10.97, SD = 2.92$), $t(98) = -2.12,$
Chapter 3 – Psychometric Properties of the Knowledge Measures

$p < .05$, and reported less agoraphobic avoidance when accompanied ($M = 54.01, SD = 21.51$ vs. $M = 63.87, SD = 21.02$), $t(98) = 2.22$, $p < .05$. Sample C differed significantly from Sample B only on benzodiazepine use (17.1% vs. 39.0%), $\chi^2(1, N = 76) = 4.40$, $p < .05$. No other differences between Samples C and B or Samples C and A were found for other pretreatment demographic variables, comorbid diagnoses, intelligence or pretreatment self-report measures of psychopathology ($p > .05$).

**Clinician Samples**

Three clinician samples were recruited to validate the MC-PTKQ: (1) Clinical Psychologists, (2) Intern Clinical Psychologists in their second or third year of a post-graduate degree in clinical psychology, and (3) Entry level Intern Clinical Psychologists at the beginning of their first year of a Doctorate of Clinical Psychology degree. A small number of the clinical psychologists participated in the earlier phase described in chapter 2.

Eighteen Clinical Psychologists were recruited from three sources:

1. Clinical academics from the School of Psychology at the University of Sydney ($n = 4$)
2. Clinical psychologists experienced in CBT for anxiety disorders employed within the Department of Medical Psychology, Westmead Hospital ($n = 4$)
3. Clinical psychologists specialising in psychotherapy for other disorders (e.g., depression, eating disorders, illness adjustment, personality disorders, psychosis) employed within the Department of Medical Psychology, Westmead Hospital ($n = 10$)
In total, 16 (88.9%) of the 18 clinical psychologists returned the MC-PTKQ. They comprised three academics (75%), four clinicians specialising in anxiety disorders (100%) and nine (90%) clinicians specialising in other disorders.

Twenty-eight intern clinical psychologists in their second or third year of a post-graduate degree in clinical psychology who had also completed a 6-month field placement at the SWAHS Anxiety Treatment and Research Unit were recruited. All 28 (100%) interns completed and returned the MC-PTKQ.

A total of 35 entry-level intern clinical psychologists recruited from students enrolled in the Doctorate of Clinical Psychology program at the University of Sydney at the commencement of their first year were invited to participate in the study. Of these 35 interns, 20 (57%) returned useable questionnaires. This lower return rate likely reflects participants’ discomfort in completing a treatment knowledge questionnaire of which they have little experience.

Brief demographic information was collected from clinician samples (see Table 3.5) regarding years of clinical experience, primary areas of expertise, treatment modalities and self-rated expertise in CBT for Panic-Ag ranging from 1 (very low) to 5 (very high). As expected, clinical psychologists had significantly greater years of clinical experience than second/third year interns, $t(42) = 6.24, p < .001$, who in turn had more clinical experience than entry-level interns, $t(46) = 4.50, p < .001$. Similarly, clinical psychologists reported higher Panic-Ag expertise ratings than second/third year interns, $t(42) = 4.07, p < .001$, who in turn reported higher ratings than entry-level interns, $t(46) = 8.60, p < .001$. 


### Table 3.5 Demographic Characteristics for Clinician Samples

<table>
<thead>
<tr>
<th></th>
<th>Clinical psychologists (N = 16)</th>
<th>Second/third year interns (N = 28)</th>
<th>Entry-level interns (N = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Years experience</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(M ± SD)</td>
<td>10.6 ± 0.8</td>
<td>1.5 ± 0.5</td>
<td>0.6 ± 0.8</td>
</tr>
<tr>
<td><strong>Areas of expertise</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety Disorders</td>
<td>8 (50.0%)</td>
<td>28 (100.0%)</td>
<td>3 (15.0%)</td>
</tr>
<tr>
<td>Mood Disorders</td>
<td>6 (37.5%)</td>
<td>5 (55.6%)</td>
<td>1 (5.0%)</td>
</tr>
<tr>
<td>Eating Disorders</td>
<td>3 (18.8%)</td>
<td>0 (0.0%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Other</td>
<td>8 (50.0%)</td>
<td>3 (10.7%)</td>
<td>4 (20.0%)</td>
</tr>
<tr>
<td><strong>Treatment modality</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CBT</td>
<td>16 (100.0%)</td>
<td>28 (100.0%)</td>
<td>9 (45.0%)</td>
</tr>
<tr>
<td>Schema</td>
<td>4 (25.0%)</td>
<td>3 (10.7%)</td>
<td>1 (5.0%)</td>
</tr>
<tr>
<td>IPT</td>
<td>2 (12.5%)</td>
<td>1 (3.6%)</td>
<td>1 (5.0%)</td>
</tr>
<tr>
<td>DBT</td>
<td>2 (12.5%)</td>
<td>0 (0.0%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Other</td>
<td>1 (6.3%)</td>
<td>1 (3.6%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td><strong>Panic-Ag expertise</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1 - 5) (M ± SD)</td>
<td>3.8 ± 0.6</td>
<td>3.0 ± 0.6</td>
<td>1.4 ± 0.6</td>
</tr>
</tbody>
</table>

*Note.* CBT = Cognitive Behaviour Therapy; IPT = Interpersonal Therapy; DBT = Dialectical Therapy.

### Measures

In addition to the MCLPTKQ and Int-PTK, the measures used to examine the construct validity of the knowledge measures were the Personal Details Questionnaire, Matrix Reasoning and the WTAR described in chapter 2.

### Procedure

The procedure for Sample A and B participants was previously reported in chapter 2. Sample C participants were contacted by telephone 6 to 12 months post-treatment to organise follow-up assessments at the clinic as part of routine clinical procedures. During this telephone call they were also invited to participate in
the research study and informed of its purpose and requirements. A battery of routine psychosocial measures was mailed to participants for them to complete at home and return at the follow-up assessment. Follow-up clinical and research assessments were conducted at the clinic and combined in the same appointment. Sample C participants were initially re-assessed with the ADIS-IV and subsequently completed the same battery of measures as Sample A participants in the following order: WTAR, Matrix Reasoning, Int-PTK, MC-PTKQ and questionnaires assessing beliefs about Panic-Ag and its treatment (described in chapter 4). The Int-PTK was administered prior to the MC-PTKQ to ensure participants’ recall on the knowledge interview was not influenced by recognition of correct information contained within the multiple choice questionnaire answers. Pretreatment data for the MC-PTKQ, Int-PTK and belief questionnaires were not available for Sample C as these participants were only assessed at posttreatment.

In order to establish test-retest reliability, 30 consecutive Sample A participants and 20 consecutive Sample C participants completed the MC-PTKQ and the belief scales again at home (in the absence of study aids) 1 to 2 weeks after the initial administration and returned it in a reply-paid envelope. A total of 24 of the 30 Sample A participants (80.0%) and 15 of the 20 Sample C participants (75.0%) returned useable data (return rate = 78.0%).

**Statistical Analyses**

Internal consistency was assessed using Cronbach’s alpha. Pearson correlations were used to examine test-retest reliability, convergent and discriminant validity. Differences in treatment knowledge within patient and clinician samples were assessed using independent samples t-tests. Repeated measures t-tests were
used to assess the knowledge measures’ sensitivity to change and to investigate treatment efficacy. Treatment effect sizes (Cohen’s $d$) were calculated to determine magnitude of change. Effect sizes are defined as small: $d = 0.2$, medium: $d = 0.5$, and large: $d = 0.8$.

**Results**

**Reliability**

Table 3.6 displays the internal consistency and test-retest reliability of the MC-PTKQ and Int-PTK. Using data from Sample A, the MC-PTKQ and Int-PTK were both highly reliable instruments with internal consistency estimates above .90, indicating items assessed a unitary concept. The MC-PTKQ also appeared to be stable over a test-retest interval of 7 to 14 days. Replication with Sample C data yielded virtually identical results; internal consistency was slightly lower for the MC-PTKQ although remaining within the desired range.

**Table 3.6 Internal Consistency and Test-Retest Reliability Coefficients for the Treatment Knowledge Measures for Samples A and C**

<table>
<thead>
<tr>
<th></th>
<th>Sample A</th>
<th>Sample C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Internal consistency</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MC-PTKQ ($n$)</td>
<td>.91 (65)</td>
<td>.85 (35)</td>
</tr>
<tr>
<td>Int-PTK ($n$)</td>
<td>.91 (65)</td>
<td>.91 (34)$^a$</td>
</tr>
<tr>
<td><strong>Test-retest reliability</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MC-PTKQ ($n$)</td>
<td>.93 (24)</td>
<td>.93 (15)</td>
</tr>
</tbody>
</table>

Note. MC-PTKQ = Multiple-Choice Panic-Ag Treatment Knowledge Questionnaire; Int-PTK = Interview of Panic-Ag Treatment Knowledge. $^a$Int-PTK data was missing for one participant.
Chapter 3 – Psychometric Properties of the Knowledge Measures

Construct Validity

Convergent Validity

To assess the extent the two knowledge instruments measured the same construct, total scores on the MC-PTKQ and Int-PTK were compared. High correlations between the measures were found for Sample A at pretreatment (r = .69, p < .001), Sample B at posttreatment (r = .72, p < .001) and replicated at posttreatment in Sample C (r = .72 p < .001). The lack of shared measurement method between the two knowledge measures (written vs. oral) further strengthens their convergent validity.

Pearson correlations were computed for age, education and intelligence as these demographic variables are theoretically and empirically related to patient knowledge. These associations were examined in Sample B using pretreatment and posttreatment data. As displayed in Table 3.7, age was modestly but not significantly associated with treatment knowledge at pretreatment. However, significant negative age effects emerged with the provision of treatment whereby younger participants were better at learning and retaining information presented during treatment than their older counterparts.

Years of education was significantly positively related to increased knowledge, indicating educated participants demonstrated higher knowledge at pretreatment and posttreatment on both the MC-PTKQ and Int-PTK, although the association with the MC-PTKQ failed to reach significance (r = .27, p = .09) due to insufficient power (only 60% power to detect small-to-medium effects). Intelligence, whether assessed by the WTAR or Matrix Reasoning, was also significantly positively associated with knowledge scores at pretreatment and posttreatment for both knowledge measures.
<table>
<thead>
<tr>
<th></th>
<th>Pretreatment</th>
<th>Posttreatment</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>MC-PTKQ</td>
<td>Int-PTK</td>
<td>MC-PTKQ</td>
<td>Int-PTK</td>
<td>Age</td>
<td>Education</td>
<td>WTAR</td>
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<tr>
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<td></td>
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<tr>
<td>MC-PTKQ</td>
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<tr>
<td>Int-PTK</td>
<td>.67***</td>
<td>-</td>
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<tr>
<td>Posttreatment</td>
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<td>.70***</td>
<td>-</td>
<td></td>
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<tr>
<td>MC-PTKQ</td>
<td>.49**</td>
<td>.61***</td>
<td>.72***</td>
<td>-</td>
<td></td>
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<td></td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Age</td>
<td>-.15</td>
<td>-.29</td>
<td>-.31*</td>
<td>-.43**</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>.31*</td>
<td>.35*</td>
<td>.27</td>
<td>.45**</td>
<td>-.12</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>WTAR</td>
<td>.43**</td>
<td>.45**</td>
<td>.51**</td>
<td>.43**</td>
<td>.22</td>
<td>.38*</td>
<td>-</td>
</tr>
<tr>
<td>Matrix Reasoning</td>
<td>.48**</td>
<td>.49**</td>
<td>.61***</td>
<td>.46**</td>
<td>-.18</td>
<td>.51**</td>
<td>.60***</td>
</tr>
</tbody>
</table>

*Note. MC-PTKQ = Multiple-Choice Panic-Ag Treatment Knowledge Questionnaire; Int-PTK = Interview of Panic-Ag Treatment Knowledge; WTAR = Wechsler Test of Adult Reading. *p < .05. **p < .01. ***p < .001.*
Finally, among clinician samples, self-rated expertise in CBT for Panic-Ag was expected to be positively associated with knowledge scores. Collapsing across the three clinician samples, MC-PTKQ scores correlated .63 ($p < .001$) with expertise ratings, thereby supporting this prediction.

Known-Group Validity

Assuming the provision of treatment improves knowledge, scores for both knowledge measures were predicted to be significantly higher for participants at posttreatment than pretreatment. This prediction was confirmed. The mean MC-PTKQ score for Sample A was 18.34 ($SD = 9.27$) compared with 31.34 ($SD = 5.74$) for Sample C, $t(98) = 7.54, p < .001$. For the Int-PTK, Sample A scored 23.85 ($SD = 15.43$) in comparison to 65.41 ($SD = 16.10$) for Sample C, $t(97) = 12.54, p < .001$.

Further evidence of construct validity would be shown if Sample A participants reporting previous CBT treatment ($n = 18$) demonstrated higher knowledge scores than those without prior CBT exposure ($n = 47$). This prediction was supported for the MC-PTKQ (Previous CBT: $M = 23.06, SD = 8.73$, No-previous CBT: $M = 16.53, SD = 8.91$), $t(63) = 2.66, p < .05$, and the Int-PTK (Previous CBT: $M = 37.17, SD = 14.63$, No-previous CBT: $M = 18.74, SD = 12.49$), $t(63) = 5.07, p < .001$.

It was also expected that clinicians with experience in CBT would score higher on the MC-PTKQ than entry-level intern clinical psychologists lacking such experience. Examination of total MC-PTKQ scores among the clinician samples confirmed this expectation. Clinical psychologists ($M = 37.31, SD = 2.63$) and second/third year interns ($M = 38.11, SD = 1.57$) scored significantly higher on the MC-PTKQ than entry-level interns ($M = 30.95, SD = 4.97$), $t(34) = 4.62, p < .001$;
Clinical psychologists did not differ significantly from second/third year interns, \( t(42) = -1.26, p > .05 \). This latter finding is not surprising as second/third year interns gain extensive experience in treating anxiety disorders while on placement at the Clinic. These findings further support the validity of the MC-PTKQ and Int-PTK.

**Sensitivity to Change**

In order to examine whether improvement in treatment knowledge is related to treatment outcome, the knowledge measures must be sensitive to change. To assess sensitivity to treatment effects, repeated measures \( t \)-tests were applied using Sample B data. Although treatment integrity was unable to be verified, pre- to posttreatment changes across all self-report measures of psychopathology were significant in the desired direction with large effect sizes indicating treatment was efficacious (see Table 3.8).

As predicted, knowledge scores increased following treatment. At pretreatment, the mean MC-PTKQ score was 19.27 (\( SD = 10.14 \)) which increased significantly to 30.07 (\( SD = 6.37 \)) at posttreatment, \( t(40) = -8.14, p < .001 \). Similarly, scores on the Int-PTK increased significantly from 26.24 (\( SD = 16.44 \)) at pretreatment to 62.98 (\( SD = 16.44 \)) at posttreatment, \( t(40) = -16.03, p < .001 \). These results indicate both knowledge measures are sensitive to treatment effects. No significant floor or ceiling effects were present on either the MC-PTKQ or Int-PTK.
Table 3.8 Pre- and Posttreatment Means, Standard Deviations and Effect Sizes for Self-Report Measures of Psychopathology for Sample B (N = 41)

<table>
<thead>
<tr>
<th>Treatment variable</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
<th>$t$ (40)</th>
<th>$d$</th>
</tr>
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<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>nPA-ADIS$^a$</td>
<td>10.22</td>
<td>10.69</td>
<td>1.15</td>
<td>1.65</td>
</tr>
<tr>
<td>PASS-ADIS</td>
<td>62.56</td>
<td>20.15</td>
<td>30.45</td>
<td>22.62</td>
</tr>
<tr>
<td>ACQ–Frequency</td>
<td>35.02</td>
<td>9.92</td>
<td>26.12</td>
<td>9.03</td>
</tr>
<tr>
<td>ACQ-Belief</td>
<td>19.51</td>
<td>12.35</td>
<td>8.22</td>
<td>8.06</td>
</tr>
<tr>
<td>MI-Accompanied</td>
<td>64.07</td>
<td>21.48</td>
<td>43.06</td>
<td>17.88</td>
</tr>
<tr>
<td>MI-Alone</td>
<td>80.85</td>
<td>26.70</td>
<td>54.16</td>
<td>26.92</td>
</tr>
<tr>
<td>BSQ</td>
<td>51.95</td>
<td>12.63</td>
<td>35.24</td>
<td>13.99</td>
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<tr>
<td>PBI</td>
<td>123.22</td>
<td>32.93</td>
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<td>31.90</td>
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<tr>
<td>BDI-II</td>
<td>25.68</td>
<td>13.06</td>
<td>15.56</td>
<td>10.47</td>
</tr>
<tr>
<td>WSAS</td>
<td>24.50</td>
<td>9.69</td>
<td>13.50</td>
<td>8.38</td>
</tr>
</tbody>
</table>

*Note. nPA-ADIS = Number of panic attacks in the last month assessed on the ADIS-IV; PASS-ADIS = Panic attack sensation severity assessed on the ADIS-IV; ACQ-Frequency = Agoraphobic Cognitions Questionnaire-Frequency Score; ACQ-Belief = Agoraphobic Cognitions Questionnaire-Belief Score; MI-Accompanied = Mobility Inventory for Agoraphobia-Accompanied subscale; MI-Alone = Mobility Inventory for Agoraphobia-Alone subscale; BSQ = Body Sensations Questionnaire; PBI = Panic Beliefs Inventory; BDI-II = Beck Depression Inventory – II; WSAS = Work and Social Adjustment Scale.

$^a$Cohen’s effect size = $M_{pre} - M_{post} / SD_{pooled}$, where $SD_{pooled} = \sqrt{(SD_{pre}^2 + SD_{post}^2)/2}$.

*$p < .001$.

Discussion

This chapter described the psychometric properties of the Int-PTK and MC-PTKQ. The psychometric properties of the MC-PTKQ and Int-PTK were found to be acceptable. Internal consistency and test-retest reliability of both measures was high across two separate patient samples. The MC-PTKQ and Int-PTK were highly intercorrelated and further evidence of construct validity was obtained using patient and clinician samples. Both measures were sensitive to change.
Although sound, the psychometric properties of the MC-PTKQ and Int-PTK need to be considered in light of several methodological limitations. First, many of the psychometric analyses were based on the same participants (Sample A) used to develop the MC-PTKQ and Int-PTK which may have inflated the observed reliability and validity coefficients. However, similar reliability estimates obtained from an independent patient sample (Sample C) and validity analyses incorporating Sample C and clinician samples corroborated the knowledge measures’ psychometric properties.

Second, as previously discussed in relation to the MC-PTKQ, analyses employing Sample B participants are based on only half the total eligible sample, hence the psychometric properties may not be as robust for the general Panic-Ag population. As Sample B consisted of higher functioning patients, a broader sample of Panic-Ag patients may find the Int-PTK and MC-PTKQ more difficult and show less improvement following treatment. However, arguing against this interpretation is the lack of significant differences between Sample B and treatment dropouts/non-starters on other important indices including age, education, intelligence, level of comorbidity, social functioning and severity of Panic-Ag and depressive symptoms.

Third, readability analysis of the MC-PTKQ indicated it was fairly difficult to read and as such may not have accurately assessed knowledge for less educated individuals or those with reduced verbal skills. Development of treatment knowledge measures containing items with fewer words per sentence and fewer syllables per word would be useful for future investigations involving treatment knowledge.

Notwithstanding these limitations, the knowledge measures developed in this study address many of the methodological weaknesses inherent in research investigating patient treatment knowledge. Firstly, many studies used measures with
poor or unknown psychometric properties (Durose et al., 2004; Heisler et al., 2002; Miller et al., 2003; Weiss et al., 2003), whereas the MC-PTKQ and Int-PTK were subjected to rigorous psychometric testing, demonstrating high reliability, sensitivity to change and good evidence of convergent, divergent and known-group validity.

Second, several studies (Ho et al., 2003; Wigal et al., 1993) combined multiple aspects of knowledge (treatment, symptoms, prevalence) in the same measure ignoring conceptual distinctions which obfuscate relationships between treatment knowledge and outcome. The development of the MC-PTKQ and Int-PTK ensured items assessed only knowledge specific to treatment and thus represent pure measures of treatment knowledge. Third, the MC-PTKQ provided an objective measure of patients’ treatment knowledge free from therapist bias in contrast to therapist ratings of patient knowledge used by Abramowitz et al. (2002). Less objective measures can inflate associations between knowledge and outcome, hence the MC-PTKQ allows a more accurate examination of this relationship.

Finally, the shortcomings of multiple-choice measures were addressed with the Int-PTK. Whereas multiple-choice measures can overestimate patients’ true knowledge by providing cues and reminders, the Int-PTK may offer a more ecologically valid assessment of patients’ treatment knowledge by focussing on recall rather than recognition of information and as such may be more relevant to real life situations reliant on information recall. The Int-PTK also allowed a more sensitive assessment of patient knowledge whereby partial knowledge of concepts could be differentiated from complete knowledge, making the measure less prone to ceiling effects sometimes observed in multiple-choice measures (Beeney et al., 1994).
In summary, the MC-PTKQ and Int-PTK offer two reliable and valid methods of assessing patients’ treatment knowledge enabling examination of its relationship with treatment outcome. Before examining this relationship, the next chapter describes the development of the treatment beliefs scales.
Chapter 4

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Development of the Treatment Belief Scales

This chapter describes the development of three belief scales assessing acceptance of the treatment rationale, expectancies of treatment outcome, and treatment self-efficacy for Panic-Ag. Construction of scales assessing treatment expectancy and rationale acceptance was necessary to address the limitations of existing measures which included single-item scales (Addis & Jacobson, 1996, 2000; Borkovec & Nau, 1974), combination ratings (Addis & Jacobson, 1996; Borkovec & Nau, 1974) and measures with unknown psychometric properties (Kennardy et al., 2003; Stern & Marks, 1973). For treatment self-efficacy, existing self-efficacy scales used for other disorders were inappropriate because self-efficacy is situation specific. Hence, a self-efficacy scale tailored to assessing patients’ confidence in applying therapy skills for Panic-Ag was needed.

Scale Construction

Acceptance of the Treatment Rationale for Panic-Ag (ATR-PA)

In constructing a measure of acceptance of the treatment rationale for Panic-Ag, items assessing rationale knowledge were derived from the MC-PTKQ described in chapter 2 and rated according to belief strength. The MC-PTKQ comprised 68 treatment facets which were phrased as statements to form the initial version of the Acceptance of the Treatment Rationale for Panic-Ag Scale (ATR-PA-68). Acceptance of each statement was assessed on a 5-point Likert scale from 0 (do not believe at all), 1 (slightly believe), 2 (somewhat believe), 3 (mostly believe) and 4 (completely believe). The ATR-PA-68 covered 17 psychoeducation items (9 reverse scored), 23 cognitive therapy items (8 reverse scored), 11 role of avoidance items (5
reverse scored) and 17 exposure therapy items (4 reverse scored) (see Appendix D). Items across categories were randomly distributed within the measure.

**Expectancy of Treatment Outcome for Panic-Ag (ETO-PA)**

Construction of expectancy of treatment outcome items was guided by literature surrounding patients’ beliefs about treatment (Arnkoff et al., 2002; Frank, 1982; Kazdin, 1979; Leventhal et al., 1992) and existing measures containing items assessing therapy expectancies including the Nijmegen Motivation List 2 (Keijsers et al., 1999) and the Treatment Credibility/Expectancy Questionnaire (Devilly & Borkovec, 2000). The Expectancy of Treatment Outcome for Panic-Ag Scale (ETO-PA) comprised nine items (four reverse scored) pertaining to expectancies of CBT for improving anxiety and panic (see Appendix E). All items were expressed as statements worded in the first person and rated on the above 5-point Likert scale from 0 (do not believe at all) to 4 (completely believe).

