Etiological Beliefs about Illness in Panic Disorder:

Relationship with Baseline Demographic and Clinical Characteristics and

Impact on Treatment Response

By

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Abstract

Purpose: The relation between the causal attributions of individuals with panic disorder (PD) and their health outcomes remains relatively unexplored. Therefore we examined 1) the relationship between participants' etiological beliefs about PD and baseline demographic and clinical characteristics and 2) whether participants' etiological beliefs about PD predicted compliance, clinical response, and side effect profiles with the treatments they were assigned. **Method:** The study included 251 participants. A series of multiple linear regressions were used to evaluate the relationship between participants' causal attributions, measured by the Etiological Model Questionnaire, and their baseline characteristics. To determine whether these beliefs predicted treatment outcome, logistic and linear regressions were conducted. **Results:** Our results revealed that participants with a family history of psychiatric illnesses were more likely to endorse biological etiological beliefs whereas those with a younger age, comorbid psychiatric disorders, and a history of suicide attempts were more likely to attribute their illness to psychological causes. Participants experiencing impairment in family life endorsed both psychological and environmental causal beliefs, while those reporting higher fear of body sensations and agoraphobic cognitions were more likely to attribute their illness to biological and psychological causes. With regards to treatment outcome, results indicated that participants who endorsed psychological and environmental etiological beliefs experienced more severe symptoms 12 weeks following treatment; irrespective of the type of treatment they received. **Implications:** The consideration of individuals' causal attributions might help health-care professionals better assist clients by communicating a more balanced perspective of the causes of PD and deliver interventions that are in line with clients' individual beliefs.

Résumé

Objectifs: La relation entre les attributions de cause des personnes atteintes de trouble panique (TP) et leurs effets sur la santé reste relativement inexplorée. Nous avons donc examiné 1) la relation entre les croyances étiologiques des participants au sujet de leur TP et leurs caractéristiques démographiques et cliniques de base et 2) si les croyances des participants permettent de prédire leurs résultats cliniques à l'égard des traitements attribués. **Méthodologie:** L'étude comprenait 251 participants. Une série d'analyses de régressions linéaires multiples a été utilisée pour évaluer la relation entre les croyances étiologiques des participants, mesurées par l'*Etiological Model Questionnaire*, et leurs caractéristiques de base. Pour déterminer si ces croyances prédisent les résultats du traitement, des régressions logistiques et linéaires hiérarchiques ont été effectuées. **Résultats:** Nos résultats ont révélé que les participants ayant des antécédents familiaux psychiatriques étaient plus susceptibles d'endosser des croyances étiologiques biologiques alors que ceux avec un plus jeune âge, des troubles concomitants, et une tentative de suicide au passé étaient plus susceptibles d'endosser des causes psychologiques. Les participants ayant un trouble dans la vie familiale endossaient à la fois des causes psychologiques et environnementales, tandis que ceux avec des niveaux plus élevés de peur de sensations physiques et de cognitions agoraphobiques étaient plus susceptibles d'attribuer leur condition à des causes à la fois psychologiques et biologiques. Concernant les résultats du traitement, nos analyses ont révélé que les participants qui endossaient des croyances environnementales et psychologiques ont éprouvé des symptômes plus sévères 12 semaines après le traitement, indépendamment du type de traitement attribué. **Implications:** Examiner les attributions de cause des individus pourrait assister les professionnels de santé à mieux aider les clients en communiquant une perspective plus équilibrée des causes du TP et concevoir des interventions en ligne avec les croyances individuelles des clients.

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Etiological Beliefs about Illness in Panic Disorder

Etiological beliefs are defined as individuals' perceptions about the origins of their illness (Franz et al., 2014). Although a few studies have explored the impact of illness perceptions on individuals' health outcomes, little attention has been given to individuals' causal attributions of illness, particularly anxiety disorders such as panic disorder (PD). The research described in this thesis examined the relationship between individuals' etiological beliefs about PD and baseline demographic and clinical characteristics, as well as their impact on treatment outcome. The thesis is organized as follows: In the Introduction section, I present an overview of PD, its treatment and etiology, as well as a review of the research literature on causal attributions that informed the current study. In the Methods section, I describe in detail the study context, participant characteristics and procedures, data collection methods, measures, and statistical approach. In the ensuing section (Results), I describe the findings of the study and finally, in the Discussion section, I discuss the findings as they relate to the literature on causal attributions as well as the study limitations and suggestions for future research.

Panic Disorder Overview

Panic Disorder is a disabling condition that affects about three out of every 100 individuals at some point in their lives (Canadian Psychological Association [CPA], 2014). The disorder is characterized by the repeated occurrence of unexpected panic attacks and anticipatory fear of future attacks (American Psychiatric Association [APA], 2013). Panic attacks are discrete episodes of intense anxiety that are accompanied by intense affective (anxiety, fear, apprehension) and somatic (e.g., dyspnea, palpitations, choking, sweating) symptoms. Approximately one-third of individuals with PD eventually develop agoraphobia (Keller & Hanks, 1993), which is characterized by fear and avoidance of situations where the dreaded panic attack might occur (APA, 2013). Typical agoraphobic situations include those where it may be embarrassing to panic (e.g., at a social event), where escape might be difficult (e.g., being in a subway) or where help may not be available in the event of an attack (e.g., being alone at home).

Epidemiological research indicates that the onset of PD typically occurs in middle adolescence to early adulthood (Keller & Hanks, 1993), with girls and women being twice as likely to develop the disorder than boys and men (Wittchen, Nelson, & Lachner, 1998). In many, but not all cases, the onset of PD is triggered by a stressful life event (Moitra et al., 2011). Longitudinal studies indicate that PD tends to be a chronic condition, with a course marked by

periods of remission and relapses (Roy-Byrne, Stand, Wittchen et al, 2000; Roy-Byrne & Cowley, 1995). The disorder is associated with a high prevalence of comorbid psychiatric disorders, the most notable being depressive disorders, other anxiety disorders, and substance use disorders, as well as increased risk for suicidal ideation and suicide attempts (APA, 2013; Beamish et al., 1996). PD is also frequently associated with medical comorbidities such as heart disease, chronic heart failure, irritable bowel syndrome, fibromyalgia, gastritis, stomach ulcers, arthritis, obesity, and asthma (Davidoff et al., 2012), although the nature of this association remains unclear.

PD is a costly illness, with the economic burden primarily attributed to a high use of medical services, diminished productivity and excessive absenteeism at work (Combs & Markam, 2014; Davidoff et al., 2012; Katon & Roy-Byrne, 1989). Individuals with PD also report a poor quality of life and significant impairment in psychosocial functioning (Carrera et al., 2006; Mendlowicz & Stein, 2000; Rubin et al., 2000; Markowitz et al., 1989), particularly when it is comorbid with other diagnoses (Bonham & Uhlenhuth, 2014). Compared to individuals with medical illnesses, individuals with PD have been found to have lower social functioning than those with hypertension, and worse quality of life than individuals with diabetes and heart disease (Srivastava, Shekhar, Bhatia, & Dwivedi, 2017; Candilis et al., 1999; Sherbourne, Wells, & Judd, 1996). Role limitations in daily activities due to emotional problems for individuals with PD have been found to be comparable to individuals with depression, yet more limited than those with other medical conditions, such as congestive heart failure (Srivastava et al., 2017; Candilis et al., 1999; Sherbourne et al., 1996). Finally, individuals with PD report more significant impairment with regards to social and familial relationships, leisure, and ability to function than those with other anxiety or affective disorders (except social phobia and obsessive compulsive disorder (OCD); Rapaport, Clary, Fayyad, & Endicott, 2005).

Fortunately, the morbidity and functional disability associated with PD can be reduced with effective pharmacological and psychological treatments. The next section will provide an overview of current evidence-based treatments for PD.

Treatment of Panic Disorder

Pharmacological interventions. Pharmacotherapy is one of the mainstay treatments for PD. The pharmacological interventions with the strongest evidence for efficacy in the treatment of PD include different classes of antidepressants (serotonin reuptake inhibitors, serotonin

norepinephrine reuptake inhibitors, monoamine oxidase inhibitors) and high potency benzodiazepines (Katzman et al., 2014).

First-line agents. Selective serotonin reuptake inhibitors (SSRIs) and serotonin norepinephrine reuptake inhibitors (SNRIs) are currently considered the front-line pharmacological treatments for PD because of their established efficacy, favourable side effect profile, and ease of use (McHugh, Smits, & Otto, 2009). SSRIs are believed to diminish symptoms of PD by selectively inhibiting the reuptake of serotonin in the brain, whereas SNRIs work by inhibiting the reuptake of both serotonin and norepinephrine (Apter, 1998).

Several meta-analyses (Andrisano, Chiesa, & Serretti, 2013; Mitte, 2005; Otto et al., 2001; Boyer, 1995) have demonstrated the superiority of SSRIs and SNRIs to placebo and other antidepressants for treating PD, with moderate effect sizes. These interventions have been associated with improvements in the frequency of panic attacks, agoraphobic avoidance, and general anxiety (Andrisano et al., 2013; Pollack et al., 2007; Bakker, Balkom, & Spinhoven, 2002). Bakker, Balkom, and Spinhoven (2002), reviewed the results of 43 studies on the efficacy of SSRIs and tricyclic antidepressants. Their results, as well as those of several other meta-analyses, have revealed that SSRIs and SNRIs were associated with lower dropout rates (Andrisano et al., 2013) and higher tolerability relative to the older tricyclic antidepressants and decreased risk for dependency relative to benzodiazepines (Lepola, Arato, & Austin, 2003; Pollack et al., 2000; Apter, 1998).

The side effects associated with SSRIs include restlessness and insomnia in the first days or weeks of treatment and fatigue, dizziness, nausea or weight gain, and sexual dysfunctions in long-term treatment (Bandelow, & Kaiya, 2006). In general, the side effect profile of SSRIs tends to be benign (Bandelow, & Kaiya, 2006).

Second-line agents. There is good evidence from randomized controlled trials (RCTs) to support the use of tricyclic antidepressants (TCAs) for the treatment of PD (Lecrubier, Bakker, & Dunbar, 1997; Modigh, Westberg, & Eriksson, 1992). Many studies (Lepola et al., 2003; Barlow, Gorman, Shear, & Woods, 2000), including meta-analyses (e.g., Bakker et al., 2002; Otto et al., 2001), have shown that clomipramine and imipramine have similar efficacy to SSRIs in treating panic symptoms, agoraphobic avoidance, depressive symptomatology, and general anxiety (Katzman et al., 2014). However, as noted above, TCAs have a more problematic side effect profile (Farach et al., 2012), resulting in a higher rate of treatment discontinuation relative to

SSRIs (Bakker at al., 2002). Due to the higher discontinuation rates associated with TCAs and their lower tolerability, this class of medication is recommended as second-line options for PD (Katzman et al., 2014), prescribed when first-line agents (e.g., SSRIs) do not result in optimal clinical improvement (Farach et al., 2012).

Other useful second-line treatments for PD include noradrenaline reuptake inhibitors (Seedat et al., 2003; Versiani et al., 2002) and benzodiazepines (Moylan et al., 2011). Benzodiazepines have been found to be particularly useful for the short-term management of acute or severe agitation or anxiety (Katzman et al., 2014) and to facilitate early improvement of panic symptoms at the initiation of SSRI treatment (Goddard et al., 2001). The chronic use of benzodiazepines has nevertheless been associated with physiological dependence (Bandelow, & Kaiya, 2006), sedation (Tiller, 2000), short-term cognitive and psychomotor impairment, and rebound anxiety once treatment is discontinued (Farach et al., 2012).

Third-line agents. Monoamine oxidase inhibitors (MAOIs) and reversible inhibitors of monoamine oxidase (RIMAs) are currently considered third-line options for the treatment of PD (Katzman et al., 2014). MAOIs and RIMAs are thought to be particularly useful for individuals with PD who have had suboptimal responses to first and second-line agents, and for severe, treatment-resistant anxiety disorders (Farach et al., 2012). Phenelzine, the most common MAOI for PD, has been found, in an earlier study, to lead to superior improvement compared to placebo in symptom severity, avoidance, and work and social disability (Sheehan, Ballenger, & Jacobsen, 1980). However, the use of this class of antidepressant is fairly restricted due to safety concerns (Bandelow, & Kaiya, 2006). MAOIs are associated with serious side effects such as dangerous hypertensive reactions, dietary restrictions (Bakish, Saxena, Bowen, & D'Souza, 1993), weight gain, sleep loss, and low tolerance (Farach et al., 2012).

Cognitive-behavior therapy. Cognitive behavior therapy (CBT) is recognized by several treatment guidelines as the gold standard psychological intervention for PD (Gloster et al., 2013; National Institute for Health and Clinical Excellence, 2011; Otto & Deveney, 2005). This intervention is based on a cognitive model of panic, which emphasizes the role of catastrophic cognitions and fear of anxiety sensations in the psychogenesis and maintenance of PD (Telch, Schmidt, Jaimez, Jacquin, & Harrington, 1995). CBT treatment protocols for PD typically include extensive psychoeducation about the nature of panic anxiety, monitoring and restructuring of maladaptive cognitions, abdominal breathing exercises, interoceptive exposure to

feared somatic cues of panic, and exposure to agoraphobic situations (Katzman et al., 2014; Pollack et al., 2003; Telch et al., 1995).

An extensive body of evidence exists in support of the efficacy of individual CBT for PD (Hofmann et al., 2012; Roshanaei-Moghaddam et al., 2011; Hendriks et al., 2010; Barlow et al., 2000; Gould, Otto, Pollack, 1995; Clum, Clum, & Surls, 1993). In a meta-analysis of 124 studies, Mitte (2005) found that CBT was more effective than a no-treatment and a placebo control, with large effects. Another meta-analysis of 21 studies comparing pharmacotherapy to CBT in anxiety disorders by Roshanaei-Moghaddam et al. (2011) revealed that CBT fared better than medications for PD, with a moderate effect size. Specifically, Sanchez-Meca et al. (2010) indicated that the CBT techniques of exposure (both interoceptive and in vivo), combined with breathing retraining, and relaxation or anxiety management training provided the greatest benefits for the treatment of PD, especially when these interventions included homework and a follow-up in which treatment is extended out of the therapeutic environment to more natural contexts (Sanchez-Meca et al., 2010). Numerous studies indicate that CBT for PD is a cost-effective modality (McHugh et al., 2009) that offers a strong maintenance of treatment benefits, superior symptom control, and higher treatment tolerability than certain pharmacological interventions, including benzodiazepines and antidepressants such as imipramine (Manfro, Heldt, Cordioli, & Otto, 2008; Otto & Deveney, 2005; Rayburn & Otto, 2003; Telch et al., 1995). Moreover, CBT has been found to result in significantly less impairment in quality of life (Mitte, 2005), including working inside and outside the home, leisure activities, and family and marital relationships, compared to no-treatment controls (Telch et al., 1995).

Cognitive-behavior group therapy (CBGT) also appears to be effective in the treatment of PD (Manfro et al., 2008), resulting in increases in emotional and physical aspects of quality of life and a significant decrease in PD symptoms (Rufer et al., 2010). In a randomized controlled trial (Marchand, Roberge, Primiano, & Germain, 2009), CBGT was as effective as individual CBT in reducing symptom intensity, relapse rates and maintaining treatment gains in the medium and long term. Another RCT (Roberge, Marchand, Reinharz, & Savard, 2008) found that CBGT was associated with lower treatment costs and a higher cost-effectiveness ratio, compared with individual CBT. Nevertheless, CBGT has been associated with a few challenges, including individuals' hesitance to share their personal experiences within a group format, concerns about confidentiality, and social fears in general (Norton, 2012). Additionally, some group members

convey a fear over "contagion"; the acquisition of fears expressed by other members (White, & Freeman, 2000).

Finally, there is evidence that self-administered CBT (SCBT) is beneficial for PD (Haug, Nordgreen, Ôst, & Havik, 2012; Lewis et al., 2003). SCBT in the form of written manuals, the Internet, and audio or video recordings incorporates standard cognitive-behavioural techniques used in face-to-face sessions (e.g., psychoeducation, relaxation, graded exposure, cognitive restructuring, and anxiety management) (Van't Hof, Cuijpers, & Stein, 2009). Typically, SCBT is administered with minimal therapist contact in stepped care models of care (Haug et al., 2012; Kiropoulos et al., 2008; Barlow et al., 2005), although it can also be used without any therapist support (Lewis et al., 2003). SCBT has been found to lead to significant improvements in panic frequency and severity, panic-related cognitions, agoraphobic avoidance (Kiropoulos et al., 2008; Barlow, Ellard, Hainsworth, Jones, & Fisher, 2005), bodily sensations related to anxiety arousal, anxiety symptoms, and depression (Carlbring et al., 2005). Several meta-analytic reviews of randomized controlled trials have concluded that SCBT is an effective treatment option for PD with moderate to large effects (Lewis, Pearce, & Bisson, 2012; Spek et al., 2007), and comparable in efficacy to therapist-delivered CBT (Haug et al., 2012; Cuijpers et al., 2010).

SCBT is thus considered to be a therapist-time-efficient alternative treatment for PD (Côté et al., 1994), that is cost-effective, fits clients' individual agendas, and provides those who cannot access or afford therapist-directed CBT, an evidence-based psychological treatment (Van't Hof et al., 2009; Carlbring et al., 2005). However, certain limitations have been identified with this treatment method, most notably non-compliance with treatment (MacLeod, Martinez, & Williams, 2009) and higher dropout rates (Waller, & Gilbody, 2009). Due to the importance of compliance in SCBT, it has been suggested that this factor may be improved with some degree of therapist contact (Gellatly et al., 2007). Other problems associated with SCBT include a lack of detection of a worsening of individuals' clinical state as a result of reduced therapist contact, missed opportunity for non-specific therapy factors (e.g., the therapeutic relationship), lack of confidentiality, and motivation on the client's part (MacLeod et al., 2009).

Combination of antidepressants and CBT. The results of a large number of studies investigating the effectiveness of antidepressants and CBT for the treatment of PD suggest that a combination of both treatment modalities can lead to a better outcome than placebo (Furukawa et al., 2007; Furukawa, Watanabe, & Churchill, 2006) and either intervention alone (Koszycki,

Taljaard, Segal, & Bradwejn, 2011; Craske et al., 2005; Ham et al., 2005; Roy-Byrne et al., 2005). A recent meta-analysis of 52 randomized trials by Cuijpers et al. (2014) has demonstrated the efficacy of combined treatment with psychotherapy and antidepressant medication, versus antidepressants alone for PD, with a moderately large effect. The superior effects of the combination of antidepressants and psychotherapy (mainly cognitive and behavioural therapies) were significant and relevant up to two years after treatment (Cuijpers et al., 2014). The use of combined cognitive-behavioural and pharmacotherapeutic interventions has been shown to result in higher rates of remission, lower levels of disability and anxiety sensitivity, and improvements in psychosocial functioning and social avoidance, compared to anti-panic medications alone (Craske et al., 2005; Roy-Byrne et al., 2005).

      Summary of treatments for PD. The above review of the treatment literature suggests that there are several effective treatment modalities for PD. The choice of treatment for PD is often based on side effect profile, past treatment response, cost (Bystritsky, Khalsa, Cameron, & Schiffman, 2013), and importantly, client preference (McHugh, Whitton, Peckham, Welge, & Otto, 2013; Steidtmann et al., 2012). Models of evidence-based practice highlight the importance of client preference in the selection of treatment (Pilling, Whittington, Taylor, & Kendrick, 2011), particularly considering the similar efficacy of pharmacological and psychological treatments for anxiety disorders such as PD (McHugh et al., 2013). The factors that influence client preference for one treatment modality over another is not well understood, but may reflect beliefs clients and their health care providers have about the etiology of PD. In the ensuing section, I review the literature on the etiology of PD.

Etiology of Panic Disorder

      To date, the etiology of PD remains unclear. The literature on the causes of PD is conflicting, with some studies suggesting that the disorder may be the result of abnormal brain activity and biochemistry and others proposing a predominant psychological etiology (CPA, 2014). Biological theories attribute the origin of PD to biochemical imbalances, genetic factors, and pathophysiology of the brain (Lam, Salkovskis, & Warwick, 2005). The neuroanatomical hypothesis proposed by Gorman and colleagues (Gorman, Kent, Sullivan, & Coplan, 2000) postulates that people with PD have a significantly lower threshold for the activation of the "fear network" in their brains than healthy controls. This hypothesis suggests that deficits in cognitive processing pathways within the cortex lead to the "misinterpretation" of bodily sensations and the

release of neurotransmitters (GABA, serotonin, and norepinephrine) that result in physiological responses associated with autonomic arousal (high respiratory rate, blood pressure, and heart rate) commonly observed in individuals with PD (Fava & Morton, 2009).

Panic disorder is also believed to run in families (Biederman et al., 2001; Goldstein, Wickramaratne, Horwath, & Weissman, 1997; Noyes et al., 1986; Crowe, Noyes, Pauls, & Slymen, 1983) and has a moderate heritable component (Na, Kang, Lee, & Yu, 2011; Finn & Smoller, 2001). A large meta-analysis of twin studies has suggested that PD heritability is estimated at 0.43 (Hettema, Neale, & Kendler, 2001). However, molecular genetic research has failed to reliably identify genetic variants that confer risk for PD, possibly due to the substantial heterogeneity associated with the disorder and contribution of multiple individual genes with minor effects (Na et al., 2011; Maron, Hettema, & Shlik, 2010; Knowles et al., 1998; Vieland, Goodman, Chapman & Fyer, 1996). Family context must also be considered in the familial transmission of PD. For example, parents with anxiety disorders have been found to exhibit a rejecting and/or overprotective parenting style compared to non-anxious control parents (Challacome & Salkovskis, 2009; Lindhout et al., 2006; Whaley, Pinto, & Sigman 1999). However, not all studies report that anxious parents, including those with PD, exhibit a negative parenting style (Koszycki et al., 2013; Woodruff-Borden, Morrow, Bourland, & Cambron, 2002; McClure, Brennan, Hammen, & Le Brocque, 2001) and the amount of variance in PD risk due to parenting versus genetic and other environmental risk factors remains unclear (Koszycki et al., 2013).

Clinical and epidemiological studies (Klauke, Deckert, Reif, Pauli, & Domschke, 2010; Spatola et al., 2010; Faravelli et al., 2007; Manfro et al., 1996) suggest that PD can be triggered by life events and adversities (e.g., interpersonal conflicts and physical illnesses), especially in individuals who are genetically susceptible to developing the disorder (i.e., stress diathesis model of PD; Barlow, 1988). There is also evidence to suggest that endophenotypes, which are observable characteristics with a genetic origin that can be reliably measured and confer risk for PD (Gottesman & Gould, 2003), might moderate the relationship between environmental stressors and subsequent emergence of spontaneous panic attacks. For example, anxiety sensitivity, an intermediate phenotype explaining some cognitive vulnerability to PD (Reiss, Peterson, Gursky, McNally, 1986; Reiss & McNally, 1985), can be modified by stressful experiences in adulthood (Klauke et al., 2010; Scher & Stein, 2003; Schmidt, Lerew, & Joiner,

2000; Schmidt, Lerew, & Jackson, 1997). Likewise, in a study involving 712 general population twins, sensitivity to the panic-inducing effects of carbon dioxide inhalation was found to be moderated by stressful life events, particularly events that occurred within the childhood-adolescence window of risk (Spatola et al. (2010).

Psychological theories of PD focus on individuals' information processing, reactions to stressors, and distorted beliefs about the consequences of symptoms as causes of panic attacks (Lam et al., 2005). Two of the most prominent psychological models of PD are cognitive theories and the anxiety sensitivity theory (Bouton, Mineka, & Barlow, 2001). Cognitive theories, based primarily on the work of Clark and Beck (Clark, 1986; Beck, & Emery, 1985), consider individuals' catastrophic thoughts, formed about the meaning of their internal bodily sensations related to PD, as the primary cause of the disorder (Fava & Morton, 2009). The result is a vicious cycle in which "catastrophic misinterpretations" lead to the experience of additional bodily sensations of arousal, which in turn, leads to higher levels of anxiety and apprehension (Fava & Morton, 2009) that spiral into a panic attack (Bouton et al., 2001). The theory however, does not take into account the fact that not all individuals who experience panic attacks report catastrophic cognitions (Bouton et al., 2001).

The Anxiety Sensitivity (AS) theory proposed by Reiss and McNally (1985), claims that people with PD develop a fear of anxiety-related sensations based on inherent beliefs about their symptoms' harmful physical, psychological, or social consequences (Dixon, Sy, Kemp, & Deacon, 2013; Bouton et al., 2001). Anxiety sensitivity is thus considered a premorbid cognitive risk factor for the development of PD (Bouton et al., 2001; Reiss, 1991). Unlike the cognitive model of panic, proposed by Clark and Beck (Clark, 1986; Beck, & Emery, 1985), the AS perspective refers solely to the tendency to respond fearfully to anxiety, without necessarily mistaking the sensations for something else (e.g., impending insanity, heart attack; McNally, 2002). Thus, although individuals with high AS may have the inclination to make catastrophic misinterpretations, they may still hold a belief that the sensations they are experiencing are dangerous even if they are fully aware of the causes of their symptoms (Nutt, Ballenger, & Lepine, 1999). Questions remain however about whether AS is a better predictor of panic attacks than of negative affectivity (Hayward, Killen, Kraemer, & Taylor, 2000).