**Treatment Self-Efficacy for Panic-Ag (TSE-PA)**

Treatment self-efficacy items for Panic-Ag were generated from the self-efficacy subscale of the psychometrically sound Knowledge, Attitudes and Self-Efficacy Asthma Questionnaire (Wigal et al., 1993) modified for Panic-Ag, and literature on self-efficacy in relation to illness management (Bandura, 1977; Kobau & DiIorio, 2003; Scherer & Bruce, 2001; Waldrop, Lightsey, Ethington, Woemmel, & Coke, 2001). Due to symptom idiosyncrasies, specific CBT techniques (e.g., interoceptive exposure, cost experiments) can be irrelevant for particular Panic-Ag patients. Therefore, a self-efficacy scale for CBT in general was preferred as opposed to a scale assessing self-efficacy for specific individual CBT techniques. The
Treatment Self-Efficacy for Panic-Ag Scale (TSE-PA) consisted of nine items (two reverse scored) phrased as statements in the first person (see Appendix E). Belief in each item was rated on the aforementioned 5-point Likert scale (0 = do not believe at all to 4 = completely believe).

Initial Item Refinement

Items across the ATR-PA-68, ETO-PA and TSE-PA scales were analysed to identify unreliable items using data from Sample A. Poor items were detected by examining alpha coefficients. Items exceeding alpha by .01 or more when deleted were considered unreliable. No item on the ATR-PA-68 scale significantly exceeded alpha by .01 when deleted (Cronbach’s alpha = .912, alpha if item deleted = .908 – .914). However, with 68 items, attempts were made to reduce scale length to a more manageable 15 item version without loss of reliability. Accordingly, only those with corrected item-total correlations above .50 were retained. This cut-off yielded 14 items (referred to as ATRLPA, see Appendix E) consisting of two psychoeducation items (one reverse scored), three cognitive therapy items (one reverse scored), one role of avoidance item and eight items focussed on exposure (in vivo and interoceptive exposure and behavioural experiments). Total scores ranged from 0 to 56, with higher scores reflecting greater acceptance. Example items from the 14-item ATR-PA scale are displayed in Figure 4.1.

For the ETO-PA and TSE-PA, no item when deleted significantly exceeded alpha by more than .01 (ETO-PA: Cronbach’s alpha = .847, alpha if item deleted = .817 – .848; TSE-PA: Cronbach’s alpha = .747, alpha if item deleted = .694 – .754), hence all items for both scales were retained. Example ETO-PA and TSE-PA items are displayed in Figure 4.1. Total scores for ETO-PA and TSE-PA each ranged from
0 to 36 with higher scores reflecting more positive expectancies and greater self-efficacy, respectively.

**Figure 4.1.** Example items from the Acceptance of the Treatment Rationale, Treatment Outcome Expectancies and Treatment Self-Efficacy Scales.

<table>
<thead>
<tr>
<th>Scale</th>
<th>Example Items</th>
</tr>
</thead>
</table>
| ATR-PA (14 items) | ▪ A panic attack is just the fight or flight response coming on when there is no real danger.  
                         ▪ Testing out the way I interpret my panic symptoms is a sensible approach for overcoming my panic.  
                         ▪ Facing my fears helps me to learn that panic symptoms are harmless even if they are unpleasant. |
| ETO-PA (9 items)  | ▪ *I do not believe CBT will be helpful for me  
                         ▪ CBT will help me overcome my panic  
                         ▪ *CBT is too simplistic to be helpful for treating my panic |
| TSE-PA (9 items)  | ▪ I feel I can implement the techniques as recommended by my therapist  
                         ▪ I feel I have learned strategies to effectively manage my anxiety and panic  
                         ▪ During the early stages of a panic attack I can apply the skills I have learned to reduce the attack |

*Note: ATR-PA = Acceptance of the Treatment Rationale for Panic-Ag; ETO-PA = Expectancies of Treatment Outcome for Panic-Ag; TSE-PA = Treatment Self-Efficacy for Panic-Ag.  
*Reverse scored.

Readability of the treatment belief scales was assessed with the Flesch Reading Ease Index (Flesch, 1948). Based on average syllables per word, the ATR-PA was regarded as “difficult” to read (Reading Ease score (RE) = 30 – 50) while the ETO-PA and TSE-PA were rated as “fairly difficult” (RE = 50 – 60). However, when defined by average number of words per sentence, the ETO-PA was rated as “easy” (RE = 80 – 90), TSE-PA as “fairly easy” (RE = 70 – 80) and ATR-PA as
“standard” (RE = 60 – 70). On average, these ratings imply the treatment belief scales had the equivalent readability of a digest-style magazine or easier.

Summary

This phase of the study involved developing scales assessing acceptance of the treatment rationale, expectancies of treatment outcome and treatment self-efficacy for Panic-Ag (ATRLPA, ETOLPA, TSELPA, respectively). Items for the ATRLPA assessed acceptance of treatment knowledge derived from the MC-PTKQ and initially comprised 68 items. Items for the ETOLPA and TSELPA were constructed from scientific literature and published questionnaires adapted for Panic-Ag and each contained nine items. Sixty-five pretreatment Panic-Ag participants completed each scale to identify unreliable items. Examination of alpha coefficients within each scale indicated all items were internally consistent, hence all items were retained. However, the ATRLPA scale was reduced to 14 items on the basis of corrected item-total correlations above .50 to decrease scale length.

In order to examine the convergent validity of the ATRLPA, ETOLPA and TSELPA scales, they must be compared with measures assessing theoretically related constructs. To this end, three additional belief scales were constructed as described in the next section.
Development of the Aetiology, Alternative Non-CBT Treatments and Treatment Barriers Belief Scales

The purpose of this component of the study was to develop a set of measures to establish the convergent validity of the ATRLPA, ETOLPA and TSELPA scales. Three theoretically related constructs were identified. Firstly, Addis and Carpenter (1999) found that decreased acceptance of an action-oriented treatment rationale for depression was associated with endorsement of more reasons offered for depression inconsistent with that rationale. It was therefore hypothesised that the more reasons offered for the cause of Panic-Ag incompatible with a treatment rationale focussed on correcting catastrophic cognitions, the less accepting participants will be of that rationale. Hence, a scale assessing aetiology beliefs for Panic-Ag was developed.

Secondly, stronger belief in non-CBT based treatments would indicate decreased acceptance of the treatment rationale for Panic-Ag, hence a scale assessing belief in alternative non-CBT treatments was constructed. Conceptually, belief in non-CBT treatments should also be associated with reduced treatment outcome expectancies of CBT for Panic-Ag. Finally, factors believed to interfere in one’s ability to respond to treatment should be associated with reduced expectancies of treatment outcome and poorer treatment self-efficacy. Therefore a treatment barriers scale was developed.

Scale Construction

In order to develop a representative and comprehensive set of items for the aetiology, alternative non-CBT treatment and treatment barriers belief scales, a two-phase procedure was used. In phase one, a checklist of beliefs was constructed with
items derived from the author’s eight years clinical experience conducting diagnostic assessments and treatment of patients with Panic-Ag, and literature on patient treatment and illness representations (Addis, Truax, & Jacobson, 1995; Atkinson, Worthington, Dana, & Good, 1991; Foulks et al., 1986; Johnson et al., 2000; Leventhal et al., 1992; Moss-Morris et al., 2002; Weinman, Petrie, Moss-Morris, & Horne, 1996). However, as individuals present to treatment with pre-existing beliefs about their illness and treatment (Donovan et al., 1989), views of Panic-Ag patients were also incorporated. Hence phase two involved semi-structured interviews with Panic-Ag patients designed to elicit a representative range of beliefs not captured by the above beliefs checklist or previous studies. Interview methods are useful for identifying patient attitudes and beliefs to generate items for questionnaire development (Cooper-Patrick et al., 1997; Horne, Weinman, & Hankins, 1999).

Method

Participants

Participants comprised 25 consecutive Panic-Ag patients on the waiting list for treatment and 15 consecutive Panic-Ag patients who completed treatment within 6 months of assessment at the SWAHS Anxiety Treatment and Research Unit, Cumberland Hospital, between November 2004 and August 2005. (This study was conducted prior to the evaluation of the treatment knowledge and belief scales.) Of these 40 participants, 19 (47.5%) participated in other aspects of the study and comprised 54.3% of Sample C. The remaining 21 (52.5%) participants represented an independent sample of Panic-Ag patients who had no further involvement in any aspect of the study. Of the 40 participants, 26 (65.0%) were female and 14 (35.0%) were male. Participants were aged between 21 and 67 years (M = 40.2 years, SD =
13.0 years). Additional demographic characteristics of these participants are presented in Table 4.1

**Table 4.1** Demographic Characteristics of Panic-Ag Participants Completing the Beliefs Checklist and Beliefs Interview (N = 40)

<table>
<thead>
<tr>
<th>Marital status</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Never married</td>
<td></td>
<td>13 (32.5%)</td>
</tr>
<tr>
<td>Married/de facto</td>
<td></td>
<td>23 (57.5%)</td>
</tr>
<tr>
<td>Divorced/separated</td>
<td></td>
<td>4 (10.0%)</td>
</tr>
</tbody>
</table>

**Education in years (M ± SD)**

12.8 ± 2.8

Range = 6 – 20

**Employed**

30 (75.0%)

**Country of origin**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>29 (72.5%)</td>
</tr>
<tr>
<td>Other</td>
<td>11 (27.5%)</td>
</tr>
</tbody>
</table>

**Duration of anxiety disorder in years (M ± SD)**

9.5 ± 8.9

Range = 0.25 – 30

**Previous treatments**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Medication</td>
<td>33 (82.5%)</td>
</tr>
<tr>
<td>Counselling</td>
<td>20 (50.0%)</td>
</tr>
<tr>
<td>Self-help books</td>
<td>15 (37.5%)</td>
</tr>
</tbody>
</table>

**Measures**

*The Beliefs Checklist* was developed for this study and comprised 12 aetiological beliefs, 15 alternative non-CBT treatment beliefs and eight beliefs about treatment barriers. Each item was worded in the first person using a stem and leaf format (see Figure 4.2). Space was allocated for patients to add additional beliefs not listed in the checklist. Example items from each belief domain are presented in Figure 4.2.
Figure 4.2. Example items from each belief domain of the Beliefs Checklist.

<table>
<thead>
<tr>
<th>Belief domain</th>
<th>Example belief item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aetiology</td>
<td>I believe my problem is caused by:</td>
</tr>
<tr>
<td></td>
<td>▪ Something physically wrong with me</td>
</tr>
<tr>
<td></td>
<td>▪ Inheriting anxious genes from my parents (genetics)</td>
</tr>
<tr>
<td></td>
<td>▪ A curse or supernatural force</td>
</tr>
<tr>
<td>Alternative non-CBT treatments</td>
<td>In order to treat myself for my problem I believe I need to:</td>
</tr>
<tr>
<td></td>
<td>▪ Rely on tranquilizers (e.g., Xanax, Valium, Ativan)</td>
</tr>
<tr>
<td></td>
<td>▪ Treat the underlying medical problem</td>
</tr>
<tr>
<td></td>
<td>▪ Avoid people, places or situations that trigger my anxiety</td>
</tr>
<tr>
<td>Treatment barriers</td>
<td>I believe the following factors will interfere in my ability to respond to treatment:</td>
</tr>
<tr>
<td></td>
<td>▪ The intensity of my symptoms</td>
</tr>
<tr>
<td></td>
<td>▪ The length of time I have had the problem</td>
</tr>
<tr>
<td></td>
<td>▪ My previous unsuccessful attempts with treatment</td>
</tr>
</tbody>
</table>

The Beliefs Interview, also developed for this study, consisted of three open-ended questions assessing the same belief domains assessed by the Beliefs Checklist. Participants were asked:

1. What do you believe has caused your problem?
2. What do you believe needs to happen for you to overcome your problem?
3. Do you believe there is anything about you or your situation that will interfere in the success of treatment?

Procedure

Ethics approval for this phase of the study was provided from the Western Sydney Area Health Service (WSAHS) Human Research Ethics Committee [HREC2004/8/4.10(1924)]. Participants were invited to attend a research appointment where the purpose of the study was explained and informed consent was obtained. During this research appointment, patients firstly completed the Beliefs
Checklist alone in a quiet office. The following instructions were provided and read aloud:

The following are some beliefs about anxiety and panic that some people have. Please place a tick in the box if YOU have EVER had the belief (even if you only believed it for a moment). Try to be as honest as possible when answering. There are no right or wrong answers. We are interested in YOUR beliefs.

Participants were subsequently interviewed about their beliefs via the Beliefs Interview where they were probed until an exhaustive list of beliefs was obtained. The Beliefs Checklist and the Beliefs Interview each took approximately 10 minutes to complete. At the conclusion of the interview, participants were thanked for their time and effort.

Results

Beliefs Checklist Data

Participant endorsement of beliefs about aetiology, alternative treatments, and treatment barriers are displayed in Table 4.2. All items were endorsed by at least one participant indicating the range of beliefs were relevant to Panic-Ag patients. Furthermore, no item was endorsed by 100% of the sample, indicating good variability of responses. Endorsement of aetiology beliefs ranged from 8% (“Exposure to environmental contaminants”) to 73% (“Chemical imbalance in my brain”). Of the 12 aetiology belief items, seven were endorsed by at least 50% of the sample and five by less than 50%. 
Table 4.2 Endorsement of Beliefs About Aetiology, Alternative Non-CBT Treatments and Treatment Barriers in 40 Patients with a History of Panic-Ag

<table>
<thead>
<tr>
<th>Belief Items</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aetiology beliefs</strong></td>
<td></td>
</tr>
<tr>
<td><em>I believe my panic/anxiety is caused by…</em></td>
<td></td>
</tr>
<tr>
<td>1. A chemical imbalance in my brain</td>
<td>29 (73%)</td>
</tr>
<tr>
<td>2. Something physically wrong with me</td>
<td>24 (60%)</td>
</tr>
<tr>
<td>3. Inheriting anxious genes from my parents (genetics)</td>
<td>23 (58%)</td>
</tr>
<tr>
<td>4. A hormonal imbalance</td>
<td>22 (55%)</td>
</tr>
<tr>
<td>5. Early traumatic experiences from my childhood or adolescence</td>
<td>22 (55%)</td>
</tr>
<tr>
<td>6. A medical condition that the doctors haven’t found yet</td>
<td>20 (50%)</td>
</tr>
<tr>
<td>7. A traumatic experience (e.g., assault, rape, death of a family member/friend, relationship break-up)</td>
<td>20 (50%)</td>
</tr>
<tr>
<td>8. Using drugs and/or alcohol</td>
<td>9 (23%)</td>
</tr>
<tr>
<td>9. Punishment from God for my past sins</td>
<td>8 (20%)</td>
</tr>
<tr>
<td>10. Taking too much caffeine</td>
<td>7 (18%)</td>
</tr>
<tr>
<td>11. A curse or supernatural force</td>
<td>4 (10%)</td>
</tr>
<tr>
<td>12. Exposure to environmental contaminants</td>
<td>3 (8%)</td>
</tr>
<tr>
<td><strong>Alternative non-CBT treatment beliefs</strong></td>
<td></td>
</tr>
<tr>
<td><em>In order to treat myself for my problem I believe I need to…</em></td>
<td></td>
</tr>
<tr>
<td>1. Think positively</td>
<td>36 (90%)</td>
</tr>
<tr>
<td>2. Talk about my personal problems with a counsellor</td>
<td>34 (85%)</td>
</tr>
<tr>
<td>3. Talk about my problem with someone who has had similar experiences with anxiety</td>
<td>31 (78%)</td>
</tr>
<tr>
<td>4. Slow my breathing down or practice breathing exercises</td>
<td>31 (78%)</td>
</tr>
<tr>
<td>5. Stay away from stressful things</td>
<td>26 (65%)</td>
</tr>
<tr>
<td>6. Avoid people, places or situations that trigger my anxiety</td>
<td>25 (63%)</td>
</tr>
<tr>
<td>7. Distract myself</td>
<td>24 (60%)</td>
</tr>
<tr>
<td>8. Treat the underlying medical problem</td>
<td>23 (58%)</td>
</tr>
<tr>
<td>9. Practice yoga, meditation or exercise</td>
<td>22 (55%)</td>
</tr>
<tr>
<td>10. Rely on antidepressant medication (e.g., Zoloft, Prozac, Aropax, Cipramil, Avanza, Efexor-XR, Aurorix, Prothiaden)</td>
<td>19 (48%)</td>
</tr>
<tr>
<td>11. Address underlying issues from my childhood/adolescence</td>
<td>18 (45%)</td>
</tr>
<tr>
<td>12. Rely on tranquilizers (e.g., Valium, Xanax, Serapax, Ativan, Lexotan)</td>
<td>12 (30%)</td>
</tr>
<tr>
<td>13. Undergo spiritual cleansing</td>
<td>11 (28%)</td>
</tr>
<tr>
<td>14. Rely on alcohol</td>
<td>3 (8%)</td>
</tr>
<tr>
<td>15. Have my sins forgiven by a religious/spiritual leader</td>
<td>3 (8%)</td>
</tr>
</tbody>
</table>
Table 4.2 (continued) Endorsement of Beliefs About Aetiology, Alternative Non-CBT Treatments and Treatment Barriers in 40 Patients with a History of Panic-Ag

<table>
<thead>
<tr>
<th>Belief items</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment barrier beliefs</strong></td>
<td></td>
</tr>
<tr>
<td><em>I believe the following factors will interfere in my ability to respond to treatment:</em></td>
<td></td>
</tr>
<tr>
<td>1. My anxiety level</td>
<td>24 (60%)</td>
</tr>
<tr>
<td>2. The length of time I have had the problem</td>
<td>19 (48%)</td>
</tr>
<tr>
<td>3. The intensity of my symptoms</td>
<td>18 (45%)</td>
</tr>
<tr>
<td>4. My previous unsuccessful attempts with treatment</td>
<td>14 (35%)</td>
</tr>
<tr>
<td>5. My physical health</td>
<td>9 (23%)</td>
</tr>
<tr>
<td>6. My age</td>
<td>6 (15%)</td>
</tr>
<tr>
<td>7. I am not intelligent enough</td>
<td>3 (8%)</td>
</tr>
<tr>
<td>8. Presence of my (diagnosed) medical problem</td>
<td>1 (3%)</td>
</tr>
</tbody>
</table>

Endorsement of alternative non-CBT treatment beliefs ranged from 8% (e.g., “Have my sins forgiven by a religious/spiritual leader”) to 90% (“Think positively”). Of the 15 alternative non-CBT treatment beliefs, nine were endorsed by at least 50% of the sample and six by less than 50%. Endorsement of beliefs about treatment barriers ranged from 3% (“Presence of my diagnosed medical problem”) to 60% (“My anxiety level”). Of the eight treatment barriers, only one was endorsed by at least 50% of the sample, while the remaining seven were endorsed by less than 50%. One item (“Presence of my diagnosed medical problem”) was endorsed by only one participant and was subsequently omitted from the scale.

**Belief Interview Data**

Qualitative data from the Belief Interview corroborated items from the Beliefs Checklist. However, interview responses prompted several minor modifications and additional items. With regard to aetiology beliefs, the item “Using drugs or alcohol” from the Beliefs Checklist was split into three separate items.
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(“Using prescription drugs”, “Using illicit drugs”, “Using alcohol”) highlighting the differences between substances. Similarly, an item relating to traumatic experiences (assault, rape, death of a family member/friend, relationship break-up) was separated into two items, one reflecting more violent traumas (assault, rape, war), the other relating to stress from personal/family problems (death of a family member, relationship break-up, financial problems). A further item relating to physical stress to the body (illness, virus, fatigue, childbirth) was also added.

Interview responses added three items to the alternative non-CBT treatment beliefs scale (“Have further medical tests conducted”, “Probe into my past to discover the cause of my fear”, and “Avoid foods or substances that trigger my anxiety”). However one item, “Think positively”, was removed due to ambiguity in meaning with some participants interpreting thinking realistically as equivalent to thinking positively. In addition, five items were added to the treatment barriers scale (“My depression”, “Presence of my other emotional/psychological problem(s)”, “Chemical imbalance in my brain”, “The hereditary nature of my problem (genetics)” and “The previous effects of drugs/alcohol/toxins on my system”) and several items were reworded to improve their comprehensibility (e.g., “My previous unsuccessful attempts with treatment” changed to “My previous failure to respond to treatment”).

Refined Belief Scales

On the basis of participant feedback from the Beliefs Checklist and Beliefs Interview, the Aetiology scale comprised 16 items, the Alternative Non-CBT Treatments scale comprised 17 items and the Treatment Barriers scale consisted of
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12 items (see Appendix F). Items across the three scales were rated on a 5-point Likert scale ranging from 0 (do not believe at all) to 4 (completely believe).

Reliability

Internal consistency (Cronbach’s alpha) and test-retest reliability (retest-interval = 1-2 weeks; Pearson correlations) were assessed using Samples A and C. Reliability of the Aetiology, Alternative Non-CBT Treatments and Treatment Barriers belief scales was sound as displayed in Table 4.3.

<table>
<thead>
<tr>
<th>Belief Scales</th>
<th>Aetiology</th>
<th>Alternative non-CBT treatments</th>
<th>Treatment barriers</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Internal consistency (n)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sample A (64)</td>
<td>.86</td>
<td>.86</td>
<td>.91</td>
</tr>
<tr>
<td>Sample C (35)</td>
<td>.84</td>
<td>.87</td>
<td>.89</td>
</tr>
<tr>
<td><strong>Test-retest reliability (n)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sample A (24)</td>
<td>.85</td>
<td>.87</td>
<td>.92</td>
</tr>
<tr>
<td>Sample C (15)</td>
<td>.86</td>
<td>.92</td>
<td>.78</td>
</tr>
</tbody>
</table>

Summary

To evaluate the convergent validity of the ATR-PA, ETO-PA and TSE-PA scales, three additional belief scales were developed consisting of a 16-item
Aetiology scale, a 17-item Alternative Non-CBT Treatments scale and a 12-item Treatment Barriers scale. Scale items were constructed from clinical experience, scientific literature and feedback from 40 Panic-Ag patients. Each belief scale was found to be very reliable in a pretreatment and posttreatment sample.

**Evaluation of the Psychometric Properties of the Treatment Belief Scales**

This section describes the psychometric properties of the treatment belief scales assessing acceptance of the treatment rationale (ATR-PA), expectancies of treatment outcome (ETO-PA) and treatment self-efficacy (TSE-PA) for Panic-Ag. Reliability of these scales was assessed in a pretreatment sample and replicated in a posttreatment sample. The treatment belief scales were compared with other belief scales listed below to establish convergent validity. The ability of the ATR-PA and TSE-PA scales to differentiate between pretreatment and posttreatment participants and be sensitive to change was also examined.

**Method**

**Participants**

The psychometric properties of the treatment belief scales were established using Samples A, B and C.
Measures

In addition to the ATRLPA, ETO-PA, TSE-PA and the Aetiology, Alternative Non-CBT Treatments and Treatment Barriers belief scales described above, the following measures were administered.

Personal Details Questionnaire. Described in chapter 2, this questionnaire was administered to collect demographic information on age and gender for the purpose of establishing discriminant validity.