Further contributing to the enigma surrounding PD's etiology is the myriad of symptoms (somatic, cognitive, emotional, and behavioural) associated with the disorder (Waikar,

Bystritsky, Craske, & Murphy, 1995). Thus, most experts indicate that the true etiology of PD is multifactorial: a combination of biological, psychological, and environmental factors (CPA, 2014). Certain theories, such as the diathesis-stress model, which suggests that PD is the result of an interaction between predispositional vulnerability (e.g., a biological and/or generalized psychological vulnerability) and precipitating circumstances (Calkins et al., 2009; Zvolensky, Kotov, Antipova, & Schmidt, 2005; Barlow, Chorpita, & Turovsky, 1996; Barlow, 1991, Barlow, 1988), highlight the contribution of environmental factors such as stressful life events to the onset of PD for some individuals (Moitra et al., 2011; Fava & Morton, 2009; Venturello, Barzega, Maina, & Bogetto, 2002; Rapee, Litwin, & Barlow, 1990). Life events, particularly on an interpersonal level, seem to increase the risk of PD as well as the risk for comorbid psychiatric disorders, such as major depression (Klauke et al., 2010; Servant, Bailly, Allard, & Parquet, 1991; Roy-Byrne, Geraci, & Uhde, 1986). Other studies have suggested that adverse life events early in life, such as disruptions in early attachment to caregivers and traumatic childhood experiences (Tibi et al., 2013; Bandelow et al., 2002; Friedman et al., 2002) can increase the risk for the development of PD, possibly by causing tonic autonomic hyperactivity or a neurocognitive defect that would prevent individuals from appropriately interpreting fear network signals (Gorman et al., 2000).

In summary, there remains an evident divergence of views concerning the etiology of PD, which can influence the ways in which clinicians educate clients about the nature of this disorder, and shape clients' own perceptions of their illness (Lam et al., 2005). The next section will describe theoretical frameworks of etiological beliefs and provide a review of the literature on individuals' causal attributions about illness.

Causal Attributions about Illness

Theoretical frameworks. David Hume (1739) stated, in *A Treatise of Human Nature*, that understanding the causes of events is "an essential part in all our reasonings" (Sweeton & Deerrose, 2010). Although there is no single attribution theory, several models and theories about the process by which individuals form inferences about the cause of events and the consequences of causal attributions have emerged a long time ago. Attribution theory thus refers to the collection of these models, concerned primarily with people's perceptions of causation rather than actual causes, and how these perceptions influence subsequent cognitive, affective, and behavioural responses (Voci, 2014).

The first theory of attribution was developed by Fritz Heider in 1958. His theory suggested that people believe there are causes behind behaviour, causes that can be within a person or a situation, and that people have an inherent need to understand why others behave the way they do (Sweeton & Deerrose, 2010). Understanding the causes behind one's behaviour, in this theory, provides individuals with a higher sense of stability and predictability. Heider's theory suggested that individuals pursue a three-step process through which they first observe an event, then determine its intention, and lastly, make an attribution about the event (Heider, 1958). The attributions generated by individuals, Heider proposed, could be internal or external in nature (Heider, 1958). That is, people's behaviour could be attributed to their disposition or their environment, or to both causes (Sweeton & Deerrose, 2010). Heider's theory was further advanced by psychologists such as Kelley (1967) and Weiner (1974).

Kelley (1967) examined people's decision to make either external or internal attributions. In *Attribution Theory in Social Psychology* (1967), Kelley suggested that individuals make attributions based on the information they have about the consistency, distinctiveness, and consensus of a person's behaviour (Sweeton & Deerrose, 2010). His covariation model proposes that external attributions can be made when a person's behaviour is consistent with how other individuals would behave in a similar situation, whereas an internal attribution is usually made when a person's behaviour does not meet expectations (Sweeton & Deerrose, 2010). According to Kelley's theory, causal attributions represent people's effort to understand events and experiences as well as have some control over what happens in their world (Mumma & Mccorkle, 1982).

Weiner's attribution theory of motivation and emotion (1985) has classified attributions along three causal dimensions: locus of causality, stability, and controllability. According to Weiner, a perceived locus of causality can affect individuals' reaction to positive or negative events (Weiner, 1985). An internal locus of causality implies that the event or behaviour is a result of dispositional factors of the individual whereas an external locus of causality infers that contextual factors compel people to behave a certain way (Voci, 2014). The controllability dimension suggests that certain causes are under individuals' control and others are uncontrollable (Weiner, 1985). Weiner proposed that if a cause is perceived to be out of an individual's control, he or she is less likely to make persistent efforts in the future (Weiner, 1985). The last dimension, stability, refers to the cause's perceived degree of permanence over

time (Voci, 2014). According to the theory, stability inferences can influence people's future expectations, with events being more easily predictable if they are repeatedly due to the same cause rather than precipitated by multiple causes (Sweeton & Deerrose, 2010).

While Weiner focused on the perceived causes of success and failure, the author suggested that the model had a wider applicability and that the influence of causal attributions on behavioural outcomes and their function as a mechanism of emotion regulation could be observed in any situation of goal attainment or nonattainment (Weiner, 1985). Following Weiner's three-dimensional attribution theory, several studies have explored individuals' understanding of positive and negative events and the influence of causal perceptions on motivations and future behaviour.

In 2001, Roesch and Weiner proposed a theoretical model based on Weiner's attribution theory as a conceptual framework, suggesting that causal attributions guide some motivated cognitions and behaviour within the context of illness and are related to specific coping strategies. In a meta-analytic review testing the relation between causal attributions, coping, and psychological adjustment in individuals with physical illnesses or undergoing medical procedures, the authors concluded that internal, unstable and controllable attributions were indirectly associated with positive psychological adjustment, through the use of Approach and Emotion-Focused coping (Roesch & Weiner, 2001). Stable and uncontrollable attributions however, were indirectly associated with negative psychological adjustment, through the use of Avoidance coping (Roesch & Weiner, 2001).

One of the most widely known theoretical frameworks however, is Leventhal's Common Sense Model (CSM) of self-regulation of health and illness (Leventhal, Brissette, & Leventhal, 2003; Leventhal, Meyer, & Nerenz, 1980). This model of illness representations suggests that individuals form mental representations of health threats in order to understand them and manage them better (Riedl et al., 2009). The illness representations develop as individuals become increasingly aware of their symptoms and as they gain information from different sources regarding health risks (Riedl et al., 2009).

With regards to the content of illness representations, Leventhal et al. (2003) suggested that individuals form ideas about their illness around five key dimensions, namely, illness identity (symptoms and label associated with the illness), cause (etiological attribution), timeline (expected course of illness), consequences (personal, social, and financial repercussions), and

controllability (perceived level of control over illness). A main focus of illness representations according to the model is the search for the origin of the illness as well as causal attributions (Riedl et al., 2009). The cause dimension of the model refers to people's individualistic ideas about the perceived cause of their condition, based on information gained from personal experience and external sources (Hale, Treharne, & Kitas, 2007). The Illness Perception Questionnaire (IPQ; Weinman et al., 1996) was developed to measure the illness perceptions proposed in Leventhal's model and several studies have confirmed the validity and consistency of these constructs (Watson et al., 2006; Hagger & Orbell, 2003).

According to the model, these representations guide actions to manage health threats and regulate emotions evoked by these threats (Leventhal et al., 2003). An appraisal of these actions generates information that is fed back into the model (Severtson, Baumann, & Brown, 2008). Thus, the theory posits that there is a mediational relationship between illness representations, coping strategies, and illness outcome (Riedl et al., 2009), as demonstrated in Figure 1.
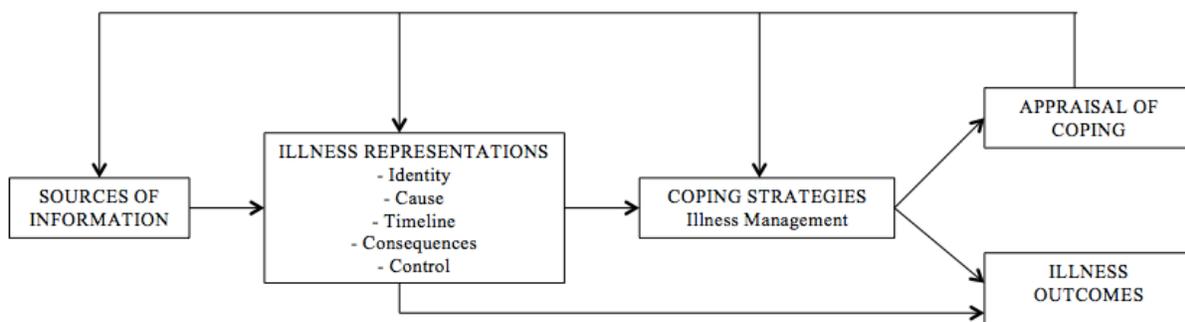
Figure 1. Adapted version of Leventhal's Common Sense Model of self-regulation of health and illness.

Etiological beliefs about illness. Individuals' subjective illness perceptions include assumptions related to their disease's symptoms, causes, consequences, and duration (Franz et al., 2014). These etiological models of illness also involve perceptions with regards to self-efficacy, the effectiveness of treatment, and emotional representations of the disorder (Franz et al., 2014). Although a few studies have researched the impact of illness perceptions on individuals' health outcomes, especially within the medical domain, little attention has been given to people's causal attributions of anxiety disorders such as PD.

People with medical or psychological conditions tend to develop perceptions about the origins of their illness, particularly when their disease disrupts their health and functioning (Franz et al., 2014). Individuals' beliefs about whether their psychological symptoms are attributed to a brain disease or a psychological reaction are influenced by a number of factors, including the particular theoretical approach adopted by their health care provider, media advertisement, or information provided in self-help books or the Internet (Lam et al., 2005). Only a small number of studies have examined the effects of people's beliefs about the nature of their illness on treatment outcomes, particularly in people with mental health problems. Nevertheless, results of these studies suggest that causal attributions can have notable implications on the course of treatment of various mental illnesses (Lam, & Salkovskis, 2007; Kuppin & Carpiano, 2006, Johnson et al., 2000; Waikar, Bystritsky, Craske & Murphy, 1995).

Waikar et al. (1995) explored the effect of etiological beliefs of individuals with anxiety disorders on their decisions to seek treatment and its perceived efficacy. The study also considered the relationship between these beliefs and individuals' past use of medications, family psychiatric history, and current symptoms. The sample comprised 61 participants with a variety of anxiety disorders including PD, OCD, generalized anxiety disorder (GAD), social phobia and post-traumatic stress disorder (PTSD), who were treated at an outpatient clinic. In order to measure participants' beliefs about the pathogenesis of their anxiety symptoms, the researchers developed an Etiological Beliefs Questionnaire (ETBQ; Waikar et al., 1995), which included both psychological and biological scales. Their results suggested that etiological beliefs and treatment preferences were significantly associated, with people endorsing psychological beliefs favouring psychological treatment and those embracing a belief in multiple etiology indicating a preference for multiple treatment modes. Furthermore, the authors found that several factors affected participants' etiological beliefs. Past or present use of multiple prescription drugs, medical symptomatology, and use of medical services were associated with higher biological beliefs, whereas past family psychiatric history led to greater psychological beliefs.

In a cross-sectional observational study, Johnson et al. (2000) investigated the relationship between the causes that individuals with PD in primary care use to "explain" their symptoms and their willingness to accept treatment from specialty health care professionals (medical specialists, psychiatrists, and psychotherapists), take psychotropic medication, and undergo additional medical testing. The study included 73 adult participants who either received

primary and mental health care, primary care only, or were recruited from clinical trials of pharmacotherapies. Their results indicated that the majority of participants attributed their panic symptoms to psychological causes (78-90%), particularly stress. Participants who had received primary care and mental health care were more apt to attribute their symptoms to medical causes and participants recruited from clinical trials more commonly attributed their panic attacks to chemical imbalances. Interestingly, most participants, regardless of treatment group, were willing to seek treatment from a psychiatrist (84-94%) or psychotherapist (95-100%), and take psychotropic medications (87-100%).

Similarly, Lam and Salkovskis (2007) examined the extent to which anxious individuals' biological or psychological explanations of PD impacted their impressions about being assessed for panic attacks and agoraphobia, including their expectation of change, engagement in treatment, and response to treatment. Their sample consisted of 49 participants with various anxiety and depressive disorders who were randomly allocated to three experimental conditions. The participants were told, prior to watching a video of a person diagnosed with PD, that research indicated that panic attacks were caused either by unclear, biological, or psychological factors. The results of this study revealed that participants in the biological etiology condition rated the individual with PD as significantly less likely to make progress following treatment, were more pessimistic regarding the individual's recovery, and viewed the person with PD as being at a higher risk for self-harm than did participants in the psychological cause condition.

A more recent study by Cohen et al. (2015) examined the relationship between etiological attributions and baseline symptom severity, as well as response to pharmacotherapy in a sample of individuals seeking treatment for social anxiety disorder (SAD). The study included 137 treatment-seeking outpatients. Individuals were recruited to participate in an open trial of paroxetine, followed by randomization to augmentation with CBT or continuation of paroxetine. The Attributions for the Etiology of Social Anxiety Scale (AESAS), a scale specifically developed for this study, was used to measure participants' etiological beliefs. The scale assessed two causes of social anxiety: a genetic and biological dimension, and a psychosocial dimension. The study revealed that psychosocial attributions were associated with more severe symptoms at baseline than biological or genetic attributions. With regards to response to treatment, individuals endorsing genetic and environmental family-related attributions achieved the fastest response to

pharmacotherapy, exhibiting greater reductions in severity of symptoms at week 4 of treatment than individuals endorsing other types of attributions.