Expectancy Factor of the Credibility/Expectancy Questionnaire (CEQ, Devilly & Borkovec, 2000). The CEQ is a 6-item self-report questionnaire designed to measure treatment outcome expectancy and rationale credibility. It consists of two factors each comprising three items: Factor 1 (credibility) examines how credible the patient thinks therapy is, and Factor 2 (expectancy) concerns the patient’s emotional expectations about the effectiveness of therapy. The psychometric properties of the expectancy factor demonstrated sound internal consistency (Cronbach’s alpha = .79 – .90) and high 1-week test-retest reliability ($r = .82, p < .001$). As the CEQ is administered prior to the conclusion of therapy, data for this measure is not available for Sample B at posttreatment or for Sample C.

Doubt Factor of the Nijmegen Motivation List 2 (NML2, Keijsers et al., 1999). The NML2 is a 24-item self-report questionnaire designed to assess patients’ motivation for commencing psychotherapy. Factor analysis revealed the measure consisted of three factors: preparedness, distress and doubt. The preparedness subscale assesses a “patient’s preparedness to actively invest in treatment and to make sacrifices” (p. 171). The distress subscale captures the level of distress experienced by the patient as a result of their problems. The doubt factor consists of six items assessing “doubt about the investment in treatment, the treatment itself and
the possibility of gaining from it” (p. 171). An example item from the doubt factor is, “I do not believe that this is the right treatment for me”. Responses are rated on a 6-point scale from 1 (not at all applicable) to 6 (very applicable). The doubt factor had sound test-retest reliability over a 1-week interval ($r = .73, p < .001$) and a Cronbach’s alpha of .69 (following the removal of one item). As this measure assesses attitudes prior to commencing therapy it was only administered at pretreatment, hence data is unavailable for Sample B (posttreatment) and Sample C.

**Self-deprecation Subscale of the Panic Belief Inventory** (PBI, Greenberg, 1989; Wenzel et al., 2006). The self-deprecation subscale of the PBI (described in chapter 2) comprises six statements (e.g., “Having panic attacks means I’m weak, defective, or inferior”). The psychometric properties of the self-deprecation subscale revealed good internal consistency ($\alpha = .82$) and significant moderate correlations were observed between this subscale and other self-report measures of cognitions at pretreatment ($r = .61, p < .001$) and posttreatment ($r = .52 - .77, p < .05$), demonstrating evidence of convergent validity. Evidence of discriminant validity was achieved with a lack of association between this subscale and a measure of suicidal ideation. The self-deprecation subscale is also sensitive to treatment gains with scores decreasing significantly from pretreatment to posttreatment ($p < .001$).

**Procedure**

Assessment procedures for participants from Samples A, B and C were identical to those described in chapter 2 and chapter 3. In brief, prior to treatment all participants were assessed with the ADIS-IV and completed a battery of self-report questionnaires containing the Panic Beliefs Inventory. Sample A participants then attended a pretreatment research appointment where their treatment knowledge and
beliefs were assessed. The order of measures was as follows: Int-PTK, Personal Details Questionnaire, CEQ, NML2 and MC-PTKQ. The Aetiology, Alternative Non-CBT Treatments and Treatment Barriers belief scales were then administered followed by the ATR-PA, ETO-PA and TSE-PA with items from the latter three scales intermixed. These six belief scales took approximately 30 minutes in total to complete. Prior to administration of the belief scales the following instructions were provided and read aloud:

Below is a list of beliefs that some people have about Panic Disorder and Agoraphobia. Sometimes people’s beliefs match what they have previously been told, and sometimes they differ. We are interested in what you truly or secretly believe (not what you think you should believe). Please read each item and circle the number using the scale below to rate the extent YOU believe the item to be true for you. Do not spend too long on any item. There are no right or wrong answers. We are interested in what you really believe.

Sample B and C participants were contacted 6 to 12-months posttreatment to organise a routine clinical follow-up assessment and invite them to participate in the study. Prior to this assessment, participants were mailed a battery of self-report questionnaires including the PBI. During the follow-up assessment, they were reassessed with the ADIS-IV, after which the abovementioned knowledge and belief measures were administered, with the exception of the CEQ and NML2.

**Statistical Analyses**

Internal consistency of the belief scales was evaluated using Cronbach’s alpha. Test-retest reliability, concurrent, convergent and discriminant validity was assessed with bivariate Pearson correlations. Independent samples t-tests were
performed to examine differences in beliefs between pretreatment and posttreatment participants. Repeated measures t-tests were used to investigate pre- to posttreatment belief change within Sample B.

Scores on the Aetiology, Alternative Non-CBT Treatments and Treatment Barriers beliefs scale items were recoded dichotomously such that items were either endorsed or not. Responses indicating any level of endorsement (scores of 1–4) were classified as item endorsement. Total scores for these scales were calculated by summing the number of items endorsed. Therefore, total scores ranged from 0 to 16 for Aetiology, 0 to 17 for Alternative Non-CBT Treatments and 0 to 12 for Treatment Barriers.

**Results**

**Reliability**

As displayed in Table 4.4, internal consistencies of the treatment belief scales were satisfactory for Sample A. The TSE-PA scale yielded slightly lower ratings, yet still above the minimum acceptable level for internal consistency of .5 to .7 (Bowling, 2002). Replication with Sample C participants demonstrated internal consistency estimates in the desired range. Test-retest reliability of the ATR-PA, ETO-PA and TSE-PA was sound (see Table 4.4). Moreover, ATR-PA and TSE-PA beliefs appeared to become more stable after treatment (Sample C: \( r > .9 \)).
Table 4.4 Internal Consistency and Test-Retest Reliability Scores for the Treatment Beliefs Measures for Samples A and C

<table>
<thead>
<tr>
<th>Internal Consistency</th>
<th>Sample A</th>
<th>Sample C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretreatment</td>
<td>Posttreatment</td>
</tr>
<tr>
<td>ATR-PA (n)</td>
<td>.88 (64)</td>
<td>.92 (35)</td>
</tr>
<tr>
<td>ETO-PA (n)</td>
<td>.85 (64)</td>
<td>-</td>
</tr>
<tr>
<td>TSE-PA (n)</td>
<td>.75 (64)</td>
<td>.88 (35)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Test-retest Reliability</th>
<th>Sample A</th>
<th>Sample C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretreatment</td>
<td>Posttreatment</td>
</tr>
<tr>
<td>ATR-PA (n)</td>
<td>.72 (24)</td>
<td>.97 (15)</td>
</tr>
<tr>
<td>ETO-PA (n)</td>
<td>.81 (24)</td>
<td>-</td>
</tr>
<tr>
<td>TSE-PA (n)</td>
<td>.77 (24)</td>
<td>.91 (15)</td>
</tr>
</tbody>
</table>

Note. ATR-PA = Acceptance of the Treatment Rationale for Panic-Ag; ETO-PA = Expectancies of Treatment Outcome for Panic-Ag; TSE-PA = Treatment Self-Efficacy for Panic-Ag.

Construct Validity

Convergent Validity

The ATR-PA scale assesses acceptance of a treatment rationale based on correcting catastrophic misinterpretations of physical symptoms. Theoretically, participants endorsing more reasons for panic incompatible with this rationale should demonstrate reduced acceptance of the rationale. This prediction was supported through significant negative correlations between the Aetiology and ATR-PA scales (Sample A: $r = -.32, p < .05$; Sample B-posttreatment: $r = -.50, p < .01$; Sample C: $r = -.44, p < .01$). Conceptually, greater acceptance of the treatment rationale should be associated with endorsement of fewer non-CBT based treatments. Significant negative correlations between the ATR-PA and Alternative Non-CBT Treatments scales supported this prediction (Sample A: $r = -.30, p < .05$; Sample B-
posttreatment: $r = -0.55, p < .01$; Sample C: $r = -0.60, p < .001$), thus providing further evidence of convergent validity for the ATR-PA scale.

The ETO-PA was only completed at pretreatment by Sample A. Concurrent validity of the ETO-PA scale was demonstrated through significant correlations with other measures assessing treatment expectancy, namely the expectancy factor of the CEQ ($r = 0.55, p < .001$) and the NML2 doubt factor ($r = -0.30, p < .05$). The difference in magnitude of the two correlations also supported the construct validity of the ETO-PA because, unlike the expectancy factor of the CEQ, the NML2 doubt factor does not purely assess outcome expectancies and hence yielded a lower correlation.

Evidence of convergent validity for the ETO-PA would also be shown if participants endorsing more non-CBT treatments and treatment barriers expressed lower treatment outcome expectancies. These predictions were confirmed. Significant negative correlations between ETO-PA were observed with the Alternative Non-CBT Treatments and Treatment Barriers scales ($r = -0.32, p < .001$, $r = -0.56, p < .001$, respectively). In addition, greater acceptance of the treatment rationale should be associated with higher expectancies of treatment outcome. Further strengthening its construct validity, significant positive correlations were observed between ATR-PA and ETO-PA ($r = 0.42, p < .001$).

Regarding the TSE-PA scale, participants endorsing more treatment barriers should theoretically report reduced treatment self-efficacy. Significant negative correlations between the TSE-PA and the Treatment Barriers scale supported this prediction (Sample A: $r = -0.36, p < .01$; Sample B-posttreatment: $r = -0.72, p < .001$; Sample C: $r = -0.51, p < .01$). Furthermore, highly self-deprecating participants were expected to report lower treatment self-efficacy. This relationship was borne out in
significant negative correlations between TSE-PA and the self-deprecation subscale of the PBI (Sample A: $r = -0.42, p < .01$; Sample B-posttreatment: $r = -0.50, p < .01$; Sample C: $r = -0.57, p < .01$). These results strengthen the convergent validity of the TSE-PA scale.

**Known-Group Validity**

Theoretically, treatment should increase patients’ acceptance of the treatment rationale and treatment self-efficacy. Posttreatment participants were therefore predicted to score significantly higher on the ATR-PA and TSE-PA than pretreatment participants. Comparison of data from Sample A and Sample C revealed posttreatment participants scored significantly higher than pretreatment participants on the ATR-PA (Sample A: $M = 32.80, SD = 10.36$, Sample C: $M = 48.29, SD = 9.98$), $t(97) = -7.20, p < .001$, and TSE-PA (Sample A: $M = 18.83, SD = 5.85$, Sample C: $M = 31.03, SD = 6.18$), $t(97) = -9.23, p < .001$, thus providing supportive evidence of known-group validity.

**Sensitivity to Change**

Using Sample B data, the ATR-PA and TSE-PA were shown to be sensitive to treatment effects with scores increasing significantly from pretreatment to posttreatment: ATR-PA (pretreatment $M = 33.05, SD = 10.36$, posttreatment $M = 45.66, SD = 9.60$), $t(40) = -7.22, p < .001$; TSE-PA (pretreatment $M = 20.02, SD = 6.34$, posttreatment $M = 28.29$), $SD = 6.14$, $t(40) = -6.79, p < .001$. 164
Discussion

This chapter aimed to develop reliable and valid measures assessing acceptance of the treatment rationale, expectancies of treatment outcome and treatment self-efficacy for Panic-Ag (referred to as ATR-PA, ETO-PA and TSE-PA, respectively). The ATR-PA, ETO-PA and TSE-PA demonstrated adequate levels of internal consistency and test-retest reliability in two independent samples of Panic-Ag patients. The convergent and discriminant validity of each measure was supported. The ATR-PA and TSE-PA effectively discriminated between pretreatment and posttreatment participants and were sensitive to change, further supporting the construct validity of the scales.

The ATR-PA, ETO-PA and TSE-PA represent an advancement over similar measures used in previous research for the purpose of assessing relationships with treatment outcome. Firstly, as previously described, some studies used single-item measures of acceptance of the treatment rationale (Addis & Jacobson, 1996, 2000) and treatment outcome expectancies (Clark et al., 1999; Hansson & Berglund, 1987; Vogel et al., 2006). Single-item scales are unreliable and prone to measurement error; the ATR-PA, ETO-PA and TSE-PA in contrast are all multi-item measures.

Second, other studies have combined related but distinct variables into one measure (Addis & Jacobson, 1996; Chambless et al., 1997; Goosens et al., 2005; Safren et al., 1997) when investigating associations between treatment outcome and acceptance of the rationale and treatment expectancies, thereby obscuring true relationships. Acknowledging this methodological problem, the ATR-PA was constructed purely from items assessing the treatment rationale, while ETO-PA items solely focussed on outcome expectancy without assessing treatment credibility.
Third, as self-efficacy is domain specific, existing self-efficacy measures developed for treatment of other disorders (e.g., asthma, diabetes, HIV, insomnia) were unsuited to the Panic-Ag domain. As treatment self-efficacy for Panic-Ag has not been investigated, the TSE-PA scale represents the first attempt to develop a valid and reliable measure of self-efficacy for implementing CBT in Panic-Ag.

Finally, numerous studies have used measures unsubjected to rigorous psychometric testing (Addis & Jacobson, 1996, 2000; Emmelkamp & Emmelkamp-Benner, 1975; Joyce et al., 2003; Kenardy et al., 2003; Stern & Marks, 1973). In contrast, the ATR-PA, ETO-PA and TSE-PA scales possess sound internal consistency, test-retest reliability and favourable evidence supporting construct validity in two independent samples of Panic-Ag patients. On the basis of these measurement improvements, relationships between acceptance of the treatment rationale, treatment expectancy, treatment self-efficacy and treatment outcome can be more accurately ascertained.

Despite their advantages, the development of the treatment belief scales are not without limitations. The most notable limitation involved sample overlap between scale development and validation. The sample used to develop and refine ATR-PA, ETO-PA and TSE-PA items (Sample A) was the same one used to evaluate the scales’ construct validity, which may have affected the way these scales correlated with other measures. However, similar reliability and validity coefficients were obtained with an independent sample (Sample C). In a similar vein, approximately half the participants used to develop the Aetiology, Alternative Non-CBT Treatments and Treatment Barriers scales went on to form Sample C which may have inflated correlations between these measures and the ATR-PA and TSE-
PA. However, correlations of similar magnitude were observed in Sample B, strengthening the validity of the scales.

As previously stated, Sample B comprised only 50% of eligible participants, hence the psychometric properties (e.g., sensitivity to change) of the treatment belief measures obtained from Sample B may not generalise to the wider population of Panic-Ag patients. As reported in chapter 2, Sample B participants tended to be higher functioning, hence a more representative sample of Panic-Ag patients may exhibit less pre- to posttreatment change on the ATR-PA and TSE-PA. Finally, all measures used to assess the validity of the scales were self-report paper and pencil measures, thus shared measurement methods may have contributed to the significant correlations obtained in this study, potentially over-inflating the true level of association.
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**Relationships Between Treatment Knowledge, Beliefs and Outcome**

This chapter examines the final aspect of the study investigating relationships between treatment knowledge, beliefs and treatment outcome. It assesses relative contributions of treatment knowledge and beliefs to outcome and also explores whether belief in catastrophic cognitions mediate observed relationships. On the basis of literature reviewed in chapter 1, hypotheses regarding associations between treatment knowledge, beliefs and outcome were as follows:

1. Improving treatment knowledge will be associated with reduced Panic-Ag severity.
2. Greater posttreatment acceptance of the treatment rationale will be associated with reduced Panic-Ag severity.
3. Stronger treatment self-efficacy at posttreatment will be associated with reduced Panic-Ag severity.
4. Recovered participants will show greater treatment knowledge, pretreatment outcome expectancies, acceptance of the treatment rationale and treatment self-efficacy than non-recovered participants.
5. Relationships between treatment knowledge and outcome will be mediated by belief in catastrophic cognitions.
6. Relationships between treatment beliefs and outcome will be mediated by belief in catastrophic cognitions.

Additionally, exploratory analyses investigated associations between pretreatment beliefs (acceptance of the rationale, expectancies of outcome and treatment self-efficacy) and outcome. Finally, the influence of benzodiazepine use on acquisition of treatment knowledge was explored.
Method

Participants

Sample B (N = 41) and Sample C (N = 35) participants participated in this phase of the study.

Measures

Outcome measures were selected to assess four fundamental aspects of Panic-Ag symptoms: panic attack frequency, panic attack sensation severity, frequency of catastrophic cognitions and frequency of agoraphobic avoidance. These measures, detailed fully in chapter 2, are briefly described again here.

*Number of Panic Attacks Assessed on the ADIS-IV* (nPA-ADIS). The number of DSM-IV defined panic attacks experienced in the previous month was assessed using the Panic Disorder module of the ADIS-IV (Brown et al., 1994).

*Panic Attack Sensation Severity Assessed on the ADIS-IV* (PASS-ADIS). The PASS-ADIS assesses the severity of 14 physical and mental sensations experienced during an unexpected panic attack. Items were rated on a 9-point visual analogue scale ranging from 0 (*none*) to 8 (*very severe*). Scores ranged from 0 to 112 with higher scores reflecting greater symptom severity.

*Agoraphobic Cognitions Questionnaire – Frequency* (ACQ-Frequency, Chambless et al., 1984). Frequency of catastrophic cognitions was measured using the Agoraphobic Cognitions Questionnaire (Chambless et al., 1984). In brief, participants rated how often they experienced 14 catastrophic cognitions when anxious from 1 (*never*) to 5 (*always*). Scores on the ACQ-Frequency scale ranged from 14 to 70.
Mobility Inventory for Agoraphobia – Alone Subscale (MI-Alone, Chambless et al., 1985). The alone subscale of the Mobility Inventory of Agoraphobia (Chambless et al., 1985) was used to assess frequency of agoraphobic avoidance. The alone subscale was used in preference to the accompanied subscale, as degree of avoidance without the presence of a trusted companion was considered a more accurate measure of functional improvement. Participants rated how often they avoided 26 situations on a 5-point scale from 1 (never avoid) to 5 (always avoid). Scores on the MI-Alone ranged from 26 to 130 with higher scores indicating greater severity.

Agoraphobic Cognitions Questionnaire – Belief (ACQ-Belief). Belief in catastrophic cognitions was assessed using a modified version of the ACQ. Belief in the 14 ACQ items was assessed on a 5-point Likert scale from 0 (do not believe at all) to 4 (completely believe). Scores ranged from 0 to 56, with higher scores reflecting stronger beliefs.

Measures assessing treatment knowledge and beliefs were previously described in chapters 2, 3 and 4. Treatment knowledge was assessed with the Multiple-Choice Panic Disorder-Agoraphobia Treatment Knowledge Questionnaire (MC-PTKQ) and the Interview of Panic Disorder-Agoraphobia Treatment Knowledge (Int-PTK). Acceptance of the treatment rationale, expectancies of treatment outcome and treatment self-efficacy for Panic-Ag, were assessed with the ATR-PA, ETO-PA and TSE-PA, respectively.

Procedure

Assessment procedures for Samples B and C were identical to those described in chapters 2, 3 and 4. In summary, prior to treatment, all participants
initially completed an ADIS-IV assessment and a battery of self-report questionnaires assessing psychosocial functioning. Sample B participants then attended a pretreatment research appointment assessing treatment knowledge and beliefs after which they completed an 8-week group-based CBT program for Panic-Ag. At 6-months posttreatment, participants were mailed the same battery of self-report measures of psychosocial functioning which they completed and returned at a posttreatment assessment conducted at the clinic. During this posttreatment assessment the ADIS-IV was readministered, followed by measures assessing treatment knowledge and beliefs. Sample C participants completed an identical assessment procedure except they did not attend the pretreatment research appointment, hence pretreatment knowledge and belief data is unavailable for this sample.

**Statistical Analyses**

To examine treatment efficacy, repeated measures \( t \)-tests were applied to assess changes in the four Panic-Ag symptom domains (panic attack frequency, panic attack sensation severity, frequency of catastrophic cognitions, frequency of agoraphobic avoidance), treatment knowledge and beliefs (acceptance of the treatment rationale, treatment self-efficacy and belief in catastrophic cognitions) and effect sizes (Cohen’s \( d \)) were used to compare degree of change. Effect sizes are defined as follows: small: \( d = 0.2 \), medium: \( d = 0.5 \), large: \( d = 0.8 \). Analyses of covariance (ANCOVAs) that included pretreatment knowledge as a covariate were performed to investigate differences in posttreatment knowledge (MC-PTKQ, Int-PTK) between participants using and not using benzodiazepines.
Partial correlations were performed to investigate relationships between treatment knowledge, beliefs and Panic-Ag outcome, controlling for pretreatment Panic-Ag severity. Zero-order Pearson correlations were used to examine inter-relationships between treatment knowledge and beliefs at pre- and posttreatment.

Univariate hierarchical multiple regression analyses investigating relative contributions (Δ\(R^2\)) of treatment knowledge and beliefs to the four indices of Panic-Ag outcome were conducted. Posttreatment Panic-Ag severity scores (nPA-ADIS, PASS-ADIS, ACQ-Frequency, MI-Alone) were the dependent variables. To control for initial severity, pretreatment Panic-Ag scores were entered as the independent variable in step 1. For analyses involving treatment knowledge or belief in catastrophic cognitions, pretreatment MC-PTKQ/Int-PTK and ACQ-Belief scores were also entered at step 1 to control for pretreatment knowledge and catastrophic beliefs, respectively. Posttreatment knowledge (MC-PTKQ, Int-PTK), acceptance of the rationale (ATR-PA), treatment self-efficacy (TSE-PA) and pretreatment expectancies of treatment outcome (ETO-PA) were entered separately as independent variables in step 2.

Additional univariate hierarchical multiple regressions were conducted to determine whether relationships between treatment knowledge, beliefs and outcome were mediated by belief in catastrophic cognitions. As before, analyses involving Panic-Ag outcome, treatment knowledge or belief in catastrophic cognitions controlled for respective pretreatment scores in step 1.

Univariate analyses were selected over multivariate analyses for two reasons. Firstly, it was of interest to determine which specific aspects of Panic-Ag outcome were related to knowledge and beliefs. Secondly, as there were four outcome variables and analyses controlled for pretreatment Panic-Ag severity, multivariate
analyses therefore required control of four pretreatment severity variables (as opposed to only one for univariate analyses). The inclusion of an additional three predictors reduced power to unacceptable levels.

Finally, a series of univariate analyses of covariance (ANCOVA) investigated differences in treatment knowledge and beliefs between recovered and non-recovered participants, controlling for pretreatment severity. Analyses involving treatment knowledge and belief in catastrophic cognitions also controlled for pretreatment MC-PTKQ/Int-PTK and ACQ-Belief scores, respectively. To control for pretreatment symptom severity, a Panic-Ag severity composite was constructed from significantly inter-correlating pretreatment Panic-Ag variables. Of the four Panic-Ag measures, three shared significant inter-correlations: ACQ-Frequency correlated with PASS-ADIS \( (r = .44, p < .01) \) and MI-Alone \( (r = .42, p < .01) \). Frequency of panic attacks \( (nPA-ADIS) \) did not correlate significantly with any other Panic-Ag variable and therefore was excluded from the composite. To produce the composite, ACQ-Frequency, PASS-ADIS and MI-Alone scores were converted to z-scores to obtain a common metric and then averaged.

**Power Analyses**

A sample size of 41 and a significance level of \( p < .05 \) (two-tailed test) was used for analyses based on Sample B. As argued by Cohen (1994), adjusting the alpha error for multiple tests to reduce the Type I error rate would result in unacceptably low power (hence inflating the Type II error rate) and any result significant at this level would represent a gross overestimation of the population effect size.
Estimates of power to detect significant effects were extracted using formulas and power tables provided by Cohen (1988). By convention, at least 80% power is considered desirable to detect effects. Only medium and large effects were of interest as small effects are clinically unimportant. For repeated-measures analyses, there was 90% power for detecting medium effects. For correlations between knowledge, beliefs and outcome, there was 88% power to detect medium effects indicating sufficient power to detect meaningful relationships for these analyses.

Based on power graphs provided by Miles and Shelvin (2001), there was sufficient power for detecting large effects for regression analyses involving up to four predictors; medium effects required a sample size of \( N = 90 \). Regressions involving five predictors had 75% power to detect large effects.