Finally, Kemp, Lickel, and Deacon (2014) investigated the effect of a biomedical causal explanation of depression (i.e., "chemical imbalance" theory) on depressed individuals' perceptions of themselves and their symptoms. Participants were 91 undergraduate psychology students who endorsed a past or current depressive episode. The students were assigned to either a chemical imbalance condition, in which they were informed that a biological test they completed for the study indicated that their depression was caused by an imbalance of the neurotransmitter serotonin, or a control condition, in which they were told that their past and current depression was not related to a chemical imbalance. Etiological beliefs were measured using the Causal Attributions for Depression (PDS). The study results showed that chemical imbalance test feedback increased prognostic pessimism, lowered negative mood regulation expectancies, and led participants to view pharmacotherapy as more credible and effective than psychotherapy. Moreover, people who believed their disorder was a result of a chemical imbalance had a lower perceived ability to successfully regulate their depressed mood. Chemical imbalance feedback however, had no effect on self-blame.

Although the study by Kemp et al. (2014) focused on individuals with depression, their results, as well as those described above, highlight the need to further explore the ways in which individuals' causal attributions of their illness affect the course of treatment. Etiological beliefs, as the above research points out, may have a substantial impact on people's expectations of change (Lam & Salkovskis, 2007), their motivation (Lam et al., 2005), treatment preferences (Waikar et al., 1995), acceptability (Johnson et al., 2000), and response to treatment (Lam & Salkovskis, 2007; Phelan, Yanf, & Cruz-Rojas, 2006). The current study contributes to the literature by examining casual attributions of illness in individuals with PD.

The Present Study

Despite the growing interest in this area of research, the relation between individuals' etiological beliefs about their illness and health outcomes remains relatively unexplored (Frostholm et al., 2007), particularly in individuals with mental health conditions. Although there is a large literature on the etiology of PD, results are relatively conflicting. As a result, people receive inconsistent messages about the nature and origin of their panic attacks, which become internalized and subsequently, as the aforementioned studies have shown, individuals tend to

self-regulate their treatment behaviour according to their subjective views of illness (Chen, Tsai, & Chou, 2011).

The purpose in conducting the current study was two-fold: 1) to explore the relationship between individuals' etiological beliefs (biological, psychological, or environmental) about PD and baseline demographic and clinical characteristics including age, gender, treatment history, family history of psychiatric illness, baseline severity of illness, the presence agoraphobia, presence of comorbid psychiatric disorders, level of agoraphobic avoidance, dysfunctional and panic-related cognitions, and impairment and 2) to investigate whether individuals' etiological beliefs about PD predicted compliance, clinical response, and side effect profiles with the treatments they were randomly assigned to.

Due to the preliminary nature of this research, it was not possible to form specific hypotheses regarding the impact of individuals' etiological beliefs on all study measures. However, like Waikar et al. (1995), it was expected that biological etiological beliefs would be significantly associated with a history of medication use. Moreover, since biological beliefs have been associated with a higher endorsement and acceptance of medical treatments (Kemp, Lickel, & Deacon, 2014; Phelan, Yang, & Cruz-Rojas, 2006; Kuppin, & Carpiano, 2006; Iselin, & Addis, 2003), it was postulated that individuals endorsing biological attributions for the cause of PD would more likely respond well and comply with the pharmacological intervention than those endorsing non-biological beliefs.

Finally, because people who attribute mental health issues to psychological causes have been found to have a preference for psychological treatment (Iselin, & Addis, 2003; Waikar et al., 1995), to believe that one can cope with their condition on their own (Goldstein, & Rosselli, 2003) and require less professional help (Lam, Salkovskis, & Warwick, 2005), it was predicted that individuals who endorsed non-biological etiological beliefs would be more likely to respond well and comply with the SCBT than individuals with biological etiological attributions.

Method

The present study used archival data collected as part of a large randomized placebo-controlled trial (RCT) that evaluated the efficacy of sertraline, SCBT, and their combination for PD (Koszycki et al., 2011). Briefly, the main finding of this RCT was that sertraline plus SCBT fared better than SCBT (with placebo) and sertraline alone in reducing fear of bodily sensations and better than placebo in reducing agoraphobic avoidance, dysfunctional cognitions, functional

impairment, and symptom improvement. Conversely, neither sertraline alone nor SCBT (with placebo pill) was found to be superior to placebo in improving outcome.

Participants

Two hundred and fifty-one outpatients who met DSM-IV (APA, 2000) criteria for PD (with or without agoraphobia) assessed by a psychiatric interview and the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1997) participated in the study. The sample was recruited through self and practitioner referrals and media advertisements in 15 academic health centers. Participants were eligible if they had a minimum of six panic attacks in the four-week period before the screen visit, and at least two panic attacks per week in the two-week period before the baseline visit. Exclusion criteria included a lifetime history of psychosis, mental retardation, organic mental disorder, bipolar disorder, post-traumatic stress disorder, a current diagnosis of obsessive-compulsive disorder, substance use disorders, eating disorders, and significant suicide risk. Individuals with a current diagnosis of generalized anxiety disorder, social phobia, specific phobia, somatization disorder and depression (score ≤ 17 on the 21-item Hamilton Depression Rating Scale; Hamilton, 1960) were allowed to participate in the study as long as these conditions were secondary to their PD diagnosis.

Individuals with a history of psychosurgery, thyroid disease without controlled medication, hypersensitivity to serotonin reuptake inhibitors, lactose intolerance, and significant medical conditions, as well as those using psychotropic medications within 14 days of the baseline visit, or treated with CBT during the past 12 months were also excluded from the study. Participants using the benzodiazepine oxazepam, with a daily dose above 15 mg and a weekly dose over 60 mg, and women who were pregnant, lactating or not using adequate contraception were also not included.

Procedure

The ethics committees at each of the hospitals approved the study and participants provided written informed consent to participate in the study. The study consisted of three phases: an acute, an extension, and a follow-up phase. This thesis focused on the data obtained from the acute phase of the trial. This phase included the screening visit, in which participants' psychiatric history was obtained and a physical examination (vital signs, urine drug screen, pregnancy test) was conducted. Participants who met the inclusion criteria began a 14-day lead-in period and if deemed necessary, were asked to stop taking psychotropic or disallowed

medications under the supervision of the study investigator. Participants also began recording their panic attacks in a diary and if, during the baseline visit (following the lead-in period), the frequency of their panic attacks was less than two attacks per two weeks, the lead-in period was extended for another two weeks. Participants whose panic attack frequency did not meet the entrance criteria after these two weeks were excluded from the study. The flow of participants during the acute phase of the study is summarized in Figure 2. Of the 289 participants who were screened for the study, 251 met the inclusion and were randomly assigned to one of the four treatment cells. During the acute treatment phase, efficacy and safety measurements were obtained at the baseline visit and at weeks 1, 2, 3, 4, 6, 8, 10, and 12. Toxicology screening was also performed at baseline and repeated at weeks 6 and 12. Compliance with medication was monitored at each visit and participants were given their CBT packages each week following the baseline visit. The evaluation of efficacy and treatment tolerance was also done each week. A total of 176 participants completed the 12-week acute treatment phase and 71 participants (28.7%) discontinued acute treatment prematurely.

　　　　Treatments. Participants were randomly assigned to one of four treatment groups: placebo drug alone (PBO), placebo drug plus SCBT (PBO/SCBT), sertraline alone (SERT), or sertraline plus SCBT (SERT/SCBT). Sertraline and placebo were administered double-blind and were provided as matching capsules to be taken initially at 25 mg daily and after the first week, increased to 50 mg daily until the end of week 4 of the study. Participants were withdrawn from the study if they experienced significant side effects that prevented the increase of the dosage to 50 mg. After the fourth week, if participants did not develop dose-limiting side effects, the dose level was increased by 50 mg every 2 weeks or more (maximum allowed was 200 mg/day) until the maximum improvement had been seen in the CGI scale (Guy, 1976).

　　　　The SCBT program consisted of a combination of cognitive and behavioural techniques described in therapist and self-help manuals that correspond with standard cognitive-behavioural treatment. The treatment approach included client education about anxiety and the cognitive model of PD, breathing and relaxation skills, cognitive restructuring interventions that addressed misappraisal of panic symptoms, and interoceptive and situational exposure to panic-provoking stimuli. These components were administered in chronological order and specifically addressed the configuration of PD, including the panic attacks, the anticipatory anxiety, and the agoraphobic avoidance. The program included 12 audiotapes and a workbook developed for the

study by Drs. Zindel Segal and Diana Koszycki, clinical psychologists who specialize in CBT interventions. The tapes described the principles of treatment and contained detailed instructions and homework. Participants did not receive monetary compensation for their participation in this study.

Measures

       Etiological Model Questionnaire. Participants' etiological beliefs were measured using the Etiological Model Questionnaire (ETMQ), adapted from Waikar et al.'s (1995) Etiological Beliefs Questionnaire (ETBQ). The ETBQ was developed by psychiatrists and psychologists with expertise in anxiety disorders in order to assess individuals' beliefs about the cause of their anxiety symptoms. The ETBQ consisted of 22-items divided into biological and psychological scales. The items were chosen for their face validity, ease of classification and relevance, as judged by a small sample of participants. The adapted ETMQ used in the current study consisted of 32 items that were rated on a 0 ("not important at all") to 8 ("extremely important") scale. Participants indicated the extent to which they believed each item caused their initial panic attack and their current problems with anxiety and panic. Sample items included: "I have a chemical imbalance in my brain", "My problems result from a difficulty expressing my true feelings" and "My problems result from the stress of a major lifestyle change". The questionnaire was divided into biological, psychological, and environmental subscales. The ETMQ was found to have a good level of internal consistency (Cronbach's alpha = .86) in the current sample.

       Structured Clinical Interview for DSM-IV. The Structured Clinical Interview for DSM-IV (SCID; First et al., 1997) was used in combination with a psychiatric interview to confirm whether participants met DSM-IV criteria for PD with or without agoraphobia. The SCID is a semi-structured diagnostic interview that provides excellent coverage of psychiatric disorders and yields highly reliable diagnosis for most disorders (Babor, & First, 2001). It is considered the "gold standard" for the assessment of psychiatric disorders in clinical settings (Lobbestael, Leurgans, & Arntz, 2011). The inter-rater reliability for PD ranges between .67 (Lobbestael et al., 2011) and .88 (Skre et al., 1991; Zanarini et al., 2001). Inter-rater reliability was not assessed in the current sample.

       Clinical Global Impressions. The Clinical Global Impressions (CGI; Guy, 1976) is a clinician-rated scale that rates the overall severity of illness, change over time, and response to treatment. With regards to PD, the CGI takes into account the intensity and frequency of panic

attacks, the level of phobic avoidance, the degree of anticipatory anxiety, level of impairment, and the need of treatment adjustments (Heldt et al., 2006). The CGI consists of a measure of illness severity (CGI-S), which is rated on a scale from 1 ("Normal, not at all ill") to 7 ("Among the most extremely ill patients"), and a measure of treatment-related improvement (CGI-I), which is rated on a scale from 1 ("Very much improved") to 7 ("Very much worse") (Guy, 1976). This outcome measure has been widely used in clinical research (e.g., Koszycki, Benger, Shlik, & Bradwejn, 2007), primarily because it has been shown to be sensitive to change with interventions for PD treatment (Gloster et al., 2011; Koszycki et al., 2007; Barlow et al., 2000).

Mobility Inventory for Agoraphobia. The Mobility Inventory for Agoraphobia (MI; (Chambless, Caputo, Jasin, Gracely, & Williams, 1985) is a 27-item inventory designed to measure agoraphobic avoidance behaviour. The self-report scale lists numerous situations that are generally avoided by individuals with agoraphobia (e.g., theatres, classrooms, high places, staying at home alone) (Craske, Rachman, & Tallman, 1986). The MI consists of four global measures: avoidance alone (MI-AAL), avoidance accompanied (MI-AAC), discomfort alone (MI-DAL) and discomfort accompanied (MI-DAC) (Chambless et al., 1985). Participants were asked to rate the degree to which they avoided agoraphobic situations due to discomfort or anxiety on a five-point scale ranging from 1 ("Never avoid") to 5 ("Always avoid") (Chambless et al., 1985). The present study used the avoidance-alone (MI-AAL) subscale of the MI. The MI-AAL subscale has been shown to have good psychometric properties including internal consistency (Cronbach's alpha = .94) (Chambless et al., 1985; Craske et al., 1986), test-retest reliability (*r* = .90) and discriminant validity (Chambless et al., 2011). In the current sample, the internal consistency of the MI-AAL was excellent (Cronbach's alpha = .94).

Body Sensations Questionnaire. The Body Sensations Questionnaire (BSQ; Chambless, Caputo, Bright, & Gallagher, 1984) is a 17-item self-report scale that assesses the degree to which participants fear somatic sensations associated with panic (e.g., heart palpitations, dizziness, nausea, and sweating) (Chambless, & Gracely, 1989). Items on the scale were rated on a five-point scale ranging from 1 ("Not frightened or worried by this sensation") to 5 ("Extremely frightened by this sensation") (Chambless, & Gracely, 1989). The BSQ has been shown to have good psychometric properties including high internal consistency (Cronbach's alpha = .88), acceptable test-retest reliability (*r* = .67), and a positive construct validity (Chambless, & Gracely, 1989). In the current sample, the BSQ had a good internal consistency (Cronbach's

alpha = .86).