**Results**

**Treatment Efficacy**

Table 5.1 displays pretreatment and posttreatment means, standard deviations and effect sizes for Panic-Ag symptom severity, treatment knowledge and beliefs for Sample B. As the distribution of panic attack frequency scores was skewed, a square-root transformation was applied and subsequent analyses used these transformed scores. Results revealed significant pre- to posttreatment reductions in Panic-Ag symptoms and catastrophic beliefs as well as significant pre- to posttreatment improvements in treatment knowledge, acceptance of the treatment rationale and treatment self-efficacy. These findings demonstrate treatment was efficacious in reducing Panic-Ag symptom severity and improving treatment knowledge and beliefs. As seen in Table 5.1, effect sizes were large (\( M = 1.39 \)).
Table 5.1 Pre- and Posttreatment Means, Standard Deviations and Effect Sizes for Panic-Ag Symptoms, Treatment Knowledge and Belief Measures for Sample B (N = 41)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
<th>t(40)</th>
<th>d*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Panic-Ag symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>nPA-ADIS(^a)</td>
<td>10.22</td>
<td>10.69</td>
<td>1.15</td>
<td>1.65</td>
</tr>
<tr>
<td>PASS-ADIS</td>
<td>62.56</td>
<td>20.15</td>
<td>30.45</td>
<td>22.62</td>
</tr>
<tr>
<td>ACQ–Frequency</td>
<td>35.02</td>
<td>9.92</td>
<td>26.12</td>
<td>9.03</td>
</tr>
<tr>
<td>MI-Alone</td>
<td>80.85</td>
<td>26.70</td>
<td>54.16</td>
<td>26.92</td>
</tr>
<tr>
<td>Catastrophic beliefs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACQ-Belief</td>
<td>19.51</td>
<td>12.35</td>
<td>8.22</td>
<td>8.06</td>
</tr>
<tr>
<td>Treatment knowledge</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Int-PTK</td>
<td>26.24</td>
<td>16.44</td>
<td>69.98</td>
<td>16.65</td>
</tr>
<tr>
<td>Treatment beliefs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ATR-PA</td>
<td>33.05</td>
<td>10.36</td>
<td>45.66</td>
<td>9.60</td>
</tr>
<tr>
<td>ETO-PA</td>
<td>27.10</td>
<td>5.79</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>TSE-PA</td>
<td>20.02</td>
<td>6.34</td>
<td>28.29</td>
<td>6.14</td>
</tr>
</tbody>
</table>

Note. nPA-ADIS = Number of panic attacks in the last month assessed on the ADIS-IV; PASS-ADIS = Panic attack sensation severity assessed on the ADIS-IV; ACQ-Frequency = Agoraphobic Cognitions Questionnaire-Frequency Score; MI-Alone = Mobility Inventory for Agoraphobia-Alone subscale; ACQ-Belief = Agoraphobic Cognitions Questionnaire-Belief Score; MC-PTKQ = Multiple-Choice Panic-Ag Treatment Knowledge Questionnaire; Int-PTK = Interview of Panic-Ag Treatment Knowledge; ATR-PA = Acceptance of the Treatment Rationale for Panic-Ag; ETO-PA = Expectancies of Treatment Outcome for Panic-Ag; TSE-PA = Treatment Self-Efficacy for Panic-Ag.

\(^a\)To achieve normality, analyses were performed on square-root transformed panic frequency scores.

\(^b\)Cohen’s effect size = \(\frac{M_{\text{pre}} - M_{\text{post}}}{SD_{\text{pooled}}}\), where \(SD_{\text{pooled}} = \sqrt[2]{\frac{(SD_{\text{pre}}^2 + SD_{\text{post}}^2)}{2}}\).

*\(p < .001\).

Significant reductions in Panic-Ag symptom severity were replicated in Sample C for nPA-ADIS: pretreatment: \(M = 6.80, SD = 8.63\), posttreatment: \(M = 2.29, SD = 3.98\), \(t(34) = 3.17, p = .003\), \(d = 0.68\), PASS-ADIS: pretreatment: \(M = 59.00, SD = 20.93\); posttreatment: \(M = 26.23, SD = 23.23\), \(t(34) = 7.37, p < .001\), \(d = 1.48\), ACQ-Frequency: pretreatment: \(M = 35.11, SD = 10.85\), posttreatment: \(M = \ldots\)
Chapter 5 – Relationships Between Treatment Knowledge, Beliefs and Outcome

23.97, SD = 8.47, \( t(34) = 6.62, p < .001, d = 1.14 \), and MI-Alone: pretreatment: \( M = 76.06, SD = 26.97 \), posttreatment: \( M = 47.24, SD = 24.40, t(34) = 7.93, p < .001, d = 1.12 \). The mean effect size was 1.11, further supporting the efficacy of the treatment protocol in reducing Panic-Ag symptoms.

Influence of Benzodiazepine Use on Treatment Knowledge

As stated earlier, several studies associated benzodiazepine use with learning and memory impairments. As such, ANCOVAs were performed to assess whether Sample B participants reporting benzodiazepine (BZ) use acquired less knowledge relative to their non-LBZ using counterparts. After controlling for pretreatment knowledge, no significant differences existed between the two groups on the MC-PTKQ (BZ: \( M = 30.44, SD = 5.25 \), non-LBZ: \( M = 29.84, SD = 7.09 \)), \( F(1, 38) = 0.12, p > .05, d = 0.10 \), or Int-PTK (BZ: \( M = 60.75, SD = 13.68 \), non-LBZ: \( M = 64.24, SD = 18.81 \)), \( F(1, 38) = 2.96, p > .05, d = 0.22 \). As such, benzodiazepine use was disregarded in subsequent analyses.

Relationships Between Treatment Knowledge and Treatment Outcome

To examine associations between treatment knowledge and outcome, partial correlations between posttreatment knowledge and posttreatment measures of Panic-Ag severity were computed for Sample B, controlling for pretreatment severity and pretreatment knowledge scores. Greater MC-PTKQ scores were significantly associated with decreased frequency of catastrophic cognitions (\( r = -.39, p = .015 \)) and agoraphobic avoidance (\( r = -.46, p = .003 \)). However, the MC-PTKQ was unrelated to panic attack frequency (\( r = -.06, p > .05 \)) or panic attack sensation severity (\( r = -.09, p > .05 \)). Higher scores on the Int-PTK were significantly
correlated with reduced frequency of agoraphobic cognitions \((r = -.40, p = .012)\), yet unrelated to other Panic-Ag domains (nPA-ADIS: \(r = .08\); PASS-ADIS: \(r = -.10\), MI-Alone: \(r = -.14, p > .05\)).

At posttreatment, three MCLPTKQ items were answered incorrectly by more than 70% of Sample B participants (Items 7, 17 and 18). Item 7 assessed understanding that panic attacks were maintained by threatening interpretations of physical sensations. Item 17 required comprehension of “overestimating the probability” and discriminating it from “overestimating the cost”. Item 18 concerned methods for reducing the difficulty of exposure tasks. Partial correlations between these three items and indices of treatment outcome, controlling for pretreatment severity and knowledge of respective items were not significant, when analysed individually \((r = .02 – -.21, p > .05, \text{mean } r = -.09)\) or combined \((r = -.01 – -.14, p > .05, \text{mean } r = -.07)\).

On the Int-PTK, more than 70% of participants responded at least partially incorrectly (i.e., scores of 3 or less out of 4) on nine items. Partial correlations between the sum of these nine items and treatment outcome, controlling for pretreatment severity and knowledge of such items, revealed no significant associations \((r = .00 – -.31, p > .05, \text{mean } r = -.13)\). Of these nine items, two correlated significantly with frequency of catastrophic cognitions: item 1b concerning symptoms of the fight/flight response and their function \((r = -.36, p = .025)\) and item 4 involving the identification of underlying catastrophic cognitions \((r = -.39, p = .015)\). However, with 36 separate partial correlations (9 items by 4 outcome variables), these significant results likely reflect chance findings (Type I errors).
Relationships Between Treatment Beliefs and Treatment Outcome

Pretreatment Beliefs

Controlling for pretreatment symptom severity, indices of treatment outcome (panic attack frequency, panic attack sensation severity, frequency of catastrophic cognitions and agoraphobic avoidance) were not significantly associated with pretreatment acceptance of the rationale ($r = .01 - .22$, $p > .05$), expectancies of treatment outcome ($r = .05 - .13$, $p > .05$), or treatment self-efficacy ($r = .00 - .15$, $p > .05$).

Posttreatment Beliefs

Partial correlations between posttreatment acceptance of the treatment rationale, treatment self-efficacy and treatment outcome were performed, controlling for pretreatment Panic-Ag severity. Associations were stronger for Sample C than Sample B. Outlier analysis was performed revealing the presence of one outlier in Sample C which was subsequently removed from all further analyses. Consequently, as displayed in Table 5.2, similar correlations for Sample B and Sample C were observed. Acceptance of the treatment rationale was not significantly associated with treatment outcome for Samples B or C, although correlations were generally in the predicted direction. Treatment self-efficacy was significantly related to all indices of treatment outcome (with the exception of frequency of panic attacks in Sample B), where higher self-efficacy was associated with less severe symptoms.
Table 5.2 Partial Correlations Between Posttreatment Acceptance of the Treatment Rationale, Treatment Self-Efficacy and Outcome, Controlling for Pretreatment Severity for Samples B and C

<table>
<thead>
<tr>
<th>Outcome variable</th>
<th>Sample B</th>
<th>Sample C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 41</td>
<td>N = 34*</td>
</tr>
<tr>
<td></td>
<td>ATR-PA</td>
<td>TSE-PA</td>
</tr>
<tr>
<td>nPA-ADIS</td>
<td>.02</td>
<td>-.21</td>
</tr>
<tr>
<td>PASS-ADIS</td>
<td>-.16</td>
<td>-.46**</td>
</tr>
<tr>
<td>ACQ-Frequency</td>
<td>-.30</td>
<td>-.54***</td>
</tr>
<tr>
<td>MI-Alone</td>
<td>-.22</td>
<td>-.46**</td>
</tr>
</tbody>
</table>

Note. nPA-ADIS = Number of panic attacks in the last month assessed on the ADIS-IV; PASS-ADIS = Panic attack sensation severity assessed on the ADIS-IV; ACQ-Frequency = Agoraphobic Cognitions Questionnaire-Frequency Score; MI-Alone = Mobility Inventory for Agoraphobia-Alone subscale; ATR-PA = Acceptance of the Treatment Rationale for Panic-Ag; TSE-PA = Treatment Self-Efficacy for Panic-Ag.

*aOne outlier was removed from analyses.

*bPosttreatment variable, controlling for pretreatment scores.

*p < .05. **p < .01. ***p < .001.

Using Sample B, partial correlations were also computed between belief in catastrophic cognitions and treatment outcome, controlling for pretreatment severity and pretreatment catastrophic beliefs. Belief in catastrophic cognitions was significantly related to all outcome measures, with stronger catastrophic beliefs associated with greater symptom severity: frequency of panic attacks, \( r = .32, p = .048 \); panic attack sensation severity, \( r = .39, p = .013 \); frequency of catastrophic cognitions, \( r = .56, p < .001 \); and agoraphobic avoidance, \( r = .57, p < .001 \). These analyses were unable to be performed for Sample C due to reliance on pretreatment catastrophic belief data which was unavailable for this sample.
Inter-relationships Between Treatment Knowledge and Beliefs

Zero-order Pearson correlations between pretreatment and posttreatment knowledge and belief variables for Sample B are displayed in Table 5.3. Significant relationships between variables were conceptually consistent. Treatment knowledge (MC-PTKQ, Int-PTK) and acceptance of the treatment rationale were significantly positively correlated when assessed concurrently such that greater knowledge was associated with increased rationale acceptance. In addition, greater posttreatment knowledge on the MC-PTKQ was significantly associated with higher posttreatment self-efficacy, although this relationship failed to reach significance with the Int-PTK ($r = .30, p < .06$). Of note, treatment knowledge was not significantly related to belief in catastrophic cognitions (ACQ-Belief).

Higher pretreatment acceptance of the rationale was significantly related to higher expectations of treatment outcome and greater treatment self-efficacy (at pre- and posttreatment). Stronger posttreatment acceptance of the rationale correlated significantly with higher posttreatment self-efficacy and lower posttreatment belief in catastrophic cognitions.

Significant positive associations existed between expectancy of treatment outcome and treatment self-efficacy such that participants who expected treatment to be helpful reported higher self-efficacy at pretreatment and posttreatment. Belief in catastrophic cognitions was significantly negatively associated with concurrent ratings of treatment self-efficacy, whereby participants endorsing strong belief in catastrophic cognitions expressed lower self-efficacy. Interestingly, pretreatment self-efficacy was not significantly associated with posttreatment self-efficacy indicating self-efficacy perceptions are flexible across treatment.
## Table 5.3 Zero-Order Pearson Correlations Between Pre- and Posttreatment Treatment Knowledge and Beliefs for Sample B (N = 41)

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. MC-PTKQ-Pre</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. MC-PTKQ-Post</td>
<td>.55***</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Int-PTK-Pre</td>
<td>.67***</td>
<td>.70***</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Int-PTK-Post</td>
<td>.49**</td>
<td>.72***</td>
<td>.61***</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. ATR-PA-Pre</td>
<td>.51**</td>
<td>.27</td>
<td>.36*</td>
<td>.37*</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. ATR-PA-Post</td>
<td>.27</td>
<td>.55***</td>
<td>.28</td>
<td>.55***</td>
<td>.37*</td>
<td>-</td>
<td></td>
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<td>7. ETO-PA-Pre</td>
<td>-.04</td>
<td>-.13</td>
<td>-.22</td>
<td>.03</td>
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<tr>
<td>8. TSE-PA-Pre</td>
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<td>-.16</td>
<td>-.07</td>
<td>.44**</td>
<td>.06</td>
<td>.53***</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. TSE-PA-Post</td>
<td>.12</td>
<td>.38*</td>
<td>.02</td>
<td>.30</td>
<td>.31*</td>
<td>.78***</td>
<td>.41**</td>
<td>.22</td>
<td>-</td>
<td></td>
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<tr>
<td>10. ACQ-Belief-Pre</td>
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<td>-.05</td>
<td>-.01</td>
<td>.03</td>
<td>-.30</td>
<td>-.27</td>
<td>-.29</td>
<td>-.35*</td>
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<td>-</td>
</tr>
<tr>
<td>11. ACQ-Belief-Post</td>
<td>-.12</td>
<td>-.25</td>
<td>-.12</td>
<td>-.07</td>
<td>-.16</td>
<td>-.42**</td>
<td>-.11</td>
<td>-.21</td>
<td>-.54***</td>
<td>.59***</td>
</tr>
</tbody>
</table>

*Note.* MC-PTKQ = Multiple-Choice Panic-Ag Treatment Knowledge Questionnaire; Int-PTK = Interview of Panic-Ag Treatment Knowledge; ATR-PA = Acceptance of the Treatment Rationale for Panic-Ag; ETO-PA = Expectancies of Treatment Outcome for Panic-Ag; TSE-PA = Treatment Self-Efficacy for Panic-Ag; ACQ-Belief = Agoraphobic Cognitions Questionnaire-Belief Score.

*p < .05. **p < .01. ***p < .001.
For Sample C, correlations between posttreatment knowledge and beliefs were largely similar to those observed for Sample B (see Table 5.4).

Table 5.4 Zero-Order Pearson Correlations Between Posttreatment Treatment Knowledge and Beliefs for Sample C (N = 34)

<table>
<thead>
<tr>
<th></th>
<th>MC-PTKQ</th>
<th>Int-PTK</th>
<th>ATR-PA</th>
<th>TSE-PA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Int-PTK</td>
<td>.62***</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ATR-PA</td>
<td>.70***</td>
<td>.34</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>TSE-PA</td>
<td>.21</td>
<td>-.06</td>
<td>.72***</td>
<td>-</td>
</tr>
<tr>
<td>ACQ-Belief</td>
<td>-.14</td>
<td>.03</td>
<td>-.50**</td>
<td>-.73***</td>
</tr>
</tbody>
</table>

*Note. MC-PTKQ = Multiple-Choice Panic-Ag Treatment Knowledge Questionnaire; Int-PTK = Interview of Panic-Ag Treatment Knowledge; ATR-PA = Acceptance of the Treatment Rationale for Panic-Ag; TSE-PA = Treatment Self-Efficacy for Panic-Ag; ACQ-Belief = Agoraphobic Cognitions Questionnaire-Belief Score.

One outlier was removed from analyses.

*p < .05. **p < .01. ***p < .001.

Contribution of Treatment Knowledge and Beliefs to Treatment Outcome

Table 5.5 summarises results of hierarchical multiple regression analyses examining contributions of knowledge and beliefs to variance across the four Panic-Ag outcome domains for Samples B and C. Analysis of residuals indicated no violations of assumptions. Analyses involving treatment knowledge and belief in catastrophic cognitions were unable to be performed in Sample C due to their reliance on pretreatment data which was unavailable for this sample.

Treatment knowledge, when assessed by the MC-PTKQ, exerted significant effects on frequency of catastrophic cognitions and agoraphobic avoidance, explaining an additional 9.6% and 14.8% variance after controlling for pretreatment severity, respectively. The Int-PTK also had a significant effect on frequency of
catastrophic cognitions, explaining an additional 10.1% variance, however it did not contribute significantly to variance in agoraphobic avoidance. Neither treatment knowledge measure contributed significantly to frequency of panic attacks or panic attack sensation severity.

In Sample B, treatment self-efficacy exerted significant effects on three of four outcome indices, accounting for 17.6%, 18.9% and 15.2% additional variance in panic attack symptom severity, frequency of agoraphobic cognitions and agoraphobic avoidance, respectively. Treatment self-efficacy did not significantly explain additional variance for panic attack frequency. For Sample C, treatment self-efficacy explained significant variance (12.3% to 24.4%) across all four outcome variables, including panic attack frequency. Acceptance of the treatment rationale did not significantly contribute to Panic-Ag outcome variance in either Sample B or C. Pretreatment expectancies of treatment outcome also failed to significantly explain such variability.

Consistent with Clark’s (1986) cognitive model, belief in catastrophic cognitions explained significant variance across the four symptom domains: 9.0% for frequency of panic attacks, 11.3% for panic attack sensation severity, 19.9% for frequency of catastrophic cognitions, and 23.3% for frequency of agoraphobic avoidance.
Table 5.5 Summary of Univariate Hierarchical Multiple Regression Analyses for Knowledge and Beliefs on Treatment Outcome, Controlling for Pretreatment Panic-Ag Severity for Samples B and C

<table>
<thead>
<tr>
<th>Dependent variable and predictor</th>
<th>Sample B (N = 41)</th>
<th></th>
<th></th>
<th>df</th>
<th>β</th>
<th></th>
<th></th>
<th></th>
<th>df</th>
<th>β</th>
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<tr>
<td></td>
<td></td>
<td>$R^2$</td>
<td>$\Delta R^2$</td>
<td>$\Delta F$</td>
<td>$df$</td>
<td>$\beta$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>nPA-ADIS</td>
<td></td>
<td>0.111</td>
<td>0.003</td>
<td>0.12</td>
<td>3, 37</td>
<td>-0.07</td>
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</tr>
<tr>
<td>MC-PTKQ$^b$</td>
<td></td>
<td>0.118</td>
<td>0.006</td>
<td>0.26</td>
<td>3, 37</td>
<td>0.10</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Int-PTK</td>
<td></td>
<td>0.083</td>
<td>0.004</td>
<td>0.17</td>
<td>2, 38</td>
<td>-0.07</td>
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<tr>
<td>ETO-PA</td>
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<td>0.080</td>
<td>0.000</td>
<td>0.01</td>
<td>2, 38</td>
<td>0.02</td>
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<td>ATR-PA</td>
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<td>0.118</td>
<td>0.039</td>
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<td>-0.20</td>
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<tr>
<td>TSE-PA</td>
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<td>0.198</td>
<td>0.090</td>
<td>4.17*</td>
<td>3, 37</td>
<td>0.37</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>ACQL-Belief$^c$</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>PASS-ADIS</td>
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<td>3, 37</td>
<td>-0.09</td>
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<tr>
<td>MC-PTKQ$^b$</td>
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<td>0.173</td>
<td>0.008</td>
<td>0.37</td>
<td>3, 37</td>
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<tr>
<td>Int-PTK</td>
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<td>0.166</td>
<td>0.002</td>
<td>0.10</td>
<td>2, 38</td>
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<tr>
<td>ETO-PA</td>
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<td>0.185</td>
<td>0.021</td>
<td>0.98</td>
<td>2, 38</td>
<td>-0.15</td>
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<td>ATR-PA</td>
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<td>0.340</td>
<td>0.176</td>
<td>10.13**</td>
<td>2, 38</td>
<td>-0.42</td>
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<tr>
<td>TSE-PA</td>
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<td>0.381</td>
<td>0.113</td>
<td>6.76*</td>
<td>3, 37</td>
<td>0.42</td>
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<tr>
<td>ACQ-Frequency</td>
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<td>0.096</td>
<td>6.56*</td>
<td>3, 37</td>
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<tr>
<td>MC-PTKQ$^b$</td>
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<td>0.465</td>
<td>0.101</td>
<td>7.01*</td>
<td>3, 37</td>
<td>-0.41</td>
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<tr>
<td>Int-PTK</td>
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<td>0.369</td>
<td>0.010</td>
<td>0.62</td>
<td>2, 38</td>
<td>-0.10</td>
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<tr>
<td>ETO-PA</td>
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<td>0.416</td>
<td>0.057</td>
<td>3.73</td>
<td>2, 38</td>
<td>-0.24</td>
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<td>ATR-PA</td>
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<td>15.93***</td>
<td>2, 38</td>
<td>-0.44</td>
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<tr>
<td>TSE-PA</td>
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<td>0.567</td>
<td>0.199</td>
<td>17.01***</td>
<td>3, 37</td>
<td>0.55</td>
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</table>

Sample C (N = 34$^d$)

<table>
<thead>
<tr>
<th>Dependent variable and predictor</th>
<th>Sample C (N = 34$^d$)</th>
<th></th>
<th></th>
<th>df</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>$\Delta R^2$</td>
<td>$\Delta F$</td>
<td>$df$</td>
<td>$\beta$</td>
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<tr>
<td>nPA-ADIS</td>
<td>0.168</td>
<td>0.068</td>
<td>2.54</td>
<td>2, 31</td>
<td>-0.27</td>
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<tr>
<td>Int-PTK</td>
<td>0.228</td>
<td>0.128</td>
<td>5.15*</td>
<td>2, 31</td>
<td>-0.38</td>
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<tr>
<td>ACQL-Belief$^c$</td>
<td>0.482</td>
<td>0.436</td>
<td>26.94***</td>
<td>2, 32</td>
<td>0.67***</td>
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<td>PASS-ADIS</td>
<td>0.126</td>
<td>0.070</td>
<td>2.48</td>
<td>2, 31</td>
<td>-0.27</td>
</tr>
<tr>
<td>ACQL-Frequency</td>
<td>0.300</td>
<td>0.244</td>
<td>10.80**</td>
<td>2, 31</td>
<td>-0.52</td>
</tr>
</tbody>
</table>

$^a$ Dependent variable

$^b$ MC-PTKQ

$^c$ ACQL-Belief

$^d$ Sample C

$^e$ ACQ-Frequency

$^f$ ACQ-Frequency

$^g$ ACQ-Frequency
## Table 5.5 (Continued) Summary of Univariate Hierarchical Multiple Regression Analyses for Knowledge and Beliefs on Treatment Outcome, Controlling for Pretreatment Panic-Ag Severity for Samples B and C

| Dependent variable and predictor | Sample B (N = 41) | | | | | | Sample C (N = 34<sup>d</sup>) | | | | | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| MI-Alone | R<sup>2</sup> | Δ R<sup>2</sup> | Δ F | df | β | R<sup>2</sup> | Δ R<sup>2</sup> | Δ F | df | β |
| MC-PTKQ<sup>b</sup> | .463 | .148 | 10.17** | 3, 37 | -.46 | | | | | | | | | |
| Int-PTK | .304 | .013 | .71*** | 3, 37 | -.15 | | | | | | | | | |
| ETO-PA | .292 | .002 | 0.09*** | 2, 38 | .04 | | | | | | | | | |
| ATR-PA | .325 | .034 | 1.93*** | 2, 38 | -.20 | | | | | | | | | |
| TSE-PA | .442 | .152 | 10.36** | 2, 38 | -.42 | | | | | | | | | |
| ACQL-Belief<sup>c</sup> | .525 | .233 | 18.15*** | 3, 37 | .63 | | | | | | | | | |
| ACQL-PA | .599 | .172 | 13.75** | 2, 31 | .43*** | | | | | | | | | |

Note. nPA-ADIS = Number of panic attacks in the last month assessed on the ADIS-IV; PASS-ADIS = Panic attack sensation severity assessed on the ADIS-IV; ACQ-Frequency = Agoraphobic Cognitions Questionnaire-Frequency Score; MI-Alone = Mobility Inventory for Agoraphobia-Alone subscale; MC-PTKQ = Multiple-Choice Panic-Ag Treatment Knowledge Questionnaire; Int-PTK = Interview of Panic-Ag Treatment Knowledge; ATR-PA = Acceptance of the Treatment Rationale for Panic-Ag; ETO-PA = Expectancies of Treatment Outcome for Panic-Ag; TSE-PA = Treatment Self-Efficacy for Panic-Ag; ACQL-Belief = Agoraphobic Cognitions Questionnaire-Belief Score.