Agoraphobic Cognitions Questionnaire. The Agoraphobic Cognitions Questionnaire (ACQ; Chambless et al., 1984) is a 14-item self-report scale that assesses thoughts individuals have regarding the negative consequences of anxiety-provoking experiences (Chambless et al., 1984). Items were rated on a five-point scale ranging from 1 ("Thought never occurs") to 5 ("Thought always occurs") (Chambless, & Gracely, 1989). Sample items included: "I am going to throw up" and "I must have a brain tumor" (Chambless et al., 1984). The instrument has good psychometric properties including test-retest reliability ($r = .86$), internally consistency (Cronbach's alpha = .80) and convergent and discriminant validity (Chambless, & Gracely, 1989). In the current sample, the internal consistency of the ACQ was acceptable (Cronbach's alpha = .78).

Sheehan Disability Scale. The Sheehan Disability Scale (SDS; Sheehan, Sheehan, & Raj, 1996) is a self-report measure of impairment. This scale, commonly used in drug trials, assesses disability across three domains: work, social life and family life (Sheehan et al., 1996). The SDS uses visual-spatial, numeric and verbal descriptive anchors to provide an accurate measurement of impairment (Sheehan et al., 1996). Developed as a treatment outcome measure, the SDS has been shown to be a useful, cost-effective instrument that is sensitive to change in drug treatment studies of PD (Sheehan, & Sheehan, 2008). The SDS asked participants to rate from 0 ("Not at all") to 10 ("Extremely") the degree to which their symptoms had disrupted their work, social, and family responsibilities (Sheehan, & Sheehan, 2008). The scale has been shown to have a fairly high correlation between its three items and an overall high internal consistency (Cronbach's alpha = .89) (Sheehan, & Sheehan, 2008). In the current sample however, the internal consistency of the SDS was questionable (Cronbach's alpha = .62).

Additional Information. During the psychiatric assessment, information on participants' age, sex, treatment history, family history of psychiatric illness, and presence of comorbid psychiatric disorders was also gathered.

Statistical Analyses

SPSS® Statistics 23.0 (SPSS Inc., Chicago, IL) was used for data analyses. Before conducting the statistical analyses, data were screened for outliers, missing values, skewness, and kurtosis. Inspection of standard z scores revealed that there were only two outliers (values greater than 3.29; Tabachnick & Fidell, 2007). Preliminary analyses were performed with and without

these outliers and results remained the same; therefore the values were included in the analyses without modification or deletion. Exploration of missing data was completed using SPSS Missing Values Analysis. Fifteen participants had one or more missing values**.** Little's Missing Completely at Random test (Little, 1988) indicated that these values were missing completely at random (p = .88). Due to the relatively small number of missing values, an Expectation-Maximization method was used to impute missing values. Not all variables were normally distributed, as assessed by Shapiro-Wilk's test. Log transformations were applied which improved the normality of the distributions. Therefore, log transformed variables were used for the analyses. Preliminary analyses revealed, nevertheless, that results were the same when transformed and untransformed variables were used.

Tests of the assumptions of linear regression with respect to the selected predictors in the study were also performed. There was linearity as assessed by partial regression plots and plots of studentized residuals against the predicted values. There was independence of residuals, as assessed by Durbin-Watson statistics (Montgomery, Peck, & Vining, 2001). There was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values and there were no extreme values, as assessed by Cook's distance values above 1. The independent variables were subjected to linear regression analysis to evaluate multicollinearity among the predictors. The results of the analysis showed that the data did not violate the multicollinearity assumption. The tolerance value of each independent variable was greater than .56, which exceeded the suggested criteria of below .1 (Pallant, 2007). Lack of multicollinearity among the independent variables was also supported by the obtained variance inflation factor (VIF) values. They were all well below the cut-off value of 10. (Field, 2005). The VIF values of the variables ranged from 1.00 to 1.79. This revealed that the data met the assumptions for multiple regression including linearity, independence of residuals (Durbin-Watson statistic), homoscedasticity, and lack of multicollinearity among the predictor variables. Additionally, the assumption of normality was met, as assessed by Q-Q Plots.

To test the hypothesis of an association between etiological beliefs and baseline demographic (age and gender) and clinical characteristics (presence of agoraphobia, presence of comorbid disorders, agoraphobic avoidance, fear of somatic sensations, panic-related cognitions, baseline severity of illness, impairment in work, social, and family life, family history of psychiatric illness, and suicide history), two types of analyses were performed. First, correlation

analysis was applied to examine bivariate correlations between the ETMQ subscale scores and baseline demographic and clinical variables. Pearson correlations were used for continuous variables and point-biserial correlations were used for categorical data (Field, 2009). Second, demographic and clinical variables that significantly ($p \leq .05$) correlated with the ETMQ subscales were subsequently included in a series of linear forced entry multiple regression analyses to examine how well they predicted each of the ETMQ subscales (Montgomery et al., 2001; Field, 2009).

To determine whether etiological beliefs predicted treatment response, compliance with the SCBT intervention, and treatment emergent side effects, hierarchical multiple linear and logistic regressions were performed. The three predictor variables (ETMQ – Biological, Psychological, and Environmental subscales) and treatment group were entered first in the regression model to examine the main effects of the predictor variables. Interactions between the treatment groups (placebo, placebo plus SCBT, sertraline, and sertraline plus SCBT) and the etiological beliefs subscales were then calculated and entered in the model as a second step (Aguinis, 2004; Jaccard & Turrisi, 2003; Aiken & West, 1991). To compute interactions between treatment group and the etiological belief subscales, treatment group was dummy coded. The dependent variables included the CGI-S and CGI-I subscales, treatment compliance defined as the % time listening to the SCBT tapes and % of homework completion, whether participants experienced adverse side effects, and whether they discontinued treatment prematurely. The predictors were the ETMQ mean scores on each dimension of the scale (biological, psychological, environmental).

Due to the exploratory nature of this study, we did not adjust for multiple testing (Bender & Lange, 2001). All analyses used an alpha level of $p \leq .05$ to determine statistical significance.

Results

The descriptive statistics and frequency distributions of the baseline demographic and clinical characteristics are presented in Table 1.

Relationship between Baseline Characteristics and Etiological Beliefs

These analyses were performed on 251 participants, who completed the baseline visit and were randomized. The results of the correlations between baseline demographic and clinical characteristics and ETMQ subscale scores are displayed in Table 2. For the ETMQ-biological subscale, significant positive correlations emerged for family history of psychiatric illness, the

presence of comorbid psychiatric disorders, and scores on the ACQ, BSQ, and SDS – family life. For the ETMQ-psychological subscale, significant correlations emerged for the presence of agoraphobia, presence of comorbid psychiatric disorders, a history of suicide attempts, and scores on the ACQ, BSQ, MI-Alone, CGI-S, SDS – work, social life, and family life. Additionally, the ETMQ-psychological subscale correlated negatively with participants' age and positively with gender, with women *(M* = 2.21, *SD* = 1.14) reporting more psychological etiological beliefs than men (*M* = 1.92, *SD* = 1.12, *d* = .26). For the ETMQ – environmental subscale, significant positive correlations were found for the presence of comorbid psychiatric disorders and scores on the ACQ, BSQ, and SDS – work and family life.

Baseline Predictors of Etiological Beliefs

Results of the regression analyses of baseline demographic and clinical predictors of etiological beliefs are displayed in Tables 3 to 5. For the ETMQ-biological beliefs subscale (Table 3), the regression model, which included five predictor variables (family history of psychiatric illness, presence of comorbidity, and scores on the BSQ, ACQ and SDS-family life), was statistically significant, $F(5, 245) = 9.11$, $p = .00$. Overall, 15.7% of the variance in biological etiological beliefs was explained when these baseline predictors were considered together ($R^2 = .16$, Adjusted $R^2 = .14$). An inspection of individual beta weights revealed that a family history of psychiatric illness and scores on the BSQ and ACQ were significant predictors ($p < .05$) of biological etiological beliefs, whereas the presence of comorbid psychiatric disorders and scores on the SDS- family life subscale had no predictive value.

For the ETMQ-psychological beliefs subscale (Table 4), the regression model, which included 11 predictor variables, was statistically significant, $F(12, 238) = 9.70$, $p = .00$ and accounted for approximately 33% of the variance in psychological etiological beliefs ($R^2 = .33$, Adjusted $R^2 = .30$). Inspection of individual beta weights revealed that age, the presence of comorbid psychiatric disorders, a history of suicide attempts and scores on the BSQ, ACQ, and SDS-family life, were significant predictors ($p < .05$) of psychological etiological beliefs. Gender, the presence of agoraphobia, and scores on the CGI –S, MI-AAL, and SDS-work and social life had no predictive value.

The regression model for the ETMQ-environmental etiological beliefs subscale, which included five predictor variables (Table 5, see above), was also significant $F(5, 245) = 7.35$, $p = .00$ and explained 13% of the variance in environmental etiological beliefs ($R^2 = .13$, Adjusted

$R^2 = .11$). Inspection of individual beta weights revealed that the SDS-family life subscale was a significant predictor of environmental etiological beliefs, whereas the other variables in the model had no predictive value.

Etiological Beliefs and Treatment Outcome

These analyses were based on 176 participants who completed the 12 weeks of treatment. Results of the hierarchical multiple linear and logistic regressions investigating the relationship between the dimensions of etiological beliefs and treatment response, treatment compliance, and presence of adverse events are presented in Tables 6 to 8, respectively.

The analyses revealed a relationship between psychological and environmental etiological beliefs and CGI- S scores following 12 weeks of treatment. For the ETMQ-psychological beliefs subscale the regression model was statistically significant, $F(2, 172) = 8.00$, $p = .00$ and accounted for 8.5% of the variance in week 12 CGI-S scores ($R^2 = .09$, Adjusted $R^2 = .07$). The interactions between treatment groups and ETMQ-psychological beliefs scores were not significant, indicating that psychological etiological beliefs predicted increased symptom severity following treatment, irrespective of which treatment participants were allocated. For the ETMQ-environmental etiological beliefs subscale, the regression model was statistically significant, $F(2, 172) = 7.25$, $p = .00$ and accounted for 7.8% of the variance in week 12 CGI-S scores ($R^2 = .08$, Adjusted $R^2 = .07$). The interactions between treatment groups and environmental etiological beliefs were not statistically significant. The regression model involving the ETMQ-biological belief subscale and its interaction with treatment group did not yield significant results.

Results of the linear regression analyses involving other treatment outcome variables indicated that none of the ETMQ subscales significantly predicted CGI-I ratings following 12 weeks of treatment, compliance with SCBT treatment, the presence of adverse events and treatment discontinuation. The interactions between treatment groups and the three dimensions of etiological beliefs were also nonsignificant for these outcome variables ($p > .05$).

Discussion

An individual's belief about the etiology of their illness has important implications for treatment behaviour and treatment outcome. To date, most research on this topic has focused on medically ill individuals. Research on etiological beliefs in psychiatric populations in general and panic disorder (PD) in particular is sparse. Accordingly, the present study addressed this gap in

the literature by exploring the baseline characteristics that could influence causal attributions of illness as well as the impact these beliefs have on treatment outcome in individuals with PD.

Influence of Baseline Demographic Variables on Etiological Beliefs

This study revealed that a number of baseline characteristics were associated with participants' etiological beliefs. However, due to the relatively small beta weights obtained for the individual characteristics, these results should be viewed with caution.

The regression analysis revealed that age was negatively associated with psychological etiological beliefs of illness, but had no predictive value in explaining biological and environmental causal attributions. A study by Bann et al. (2004) examining the etiological beliefs of individuals with major depression also found that older individuals were less likely to attribute their depression to internal factors such as stress and thought processes. The reason why psychological etiological beliefs were lower when individuals with PD's age was higher is not entirely clear. Research on aging and PD suggests that older adults with PD report less anxiety and arousal, and lower levels of depression (Sheikh, Swales, Carlson, & Lindley, 2004). The experience of higher levels of distress in relation to panic attacks might explain why younger participants may be more likely to attribute their illness to psychological causes.

It is important to recognize however, that the detection and diagnosis of anxiety disorders such as PD in older adults is further complicated by changes in life circumstances, cognitive decline, and medical comorbidities that younger adults do not face (Wolitzky-Taylor, Castriotta, Lenze, Stanley, & Craske, 2010). Therefore, the expression of anxiety symptoms may vary as a function of these and other age-related factors (Wolitzky-Taylor et al., 2010) and influence individuals' illness perceptions. In the present study, only 13% of participants were over the age of 50. Future studies involving more participants from different age groups could provide a deeper understanding of the relationship between individuals' age and their causal attributions. Future research could also consider investigating the relationship between age of onset of PD and etiological beliefs. A later age of onset of PD has been associated with less distress in relation to body sensations, panic-related cognitions and emotions during panic attacks (Sheikh et al., 2004) and therefore, could have significant implications on individuals' illness perceptions.