<sup>a</sup>Controlling for pretreatment scores.
<sup>b</sup>Controlling for pretreatment MC-PTKQ scores.
<sup>c</sup>Controlling for pretreatment ACQL-Belief scores.
<sup>d</sup>One outlier was removed from analyses.

*p < .05. **p < .01. ***p < .001.
Belief in Catastrophic Cognitions as a Mediator of Relationships Between Treatment Knowledge, Beliefs and Outcome

Baron and Kenny (1986) described a four-stage process to establish mediation using regression equations. In step 1, the independent variable (treatment knowledge, treatment beliefs) significantly predicts the mediator (belief in catastrophic cognitions). In step 2, the independent variable (treatment knowledge, treatment beliefs) significantly predicts the dependent variable (Panic-Ag outcome). In step 3, the mediator (belief in catastrophic cognitions) significantly predicts the dependent variable (Panic-Ag outcome) after controlling for the independent variable (treatment knowledge, treatment beliefs). In step 4, perfect mediation arises when the independent variable no longer affects the dependent variable after controlling for the mediator. Partial mediation is indicated when the independent variable continues to affect the dependent variable but to a lesser degree than in step 2.

Treatment Knowledge

Associations between treatment knowledge and outcome do not appear to be mediated by belief in catastrophic cognitions. The first step of mediation, that treatment knowledge (MC-PTKQ, Int-PTK) predicted belief in catastrophic cognitions, was not satisfied, $\beta = -.03 - -.25, p > .05$.

Treatment Self-Efficacy

In contrast to treatment knowledge, belief in catastrophic cognitions appears to mediate relationships between treatment self-efficacy and outcome. Treatment self-efficacy significantly predicted belief in catastrophic cognitions, $\beta = -.41, p <$
.01, supporting the first step of mediation. The second step was confirmed through significant relationships between treatment self-efficacy and Panic-Ag outcomes (excluding panic attack frequency) as reported in Table 5.5. After controlling for treatment self-efficacy, belief in catastrophic cognitions significantly predicted frequency of catastrophic cognitions, $\beta = .39, p < .05$, and agoraphobic avoidance, $\beta = .49, p < .01$, thus supporting the third step of mediation; however belief in catastrophic cognitions did not mediate the relationship with panic attack sensation severity, $\beta = .27, p = .15$. Finally, after controlling for belief in catastrophic cognitions, treatment self-efficacy no longer significantly predicted agoraphobic avoidance, $\Delta R^2 = .045, F(4, 36) = 3.74, \beta = -.26, p = .06$; however it continued to predict frequency of catastrophic cognitions, $\Delta R^2 = .054, F(4, 36) = 5.17, \beta = -.28, p = .029$, albeit to a lesser extent than for step 2. These results indicate belief in catastrophic cognitions partially mediated relationships between treatment self-efficacy and Panic-Ag outcomes, with treatment self-efficacy having an independent effect on frequency of catastrophic cognitions.

Acceptance of the treatment rationale and expectancies of treatment outcome were excluded from these analyses as they formerly made no significant contribution to outcome variance (see Table 5.5).

**Treatment Knowledge and Beliefs According to Recovery Status**

At posttreatment, 25 of the 41 (61.0%) Sample B participants no longer met DSM-IV criteria for Panic Disorder and/or Agoraphobia (referred to as recovered) while 16 participants (39.0%) continued to meet diagnostic criteria (referred to as non-recovered). Figure 5.1 displays mean standardised knowledge and belief scores according to recovery status; raw means and standard deviations are also provided.
After controlling for pretreatment symptom severity, recovered participants demonstrated significantly higher posttreatment knowledge on the MC-PTKQ, $F(1, 37) = 4.80, p = .035, d = 0.63$, acceptance of the treatment rationale, $F(1, 38) = 4.93, p = .032, d = 0.79$ and treatment self-efficacy, $F(1, 38) = 13.83, p = .001, d = 1.25$, than non-recovered participants. Moreover, recovered participants expressed significantly lower belief in catastrophic cognitions than their non-recovered counterparts, $F(1, 38) = 7.49, p = .009, d = 0.86$. The groups did not differ significantly on treatment knowledge assessed on the Int-PTK, $F(1, 37) = 0.65, p > .05, d = 0.39$, or pretreatment expectancies of outcome, $F(1, 38) = 0.06, p > .05, d = 0.17$, although group means were in the predicted direction (see Figure 5.1).

**Figure 5.1.** Mean standardised scores and raw means and standard deviations of posttreatment knowledge (MC-PTKQ, Int-PTK), acceptance of the treatment rationale (ATR-PA), treatment self-efficacy (TSE-PA), belief in catastrophic cognitions (ACQ-B) and pretreatment expectancies of treatment outcome (ETO-PA) for recovered and non-recovered Sample B participants.
Within Sample C, 23 (67.6%) participants were classified as recovered and 11 (32.4%) as non-recovered. Mean knowledge and belief scores (raw and standardised) according to recovery status are displayed in Figure 5.2. As pretreatment data for knowledge and catastrophic beliefs were unavailable for this Sample, ANCOVAs could only be performed on ATR-PA and TSE-PA. As displayed in Figure 5.2, although means were in predicted directions, after controlling for pretreatment severity, no differences existed between recovered and non-recovered participants on ATR-PA, \(F(1, 32) = 1.19, p > .05, d = 0.24\) or TSE-PA, \(F(1, 32) = 1.16, p > .05, d = 0.60\).

![Figure 5.2](image-url)

**Figure 5.2.** Mean posttreatment standardised scores and raw means and standard deviations of treatment knowledge (MC-PTKQ, Int-PTK), acceptance of the treatment rationale (ATR-PA), treatment self-efficacy (TSE-PA) and belief in catastrophic cognitions (ACQ-B) for recovered and non-recovered Sample C participants.
Discussion

This study examined associations between treatment knowledge, beliefs and outcome following CBT for patients with Panic-Ag. Treatment outcome comprised four major indices of Panic-Ag: frequency of panic attacks, panic attack sensation severity, frequency of catastrophic cognitions and agoraphobic avoidance.

Relationships between treatment knowledge and outcome revealed that greater improvement in scores on the multiple-choice treatment knowledge measure (MC-PTKQ) was associated with reductions in two of the four Panic-Ag outcome indices: frequency of catastrophic cognitions and agoraphobic avoidance. In addition, after controlling for pretreatment severity and knowledge, the MC-PTKQ significantly explained additional variance in these two domains. Consistent with these results, recovered patients demonstrated significantly greater MC-PTKQ scores than non-recovered patients.

In contrast, the interview measure of treatment knowledge (Int-PTK) only partially replicated the above findings. Increased knowledge on the Int-PTK was associated only with reduced frequency of catastrophic cognitions, significantly explaining additional variance in this Panic-Ag outcome after controlling for pretreatment severity and knowledge.

Of the treatment beliefs examined in this study, posttreatment perceptions of treatment self-efficacy demonstrated the strongest relationship with outcome. Greater treatment self-efficacy was associated with reduced panic attack sensation severity, frequency of catastrophic cognitions and agoraphobic avoidance. These results were replicated in an independent sample, which also found a significant relationship with panic attack frequency. After controlling for pretreatment severity, treatment self-efficacy significantly contributed additional outcome variance. Further supporting
these relationships, recovered participants reported higher posttreatment self-efficacy than non-recovered participants. Taken together, these results indicate the more confident patients are in their ability to implement treatment recommendations the better their treatment outcome. In contrast, pretreatment perceptions of treatment self-efficacy were unrelated to Panic-Ag outcomes.

Contrary to predictions, greater acceptance of the treatment rationale was not significantly associated with treatment outcome, whether assessed at pretreatment or posttreatment. While recovered patients showed significantly stronger posttreatment rationale acceptance than non-recovered patients, this difference was not replicated in an independent sample. Pretreatment expectancies of treatment outcome were also unrelated to Panic-Ag outcome. Hence, the results of this study suggest patients’ beliefs about treatment prior to commencing CBT have no meaningful influence on treatment outcome.

Supporting Clark’s (1986) cognitive model of panic, belief in catastrophic cognitions was significantly associated with each domain of Panic-Ag outcome, explaining significant additional variance across the four outcome measures after controlling for pretreatment severity. In accordance with this finding, recovered patients reported significantly lower belief in catastrophic cognitions than non-recovered patients.

Mediational analyses indicated relationships between treatment self-efficacy and outcome were partially mediated by belief in catastrophic cognitions while relationships between treatment knowledge and outcome were not.

Interestingly, panic attack frequency was generally not associated with treatment knowledge and beliefs. Moreover, belief in catastrophic cognitions showed the weakest relationship with panic attack frequency relative to other dimensions of
Panic-Ag. Several possibilities may account for this finding. Firstly, panic attack frequency was assessed retrospectively on the ADIS-IV and therefore may not have provided as accurate a measure as prospective ratings. Alternatively, as only DSM-IV defined panic attacks were included in the rating, incorporating subthreshold panic attacks may have provided a more realistic assessment of this aspect of Panic-Ag outcome. Cho et al. (2007) also reported reduced associations among patients’ cognitive appraisals and panic attack frequency relative to other Panic-Ag aspects using a measure that excluded subthreshold attacks.

Panic attack frequency may be a poor measure of Panic-Ag severity in that it does not always differentiate between mild and severe patients and it may be insensitive to treatment effects. To illustrate this point, highly disabled agoraphobic patients can report no panic attacks prior to treatment due to extensive avoidance behaviour and therefore resemble milder patients who rarely experience panic attacks. Furthermore, for agoraphobic patients experiencing positive treatment responses, posttreatment panic attack frequency scores may show no change or even increase in response to exposure to previously avoided situations. Anticipated panic attack frequency may offer a preferable alternative for this aspect of Panic-Ag outcome as it takes into account patients’ predictions of panic attacks (without excluding subthreshold attacks) while also incorporating the disability associated with anticipation of panic.

In summary, improved treatment knowledge and greater self-efficacy for implementing CBT techniques at posttreatment were associated with reductions in several aspects of Panic-Ag severity, namely frequency of catastrophic cognitions, agoraphobic avoidance and panic attack sensation severity. Acceptance of the rationale and treatment beliefs held prior to commencing CBT were unrelated to
outcome. Consistent with the cognitive model, reduced belief in catastrophic cognitions was highly associated with improved treatment outcome. Of theoretical importance, belief in catastrophic cognitions partially mediated relationships between treatment self-efficacy and outcome but not between treatment knowledge and outcome.
Chapter 6

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General Discussion

This thesis aimed to investigate associations between patients’ knowledge and beliefs about CBT with Panic-Ag outcomes. Specifically, relative contributions of treatment knowledge, acceptance of the treatment rationale, pretreatment expectancies of outcome and treatment self-efficacy to four major Panic-Ag symptom domains were examined. Relationships between treatment knowledge, beliefs and outcome were hypothesised to be mediated by belief in catastrophic cognitions. The initial phase of this study involved developing measures assessing treatment knowledge and beliefs about CBT for Panic-Ag from the psychotherapy and medical literature, and extensive input from expert clinical psychologists and Panic-Ag patients. The psychometric properties of these measures were investigated using patient and clinician samples and found to be sound.

This research has several noteworthy strengths. Firstly, it is the first to investigate relationships between treatment knowledge, acceptance of the rationale, treatment self-efficacy and CBT outcomes for Panic-Ag. It also extends previous research by addressing many methodological weaknesses (i.e., restricting knowledge items to the assessment of treatment knowledge, objective scoring criteria free from therapist bias, assessing treatment expectancy in the absence of credibility ratings, developing multi-item measures with sound psychometric properties) that putatively contributed to inconsistent findings among different disorders and undermined observed relationships between treatment knowledge, beliefs and outcome. Finally, the use of an independent sample allowed cross-validation of findings pertaining to relationships between treatment self-efficacy, acceptance of the rationale and outcome.
Treatment Knowledge

Partial support was provided for the hypothesis that improved treatment knowledge is associated with reduced symptom severity. Responses to the multiple-choice knowledge questionnaire revealed significant associations with frequency of catastrophic cognitions and agoraphobic avoidance, after controlling for pretreatment knowledge and severity. In addition, the hypothesis that recovered participants will demonstrate significantly greater treatment knowledge than non-recovered participants was supported through scores on the multiple-choice knowledge questionnaire. Within the psychotherapy literature, these associations are consistent with results reported by Abramowitz et al. (2002) for OCD.

Interestingly, the interview measure of treatment knowledge only partially replicated results obtained from the multiple-choice knowledge questionnaire, in finding a significant association with reduced frequency of catastrophic cognitions but not agoraphobic avoidance. Moreover, recovered participants did not score significantly higher on the knowledge interview than non-recovered participants. As the two knowledge measures were highly inter-correlated ($r = .67 - .72, p < .001$) reflecting assessment of similar constructs, these discrepant findings are likely due to differences in measurement methods, namely recall (interview) versus recognition (multiple-choice).

Recall methods were putatively postulated to offer a more valid assessment of patients’ knowledge because the knowledge must be accessed from memory without assistance from prompts or reminders that could artificially inflate scores. However, such a notion appears incorrect. Assessing knowledge via interview has previously been criticised for penalising individuals with reduced verbal skills (Beeney et al., 1994; Dunn et al., 1984) and therefore may be a less valid measure than one which
allows individuals to demonstrate knowledge through recognition of previously learned information. Difficulties with expressive language during the knowledge interview may therefore have interfered with detection of the relationship with agoraphobic avoidance and difference between recovered and non-recovered participants.

In explaining the relationship between treatment knowledge and outcome, it is tempting to invoke a causal explanation whereby the association is mediated through increased treatment compliance (specifically, decreased unintentional non-compliance). From this perspective, knowledgeable patients are more likely to conduct higher quality behavioural experiments and cognitive restructuring which in turn improve clinical outcomes. For example, patients who understand the role of safety seeking behaviours in the maintenance of catastrophic beliefs are more likely to (a) identify and eliminate safety behaviours during behavioural experiments, and (b) relate outcomes from experiments to original predictions (i.e., disconfirmation of feared catastrophes) resulting in decreased belief in catastrophic cognitions which in turn reduce agoraphobic avoidance and frequency of catastrophic cognitions. As treatment compliance was not part of the study’s initial aims and therefore not assessed, this explanation is only speculative.

Such an explanation is consistent with the results of Schmidt and Woolaway-Bickel (2000) who reported that quality of CBT homework was a stronger predictor of Panic-Ag outcome than quantity of homework. Future research should examine homework/treatment compliance to determine whether it mediates the relationship between treatment knowledge and outcome. Conceptually, greater homework quality should encourage deeper emotional processing of information inconsistent with patients’ catastrophic beliefs (Schmidt & Woolaway-Bickel, 2000). Surprisingly, no
studies have examined whether homework compliance reduces belief in catastrophic cognitions in Panic-Ag.

Contrary to expectations, the present results did not support the hypothesis that relationships between treatment knowledge and outcome are mediated by belief in catastrophic cognitions. Improving patients’ knowledge was not sufficient for decreasing belief in catastrophic cognitions. Although treatment provides realistic non-threatening information about panic symptoms, patients may continue to doubt this information until it has been personally disproved. Therefore, in addition to improving comprehension of treatment information, clinicians are encouraged to focus on assisting patients to challenge catastrophic beliefs through additional methods, for example, via behavioural experiments and/or cognitive challenging. It is likely a combination of treatment knowledge and treatment compliance is necessary to reduce belief in catastrophic cognitions; however such an assertion awaits empirical investigation.

Given the correlational nature of this study, associations between treatment knowledge and outcome could also be explained by reverse causation whereby patients with better outcomes developed a deeper understanding of treatment as a result of symptom improvement. Depending on their symptom severity, patients could make post-hoc inferences about the accuracy of treatment information. For example, treatment responders may come to learn that panic attacks do not cause heart attacks through their reduced symptom profile. Multiple repeat assessments of symptom severity and treatment knowledge over the course of treatment and statistical methods incorporating time-series analyses (e.g., Bouchard et al., 2007) are required to clarify directions of causality.
A third explanation for the association is that greater improvement in treatment knowledge may reflect other patient variables. For example, patients keen to learn new coping skills or those with stronger therapeutic alliances may be more receptive to learning new information. Indeed, willingness to learn new coping skills and therapeutic alliance have positively predicted treatment outcomes in CBT for patients with anxiety and depression (Arnow et al., 2003; Burns & Nolen-Hoeksema, 1991; Vogel et al., 2006). Inclusion of these variables in future studies should help determine whether the treatment knowledge-outcome association is mediated by such non-specific effects.

Given the observed association between improved treatment knowledge and outcome, it would seem reasonable for clinicians to regularly assess patients’ comprehension of treatment information and correct areas of confusion to further improve Panic-Ag outcomes. Consistent with past research, this study found patients’ treatment knowledge was imperfect. At 6-months posttreatment, patients scored an average of 75.2% on the multiple-choice knowledge questionnaire and 72.9% on the knowledge interview, indicating approximately one quarter of treatment information was forgotten, poorly comprehended or misunderstood.

Clinicians are advised to assume patients do not have a clear understanding of treatment information until they can explain it back in their own words (Addis & Carpenter, 2000; Pulliam, Gatchel, & Robinson, 2003; Roter & Hall, 1994; Sanson-Fisher, Campbell, Redman, & Hennrikus, 1989). Strategies such as providing written information (Cox, Tisdelle, & Culbert, 1988; Ellis et al., 1979; Helbig & Fehm, 2004; Ivens & Sabin, 2006; Ley, 1998; Raynor, 1998), audiotaping therapy sessions (Macaskill, 1996), checking comprehension (Pulliam et al., 2003; Roter & Hall, 1994; Sanson-Fisher et al., 1989) and standardised quizzes (Abramowitz et al., 2002)
have been recommended to increase patient knowledge to improve compliance and treatment outcome. Offering patients forced-choice responses (e.g., “True or false: The fight or flight response is a harmless survival response”) may prove more useful for assessing knowledge in individuals with reduced verbal fluency. In addition, clinicians are advised to pay particular attention to assessing treatment comprehension of older patients, and those with less education and lower IQs based on the associations found in this study between treatment knowledge and age, education and intelligence.

A further clinical implication arising from the treatment knowledge-outcome association concerns improving clinician-patient communication of treatment information. Guided by literature from cognitive and educational psychology (Chandler & Sweller, 1991; Kalyuga, 2007; Mayer & Moreno, 2003; Moreno, 2007), future research could investigate teaching methods that enhance clinicians’ delivery of CBT to foster patients’ understanding of treatment. Presenting CBT treatment material to patients has the potential to cause cognitive overload; patients are required to cognitively process considerable amounts of information yet may lack sufficient cognitive resources to do so, contributing to reduced treatment comprehension. When designing treatment programs, factors such as information media type (written and/or spoken words, illustrations, videos), proportion of didactic versus interactive learning, information segmentation and session duration may need to be considered to optimise engagement with treatment and minimise cognitive load.
Acceptance of the Treatment Rationale

Contrary to the hypothesis, findings across two independent samples indicated no significant relationship between patients’ acceptance of the treatment rationale and Panic-Ag outcomes. This finding is inconsistent with previous studies reporting significant associations for patients with depression (Addis & Jacobson, 1996, 2000; Fennell & Teasdale, 1987). Although it is possible that this relationship is simply weaker for Panic-Ag, an alternative explanation may be due to differences between studies regarding measurement of rationale acceptance.

The present study assessed acceptance of the treatment rationale by obtaining a sum total of patients’ beliefs about specific aspects of CBT for Panic-Ag. In contrast, the studies of Fennell and Teasdale (1987) and Addis and Jacobson (1996, 2000) assessed rationale acceptance based on patients’ overall representations of the rationale. Consistent with gestalt theory (Wertheimer, 2003) that states psychological representations of a unified whole cannot be derived from summation of its parts, patients’ overall or gestalt representations of the rationale may be more highly associated with clinical outcome than ratings derived from the sum of its components. To better determine whether acceptance of the rationale is associated with outcome for Panic-Ag, future research should incorporate multi-item measures assessing acceptance of the rationale as a whole.

Treatment Self-Efficacy

The hypothesis that stronger treatment self-efficacy at posttreatment is associated with reduced Panic-Ag severity was strongly supported. These results are consistent with those reported for other disorders (e.g., Bélanger et al., 2005; Gerber et al., 2006; Iannotti et al., 2006; Nagia, 1999). Across two independent samples,
posttreatment ratings of treatment self-efficacy explained additional variance in panic
attack sensation severity, frequency of catastrophic cognitions and agoraphobic
avoidance, after controlling for pretreatment severity. The hypothesis that recovered
participants will report higher self-efficacy than their non-recovered counterparts was
also supported. Finally, as hypothesised, mediational analyses confirmed
relationships between treatment self-efficacy and outcome were partially mediated
by belief in catastrophic cognitions. This result suggests participants who were
confident in implementing CBT directives had lower belief in catastrophic cognitions
and in turn lower symptom severity following treatment.

Associations between treatment self-efficacy, belief in catastrophic
cognitions and outcome are presumably influenced by treatment compliance. That is,
individuals with greater confidence for implementing treatment instructions are
likely to be more compliant with treatment (i.e., exhibit less intentional non-
compliance). Increased compliance in turn provides additional opportunities for
correcting catastrophic misinterpretations of physical sensations resulting in
decreased Panic-Ag symptoms. As compliance was not investigated in this study,
mediational studies are necessary for determining whether treatment compliance
mediates relationships between treatment self-efficacy, belief in catastrophic
cognitions and Panic-Ag outcomes.