With respect to gender, a significant albeit small association was found between female gender and the endorsement of psychological etiological beliefs. However, inspection of individual beta weights in the regression model indicated that gender had no predictive value in

explaining psychological beliefs of illness. This finding is consistent with that of Waikar et al. (1995) who did not find a significant association between gender and causal attributions of illness in a heterogeneous sample of individuals with anxiety disorders. However, it is interesting to note that Rief and colleagues (Rief, Nanke, Emmerich, Bender, & Zech, 2004) found that women with a diagnosis of a somatoform disorder, a psychiatric condition that is highly comorbid with PD (Battaglia, Bernardeschi, Politi, Bertella, & Bellodi, 1995; Brown, Golding, & Smith, 1990), were more likely to endorse psychological causes for their somatic symptoms than men. A more recent study examining illness attributions of individuals with depression (Schweizer et al., 2010), found a trend whereby women tended to endorse interpersonal reasons (i.e., relationships, childhood, and intimacy) as causes of their depression, whereas men were more likely to endorse achievement-related causes.

Although the current study found no association between gender and biological or environmental etiological beliefs, gender has been found to influence illness perception in other medical conditions such as cardiovascular disease. For example, men are more likely than women to attribute their illness to lifestyle (e.g., diet, overwork, alcohol) or external factors such as bacteria or lack of family support (Boruchovitch & Mednick, 2000; Green & Bird, 1986); whereas women tend to blame uncontrollable causes for their illness such as destiny (Dunkel et al., 2011) or genetics (Grace et al., 2005; Astin & Jones, 2004).

Influence of Baseline Clinical Variables on Etiological Beliefs

Family history of psychiatric illness. A family history of mood or anxiety disorders is considered an important predictor of anxiety symptoms (Katzman et al., 2014) and is associated with a more recurrent course, greater impairment, and greater service use (Milne, Harrington, Poulton, Rutter, & Moffitt, 2009). Unlike previous work by Waikar et al. (1995), who found that a past family history of psychiatric disorders was associated with psychological etiological beliefs, the present study revealed that a family history of psychiatric disorders predicted increased beliefs in a biological etiology of illness. This finding is not surprising since a family history of psychopathology is compatible with a genetic explanation of illness (Dar-Nimrod & Heine, 2011). The relationship between a family history of psychiatric disorders and beliefs in a biological etiology may reflect participants' view that genetic factors may be more important in the development of PD than psychological or environmental factors.

Prior research exploring causal attributions of individuals with medical conditions has supported a link between having a family history of cancer and identifying genetics and heredity as a cause for cancer (Hay et al., 2011). Similarly, a study examining illness perceptions among individuals with cardiovascular disease (CVD) found that those with a family history of CVD were significantly more likely to endorse biological factors (i.e., heredity and genes) as a causal factor than individuals without such a history (Grace et al., 2005).

Treatment history. We did not find that previous treatment with psychotropic medications or psychotherapy influenced participants' etiological beliefs about the cause of PD. This contrasts our hypothesis and Waikar et al. (1995)'s finding that previous use of psychotropic medication was associated with a biological explanation of anxiety disorder etiology. In a study involving individuals with PD, those who received only primary care were more likely to attribute the cause of their illness to medical reasons than those who were treated in both primary care and a mental health setting, although all were willing to undergo psychological therapies and receive medication (Johnson et al., 2000). A history of antidepressant treatment has also been associated with a greater tendency to attribute biological factors (e.g., chemical imbalance, genes, energy balance) in individuals with major depressive disorder (Bann et al., 2004). Further investigation of the relationship between past treatment modalities and causal attributions is warranted to elucidate whether perceived effectiveness of previous treatments impact or modify current etiological beliefs of illness in psychiatric disorders.

History of suicide attempts. Our results revealed that a history of suicide attempts was a significant predictor of increased psychological etiological beliefs. The relationship between causal attributions and a history of suicide attempts has not been extensively investigated. However, past suicide attempts have been associated with psychosocial deficits, including maladaptive cognitive patterns (Lewinsohn, Rohde, & Seely, 1993). Severe anxiety has been significantly related to cognitions of impending loss of control and an overwhelming urge to escape, which can contribute to feelings of helplessness (Noyes, 1991) that may lead to suicidal behaviour. Therefore, it is conceivable that individuals with PD with a history of suicide attempts may perceive themselves as more psychologically disturbed (Goggin, Range, & Brandt, 1986), providing personal attributions to the cause of their condition, including maladaptive cognitions and other psychological factors.

Presence of comorbid psychiatric disorders. Panic disorder frequently co-occurs with other psychiatric conditions, with comorbidity rates ranging from 51 to 69% (Allen et al., 2010). Among the comorbid psychiatric disorders, another anxiety disorder, mood disorders and substance use disorders are the most common (Kessler et al., 2005; Zimmermann et al., 2003). The present finding that a concurrent psychiatric disorder had predictive value in explaining psychological etiological beliefs of PD is consistent with previous research on causal illness attributions in somatoform disorders. Individuals with somatoform disorders who had a comorbid depression or anxiety disorder reported more psychological attributions (Douzenis & Seretis, 2013; Steinbrecher & Hiller, 2011; Rief et al., 2004; Hennignsen, Jakobsen, Schiltenwolf, & Weiss, 2005). Similarly, in a sample of individuals treated in primary care, the presence of a psychiatric history was associated with an increased tendency to make psychological attributions for common somatic symptoms and a decreased tendency to endorse environmental causal attributions (Robbins & Kirmayer, 1991). Consistent with our findings, Rief and colleagues (2004) did not find an association between comorbidity and biological causal attributions in individuals with somatoform disorders.

The association between psychological causal attributions and the presence of comorbid psychiatric disorders suggests that individuals diagnosed with more than one mental disorder are more likely to attribute internal causes for their illness such as stress, difficulties expressing and regulating emotions, negative thinking patterns and learned behaviour. Treatment with CBT for PD has been found to be equally efficacious for individuals with and without comorbid anxiety and unipolar mood disorders (Allen et al., 2010). Allen et al. (2010) suggest that this may be due to the treatment targeting psychological factors such as the experiencing and regulating of emotions, which may reduce the intensity of all emotional experiences, rather than just panic symptoms. However, comorbidity in individuals with PD has been associated with more severe symptoms (Allen et al., 2010). Therefore, a more thorough investigation of the relationship between psychological causal attributions and comorbid disorders in PD could shed some light into the increased severity of symptoms reported by individuals with PD and possibly elucidate the complexity of treatment for PD in individuals with comorbid diagnoses.

Presence of agoraphobia and severity of agoraphobic avoidance. Agoraphobia is a common sequela of PD and is characterized by fear and avoidance of a wide range of situations where the dreaded panic attack might occur (APA, 2013). Usually, agoraphobic situations include

those where escape might be difficult or help might not be available in the event of an attack (Wittchen et al., 2010). The regression analysis revealed that the presence of agoraphobia and severity of agoraphobic avoidance were not significantly associated with etiological beliefs of illness. A study examining individuals' understanding of the causes of agoraphobia found that stress was the most commonly endorsed causal explanation for agoraphobic symptoms (Wardle, Hayward, Higgitt, Brewin, & Gray, 1997). Overall, the study revealed that, in terms of the relative frequency of causal endorsements, the causes most often attributed by individuals with agoraphobia can be considered as "psychological" models of causation (Wardle et al., 1997). Unfortunately, the relationship between the presence and severity of agoraphobic avoidance and causal attributions has not been extensively researched. Future studies may consider investigating the association between agoraphobia and psychological etiological beliefs in order to verify whether Wardle et al. (1997)'s results can be supported.

      Agoraphobic cognitions and bodily sensations. Panic attacks are accompanied by a range of somatic symptoms (e.g., rapid heart, dizziness, dyspnea) and catastrophic misinterpretation of bodily sensations of anxiety is a hallmark of PD. The regression analysis revealed that agoraphobic cognitions and fear associated with bodily sensations of arousal were significant determinants of biological and psychological etiological beliefs of illness. The finding that agoraphobic cognitions were associated with both biological and psychological causal attributions is an interesting finding considering that the Agoraphobic Cognitions Questionnaire (ACQ), which assesses thoughts concerning the negative consequences of experiencing symptoms of panic attacks, includes items related to both physical concerns (e.g., brain tumor, heart attack, stroke) and psychological concerns (e.g., fear of going crazy, being paralyzed by fear). It is therefore possible that participants scoring higher on both the physical and psychological items of the ACQ were more likely to endorse multiple etiological beliefs about their PD.

      The relationship between elevated fear of bodily sensations of anxiety and biological and psychological etiological beliefs is also intriguing and may influence how people experience their symptoms of panic. It has been suggested that individuals who fear bodily sensations become more vigilant and develop a heightened self-focus of bodily sensations (Wells, 1997; Grant, 2010). It is therefore possible that participants reporting higher levels of fear of physical sensations were more aware of and attentive to these symptoms, and more likely to attribute their

PD to biological causes such as respiratory or cardiac problems. These participants may also have recognized that their fear stems from the catastrophic misinterpretation of these symptoms as dangerous, and therefore attributed the cause of their PD to psychological factors. Due to their increased vigilance, they may have learned from prior experience with panic attacks that biological and psychological symptoms tend to occur together, and therefore attribute their disorder to multiple causes. It is also important to note that the Body Sensations Questionnaire only measures individuals' fear of physical sensations commonly associated with panic. Future research could also use a broader measure such as the Anxiety Sensitivity Index (Peterson & Reiss, 1987), which includes psychological concerns (e.g., worrying about going mad) and social concerns (e.g., worry about appearance) about anxiety symptoms.

       Level of impairment. It is well established that PD is associated with significant impairment in multiple domains (Bonham & Uhlenhuth, 2014; Carrera et al., 2006; Mendlowicz & Stein, 2000). The current study revealed that impairment in family life, as measured by the Sheehan Disability Scale, was a significant predictor of psychological and environmental etiological beliefs of illness. On the other hand, impairment in work and social life had no predictive value in our results. The family life item of the SDS measures the extent to which individuals' family life and home responsibilities are impaired by their current psychiatric symptoms. Examples include relationships with family members, paying bills, managing their homes, and activities such as shopping and cleaning.

       Individuals with PD, especially those with agoraphobia, show less autonomy, self-confidence and affirmation, use more negative coping skills, exhibit more neurotic traits, are more irritable and hostile, and engage in self-criticism (Marcaurelle, Bélanger, & Marchand, 2003). It is possible that individuals with PD who experience functional impairment in family life internalize the above-mentioned psychological repercussions and blame themselves for interpersonal difficulties. As a result, they may be more likely to attribute the cause of their symptoms to psychological factors. Moreover, the families of individuals with PD may highlight these negative characteristics, which can sometimes accompany PD, further contributing to participants' endorsement of psychological etiological beliefs. The finding that impairment in family life was predictive of environmental causal attributions is also not surprising considering that the literature suggests that individuals with PD usually experience more major life events

during the months before and after the onset of PD, including marital and interpersonal problems (Marcaurelle et al., 2003).

Several studies involving individuals with medical conditions have found that illness perceptions correlate with impaired quality of life (Tiemensma et al., 2011; Grayson et al., 2014). Grayson et al. (2014) found that people with systemic vasculitis who believed their illness was triggered by environmental factors (i.e., weather or pollution) exhibited greater impairment in physical, social, and role functioning (Grayson et al., 2014). These researchers also found an association between psychological attributes of illness and greater fatigue and impairment in role and social functioning (Grayson et al., 2014). Similarly, Tiemensma et al. (2011) found a strong correlation between illness perceptions of individuals with long-term remission of acromegaly and impaired quality of life. In their study, psychological attributions were reported as the main perceived cause of acromegaly and included family problems or worries (Tiemensma et al., 2011).

Etiological Beliefs of Illness and Treatment Outcome

There is a paucity of research investigating the impact of causal attributions on treatment outcome, particularly in psychiatric disorders such as PD. Nevertheless, a few studies examining etiological beliefs in relation to anxiety or depressive disorders have found a significant relationship between individuals' causal attributions and their help seeking behaviour (Phelan et al., 2006; Goldstein & Rosselli, 2003), prognostic pessimism (Kemp et al., 2014) and treatment preferences (Steidtmann et al., 2012; Kuppin & Carpiano, 2006; Waikar et al., 1995), effectiveness (Lam & Salkovskis, 2007), and adherence (Sher, McGinn, Sirey, & Meyers, 2005). Therefore, our second objective was to investigate whether etiological beliefs about PD affected treatment compliance, clinical response, and side effect profiles.

Impact of etiological beliefs on treatment response. This study revealed a significant relationship between etiological beliefs and response to treatment. Specifically, participants who endorsed psychological and environmental causal attributions were more likely to be rated as more severely ill following 12 weeks of treatment. In contrast, biological causal attributions had no impact on week 12 ratings of symptom severity. Furthermore, etiological beliefs were not predictive of improvement after 12 weeks of treatment.

Although previous research has supported the association between baseline symptom severity and psychological causal attributions in individuals with social anxiety disorder (Cohen

et al., 2015), depression (Bann et al., 2004), somatoform disorders (Steinbrecher & Hiller, 2011; Rief et al., 2004), and various medical conditions (Jopson & Moss-Morris, 2003; Servaes, Verhagen, & Bleijenberg, 2002; Robbins & Kirmayer, 1991), very few studies have examined the impact of these attributions on symptom severity following treatment for psychiatric disorders, and existing findings are mixed. Bann et al. (2004) found that attributing depression to internal factors (e.g., stress, thought processes, dysfunctional relationships) was not significantly associated with reduction in depression severity after 8-weeks of treatment with mainstream and alternative pharmacological agents (i.e., sertraline versus hypericum). Beliefs in biological causes however, were associated with less improvement over 8-weeks of treatment on clinician-rated measures of depression and clinical global impressions of severity and improvement (Bann et al., 2004). Similarly, Dunlop et al (2012) found that etiological beliefs did not predict remission following treatment with CBT or escitalopram. In contrast, another study found that cognitive therapy was significantly less effective in individuals who attributed their depression to biological reasons (Leykin, DeRubeis, Shelton, & Amsterdam, 2007).