Although belief in catastrophic cognitions partially mediated relationships
between treatment self-efficacy and outcome, after controlling for belief in
catastrophic cognitions a significant independent relationship existed between
treatment self-efficacy and frequency of catastrophic cognitions. As previously
discussed in chapter 1, researchers have emphasised panic self-efficacy (confidence
in managing Panic-Ag symptoms) as central to the aetiology and maintenance of
Panic-Ag (Barlow, 1988; Casey, Oei, Newcombe et al., 2004; Rachman et al., 1986; Richards et al., 2002; Sanderson et al., 1989; Telch et al., 1996), with changes in panic self-efficacy mediating treatment outcome (Hoffart, 1995a; Williams et al., 1989; Zane & Williams, 1993). Treatment self-efficacy may promote greater panic self-efficacy, which in turn reduces Panic-Ag symptoms. Incorporation of panic self-efficacy as a mediating variable between treatment self-efficacy and outcome would be useful in elucidating this relationship. Alternatively, treatment self-efficacy may simply reflect broader underlying patient characteristics such as internal locus of control or agency expectancy (believing oneself is capable of change) which have both been predictive of CBT outcomes for patients with anxiety, depression and stress (Biswas & Chattopadhyay, 2001; Dozois & Westra, 2005; Hooke & Page, 2002).

Given the correlational nature of this study, it is also conceivable that patients reduced their level of treatment self-efficacy from their degree of symptom impairment and/or strength of catastrophic beliefs. That is, patients no longer experiencing Panic-Ag symptoms or believing previously held catastrophic thoughts are likely to have higher confidence in applying treatment recommendations involving the testing of catastrophic outcomes (behavioural experiments, graded exposure) than more symptomatic patients. Longitudinal studies incorporating multiple assessments of treatment self-efficacy, belief in catastrophic cognitions and Panic-Ag symptoms during the course of treatment would be useful to clarify directions of causality.

While posttreatment ratings of treatment self-efficacy were related to outcome, exploratory analyses revealed that pretreatment ratings were not. This latter finding is consistent with results reported by Bélanger et al. (2005) and Heapy et al.
Self-efficacy theory maintains self-efficacy is a dynamic construct that changes in response to feedback. Patients’ experiences with CBT can positively or negatively affect ongoing confidence for implementing treatment directives. On average, patients’ self-efficacy ratings increased significantly after receiving treatment, hence low pretreatment self-efficacy ratings are responsive to treatment experiences. However, treatment self-efficacy ratings which remain low and those found to decrease over treatment are cause for concern and should be addressed.

Should treatment self-efficacy exert a causal influence on treatment outcome, monitoring treatment self-efficacy ratings across therapy sessions would be useful to alert clinicians to patients’ doubts about their ability to comply with treatment recommendations (Bélanger et al., 2005). Clinicians could explore reasons contributing to low self-efficacy and implement strategies to enhance it.

To increase self-efficacy, Bandura (1977) asserted individuals’ self-efficacy beliefs are primarily derived from four sources: performance accomplishments, vicarious experience, verbal persuasion and emotional/physiological arousal. Individuals process information obtained from these sources to determine their level of self-efficacy for specific tasks. Performance accomplishments are considered to be the most influential in modifying self-efficacy beliefs, as such direct personal experiences contain highly salient and believable information about one’s capabilities.

In CBT for Panic-Ag, performance accomplishments take the form of exposure therapy or behavioural experiments. Patients’ doubts about their ability to implement treatment directives could be viewed as predictions requiring testing through behavioural experiments. When assigning and reviewing treatment recommendations (homework), pro-compliance behaviours (e.g., partial attempts)
could be highlighted as evidence to contradict patients’ negative predictions to instil
treatment self-efficacy. Principles of graduated exposure would also apply whereby
treatment directives could be graded in difficulty to promote early successes to
further increase treatment self-efficacy.

In addition to verbal persuasion, which is deemed only as strong as the
patient’s confidence in the clinician, another method for enhancing treatment self-
efficacy may involve using successfully treated past patients. These ex-patients could
function as guest speakers during early sessions of group CBT where their treatment
experiences are discussed with group members. This vicarious experience may boost
patients’ treatment self-efficacy by encouraging them to think, “If they can do it, so
can I”. Indeed, the United Kingdom National Health Service developed the *Expert
Patients Programme*, a self-management support group for individuals with chronic
diseases run exclusively by ex-patients that effectively improved treatment self-
efficacy and problems solving skills (Kennedy et al., 2007; Kennedy, Rogers, &
Gately, 2005). In addition, an earlier study by Verinis (1970) examined perceptions
of ex-patients as lay therapists in a group therapy program and found they were rated
as very helpful by patients. He argued ex-patients provide inspiration and model
appropriate behaviour more effectively than professionally trained therapists.

**Expectancies of Treatment Outcome**

Exploratory analyses revealed that treatment outcome expectancies assessed
prior to CBT were not associated with Panic-Ag treatment outcomes. As previous
research reported outcome expectancies assessed early in treatment are predictive of
clinical improvement, the present findings suggest pretreatment outcome
expectancies are not rigidly maintained but responsive to information presented early
in therapy that confirms or confutes original beliefs. Hence, pretreatment counselling to raise outcome expectancies for patients with negative beliefs about treatment helpfulness is unnecessary. Nevertheless, given research highlighting associations between outcome and treatment expectancies assessed within the first few therapy sessions, attending to factors contributing to poor outcome expectancies early in therapy is still warranted.

**Methodological Limitations**

This study has several limitations. First, small sample sizes limit the generalisability of findings. The absence of pretreatment data for Sample C prevented some findings obtained with Sample B from being replicated, although results for treatment self-efficacy and acceptance of the treatment rationale were generally consistent between the two samples. Studies using larger samples are required to replicate observed findings. In addition, larger samples would generate greater power allowing for multivariate statistical analyses.

Second, although the reliability and validity of the knowledge and belief measures were satisfactory using data from Sample B, and similar reliability estimates were found for Sample C, additional work is needed to cross-validate the psychometric properties of these measures using larger independent samples. Cross validation is particularly important because Sample B data was used throughout multiple phases of the study, from development and refinement of measures, examination of psychometric properties, to analysis of relationships with treatment outcome. Moreover, as previously discussed, Sample B was a higher functioning sample that represented only half of the eligible Panic-Ag patients referred to the
clinic, hence the psychometric properties of the knowledge and belief scales cannot be generalised to the wider Panic-Ag population.

Third, concurrent assessments of knowledge, beliefs and Panic-Ag severity and the correlational design of the study limit interpretation of causal relationships. As previously mentioned, multiple assessments of treatment knowledge and beliefs during treatment would allow more detailed analysis to determine directions of causality.

Fourth, the order of measures administered to participants was not counterbalanced. Intelligence tests were administered prior to assessment of participants’ knowledge, which in turn were administered prior to assessment of beliefs. Administration of the intelligence tests may have increased participants’ anxiety which potentially could have affected performance on the treatment knowledge tests. Furthermore, completion of the knowledge measures may have influenced participants’ treatment beliefs in some way. The issue of counterbalancing therefore needs to be addressed in future research.

Finally, treatment compliance was not assessed in this study. As previously stated, it would be important for future researchers investigating relationships between treatment knowledge, beliefs and outcome to include measures of treatment compliance to determine whether such relationships are mediated via patients’ compliance with treatment recommendations.
Conclusions

The present study found patients’ treatment knowledge and treatment self-efficacy were significantly associated with several aspects of Panic-Ag outcome. Belief in catastrophic cognitions partially mediated relationships between treatment self-efficacy and outcome, suggesting improved confidence in implementing treatment was useful in decreasing catastrophic beliefs which in turn reduced Panic-Ag symptoms. Acceptance of the treatment rationale as assessed in the present study and pretreatment outcome expectancies were unrelated to Panic-Ag severity.

The measures developed in this thesis and the obtained findings may have clinical value by assisting clinicians in identifying patients with insufficient knowledge and/or poor confidence in following treatment recommendations who consequently may be at risk of unintentional and intentional non-compliance and poor clinical outcomes. Future research using larger representative samples of Panic-Ag patients is required to further assess the measures’ psychometric properties so as to strengthen their clinical utility.

Although causal relationships cannot be ascertained, this study represents the first step in investigating patient knowledge and beliefs as means for improving treatment outcomes for Panic-Ag and paves the way for exciting new research examining associations with treatment compliance as a potential mediator of these relationships. Furthermore, in light of the findings obtained in this thesis, future research could also explore relationships between treatment knowledge, self-efficacy and outcome for other anxiety and related psychological disorders in an effort to enhance patient outcomes following CBT.
References


References


References


References


References


References


References


References


References


References


References


Surawy, C. (1989). Knowledge about diabetes in type 1 patients is related to metabolic control. *Diabetic Medicine, 6*(9), 784-786.


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Appendix A - 39-item Draft Multiple-Choice Knowledge Questionnaire

Panic Disorder and Agoraphobia Knowledge Scale

1. Title
   - Clinical Psychologist
   - Other (please specify) __________________________________________

2. Years of clinical experience ________________

3. Self-rated level of expertise with CBT for Panic Disorder and Agoraphobia (PD-Ag)
   - Very low (I would not feel at all confident in treating a person with PD-Ag)
   - Low
   - Medium
   - High
   - Very High (I would feel very confident in treating a person with PD-Ag)

Instructions for participants

Below are a number of multiple-choice questions designed to assess patient knowledge of the fundamental principles underlying cognitive behaviour therapy for Panic Disorder and Agoraphobia (PD-Ag). Please read each question carefully and using the scales provided please rate:

a) The relevance of each question to the cognitive behavioural treatment of PD-Ag
b) The comprehensibility or wording of each question and its respective answers (please feel free to make changes to the wording of items as you see fit.)
c) Whether or not you agree with the answer provided (answer highlighted in italics).

<table>
<thead>
<tr>
<th>Relevance</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all relevant to the treatment of PD-Ag</td>
<td></td>
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<tr>
<td>Very relevant to the treatment of PD-Ag</td>
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<table>
<thead>
<tr>
<th>Comprehensibility</th>
<th>0</th>
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<tbody>
<tr>
<td>Difficult for patients to understand</td>
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<td></td>
<td></td>
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<tr>
<td>Easy for patients to understand</td>
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<td></td>
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</table>

<table>
<thead>
<tr>
<th>Agreement</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>I agree with the answer provided</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I do not agree with the answer provided</td>
<td></td>
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</tbody>
</table>

There is space in each question for you to make additional comments or suggestions if you wish.
## Knowledge of CBT for Panic Disorder and Agoraphobia

<table>
<thead>
<tr>
<th>Question</th>
<th>Relevance to the treatment of PD-Ag</th>
<th>Comprehensibility of question and answers</th>
<th>Agreement with answer</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Which of the following statements about the cause of panic attacks is correct?</td>
<td>0 1 2</td>
<td>0 1 2</td>
<td>Y N</td>
<td>Comments</td>
</tr>
<tr>
<td>a. Panic attacks frequently occur following a period of physical stress</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>b. Panic attacks frequently occur following a period of psychological stress</td>
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<tr>
<td>c. People with a history of anxiety, depression or mental illness in the family are more likely to experience Panic Disorder or Agoraphobia than people without such a family history</td>
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<td></td>
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<tr>
<td>d. <strong>All of the above</strong></td>
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<td></td>
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</tr>
<tr>
<td>e. None of the above</td>
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<td></td>
<td></td>
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<tr>
<td>f. Don't know</td>
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<tr>
<th>2) Which of the following is a form of physical stress?</th>
<th>0 1 2</th>
<th>0 1 2</th>
<th>Y N</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Childbirth</td>
<td></td>
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<td></td>
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<tr>
<td>b. Sleep deprivation</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>c. Excessive use of drugs or alcohol</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>d. Being sick (e.g., flu)</td>
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<td></td>
<td></td>
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<tr>
<td>e. <strong>All of the above</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>f. Don't know</td>
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<table>
<thead>
<tr>
<th>3) Which of the following is a form of psychological stress?</th>
<th>0 1 2</th>
<th>0 1 2</th>
<th>Y N</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Running late</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>b. Relationship conflict</td>
<td></td>
<td></td>
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<tr>
<td>c. Financial pressures</td>
<td></td>
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<tr>
<td>d. Work deadlines</td>
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<tr>
<td>e. <strong>All of the above</strong></td>
<td></td>
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<tr>
<td>f. Don't know</td>
<td></td>
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</tbody>
</table>
4) **Which of the following statements is NOT correct**
   a. When anxiety levels are very low performance is low
   b. *When anxiety levels are very low performance is high*
   c. When anxiety levels are moderate performance is high
   d. When anxiety levels are very high performance is low
   e. The relationship between anxiety and performance follows an inverted U-curve
   f. Don’t know

<table>
<thead>
<tr>
<th>Relevance to the treatment of PD-Ag</th>
<th>Comprehensibility of question and answers</th>
<th>Agreement with answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2</td>
<td>0 1 2</td>
<td>Y N</td>
</tr>
<tr>
<td>Comments</td>
<td></td>
<td></td>
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</tbody>
</table>

5) **Which of the following reactions does NOT occur during the fight or flight response?**
   a. The face may go pale as blood is diverted away from parts of the body that don’t immediately require nutrition
   b. Heart rate and blood pressure increases so that oxygen and nutrients can be transported quickly to where they are needed
   c. Breathing speeds up to increase the amount of oxygen available to the muscles
   d. *Muscles relax to help you stay calm and perform at your best*
   e. Sweating increases to cool the body to prevent it from overheating during strenuous physical activity
   f. Don’t know

<table>
<thead>
<tr>
<th>Relevance to the treatment of PD-Ag</th>
<th>Comprehensibility of question and answers</th>
<th>Agreement with answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2</td>
<td>0 1 2</td>
<td>Y N</td>
</tr>
<tr>
<td>Comments</td>
<td></td>
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</table>

<p>| Comments                            |                           |                       |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Relevance to the treatment of PD-Ag</th>
<th>Comprehensibility of question and answers</th>
<th>Agreement with answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>6) Which of the following statements about the fight or flight response is correct?</td>
<td></td>
<td>0 1 2</td>
<td>0 1 2</td>
<td>Y N</td>
</tr>
<tr>
<td>a.</td>
<td>The fight or flight response is a natural biological anxiety reaction aimed to protect you from danger</td>
<td>Comments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b.</td>
<td>Symptoms from the fight or flight response are unpleasant but harmless</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c.</td>
<td>A panic attack is harmless as it is the fight or flight response being activated when there is no real danger</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>d.</td>
<td>All of the above</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>e.</td>
<td>a and b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>f.</td>
<td>Don’t know</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7) Which of the following statements about the fight or flight response is NOT correct?</td>
<td></td>
<td>0 1 2</td>
<td>0 1 2</td>
<td>Y N</td>
</tr>
<tr>
<td>a.</td>
<td>The fight or flight response only gets activated when you are physically in danger</td>
<td>Comments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b.</td>
<td>The fight or flight response gets activated whenever you think you are in danger</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c.</td>
<td>If you believe something to be dangerous even if it is not, your fight or flight response will be triggered</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d.</td>
<td>Worrying about appearing stupid or foolish in front of others can trigger off the fight or flight response.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e.</td>
<td>The body doesn’t distinguish between physical and psychological dangers when reacting with the fight or flight response</td>
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<td>f.</td>
<td>Don’t know</td>
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8) During hyperventilation, the levels of Carbon dioxide and Oxygen absorbed by the body changes in the following way:
   a. Carbon dioxide decreases and Oxygen increases
   b. Carbon dioxide increases and Oxygen increases
   c. Carbon dioxide increases and Oxygen decreases
   d. **Carbon dioxide decreases and Oxygen decreases**
   e. Carbon dioxide stays constant and Oxygen increases
   f. Don't know

9) It is often recommended that people breathe into a paper bag when they are hyperventilating. The purpose of this is to:
   a. Restore the level of Oxygen
   b. **Restore the level of Carbon dioxide**
   c. Prevent you from falling or fainting
   d. Prevent you from going crazy
   e. All of the above
   f. Don't know

10) Which of the following is NOT a symptom of hyperventilation?
    a. Dizziness
    b. Heart palpitations
    c. **Swollen feet**
    d. Blurred vision
    e. Feelings of unreality
    f. Don't know

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### 11) Which of the following is NOT a symptom of hyperventilation?

- a. Chest pain
- b. Feeling of choking or being smothered
- c. Tingling sensations (e.g., in the hands and feet)
- **d. Sore throat**
- e. Muscle stiffness
- f. Don’t know

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### 12) In individuals without a history of such problems, panic attacks are likely to cause

- a. Heart disease or heart attacks
- b. Stroke
- c. Insanity or mental illness (e.g., schizophrenia)
- d. All of the above
- **e. None of the above**
- f. Don’t know

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Comments
13) Which of the following statements about fainting and panic attacks is most correct?

- **a.** A sudden drop in blood pressure is needed to faint. Blood pressure drops during panic, so you’re more likely to faint during a panic attack.
- **b.** A sudden drop in blood pressure is needed to faint. Blood pressure rises during panic, so you’re less likely to faint during a panic attack.
- **c.** A sudden increase in blood pressure is needed to faint. Blood pressure rises during panic so you’re more likely to faint during a panic attack.
- **d.** A sudden increase in blood pressure is needed to faint. Blood pressure drops during panic, so you’re less likely to faint during a panic attack.
- **e.** Dizziness and feeling lightheaded are symptoms of a panic attack. If you experience these symptoms during a panic attack it means you are likely to faint.
- **f.** Don’t know

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Comments

**14) Which of the following statements regarding heart disease and panic attacks is correct?**

- **a.** The chest pain experienced during a panic attack is indistinguishable from the chest pain experienced during a heart attack in terms of sensations and duration.
- **b.** Symptoms of breathlessness and chest pain can occur during both a heart attack and a panic attack, but heart attack symptoms tend to be related to effort and will go away once you rest whereas symptoms of a panic attack can happen at any time.
- **c.** An ECG (electrocardiogram) is an instrument used to detect the occurrence of a heart attack. If the ECG was conducted after the panic attack had finished, it will not be able to detect whether a heart attack had occurred.
- **d.** The heart is not designed to cope with extreme anxiety and panic. Prolonged periods of anxiety cause structural changes to the heart resulting in heart disease.
- **e.** All of the above
- **f.** Don’t know

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Comments
15) Which of the following statements about hyperventilation is correct?

- a. Hyperventilation is responsible for many of the symptoms experienced during a panic attack. It is important to deliberately slow down your breathing rate during a panic attack in order to prevent something terrible from happening (e.g., collapse, stroke, heart attack, losing control) as hyperventilation is dangerous if uncontrolled.
- b. *The fight or flight response is an inbuilt survival response that protects you from danger. Hyperventilation is an important part of the fight or flight response and therefore is not dangerous. The sensations produced by hyperventilation are intense and unpleasant but completely harmless.*
- c. Hyperventilation is an important part of the fight or flight response and therefore is not dangerous. However if the fight or flight response is activated at the wrong time, hyperventilating causes intense physical sensation that may result in physical and/or mental harm.
- d. a and b
- e. a and c
- f. Don’t know

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Comments

16) According to the teachings of CBT, which of the following best explains the relationship between situations, thoughts and feeling?

- a. Situations lead to feelings. To change your feelings you need to change the situation.
- b. Situations lead to thoughts and thoughts lead to feelings. To change your feelings you need to change the situation.
- c. *Situations lead to thoughts and thoughts lead to feelings. To change your feelings you need to change your thoughts.*
- d. Situations lead to feelings and feelings lead to thoughts. To change your thoughts you need to change your feelings.
- e. Situations lead to thoughts and thoughts lead to feelings. To change your feelings you need to think positively.
- f. Don’t know

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Comments
17) According to the teachings of CBT, which of the following statements about panic attacks is NOT correct
a. If you think that panic attacks are dangerous you’ll become fearful of the sensations and will be more likely to have panic attacks in the future.
b. If you think that panic attacks are unpleasant but completely harmless, you’ll become less fearful of panic attacks and will be less likely to experience them in the future.
c. If you think panic attacks are dangerous you’ll become fearful of the sensations and will want to avoid situations that are associated with such symptoms.
d. If you think panic attacks are dangerous you’ll become fearful of the sensations and will want to avoid activities that trigger such sensations (e.g., exercise, drinking caffeine etc)
e. It is helpful to think that panic attacks are dangerous as it makes you respond in a way that keeps you safe from danger (e.g., dying, going crazy).
f. Don’t know

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Comments
### Question 18

According to the teachings of CBT, which of the following statements about the treatment of panic disorder and agoraphobia is correct?

- **a.** Treatment should be aimed at eliminating panic sensations because if you didn’t have the sensations you’d stop feeling anxious.

- **b.** *Treatment should be aimed at changing your thoughts about the dangerousness of panic symptoms as lots of normal activities and situations (e.g., exercise, intense emotions) trigger similar sensations as part of every day life.*

- **c.** Treatment should not be aimed at changing thoughts, because panic attacks can come on out of the blue even when you’re not thinking.

- **d.** Treatment should involve relaxation exercises because panic attacks are caused by excessive stress.

- **e.** Panic attacks are the result of a chemical imbalance in the brain and therefore treatment should always involve medication.

- **f.** Don’t know

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**Comments**
19) **According to the teachings of CBT, to reduce your fear of panic attacks and panic sensations:**
   a. You must firstly slow down your breathing to reduce the intensity of the symptoms.
   b. You must practice relaxation exercises regularly to reduce the stress response (the fight or flight response).
   c. **You must firstly identify the underlying thought that is causing you to be fearful about the sensations and then collect evidence to test out whether or not the thought is true.**
   d. You must probe into your past to discover issues from your early childhood that will explain the root of your fear.
   e. You must learn to identify your patterns of negative thinking and substitute them with positive thinking.
   f. Don’t know

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20) **According to the teachings of CBT, which of the following statements about the relationship between panic attacks and thoughts is NOT correct?**
   a. All people with panic disorder or agoraphobia have thoughts about their panic symptoms being dangerous in some way (either physically or psychologically dangerous).
   b. Thoughts about the dangerousness of panic symptoms keep the fear of panic attacks alive.
   c. Panic attacks can come on so quickly, even “out of the blue,” that there’s no time for thoughts. This means that thoughts aren’t always involved.
   d. If you don’t have any thoughts about your panic attacks, it means that thoughts are not involved.
   e. **c and d**
   f. Don’t know

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### Appendix A – Draft Multiple-Choice Knowledge Questionnaire

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<th>Agreement with answer</th>
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<tbody>
<tr>
<td>21) According to the teachings of CBT, if you’re having difficulty identifying your underlying thought about the dangerousness of panic…</td>
<td>0 1 2</td>
<td>0 1 2</td>
<td>Y N</td>
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<tr>
<td>a. It means there are simply no thoughts there and a medical explanation is required to explain the cause of your panic attacks and anxiety.</td>
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<td>b. It’s likely that the panic sensations are automatically associated with danger, so that you respond with fear to the sensations without needing to actually have the thought that the sensation is dangerous.</td>
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<tr>
<td>c. You can repeatedly ask yourself what would be so bad if the worst thing happened until you get to the core of the problem (e.g., If that happened what would be so bad about that? And if that happened, what would be so bad about that?).</td>
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<tr>
<td>d. You can observe your own behaviour when you’re anxious to look for clues that would help to explain the underlying thought.</td>
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<td>e. <strong>b, c and d</strong></td>
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<tr>
<td>f. Don’t know</td>
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<td>22) According to the teachings of CBT, if you’re feeling anxious in a situation that most people don’t find anxiety provoking (e.g., catching public transport or waiting in a line), it is likely to mean:</td>
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<td>0 1 2</td>
<td>Y N</td>
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<td>a. You believe there is something dangerous or scary about the situation for you</td>
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<td>b. Your beliefs about the situation are not based on the reality of the situation</td>
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<td>c. You are likely to be overestimating the likelihood of something bad happening</td>
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<td><strong>d. All of the above</strong></td>
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<td>e. None of the above</td>
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<tr>
<td>f. Don’t know</td>
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</table>
23) According to the teachings of CBT, which of the following statements about behavioural experiments is correct?