A study of individuals with schizophrenia found that attributing psychosocial and biological causal beliefs was associated with lower levels of psychotic symptoms following treatment (Caqueo-Urizan, Boyer, Baumstarck, & Gilman, 2015). Individuals endorsing higher magical and religious causal beliefs however, were found to have lower adherence to antipsychotic drugs and more severe symptoms (Caqueo-Urizan et al., 2015).

Interestingly, we did not find that the interaction between type of treatment and etiological beliefs predicted treatment outcome. Therefore, our hypothesis that participants endorsing biological causes for their PD would demonstrate a more favourable response to pharmacotherapy (i.e., sertraline alone or sertraline plus SCBT) versus psychological treatment (i.e., SCBT) was not supported. In another study on etiological beliefs in individuals with anxiety disorders, Waikar et al. (1995) also failed to detect a significant relationship between participants' current type of treatment (i.e., pharmacotherapy or psychotherapy) and their etiological beliefs about their illness.

Impact of etiological beliefs on treatment compliance. This study failed to demonstrate an effect of etiological beliefs on compliance with SCBT homework or treatment discontinuation across the four treatment groups. Although there are no published studies in PD to our knowledge, these results concur with findings in other psychiatric populations. Treatment studies

of depression have noted that etiological beliefs do not predict adherence to paroxetine or pill placebo (Sullivan et al., 2003). Similarly, a study of individuals with schizophrenia revealed that causal beliefs were not associated with treatment compliance, although those who endorsed social causes for their illness had worse therapeutic relationships than those who cited supernatural causes (McCabe & Priebe, 2004).

Other researchers, however, have noted that etiological beliefs do affect treatment adherence. Endorsing pessimism as a cause for depression was associated with an increased likelihood of completing treatment (Dunlop et al., 2012), whereas acceptance of a biological model of illness was associated with beliefs that pharmacotherapy is a more credible treatment option than psychotherapy (Kemp et al., 2014), and lower rates of treatment discontinuation with pharmacotherapy but increased prognostic pessimism (Bann et al., 2004).

In studies involving individuals with a psychotic disorder, a stronger belief in psychological causes was associated with less positive attitudes toward medication (Wiesjahn, Jung, Lamster, Rief, & Lincoln, 2014) and lower willingness to accept pharmacotherapy, although these beliefs were associated with greater perceived control over one's symptoms (Lüllmann, Berendes, Rief, & Lincoln, 2011) and improved engagement with psychotherapy (Carter, Read, & Morrison, 2016). Attributing cause to other people on the other hand was related to poor self-reported treatment adherence in individuals with a non-affective psychotic disorder (Watson et al., 2006). Other studies of individuals with a psychotic disorder have found that attributing the cause of their illness to biological factors was related to more positive attitudes toward medication (Wiesjahn et al., 2014; McCabe & Priebe, 2004) and higher satisfaction with treatment (McCabe & Priebe, 2004). In general, studies investigating causal attributions for mental illnesses suggest that people who attribute mental disorders to biological factors are more likely to endorse medication (Kuppin & Carpiano, 2006).

Impact of etiological beliefs on frequency of adverse side effects. Etiological beliefs did not have an effect on whether participants experienced adverse side effects during treatment. The interaction between the three dimensions of etiological beliefs and the four treatment conditions was also not significant with regards to whether or not participants experienced adverse effects.

Medication for PD has been associated with a number of side effects (Marcus et al., 2007). The increased serotonergic activity induced by SSRIs has been linked to the development

of anxiety, agitation, insomnia, tremors, nausea, anorexia, headache, and sexual dysfunction (Marchesi, 2008). However, in comparison to tricyclic antidepressants (TCAs) or monoamine oxidase inhibitors (MAOIs), SSRIs are considered to be safer and easier to tolerate for individuals with PD (Bighelli et al., 2016). Nonetheless, due to their heightened fear of physical sensations, individuals with PD often display a high intolerance of side effects associated with diverse pharmacological treatments (Marcus et al., 2007).

A study on medication attitudes and adherence in individuals with a psychotic disorder revealed that individuals who endorsed more biological causal beliefs, fewer psychological beliefs, and reported fewer side effects had more positive attitudes toward medication (Wiesjahn et al., 2014). However, the relationship between individuals' causal attributions and the development of treatment-related side effects has not been investigated. Since causal attributions in the present study were not associated with treatment discontinuation or improvement following treatment, it is not surprising that etiological beliefs were not predictive of whether or not participants experienced side effects.

It has also been suggested that adverse effects reported by individuals with PD are not easily distinguishable from physical symptoms associated with the underlying disorder (Marcus et al., 2007). Consequently, a medication effect could be perceived as a panic episode and could thus be reflected in individuals' severity of symptoms following treatment rather than their reported treatment side effects. Moreover, participants were withdrawn from the study if they presented side effects that prevented the increase of the dosage to 50mg. Since certain side effects associated with SSRIs such as restlessness or insomnia tend to develop in the first days or weeks of treatment (Bandelow, & Kaiya, 2006), it is possible that causal attributions may have been associated with the experiencing of immediate side effects to treatment rather than following longer-term exposure to treatment. Further investigation of the association between individuals' subjective perceptions of the cause of their disorder and the development of treatment-related side effects is warranted to provide a better understanding of the factors that may influence the frequency of adverse side effects reported by individuals with PD.

Limitations

The present study is not without its limitations. Because research on causal attributions in individuals with psychiatric disorders is limited, and due to the exploratory nature of this study, there are gaps that should be addressed in future research. We demonstrated that the etiological

beliefs of individuals with PD are associated with a number of demographic and clinical characteristics, as well as with symptom severity following treatment. However, our results did not reveal strong correlations, suggesting that the findings should be interpreted with caution. The weak correlations could possibly reflect a lack of variation in some variables, causing a limited range. It is also possible that other factors not included in this study are stronger predictors of causal attributions of illness in individuals with PD. For instance, Read and Law (1999) found that individuals who knew fewer people who had received psychiatric treatment were more likely to hold biogenetically oriented causal beliefs. Another study assessing causal beliefs in individuals with depression found that being African-American was associated with stronger beliefs in psychological and environmental causal factors (i.e., thought processes, stress, unhealthy relationships; Bann et al., 2004). It would therefore be worthwhile for future studies to explore the impact of socioeconomic status, education level and cultural background on individuals' etiological beliefs about PD.

Another limitation of this study is that the treatment outcome measure was based on clinician impressions of illness severity and improvement. It is possible that including self-report measures in the analysis would yield different findings. Additionally, this study used 12-week treatment outcome data and it is possible that an interaction between type of treatment and etiological beliefs could be observable after a more protracted period of treatment.

It should also be noted that the measure of causal attributions used in the present study is an adapted version of the Etiological Beliefs Questionnaire (ETBQ), for which the psychometric properties, have not been widely researched. Other scales such as the Illness Perception Questionnaire (IPQ; Weinman, Petrie, Moss-Morris, & Horne, 1996), which assesses five cognitive components of illness representations (identity, timeline, consequences, cause, and cure/control), have been more commonly used to measure individuals' etiological beliefs about illness. The IPQ and its revised version are theoretically derived, psychometrically validated, adaptable to different populations, and used in hundreds of published papers (Ayers et al., 2007). Therefore, future research could replicate the present study's objectives with other measures such as the IPQ in order to further support the present findings.

A finer analysis of causal attributions in individuals with anxiety disorders could also be conducted by examining the relationship between individual items or causal attributions (e.g., genes, maladaptive thoughts, family issues, etc.) and participants' demographic and clinical

variables, as well as treatment outcome measures. This analysis was beyond the scope of the present study but could yield interesting results, further clarifying the impact of individuals' etiological beliefs of illness. Lastly, our data analysis included multiple tests and we did not apply multiplicity adjustments, which raises concerns about Type I error (Keselman, Miller, & Holland, 2011). Nevertheless, it should be noted that this study was exploratory in nature and our main objectives were to identify hypotheses that could be subject to more rigorous future examination (Bender & Lange, 2001) rather than examining pre-specified hypotheses.

Despite the study's limitations, the current study has a number of strengths. Firstly, the placebo-controlled trial from which the data for this study was obtained is the first trial of the efficacy of a self-help intervention and pharmacotherapy in individuals with PD. Moreover, our analysis of the relationship between baseline characteristics and etiological beliefs included a large number of participants, which increases the confidence in our findings. With regards to the acute treatment phase, our attrition rate of 28.7% falls well within the dropout rate for studies involving pharmacotherapy for anxiety disorders (Santana & Fontenelle, 2011) and fares significantly better in comparison to other studies with self-administered therapy (Wooton et al., 2015; Titov et al., 2013; Rickwood & Bradford, 2012). Moreover, attrition was similar across the four treatment groups.

Suggestions for Future Research

A question left unexplored in the present study concerns the relationship between etiological beliefs and other variables that could potentially have a stronger effect on individuals' treatment response such as perceived helpfulness of treatments or treatment credibility. Iselin and Addis (2003) have suggested that individuals consider treatments more helpful when cause and treatment focus are congruent. They found that medical treatments for depression were considered to be significantly more helpful when individuals were presented with a physical etiological explanation of the condition, whereas psychological treatments were viewed as more helpful by individuals presented with a matching psychological etiological explanation rather than a physical one (Iselin & Addis, 2003).

Similarly, Meyer and Garcia-Roberts (2007) suggested that the causal attributions of individuals with depression were associated with a stronger motivation to engage with interventions that are congruent with their beliefs. Future research could explore the relationship between causal attributions, perceived helpfulness and treatment credibility. Furthermore, studies

could investigate the effect of etiological beliefs as a moderator of treatment outcomes rather than a predictor. Since causal attributions may not be highly predictive of treatment outcome such as compliance and clinical response, they may act as a strong moderator, changing the direction of the relationship between certain types of treatment and treatment response.

Genetic causal information has been found to influence perceived treatment effectiveness in a number of health problems by altering not only individuals' causal attributions but also their perceived controllability over their illness (Wright et al., 2012). Individuals' perceived level of control over their illness has been significantly associated with their causal attributions, as well as their information-seeking behaviour and coping strategies. Lavery and Clarke (1996) found that women with breast cancer who believed their illness was caused by factors over which they had no control became actively involved in efforts to fight their illness rather than adopt emotion-focused coping strategies. This could possibly explain why individuals who endorse psychological causal beliefs, considered as internal, controllable attributions, may experience more severe symptoms following treatment. Causal attributions could influence perceptions of control over one's illness, which have been associated with attributions of blame (Goldstein & Rosselli, 2003), perceived treatment effectiveness and credibility (Kemp et al., 2014; Wright et al., 2012) and consequently, could have a significant impact on individuals' symptom severity and response to treatment. Clear areas for future research include the relationship between etiological beliefs, perceptions of control and treatment outcomes in individuals with anxiety disorders, which could have significant implications for the treatment of PD.

Since participants did not have a choice on which treatment they were allocated, the present study did not evaluate the relationship between participants' treatment preferences and their etiological beliefs. Individuals' endorsement of psychological causes for their anxiety symptoms has been associated with a preference for psychological treatment and a belief in multiple etiology has been related to a preference for multiple treatment modes (Waikar et al., 1995). In depression, illness attributions were significantly associated with treatment assignment, with people endorsing intraindividual causes more likely to be assigned CBT and those attributing their depression to biological reasons more likely to receive psychopharmacological treatment (Schweizer et al., 2010). Similarly, another study on individuals with chronic forms of depression revealed that individuals who preferred medication only were more likely to endorse a chemical imbalance explanation for depression, whereas those desiring combined treatment were

more likely to attribute their depression to stressful experiences (Steidtmann et al., 2012). Treatment preferences have also been found to have a significant impact on treatment outcome, including lower willingness to enter antidepressant treatment or randomized clinical trials if preferences are not supported in individuals with depression (Van Schaik et al., 2004), and greater improvement and lower drop-out rates when receiving a preferred treatment in clients receiving treatment for a variety of mental diagnoses (Swift & Callahan, 2009). Therefore, since in real life individuals will seek out treatments they prefer, further investigation of the relationship between causal attributions and treatment preferences could shed light on how various client-related factors could affect response to treatment.

      In addition to causal attributions individuals have about their illness, there is interesting data suggesting that causal beliefs members of a person's social network have about illness can influence treatment adherence and outcome. A study by Cornwall, Scott, Garland, and Pollinger (2005) found that a concordance in causal attributions between individuals with depression and their partners was significantly associated with good outcome (Cornwall et al., 2005). Another study investigating etiological beliefs of caregivers and individuals with major depressive disorder suggested that the attribution of psychological causes to depression by caregivers was associated with decreased adherence to antidepressant treatment (Sher, McGinn, Sirey, & Meyers, 2005). Future studies could also consider incorporating an analysis of the influence of individuals with PD's social environment on treatment outcomes.

      Finally, several studies have found an association between causal attributions and public perception of mental illness. Biological etiological beliefs were associated with a perception that people with mental health issues are less likely to be cured, at a higher risk of harming themselves, more likely to require professional help and hospitalization (Lam et al., 2005) and more disabled than those with no mental health issues (Lam et al., 2005). With respect to PD, attributing illness to biological causes was related to a perception of people with PD as requiring significantly longer periods of treatment sessions and less likely to make progress following treatment (Lam & Salkovskis, 2007). Psychological etiological beliefs on the other hand, were associated with beliefs that people with depression are to be blamed for their condition, whereas environmental causal attributions were related to a reduced desired social distance, less agreement that depressed people are to blame for their condition, and an increased belief that people with depression are more violent than non-depressed people (Goldstein & Rosselli, 2003).

The present study has found that a number of demographic and clinical factors can influence individuals with anxiety's causal attributions and that these attributions could have an impact on their illness severity. However, perceptions regarding the impact of PD and of individuals with PD were not examined in the present study. Future research should explore these perceptions, as they could further elucidate the relationship between causal attributions and treatment outcome, as well as the stigmatization of individuals with the disorder.

Conclusion

The present study contributes to the small literature on causal attributions individuals with PD develop about their illness. The present study demonstrated that participants with a family history of psychiatric illnesses were more likely to endorse biological etiological beliefs whereas those with a younger age, comorbid psychiatric disorders, and a history of suicide attempts were more likely to attribute their illness to psychological causes. Participants experiencing impairment in family life endorsed both psychological and environmental causal beliefs, while those reporting higher fear of body sensations and agoraphobic cognitions were more likely to attribute their illness to biological and psychological causes. With regards to treatment outcome, the present study demonstrated that participants who endorsed psychological and environmental etiological beliefs experienced more severe symptoms 12 weeks following treatment; irrespective of the type of treatment they received.

As Leventhal's Common Sense Model suggests, part of the importance of assessing causal attributions lies in the fact that people act on their own lay explanations and beliefs about illness rather than on objective evidence (Leventhal et al., 2003). These findings highlight the importance of individuals' subjective perceptions about the causes of their illness, their relationship with patient characteristics, and impact on treatment response. Although causal attributions may not be directly associated with treatment behaviour, they can be considered as a way of coping with the personal and social impact of the illness (Carter et al., 2016). This consideration of the individual's perception might help health-care programs to deliver interventions for different clients that are in line with their individual beliefs. Clinically, these data may also provide an opportunity for health professionals to identify and alter maladaptive beliefs individuals may have about their disorder and develop a rapport with clients to ensure that treatments seem valid and valuable to them as well as improve functioning (Broadbent, Kydd, Sanders, & Vanderpyl, 2008). Providing psychoeducation to facilitate a shift in individuals'

causal attributions to better align with their chosen interventions may result in increased prognostic optimism, expectancies of treatment, and could improve outcome (Cohen et al., 2015).

Integrating both psychosocial and biological aspects within a causal model in psychosocial interventions has been suggested to have the potential of yielding more positive outcomes than focusing on one causal model (Caqueo-Urizar et al., 2015; Lüllmann et al., 2011). Understanding clinical and demographic differences in causal beliefs therefore provides a unique perspective through which health care providers may assist clients by communicating a more balanced perspective, in line with the biopsychosocial model, of the causes of PD and its multiple etiology. Improving clients' understanding of the causes of their illness could potentially moderate service use (Broadbent et al., 2008). The recent NICE Guideline has further emphasized the importance of providing clients with information about the nature and course of mental illness and proposed interventions, focusing on individuals' preferences in the choice of treatment and acceptability of the intervention (Pilling et al., 2011).

Furthermore, these data compel further investigation of individuals' subjective perceptions about the causes of their illness. Determining whether clients' beliefs regarding the pathogenesis of their illnesses may adversely affect their response to treatment can encourage health care professionals to find ways to minimize the negative effects of these attributions on treatment outcome (Lam, & Salkovskis, 2007). Additionally, since clinicians' adherence to a biological or psychological model of illness tends to influence how clients understand and perceive their problem (Lam, Salkovskis, & Warwick, 2005), identifying the implications of biological and psychological approaches can help raise practitioners' awareness regarding the impact of etiological information on individuals' perceptions.

Through collaboration and open communication, treatment providers could help clients in their treatment-related decision-making, address negative attitudes individuals have about their condition, as well as improve client uptake and compliance with treatment. Causal attributions have also been shown to predict people's willingness to support the allocation of funds to hypothetical cancer treatment programs (Knapp-Oliver & Moyer, 2012). Therefore, further investigation of causal attributions of illness could also have implications for agencies' willingness to support the allocation of funds to treatment programs for psychiatric illnesses, such as PD.

In conclusion, these findings extend the limited literature on etiological beliefs of illness in PD by determining their impact on severity of symptoms following treatment. The present study thus provides important insights for treatment providers regarding the factors that influence individuals with PD's subjective perceptions of illness and their treatment-related experiences.

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Table 1

Baseline Demographic and Clinical Characteristics

Variable	
Age (years)	36.26 ± 10.74
Female sex	154 (61.4)
Primary diagnosis	
Panic disorder	72 (28.7)
Panic disorder with agoraphobia	179 (71.3)
Presence of comorbid psychiatric disorders	106 (42.2)
Number of panic attacks in 2 weeks	10.08 ± 12.64
CGI–S	4.47 ± .76
MI-AAL	2.32 ± .96
BSQ	46.15 ± 11.51
ACQ	30.81 ± 8.65
SDS - Work	5.37 ± 2.66
SDS – Social life	5.78 ± 2.47
SDS – Family life	1.54 ± .98
Family history of psychiatric illnesses	169 (67.3)
Drug treatment history	113 (45)
Psychotherapy treatment history	76 (30.3)
History of suicide attempts	17 (6.8)
ETMQ	
- Biological	2.21 ± 1.36
- Psychological	2.10 ± 1.14
- Environmental	2.48 ± 1.39

Note. N = 251. Values are given as n (%) or mean± standard deviations. ETMQ = Etiological Model Questionnaire; CGI- S = Clinical Global Impression – Severity; MI-AAL = Mobility Inventory for Agoraphobia-Alone; BSQ = Body Sensations Questionnaire; ACQ = Agoraphobic Cognitions Questionnaire; SDS = Sheehan Disability Scale.

Table 2

Pearson's Product-moment/Point-biserial Correlations for Etiological Beliefs Dimensions and Baseline Demographic and Clinical Variables

	Etiological Beliefs		
	Biological	Psychological	Environmental
Age	-.08	-.17*	-.04
Gender (Female)	-.04	.13*	.12
Family history of psychiatric illnesses	.19*	.11	.12
Presence of comorbid disorders	.15*	.18*	.15*
Presence of agoraphobia	.08	.19*	-.05
MI-AAL	.06	.28*	.04
BSQ	.29*	.40*	.18*
ACQ	.31*	.45*	.22*
SDS - Work	.11	.30*	.24*
SDS - Social life	.07	.32*	.04
SDS - Family life	.20*	.30*	.29*
CGI – Severity	.05	.24*	.08
Drug treatment history	.02	-.04	.01
Psychotherapy treatment history	.03	.08	-.11
History of suicide attempts	-.03	.18*	.08

Note. N = 251. *$p \leq$ .05. MI-AAL = Mobility Inventory for Agoraphobia-Alone; BSQ = Body Sensations Questionnaire; ACQ = Agoraphobic Cognitions Questionnaire; SDS = Sheehan Disability Scale.

Table 3

Baseline Predictors of Biological Etiological Beliefs

Subscale	ETMQ – Biological beliefs				
	β	B	SE_B	t	p
Constant		-.49	.36	-1.36	.18
BSQ	.15	.01	.00	2.06	.04*
ACQ	.19	.32	.12	2.59	.01*
SDS – Family life	.08	.09	.07	1.31	.19
Family history of psychiatric illnesses	.15	.15	.06	2.53	.01*
Presence of comorbid disorders	.09	.08	.06	1.42	.16

Note. $N = 250$. *$p < .05$. ETMQ = Etiological Model Questionnaire; BSQ = Body Sensations Questionnaire; ACQ = Agoraphobic Cognitions Questionnaire; SDS = Sheehan Disability Scale.

Table 4

Baseline Predictors of Psychological Etiological Beliefs

Subscale	ETMQ – Psychological beliefs				
	β	B	SE_B	t	p
Constant		-2.75	.88	-3.14	.00
Age	-.14	-.02	.01	-2.59	.01*
Gender	.02	.04	.13	.33	.74
Presence of Agoraphobia	.01	.03	.17	.17	.87
Presence of comorbid disorders	.11	.25	.13	2.00	.05*
CGI – Severity	.07	.10	.09	1.14	.25
MI – AAL	.02	.09	.29	.33	.74
BSQ	.15	.01	.01	2.19	.03*
ACQ	.23	.99	.28	3.48	.00*
SDS – Work	.06	.02	.03	.85	.39
SDS – Social life	.06	.03	.03	.86	.39
SDS – Family life	.13	.36	.17	2.09	.04*
History of suicide attempts	.11	.49	.25	1.97	.05*

Note. N = 250. *p < .05. ETMQ = Etiological Model Questionnaire; CGI- S = Clinical Global Impression – Severity; MI-AAL = Mobility Inventory for Agoraphobia-Alone; BSQ = Body Sensations Questionnaire; ACQ = Agoraphobic Cognitions Questionnaire; SDS = Sheehan Disability Scale.

Table 5

Baseline Predictors of Environmental Etiological Beliefs

Subscale	ETMQ – Environmental beliefs				
	β	B	SE_B	t	p
Constant		-.37	1.11	-.34	.74
Presence of comorbid disorders	.12	.33	.17	1.92	.06
BSQ	.04	.00	.01	.50	.62
ACQ	.09	.46	.38	1.23	.22
SDS – Work	.12	.06	.04	1.79	.07
SDS – Family life	.20	.69	.23	2.97	.00*

Note. $N = 250$. *$p < .05$.

Table 6

Summary of Regression Analyses Predicting CGI – Improvement and Severity Scores from Mean Scores of the ETMQ

ETMQ	CGI – Improvement					CGI - Severity				
Subscale	*β*	*B*	*SE_B*	*t*	*p*	*β*	*B*	*SE_B*	*t*	*p*
Psychological	.10	.03	.02	1.41	.16	.21	.22	.08	2.91	.00*
Environmental	.11	.03	.02	1.55	.12	.19	.17	.06	2.65	.01*
Biological	.04	.03	.05	.48	.63	.11	.30	.21	1.44	.15

Note. *p* < .05. ETMQ = Etiological Model Questionnaire. CGI = Clinical Global Impression.

Table 7

Summary of Regression Analyses Predicting Compliance with SCBT and Treatment Completion from Mean Scores of the ETMQ

ETMQ Subscale	Compliance with SCBT tapes					Compliance with SCBT HW					Treatment Completion			
	β	B	SE_B	t	p	β	B	SE_B	t	p	B	SE_B	e^B	p
Psychological	.03	.00	.02	.26	.80	.03	.01	.03	.24	.81	-.02	.12	.98	.90
Environmental	-.12	-.05	.04	-1.12	.27	.01	.00	.03	.12	.91	.06	.10	1.07	.53
Biological	-.03	-.00	.01	-.24	.81	-.06	-.04	.08	-.51	.62	-.07	.31	.94	.83

Note. *p < .05. ETMQ = Etiological Model Questionnaire. SCBT = Self-administered Cognitive Behavioral Therapy. HW = homework.

Table 8

Summary of Multiple Logistic Regression Analyses Predicting the Presence of Adverse Events from Mean Scores of the ETMQ

ETMQ Subscale	Presence of adverse events			
	B	SE_B	e^B	p
Psychological	-.23	.33	.79	.49
Environmental	-.19	.29	.82	.50
Biological	.84	.94	2.31	.37

Note. *$p < .05$. ETMQ = Etiological Model Questionnaire.

| 289 SCREENED |
| 251 RANDOMIZED |

| 62 PBO | 65 PBO/SCBT | 63 SERT | 61 SERT/SCBT |

| 62 included in | 64 included in ITT | 62 included in | 59 included in ITT |

43 completed 12 wks AT	44 completed 12 wks AT	46 completed 12 wks AT	43 completed 12 wks AT
19 dropped at of AT	20 dropped out of AT	16 dropped out of AT	16 dropped out of AT
▪ 9 lack of efficacy	▪ 5 lack of efficacy	▪ 5 lack of efficacy	▪ 2 lack of efficacy
▪ 6 adverse events	▪ 3 adverse events	▪ 5 adverse events	▪ 7 adverse events
▪ 2 withdrew consent	▪ 5 withdrew consent	▪ 1 withdrew consent	▪ 2 withdrew consent
▪ 2 protocol violation	▪ 1 protocol violation	▪ 3 protocol violation	▪ 2 lost to follow –up
	▪ 2 lost to follow-up	▪ 1 lost to follow-up	▪ 3 other

Note. ITT = Intent-to-treat; AT = Acute Treatment.

Figure 2. Flow of participants during the trial.