Behavioural experiments are a method for testing your thoughts. Behavioural experiments involve putting yourself in a situation to test whether or not your prediction about a situation is true.

a. Behavioural experiments involve subjecting your thoughts to reality testing and are an effective method for making your thoughts more realistic.

b. Behavioural experiments are an effective way of changing your feelings and behaviour.

c. People with panic disorder or agoraphobia should avoid conducting behavioural experiments as it could result in serious harm (e.g., heart attack, stroke, insanity, fainting)

d. All of the above

e. a and b only

f. Don't know

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24) According to the teachings of CBT, which of the following statements is correct?

a. The purpose behind changing your thoughts is to help you think positively about situations. If you think positively, you won’t feel anxious in feared situations.  

b. **The purpose behind changing your thoughts is to help you think realistically about situations. If you think realistically, your emotion will be appropriate for the situation.**  

c. The purpose behind changing your thoughts is to help you think negatively about situations. If you think negatively, you will be prepared for the worst which will help you respond quickly if something terrible happens.  

d. There’s no point trying to change your thoughts because thoughts are uncontrollable.  

e. If negative thoughts have been around for a long time they become a bad habit and cannot be changed.  

f. Don’t know

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25) According to the teachings of CBT, “Overestimating the probability” refers to:

a. Thinking about something that has happened and making it out to be much worse than it is in reality.  

b. **Thinking that something is very likely to happen when in reality it is very unlikely to occur.**  

c. Exaggerating the importance or significance of an event.  

d. All of the above  

e. b and c  

f. Don’t know

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### 26) According to the teachings of CBT, “Overestimating the cost” refers to:

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<tbody>
<tr>
<td>a.</td>
<td>Thinking that something bad will happen when in reality this is very unlikely to occur.</td>
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<td>b.</td>
<td>Believing that if something negative was to occur it would be a disaster when in reality the consequence of that negative event has little or no effect on your life.</td>
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<td>c.</td>
<td>Blowing the importance or significance of negative events out of proportion.</td>
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<td>d.</td>
<td>All of the above</td>
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<td>e.</td>
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<td>f.</td>
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**Comments**

### 27) According to the teachings of CBT, if you are very fearful of having a panic attack in public because you believe that strangers would be thinking negatively of you, the best approach for overcoming your anxiety in this situation would be to:

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<tr>
<td>a.</td>
<td>Look for clear evidence in the strangers’ behaviour that would either confirm or disprove your belief so that you can learn whether or not your belief is actually true.</td>
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<td>b.</td>
<td>Examine the consequence of their negative opinion on your life and put their opinion in perspective with other bad things that could happen (e.g., having a car accident, becoming a paraplegic, your family being killed).</td>
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<tr>
<td>c.</td>
<td>Tell yourself that you don’t care what people think of you even if you do care (i.e. think positively).</td>
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<td>d.</td>
<td>All of the above</td>
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<td>e.</td>
<td>a and b</td>
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<td>f.</td>
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**Comments**
28) According to the teachings of CBT, to manage your anxiety in a particular situation (e.g., waiting in a line, exercise, driving in traffic), it's best to:
   a. Use positive self-talk (e.g., “Don't worry, everything’s fine”).
   b. Avoid or leave the situation in case something terrible happens.
   c. Have someone around that can support or help you, or carry something with you to keep yourself safe (e.g., mobile phone, medication, bottle of water).
   d. All of the above
   e. **None of the above**
   f. Don't know

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29) According to the teachings of CBT, which of the following is a form of avoidance?
   a. Rushing through the shopping centre to get to the car or outside as soon as possible for fear of having a panic attack.
   b. Relying on a support person (e.g., spouse, parent, child, friend) to accompany you to places that you fear may trigger a panic attack.
   c. Declining a social invitation (e.g., a party) for fear of having a panic attack.
   d. **All of the above**
   e. None of the above
   f. Don't know

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### 30) According to the teachings of CBT, which of the following is a form of avoidance?

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<td>a.</td>
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<td>Y</td>
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<td>b.</td>
<td>1</td>
<td>1</td>
<td>N</td>
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<tr>
<td>c.</td>
<td>2</td>
<td>2</td>
<td>Y</td>
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<tr>
<td>d.</td>
<td>All of the above</td>
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<tr>
<td>e.</td>
<td>None of the above</td>
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<tr>
<td>f.</td>
<td>Don't know</td>
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**Comments**

- Not engaging in physical exertion (e.g., lifting heavy objects, running up the stairs, sexual activity) for fear of triggering panic symptoms
- Distracting yourself to avoid bringing on a panic attack
- Carrying lollies, mints, snacks or a bottle of water with you all the time in case you feel anxious or panicky.

### 31) According to the teachings of CBT, which of the following is a safety seeking behaviour?

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<td>d.</td>
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<td>e.</td>
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**Comments**

- Slowing your breathing to prevent a panic attack from developing.
- Carrying (but not actually taking) anti-anxiety medication with you when you enter an anxiety provoking situation.
- Carrying a paper bag

**All of the above**

- None of the above
- Don't know
32) According to the teachings of CBT, which of the following statements about safety seeking behaviours is NOT correct?

Using a safety seeking behaviour when you are in an anxiety provoking situation is NOT a useful approach to overcoming anxiety as it serves to prevent the terrible thing from happening (e.g., heart attack, fainting, embarrassing self in public)

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<td>a.</td>
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<td>e.</td>
<td>e. is a problem because if the bad thing didn’t happen (e.g., heart attack, loss of control, fainting etc), you still believe it would have happened if you hadn’t used the safety seeking behaviour.</td>
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33) **According to the teachings of CBT, which of the following statements about avoidance and anxiety is correct?**

a. When you avoid an anxiety provoking situation, your anxiety level rapidly decreases. You learn that it feels good to avoid and so you continue to avoid when you find yourself faced with similar situations. In this way, avoidance reinforces or strengthens your fears.

b. Avoidance quickly reduces your anxiety and can prevent panic attacks from occurring. As such, avoidance reduces your anxiety in the short term.

c. Avoidance stops you from testing out whether your thoughts about panic attacks are realistic and therefore keeps your fears alive.

d. When you avoid, you notice that the bad thing you were worrying about (e.g., heart attack, fainting, losing control) doesn't occur. This teaches you that in order to keep preventing the bad thing from happening you need to keep avoiding. In this way, avoidance keeps you feeling anxious in the long term.

e. **All of the above**

f. Don't know

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34) According to the teachings of CBT, which of the following statements about exposure therapy is NOT correct?

a. Facing your fears can cause intense anxiety and even panic attacks.

b. Exposure therapy is not recommended for the treatment of Panic Disorder or Agoraphobia as during exposure therapy anxiety levels can become so severe as to cause serious physical harm (e.g., heart attack, stroke, fainting) or mental harm (e.g., schizophrenia, insanity).

c. The more often you confront your fear, the less your anxiety will rise and the faster your anxiety will fall.

d. Exposure therapy is an excellent method for testing out whether your thoughts about the dangerousness of panic attacks are correct.

e. By facing your fears you learn that the thing you feared did not happen (or was not that bad). This increases your confidence about facing your fear in the future.

f. Don’t know

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Comments

35) According to the teachings of CBT, which of the following statements about exposure therapy is correct?

The aim of exposure therapy is to:

a. Change your thoughts about the dangerousness of panic sensations

b. Learn that the sensations are unpleasant but harmless

c. Help you learn that what you fear doesn’t happen or is not that bad

d. Break the association between your fear response and the feared situation/sensation

e. All of the above

f. Don’t know

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Comments
36) According to the teachings of CBT, which of the following statements about exposure therapy is NOT correct?

When facing your fears:

a. It’s important to construct an exposure hierarchy of feared situations/sensations from mildly anxiety producing to severely anxiety producing.

b. It’s best to start exposing yourself to feared situations/sensations in a graded way, starting off with mildly anxiety provoking situations and only moving on to more difficult situations when you’ve mastered the easier ones.

c. It’s important to predict what you think will happen if you didn’t do anything to keep yourself feeling safe, and then test this prediction by conducting a behavioural experiment.

d. It’s important to remind yourself at the end of the exposure session whether or not the outcome you feared (e.g., heart attack, fainting, insanity, vomiting etc) came true.

**e. It’s best to confront your most feared situation first. Once you face your worst fear, all your other fears will disappear.**

f. Don’t know

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37) **According to the teachings of CBT, which of the following statements about exposure goals is NOT correct?**

When planning your exposure goals:
- a. Your goals should be very specific in nature (e.g., To go to the movies during a crowded performance and sit in the middle of the cinema).
- **b. Your goals should be quite broad in nature and aim to eliminate anxiety (e.g., I want to be able to go out and not feel anxious or panicky)**
- c. Your goals should vary in difficulty from mildly anxiety provoking to extremely anxiety provoking.
- d. Your goals should involve both short-term and long-term goals.
- e. Your goals should be able to be broken down into small, achievable steps.
- f. Don’t know

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Comments

38) **According to the teachings of CBT, which of the following statements is correct?**

If your anxiety is very high when you’re attempting to face one of your fears and you’re having difficulty completing it:
- a. You could vary an aspect of the situation to decrease the difficulty of the task (e.g., vary the number of people present, time of day, distance from home)
- b. You could vary the amount of time you spend in the situation (e.g., 1 minute, 3 minutes, 10 minutes, 30 minutes etc)
- c. You could initially incorporate a safety seeking behaviour (e.g., support person, mobile phone) to help get you started, remembering to take it out later on.
- d. It means that you believe panic sensations are dangerous in some way.
- **e. All of the above**
- f. Don’t know

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Comments
39) According to the teachings of CBT, which of the following statements about exposure therapy is correct?

When conducting exposure therapy to a particular task:

a. It’s not necessary to go out of your daily routine. It’s best to just fit it in around your other commitments as this make the exposure more realistic to your individual lifestyle.

b. It doesn’t matter what you do to help yourself feel safe in the situation (e.g., sit down, carry medication, paper bag, mobile phone, support person), the most important thing is that you remain in the situation.

c. After you complete an exposure activity once, there’s no need to repeat it. You are now ready to move on to the next exposure activity.

d. *The more practice you do, the easier it gets and the more progress you will make.*

e. All of the above

f. Don’t know
Did you feel some treatment components were under-represented? If so, please specify.

Did you feel some treatment components were over-represented? If so, please specify.

Are there any other pieces of information about the treatment of PD-Ag that you think are necessary for patients to know about in order to help them overcome their disorder?

Other comments about the measure?

Thank you for your participation
Appendix B – Multiple-Choice Panic Disorder-Agoraphobia Treatment Knowledge Questionnaire (MC-PTKQ)

Below are some questions exploring what people know about Cognitive Behaviour Therapy (CBT) for Panic Disorder and Agoraphobia. Some of the questions are quite difficult and you are not expected to know all of the answers. It does not matter if you do not know any of the answers or if you know them all. This is not a test or exam, we are just interested in what you currently know about CBT for Panic Disorder and Agoraphobia. The information obtained will be treated in the strictest confidence and used only for research.

Important Instructions

- Read each question carefully before answering.
- Make sure you read all the options before making your selection.
- Circle the letter of the answer that you think is most correct.
- Circle only one answer per question.
- If you think you do not know the answer to a question, circle 'Don’t know' rather than simply guess.
- Do not spend too long on any question.
1) In individuals without a history of such problems, panic attacks are likely to cause:
   a. Heart disease and/or heart attacks
   b. Stroke
   c. Insanity (e.g., schizophrenia)
   d. All of the above
   e. None of the above
   f. Don’t know

2) According to the CBT approach, to reduce your fear of having panic attacks and panic sensations:
   a. You must firstly slow down your breathing to reduce the intensity of the symptoms.
   b. You must practice relaxation exercises regularly to reduce the stress response (the fight or flight response).
   c. You must firstly identify the underlying thought that is causing you to be fearful about the sensations and then collect evidence to test out whether or not the thought is true.
   d. You must probe into your past to discover issues from your early childhood that will explain the root of your fear.
   e. You must learn to identify your patterns of negative thinking and substitute them with positive thinking.
   f. Don’t know

3) Which of the following reactions does NOT occur during the fight or flight response?
   a. The face may go pale as blood is diverted away from parts of the body that do not immediately require nutrition.
   b. Heart rate and blood pressure increases so that oxygen and nutrients can be transported quickly to where they are needed.
   c. Breathing speeds up to increase the amount of oxygen available to the muscles.
   d. Muscles relax to help you stay calm and perform at your best.
   e. Sweating increases to cool the body to prevent it from overheating during strenuous physical activity.
   f. Don’t know

4) According to the CBT approach, if you are feeling anxious in a situation that most people do not find anxiety provoking (e.g., catching public transport or waiting in a line), it is likely to mean:
   a. You believe there is something threatening about the situation for you.
   b. Your beliefs about the situation are not based on the reality of the situation.
   c. You are overestimating the likelihood of something bad happening.
   d. All of the above
   e. None of the above
   f. Don’t know
5) **According to the CBT approach, which of the following is a form of avoidance?**
   
a. Not engaging in physical exertion (e.g., lifting heavy objects, running up the stairs, sexual activity) for fear of triggering panic symptoms.
   
b. Using distraction to prevent a panic attack from occurring.
   
c. Relying on a support person (e.g., spouse, parent, child, friend) to accompany you to places that you fear may trigger a panic attack.
   
d. Carrying lollies, mints, snacks or a bottle of water with you all the time in case you feel anxious or panicky.
   
e. *All of the above*
   
f. Don’t know

6) **Which of the following statements about the fight or flight response is false?**
   
a. The fight or flight response can get activated whenever you think you are in danger.
   
b. If you believe something to be dangerous (even if it is not), your fight or flight response can be triggered.
   
c. Worrying about appearing stupid or foolish in front of others can trigger off the fight or flight response.
   
d. The body does not always distinguish between physical and psychological dangers when reacting with the fight or flight response.
   
e. *The fight or flight response only gets activated when you are in real physical danger.*
   
f. Don’t know

7) **According to the CBT approach, which of the following statements about panic attacks is false?**
   
a. *If you do not have any thoughts about your panic attacks, it means that you do not perceive panic sensations to be threatening.*
   
b. All people with panic disorder or agoraphobia interpret their panic symptoms to be dangerous or threatening in some way.
   
c. Threatening interpretations of panic symptoms are responsible for keeping the fear of panic attacks alive.
   
d. Panic attacks can come on so quickly, even “out of the blue,” that there is no time for thoughts about the sensations. This means that panic sensations have become associated with danger so that you automatically respond with fear without consciously having any thoughts.
   
e. *All of the above*
   
f. Don’t know
8) **According to the CBT approach, which of the following statements is false?**

When planning your treatment goals...

a. Your goals should be very specific in nature (e.g., to go to the movies during a crowded performance and sit in the middle of the cinema).

b. *Your goals should be quite broad in nature and aim to eliminate anxiety (e.g., to be able to go out and not feel anxious or panicky).*

c. Your goals should vary in difficulty from mildly anxiety provoking to extremely anxiety provoking.

d. Your goals should involve both short-term and long-term goals.

e. Your goals should be able to be broken down into small, achievable steps.

f. Don’t know

9) **According to the CBT approach, which of the following statements about deliberately bringing on the feared panic sensations (interoceptive exposure) is correct?**

The aim of deliberately bringing on panic sensations is to...

a. Change your thoughts about the dangerousness of panic sensations.

b. Learn that panic sensations are unpleasant but harmless.

c. Help you become less anxious when you experience such sensations (e.g., dizziness, heart palpitations) as part of your everyday life.

d. Break the association between your fear response and the feared sensation.

e. **All of the above**

f. Don’t know

10) **Which of the following is NOT a symptom of hyperventilation?**

a. Dizziness

b. Tingling sensations (e.g., in the hands and feet)

c. *Swollen feet (oedema)*

d. Blurred vision

e. Chest pain

f. Don’t know

11) **According to the CBT approach, to manage your anxiety in an anxiety provoking situation (e.g., waiting in a line, exercise, driving in traffic), it is best to:**

a. Distract yourself.

b. Avoid or leave the situation in case something terrible happens.

c. Have someone around that can support or help you, or carry something with you to keep yourself safe (e.g., mobile phone, medication, bottle of water).

d. All of the above

e. *None of the above*

f. Don’t know
12) **Which of the following statements regarding heart disease and panic attacks is correct?**

   a. The chest pain experienced during a panic attack is exactly the same as the chest pain experienced during a heart attack in terms of sensations and duration.
   
   b. *Symptoms of breathlessness and chest pain can occur during a heart attack and a panic attack, but heart attack symptoms tend to be related to effort and will go away once you rest whereas symptoms of a panic attack can happen at any time.*
   
   c. An ECG (electrocardiogram) is an instrument used to detect the occurrence of a heart attack. If the ECG was conducted after the panic attack had finished, it will not be able to detect whether a heart attack had occurred.
   
   d. The heart is not designed to cope with extreme anxiety and panic. Prolonged periods of anxiety cause structural changes to the heart resulting in heart disease.
   
   e. All of the above
   
   f. Don’t know

13) **According to the CBT approach, which of the following statements involving facing your fears is false?**

   a. Behavioural experiments that involve facing your fears can cause intense anxiety and even panic attacks.
   
   b. *Facing your fears is not recommended for the treatment of Panic Disorder or Agoraphobia as anxiety levels can become so severe as to cause serious physical harm (e.g., heart attack, stroke, fainting) or mental harm (e.g., insanity).*
   
   c. The more often you confront your fear, the less your anxiety will rise and the faster your anxiety will fall.
   
   d. Confronting a feared situation is an excellent method for testing out whether your thoughts about the dangerousness of panic attacks are correct.
   
   e. By facing your fears you learn that the thing you feared did not happen (or was not that bad). This increases your confidence about facing your fear in the future.
   
   f. Don’t know

14) **Which of the following statements about hyperventilation is false?**

   a. Hyperventilation is responsible for many of the symptoms experienced during a panic attack. The sensations produced by hyperventilation may be intense and unpleasant but are completely harmless.
   
   b. Hyperventilation is an important part of the fight or flight response and therefore is not dangerous.
   
   c. It is important to deliberately slow down your breathing rate during a panic attack in order to prevent something terrible from happening (e.g., collapse, stroke, heart attack, losing control) as hyperventilation is dangerous if uncontrolled.
   
   d. If the fight or flight response is activated at the wrong time, hyperventilating causes intense physical sensations that may result in physical and/or mental harm.
   
   e. *c and d*
   
   f. Don’t know
15) **According to the CBT approach, which of the following is a safety seeking behaviour?**

a. Slowing your breathing to prevent a panic attack from developing
b. Carrying (but not actually taking) anti-anxiety medication with you when you enter an anxiety provoking situation
c. Carrying a paper bag
d. **All of the above**
e. None of the above
f. Don't know

16) **According to the CBT approach, which of the following statements about the treatment of panic disorder and agoraphobia is correct?**

a. Treatment should be aimed at changing your interpretation of panic symptoms because if you think that they are unpleasant but harmless, you will become less fearful and less likely to experience them in the future.
b. If you think panic attacks are dangerous you will become fearful of the sensations and will feel an urge to avoid situations or activities associated with such symptoms.
c. Treatment should be aimed at changing your thoughts about the dangerousness of panic symptoms as lots of normal activities and situations can trigger similar sensations as part of everyday life.
d. Treatment should not be aimed at changing your thoughts about panic symptoms, because panic attacks can come on “out of the blue” even when you are not thinking. Instead, treatment should be aimed at eliminating panic sensations because if you did not have the sensations you would stop feeling anxious.
e. **a, b and c**
f. Don't know

17) **According to the CBT approach, “Overestimating the probability” refers to:**

a. Thinking about something that has happened and making it out to be much worse than it is in reality.
b. **Thinking that something is more likely to happen than it is in reality.**
c. Exaggerating the importance or significance of an event.
d. **All of the above**
e. b and c
f. Don’t know
18) According to the CBT approach, which of the following statements is false?

If your anxiety is very high when you are attempting to face one of your fears and you are having difficulty completing it...

a. You could vary an aspect of the situation to decrease the difficulty of the task (e.g., vary the number of people present, time of day, distance from home etc).

b. You could vary the amount of time you spend in the situation (e.g., 1 minute, 3 minutes, 10 minutes, 30 minutes etc).

b. You could initially incorporate a safety seeking behaviour to help get you started, remembering to take it out later on.

c. It means that you believe panic sensations are dangerous in some way.

d. None of the above

e. Don't know

19) According to the CBT approach, which of the following statements are correct?

If you are having difficulty identifying why you are feeling panicky...

a. It means there are simply no thoughts there and a medical explanation is required to explain the cause of your panic attacks and anxiety.

b. It is likely that the panic sensations or situation have become associated with danger (probably because of past experiences), so that you now automatically respond with fear without consciously having thoughts about the sensations.

c. You can repeatedly ask yourself what would be so bad if the worst thing happened until you get to the core of the problem (Downward Arrow Technique).

d. You can observe your own behaviour when you are anxious to look for clues that would help to explain the underlying thought.

e. b, c and d

f. Don't know

20) According to the CBT approach, which of the following statements about behavioural experiments is correct?

The aim of behavioural experiments is to...

a. Change your thoughts about the dangerousness of panic sensations.

b. Learn that panic sensations are unpleasant but harmless.

c. Help you learn that what you fear does not happen or is not that bad.

d. Break the association between your fear response and the feared situation.

e. All of the above

f. Don't know
21) **According to the CBT approach, “Overestimating the cost” refers to:**
   a. Thinking that something bad will happen when in reality this is very unlikely to occur.
   b. Believing that if the bad thing did occur it would be a disaster when in reality the consequence would have little or no effect on your life.
   c. Blowing the importance or significance of negative events out of proportion.
   d. All of the above
   e. b and c
   f. Don’t know

22) **According to the CBT approach, which of the following statements is correct?**
   a. The purpose behind changing your thoughts is to help you think positively about situations. If you think positively, you will not feel anxious in feared situations.
   b. The purpose behind changing your thoughts is to help you think realistically about situations. If you think realistically, your emotion will be appropriate for the situation.
   c. The purpose behind changing your thoughts is to help you think negatively about situations. If you think negatively, you will be prepared for the worst which will help you respond quickly if something terrible happens.
   d. Attempting to change your thoughts is pointless because thoughts are uncontrollable.
   e. If negative thoughts have been around for a long time they become a bad habit and cannot be changed.
   f. Don’t know

23) **According to the CBT approach, if you worry that other people (e.g., strangers) may be thinking negatively of you, the best approach for overcoming your anxiety in this situation would be to:**
   a. Look for clear evidence in the stranger’s behaviour that would either confirm or disprove your belief so that you can learn whether or not your belief is actually true.
   b. Examine the consequence of their negative opinion on your life and put their opinion in perspective with other bad things that could happen (e.g., having a car accident, becoming a paraplegic, your family being killed).
   c. Tell yourself that you do not care what people think of you even if you do care (i.e. think positively).
   d. All of the above
   e. a and b
   f. Don’t know
24) According to the CBT approach, which of the following statements about safety seeking behaviours is false?
Using a safety seeking behaviour when you are in an anxiety provoking situation…
   a. *is a sensible approach to overcoming anxiety as it can help prevent the terrible thing from happening* (e.g., heart attack, fainting, embarrassing self in public).
   b. is a form of avoidance.
   c. stops you from testing out your thoughts about the dangerousness of panic.
   d. keeps your fears alive.
   e. is a problem because you will still believe that something bad would have happened if you had not used the safety seeking behaviour.
   f. Don’t know

25) Which of the following statements about the fight or flight response is correct?
   a. The fight or flight response is a natural reaction aimed to protect you from danger.
   b. Symptoms from the fight or flight response may be unpleasant but they are harmless.
   c. A panic attack is harmless as it is the fight or flight response being activated when there is no real danger.
   d. The fight or flight response is a mechanism that does not need to be controlled or stopped and it will go away on its own.
   e. *All of the above*
   f. Don’t know

26) Which of the following statements about fainting and panic attacks is most correct?
   a. A sudden drop in blood pressure is needed to faint. Blood pressure drops during panic, so you are more likely to faint during a panic attack.
   b. *A sudden drop in blood pressure is needed to faint. Blood pressure rises during panic, so you are less likely to faint during a panic attack.*
   c. A sudden increase in blood pressure is needed to faint. Blood pressure rises during panic so you are more likely to faint during a panic attack.
   d. A sudden increase in blood pressure is needed to faint. Blood pressure drops during panic, so you are less likely to faint during a panic attack.
   e. Dizziness and feeling lightheaded are symptoms of a panic attack. If you experience these symptoms during a panic attack it means you are likely to faint.
   f. Don’t know
27) According to the CBT approach, which of the following statements about avoidance is correct?

a. When you avoid an anxiety provoking situation, your anxiety decreases. In this way, avoidance reinforces or strengthens your fears.

b. Avoidance can prevent panic attacks from occurring.

c. Avoidance stops you from testing out whether your thoughts about panic attacks are true and therefore keeps your unrealistic fears alive.

d. Avoidance reduces your anxiety in the short-term but increases your anxiety in the long-term.

e. All of the above

f. Don’t know

28) According to the CBT approach, which of the following statements about facing your fears is false?

a. It is important to construct a graded hierarchy of feared situations from mildly anxiety producing to severely anxiety producing.

b. It is important to predict what you think will happen if you did not do anything to keep yourself feeling safe, and then test this prediction by conducting a behavioural experiment.

c. After confronting a feared situation, it is important to remind yourself at the end whether or not your fear came true.

d. It is important to regularly and repeatedly confront a feared situation until you no longer believe the situation to be dangerous.

e. It is best to confront your most feared situation first. Once you face your worst fear, all your other fears will disappear.

f. Don’t know

29) According to the CBT approach, which of the following statements about behavioural experiments is correct?

When conducting behavioural experiments to a particular task...

a. It is not necessary to go out of your daily routine. It is best to just fit it in around your other commitments as this makes the experiment more realistic to your individual lifestyle.

b. It does not matter what you do to help yourself feel safe during the experiment (e.g., sit down, carry medication, mobile phone, support person), the most important thing is that you remain in the situation.

c. You only need to conduct the behavioural experiment once. Repeating the experiment again is unnecessary. You are now ready to move on to the next situation.

d. The more practice you do, the easier it gets and the more progress you will make.

e. All of the above

f. Don’t know
Appendix C – Interview of Panic Disorder-Agoraphobia Treatment Knowledge (Int-PTK)

A. PSYCHOEDUCATION

1. Fight Flight Response

1a. What is the Fight or Flight Response? What is its purpose?

1b. Name 3 symptoms of the Fight or Flight Response and their function.

2. Hyperventilation

2a. What is Hyperventilation?

2b. Is hyperventilation dangerous? Why/Why not?

2c. Name 5 symptoms that can be caused by hyperventilating.

B. COGNITIVE THERAPY

3. Cognitive Model

3a. According to the CBT approach, why are panic attacks/panic sensations so frightening?

3b. What is responsible for keeping the fear of panic alive over time?

3c. According to the CBT approach, what do you need to do to reduce your fear of panic attacks/panic sensations?

4. Identification of the Causal Thought

4. According to the CBT approach, describe a method for uncovering the thought that is at the core of your fears. Give an example.

5. Probability & Cost

5a. What does “Overestimating the Probability” mean?

5b. According to the CBT approach, if you are overestimating the probability, how could you go about decreasing your probability estimates?

5c. What does “Overestimating the Cost” mean?

5d. According to the CBT approach, if you were overestimating the cost, how could you go about decreasing your cost estimates?
C. ROLE OF AVOIDANCE

6a. According to the CBT approach, how does avoidance maintain fear and anxiety?

6b. According to the CBT approach, what is a safety seeking behaviour?

6c. Give an example of a safety seeking behaviour.

6d. According to the CBT approach, what is the problem with using safety seeking behaviours?

D. EXPOSURE THERAPY

7. Behavioural Experiments

7a. According to the CBT approach, what is a behavioural experiment?

7b. Give an example of a behavioural experiment.

7c. What is the purpose of behavioural experiments in the treatment of Panic Disorder and Agoraphobia?

7d. According to the CBT approach, if your behavioural experiment was too hard, describe 3 ways to make it easier for yourself.

8. Interoceptive Exposure

8a. According to the CBT approach, what is the purpose of deliberately bringing on panic sensations (e.g., dizziness, heart palpitations, lightheadedness, shortness of breath)?

8b. Give an example of an exercise you could do to deliberately bring on panic sensations and its purpose.

8c. Why is it important to reduce your fear of panic sensations (e.g., dizziness, lightheadedness etc)?
Appendix D – Acceptance of the Treatment Rationale for Panic-Ag – 68 Item Version (ATR-PA-68)

*Reverse scored

<table>
<thead>
<tr>
<th>Psychoeducation Items</th>
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<th>1</th>
<th>2</th>
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</thead>
<tbody>
<tr>
<td>1. *Panic attacks are dangerous (i.e., can cause heart attacks, stroke, insanity).</td>
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<td>2. Hyperventilation can cause symptoms such as dizziness, lightheadedness and blurred vision.</td>
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<td>3. Hyperventilation can cause tingling sensations (e.g., in hands, feet, face), breathlessness, chest pain and heart palpitations.</td>
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<td>4. *If you feel faint during a panic attack you are likely to faint.</td>
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<td>5. Hyperventilation is harmless.</td>
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<td>6. Symptoms of the fight or flight response are the same as panic attack symptoms.</td>
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<td>7. A panic attack is just the fight or flight response coming on when there is no real danger.</td>
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<td>8. The fight or flight response can get activated just by thinking of something frightening.</td>
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<td>9. *I do not believe it when people tell me that panic attacks are not dangerous. The sensations are so intense that they must be dangerous.</td>
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<td>10. *Excessive anxiety and panic causes heart problems (e.g., heart disease, heart attacks).</td>
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<td>11. *Hyperventilation cannot cause the sharp chest pain sometimes experienced during a panic attack.</td>
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<td>12. Symptoms from the fight or flight response may be unpleasant but are harmless.</td>
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<td>13. *Feelings of unreality or the inability to think clearly during a panic attack is dangerous in that it can result in loss of control or insanity.</td>
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<td>14. *Chest pain experienced during panic indicates that a heart attack is likely to occur if the panic continues uncontrolled.</td>
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<td>15. *Hyperventilation if uncontrolled is dangerous and may cause physical and/or mental harm.</td>
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<td>16. *It is important to slow your breathing rate down during a panic attack as hyperventilating can be dangerous if uncontrolled.</td>
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<td>17. Hyperventilation may cause unpleasant and intense physical symptoms (heart palpitations, dizziness etc) but they are completely harmless.</td>
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<td>Cognitive Therapy Items</td>
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<td>18. To overcome the fear of panic attacks and panic sensations, you must first identify</td>
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<td>the thought that is causing you to be fearful because panic attacks are a result of</td>
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<td>your thoughts.</td>
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<td>19. Identifying and testing out threatening interpretations of panic is the best</td>
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<td>treatment for overcoming panic disorder and agoraphobia.</td>
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<td>20. If I worry a lot about what other people are thinking of me when I am panicking,</td>
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<td>it means I am overestimating the cost of negative evaluation.</td>
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<td>21. During a panic attack, I am overestimating the likelihood of something bad</td>
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<td>happening.</td>
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<td>22. *Treatment of Panic Disorder and Agoraphobia should just focus on eliminating</td>
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<td>panic symptoms and not on changing your thoughts.</td>
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<td>23. *Interpreting panic symptoms as threatening has very little to do with panic</td>
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<td>attacks.</td>
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<td>24. *Thoughts have very little to do with keeping my panic going. A chemical</td>
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<td>imbalance in the brain is responsible for the occurrence of panic.</td>
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<td>25. Some panic symptoms are likely to occur as part of everyday life (e.g., during</td>
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<td>exercise, fatigue, hunger, temperature change), therefore it is important to change</td>
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<td>the way you think about such symptoms so that you do not fear them when they occur.</td>
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<td>26. *There is no point in identifying and testing out my underlying thoughts about</td>
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<td>my panic symptoms as the symptoms can come on even when I am not having any thoughts.</td>
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<td>27. Treatment of Panic Disorder and Agoraphobia should focus on changing your</td>
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<td>thoughts, not on eliminating panic symptoms.</td>
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<td>28. *You should be concerned by the views strangers are having of you when you are</td>
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<td>panicking.</td>
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<td>29. It is the thoughts about the physical or psychological danger associated with</td>
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<td>panic that keeps the fear of panic alive.</td>
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<td>30. Behavioural experiments are a useful method for testing out the truth about my</td>
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<td>thoughts.</td>
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<td>31. The fear of panic is kept alive by my thoughts or interpretations of my</td>
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<td>symptoms.</td>
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<td>32. Testing out my thoughts will make me more confident that what I fear will not</td>
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<td>happen.</td>
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<td>33. *There is no point trying to change my thoughts. My panic attacks sometimes come</td>
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<td>on out of the blue so they do not have anything to do with my thoughts.</td>
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<tr>
<td>Do not believe at all</td>
<td>Slightly believe</td>
<td>Somewhat believe</td>
<td>Mostly believe</td>
<td>Completely believe</td>
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</tbody>
</table>

34. Changing the way I interpret my panic symptoms will help me overcome my panic. 0 1 2 3 4

35. Comparing the consequences of negative evaluation from strangers with the consequences of having a terminal illness is a good way of putting things into perspective to reduce your anxiety about what people may think of you if they see you panic. 0 1 2 3 4

36. Testing out the way I interpret my panic symptoms is a sensible approach for overcoming my panic. 0 1 2 3 4

37. By testing out my thoughts I will be able to overcome my fears. 0 1 2 3 4

38. Attempting to change your thoughts is pointless because thoughts are uncontrollable. 0 1 2 3 4

39. If negative thoughts have been around for a long time they become a bad habit and cannot be changed. 0 1 2 3 4

40. If I feel anxious in a situation it means I perceive that something threatening could occur. 0 1 2 3 4

**Avoidance Items**

41. Avoidance is responsible for keeping my panic attacks alive. 0 1 2 3 4

42. Avoiding things that make me feel anxious and panicky is the only way to manage my anxiety. 0 1 2 3 4

43. Carrying items such as medication, lollies, water, mobile phone, support person or using slow breathing or distraction are good ways to protect yourself from something bad happening if a panic attack occurred. 0 1 2 3 4

44. Using distraction or keeping busy is a helpful treatment approach for my anxiety and panic. 0 1 2 3 4

45. Relying on a support person to accompany me to a feared situation is a form of avoidance. 0 1 2 3 4

46. Slow breathing and/or relaxation exercises are the best methods for treating my panic. 0 1 2 3 4

47. Safety seeking behaviours keep your fears alive. 0 1 2 3 4

48. Using or carrying items such as lollies, water, paper bag, medication or a mobile phone when you feel panicky are safety seeking behaviours. 0 1 2 3 4

49. Controlling my breathing keeps me safe. 0 1 2 3 4

50. Avoidance stops you from testing out whether your thoughts about panic are true and therefore keeps your fears alive. 0 1 2 3 4

51. Using a safety seeking behaviour is a problem because you will still believe that something bad would have happened if you had not used it. 0 1 2 3 4
<table>
<thead>
<tr>
<th>Item</th>
<th>Statement</th>
<th>Response</th>
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<th>1</th>
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</thead>
<tbody>
<tr>
<td>52.</td>
<td>Deliberately bringing on the sensations I fear is a helpful method for reducing my fear of the symptom.</td>
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<td>53.</td>
<td>Confronting avoided situations in a gradual fashion (from mildly to highly anxiety provoking) is a sensible approach for overcoming my fears.</td>
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<td>0</td>
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<tr>
<td>54.</td>
<td>Using a safety seeking behaviour during a behavioural experiment interferes with a person’s ability to learn how dangerous the situation actually is.</td>
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<td>55.</td>
<td>*Facing my fears could cause my anxiety to become so severe as to cause serious physical or mental harm.</td>
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<td>56.</td>
<td>*Completing a behavioural experiment once is usually sufficient to overcome your fear. Repeating the experiment is therefore unnecessary.</td>
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<td>57.</td>
<td>Deliberately bringing on symptoms of panic is a helpful way of testing out my fears.</td>
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<tr>
<td>58.</td>
<td>After completing a behavioural experiment it is important to remind yourself at the end whether or not your prediction came true.</td>
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<td>59.</td>
<td>*Deliberately bringing on panic sensations can be dangerous.</td>
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<td>60.</td>
<td>Deliberately bringing on panic sensations is a helpful way to become less anxious when I experience such sensations as part of everyday life.</td>
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<td>61.</td>
<td>Facing my fears is a necessary part of treatment.</td>
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<td>62.</td>
<td>Treatment goals should vary in difficulty from mildly anxiety provoking to extremely anxiety provoking.</td>
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<td>63.</td>
<td>In order to reduce my fear, I need to regularly and repeatedly confront the situation until I no longer believe the situation is dangerous.</td>
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<td>64.</td>
<td>*Deliberately bringing on panic sensations is unhelpful.</td>
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<td>65.</td>
<td>Deliberately bringing on panic sensations is a helpful method to learn that the sensations may be unpleasant but harmless.</td>
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<td>66.</td>
<td>When planning treatment goals it is important to make them very specific in nature.</td>
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<td>67.</td>
<td>If I am having difficulty facing one of my fears, it means I believe there is something dangerous about panic.</td>
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<td>68.</td>
<td>Facing my fears helps me to learn that panic symptoms are harmless even if they are unpleasant.</td>
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Appendix E – Treatment Belief Scales

For the purposes of administration, items from the ATRLPA, ETOLPA and TSELPA scales were intermixed.

Acceptance of the Treatment Rationale for Panic-Ag (ATR-PA)

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<tr>
<td>Do not believe at all</td>
<td>Slightly believe</td>
<td>Somewhat believe</td>
<td>Mostly believe</td>
<td>Completely believe</td>
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<tr>
<td>1.</td>
<td>Confronting avoided situations in a gradual fashion (from mildly to highly anxiety provoking) is a sensible approach for overcoming my fears.</td>
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<td>2.</td>
<td>Deliberately bringing on symptoms of panic is a helpful way of testing out my fears.</td>
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<tr>
<td>3.</td>
<td>Some panic symptoms are likely to occur as part of everyday life (e.g., during exercise, fatigue, hunger, temperature change), therefore it is important to change the way you think about such symptoms so that you do not fear them when they occur.</td>
<td>0</td>
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<td>4.</td>
<td>A panic attack is just the fight or flight response coming on when there is no real danger.</td>
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<td>5.</td>
<td>*There is no point in identifying and testing out my underlying thoughts about my panic symptoms as the symptoms can come on even when I am not having any thoughts.</td>
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<td>6.</td>
<td>Deliberately bringing on panic sensations is a helpful way to become less anxious when I experience such sensations as part of everyday life.</td>
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<td>7.</td>
<td>Facing my fears is a necessary part of treatment.</td>
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<td>8.</td>
<td>Treatment goals should vary in difficulty from mildly anxiety provoking to extremely anxiety provoking.</td>
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<tr>
<td>9.</td>
<td>In order to reduce my fear, I need to regularly and repeatedly confront the situation until I no longer believe the situation is dangerous.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10.</td>
<td>Avoidance stops you from testing out whether your thoughts about panic are true and therefore keeps your fears alive.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11.</td>
<td>Deliberately bringing on panic sensations is a helpful method to learn that the sensations may be unpleasant but harmless.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12.</td>
<td>*Chest pain experienced during panic indicates that a heart attack is likely to occur if the panic continues uncontrolled.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13.</td>
<td>Testing out the way I interpret my panic symptoms is a sensible approach for overcoming my panic.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14.</td>
<td>Facing my fears helps me to learn that panic symptoms are harmless even if they are unpleasant.</td>
<td>0</td>
<td>1</td>
<td>2</td>
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</tbody>
</table>

* Reverse scored
Appendix E – Treatment Belief Scales

Expectancies of Treatment Outcome for Panic-Ag (ETO-PA)

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Do not believe at all</td>
<td>Slightly believe</td>
<td>Somewhat believe</td>
<td>Mostly believe</td>
<td>Completely believe</td>
<td></td>
</tr>
</tbody>
</table>

1. *When it comes to my panic/anxiety, I believe there is nothing that can be done to treat my problem.* 0 1 2 3 4
2. *When it comes to my panic/anxiety, I believe I will never be able to overcome it.* 0 1 2 3 4
3. *I do not believe CBT will be helpful for me.* 0 1 2 3 4
4. I believe CBT is the right treatment approach for my panic. 0 1 2 3 4
5. I made the right decision in attending therapy. 0 1 2 3 4
6. CBT will help me overcome my panic. 0 1 2 3 4
7. CBT can be helpful to manage even the most distressing panic symptoms. 0 1 2 3 4
8. *CBT is too simplistic to be helpful for treating my panic.* 0 1 2 3 4
9. CBT helps me understand why I panic and what I can do about it. 0 1 2 3 4

* Reverse scored

Treatment Self-Efficacy for Panic-Ag (TSE-PA)

<table>
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<th></th>
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<tbody>
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</tr>
</tbody>
</table>

1. During the early stages of a panic attack I can apply the skills I have learned to reduce the attack. 0 1 2 3 4
2. I can take the necessary steps to manage my anxiety effectively. 0 1 2 3 4
3. *The only way I can feel truly safe from my panic attacks is if I take medication.* 0 1 2 3 4
4. I feel as though I am well informed about my anxiety and panic. 0 1 2 3 4
5. *I can only control my panic with medication.* 0 1 2 3 4
6. I feel that I have learned strategies to effectively manage my anxiety and panic. 0 1 2 3 4
7. I feel that understanding my panic symptoms has helped me manage my anxiety. 0 1 2 3 4
8. I feel I can implement the techniques recommended by my therapist. 0 1 2 3 4
9. I feel that I have enough information about my panic to allow me to effectively manage my anxiety. 0 1 2 3 4

* Reverse scored
Appendix F – Aetiology, Alternative Non-CBT Treatments and Treatment Barriers Belief Scales

Below is a list of beliefs that some people have about Panic Disorder and Agoraphobia. Sometimes people's beliefs match what they have previously been told, and sometimes they differ. We are interested in what you truly or secretly believe (not what you think you should believe). Please read each item and circle the number using the scale below to rate the extent YOU believe the item to be true for you. Do not spend too long on any item. There are no right or wrong answers. We are interested in what you really believe.

**Aetiology**

<table>
<thead>
<tr>
<th>Do not believe at all</th>
<th>Slightly believe</th>
<th>Somewhat believe</th>
<th>Mostly believe</th>
<th>Completely believe</th>
</tr>
</thead>
</table>

I believe my panic/anxiety is caused by...

1. Something physically wrong with me 0 1 2 3 4
2. A medical condition that the doctors have not yet found 0 1 2 3 4
3. A chemical imbalance in my brain 0 1 2 3 4
4. A hormonal imbalance 0 1 2 3 4
5. Using prescription drugs 0 1 2 3 4
6. Using illicit drugs 0 1 2 3 4
7. Using alcohol 0 1 2 3 4
8. Taking too much caffeine 0 1 2 3 4
9. Exposure to environmental contaminants (e.g., toxic fumes, radiation, asbestos) 0 1 2 3 4
10. Inheriting anxious genes from my parents (genetics) 0 1 2 3 4
11. Early traumatic experiences from my childhood or adolescence 0 1 2 3 4
12. A traumatic experience (e.g., assault, rape, accident, disaster, war) 0 1 2 3 4
13. Stress from personal or family problems/stressful circumstances (e.g., death of a family member/friend, relationship conflict/break-up, financial problems) 0 1 2 3 4
14. Physical stress to my body (e.g., illness, virus, fatigue, childbirth) 0 1 2 3 4
15. A supernatural or spiritual force 0 1 2 3 4
16. Punishment from God for my past sins 0 1 2 3 4
### Alternative Non-CBT Treatments

<table>
<thead>
<tr>
<th>Item</th>
<th>0</th>
<th>1</th>
<th>2</th>
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</thead>
<tbody>
<tr>
<td>In order to properly treat my anxiety disorder I believe I need to..</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Rely on alcohol</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
<td>2. Rely on tranquilizers (e.g., Valium, Xanax, Serapax, Ativan,</td>
<td>0</td>
<td>1</td>
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<tr>
<td>Lexotan)</td>
<td></td>
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<tr>
<td>3. Rely on antidepressant medication (e.g., Zoloft, Prozac, Aropax,</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Cipramil, Avanza, Efexor-XR, Aurox, Prothiaden)</td>
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<tr>
<td>4. Treat the underlying medical problem</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
<td>5. Have further medical tests conducted</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. Address underlying issues from my childhood/adolescence</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<td>4</td>
</tr>
<tr>
<td>7. Probe into my past to discover the cause of my fear</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<td>4</td>
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<tr>
<td>8. Talk about my personal problems with a counsellor</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. Talk about my problem with someone who has had similar</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<td>4</td>
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<tr>
<td>experiences with anxiety</td>
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<td></td>
<td></td>
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<tr>
<td>10. Stay away from stressful things</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11. Avoid people, places or situations that trigger my anxiety</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<td>4</td>
</tr>
<tr>
<td>12. Avoid foods or substances that trigger my anxiety</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<tr>
<td>13. Slow my breathing down or practice breathing exercises</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<td>4</td>
</tr>
<tr>
<td>14. Undergo spiritual cleansing</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<td>4</td>
</tr>
<tr>
<td>15. Distract myself</td>
<td>0</td>
<td>1</td>
<td>2</td>
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<td>4</td>
</tr>
<tr>
<td>16. Practice yoga, meditation or exercise</td>
<td>0</td>
<td>1</td>
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</tr>
<tr>
<td>17. Have my sins forgiven by a religious/spiritual leader</td>
<td>0</td>
<td>1</td>
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</tbody>
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Appendix F – Aetiology, Non-CBT Treatments and Treatment Barriers Belief Scales

Treatment Barriers

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<thead>
<tr>
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<td>Completely believe</td>
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</table>

I believe the following factors will interfere in the treatment of my panic/anxiety

1. My age 0 1 2 3 4
2. My physical health 0 1 2 3 4
3. My level of intelligence 0 1 2 3 4
4. The intensity/severity of my symptoms 0 1 2 3 4
5. My level of anxiety 0 1 2 3 4
6. My depression 0 1 2 3 4
7. Presence of my other emotional/psychological problem(s) 0 1 2 3 4
8. Chemical imbalance in my brain 0 1 2 3 4
9. The previous effects of drugs/alcohol/toxins on my system 0 1 2 3 4
10. The hereditary nature of my problem (genetics) 0 1 2 3 4
11. The length of time I have had the problem 0 1 2 3 4
12. My previous failure to respond to treatment 0 1 2 3 4