RUNNING HEAD: Telephone CBT for High AS

Telephone-Delivered CBT for High Anxiety Sensitivity: A Randomized Controlled Trial

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Abstract

Objective. High anxiety sensitivity (AS) is associated with the development and maintenance of anxiety and depressive symptoms, and is theorized to be a mediator of treatment outcomes for anxiety and depression. The present study tested the efficacy of a telephone-delivered cognitive behavioural treatment (CBT) in reducing high AS and its associated anxiety and depressive symptoms. **Method**. Treatment-seeking participants with high AS were recruited from the community (N = 80; M age = 36 years; 79% women; 76% Caucasian) and randomly assigned to an eight week telephone-delivered CBT program or a waiting list control. Participants completed anxiety and depression symptom and diagnostic measures at pre- and post-treatment, after a subsequent month of continued interoceptive exposure, and two months later. **Results**. Multilevel modeling showed the treatment was successful in reducing AS, as well as panic, social phobia, and posttraumatic stress symptoms, and number of DSM-IV diagnoses per participant when compared to a waiting list control. These gains were maintained at 12 week and 20 week follow-ups. Generalized anxiety and depressive symptoms, however, did not improve as a result of treatment. Mediated moderation analyses suggested that treatment-related changes in AS may mediate anxiety symptom changes. **Conclusion**. Results of the present study provide promising evidence for this transdiagnostic treatment approach. Reductions in anxiety symptoms across diagnostic categories stemming from this AS-targeted intervention may have implications for helping a broad array of clients with various anxiety disorders that share AS as a common risk or maintenance factor.

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Anxiety disorders are one of the most common mental health problems worldwide, with yearly prevalence estimates ranging from 8-18% (Alonso & Lépine, 2007; Kessler, Chiu, Demler, & Walters, 2005). They have an early onset (Kessler, 2007) and a chronic course (Bruce et al., 2005), are associated with significant functional impairment (Bijl & Ravelli, 2000), and often lead to the adoption of maladaptive strategies to reduce anxiety (Badour, Blonigen, Boden, Feldner, & Bonn-Miller, 2012). Moreover, high rates of comorbidity can exacerbate anxiety. Anxiety disorders often co-occur with each other and with mood disorders (Alonso & Lepine, 2007; Kessler et al., 2005), with rates of comorbidity in clinical samples reaching greater than 50% (Brown, Campbell, Lehman, Grisham, & Mansell, 2001).

Evidently, there is a need for treatment for anxiety and its comorbidities. It is widely recognized that cognitive behavioural therapy (CBT) is an effective treatment for various anxiety disorders, including panic disorder (PD), social phobia (SP), generalized anxiety disorder (GAD), and posttraumatic stress disorder (PTSD) (Norton & Price, 2007; Stewart & Chambless, 2009), and for depression (Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012). However, the current trend in CBT practice is to treat comorbidities consecutively or in parallel rather than in an integrated manner. Some studies show that comorbidity does not interfere with treatment outcomes (Allen et al., 2010; Tsao, Mystkowski, Zucker, & Craske, 2002) while others suggest comorbid conditions may disrupt treatment efficacy (Chambless, Renneberg, Gracely, Goldstein, & Fydrich, 2000; Farris, Epstein, McCrady, & Hunter-Reel, 2012). Given the prevalence of comorbidity, researchers are exploring integrated interventions targeted at transdiagnostic risk factors. Transdiagnostic interventions assume that mental health problems are manifestations of shared risk factors or core processes (e.g., high neuroticism); thus, treatment targeting these

underlying factors/processes could reduce symptoms across a range of disorders (Barlow, Allen, & Choate, 2004; Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010).

A salient example of a transdiagnostic intervention is Barlow's (2011) Unified Protocol which teaches neurotic individuals to respond adaptively to strong negative affect by adjusting maladaptive cognitive appraisals, modifying emotion-driven behaviour, and preventing emotional avoidance (Barlow et al., 2004). Preliminary studies of this intervention have shown it to successfully reduce anxiety and depressive symptoms across diagnostic categories (Ellard et al., 2010; Farchione et al., 2012). Given the promise of this treatment approach, the present study aimed to test a similar trans-diagnostic intervention. Instead of neuroticism, however, we targeted the underlying risk factor of anxiety sensitivity (AS).

AS is an individual difference factor implicated in the development and maintenance of anxiety disorders (Olatunji & Wolitzky-Taylor, 2009). More specifically, AS is an enduring fear of arousal-related sensations (e.g., increased heart rate) arising from the tendency to interpret these sensations catastrophically, believing that they will have serious physical, psychological, or social consequences (Reiss, 1991; Reiss & McNally, 1985). For example, an individual with high AS who experiences a racing heart might fear this sensation portends a heart attack. In contrast, those with low AS regard these sensations as unpleasant but harmless.

Clark's cognitive theory of panic (1986) provides one model for understanding the role of AS in psychopathology. In this model, when an individual interprets an otherwise benign arousal-related sensation catastrophically (similar to a person with high AS), his/her perception of threat is enhanced, leading to increased severity of arousal symptoms and maladaptive behaviours (e.g., panic attacks, avoidance). Accordingly, studies have shown that high AS predicts fearful responding to physical sensations generated by a CO_2 challenge (Zvolensky,

Feldner, Eifert, & Stewart, 2001) and that AS is high among those with PD (Taylor, Koch, McNally, & Crockett, 1992). High AS prospectively predicts the development of panic attacks (Schmidt, Lerew, & Jackson, 1997). Consistent with its theoretical role in motivating avoidance, AS is associated with agoraphobia symptoms (White, Brown, Somers, & Barlow, 2006).

Next to PD, AS levels are highest among those with PTSD (Taylor, Koch, & McNally, 1992). Studies consistently show that people with PTSD symptoms have higher AS than those without (Asmundson & Stapleton, 2008) and that AS and PTSD symptom severity are correlated (Stephenson, Valentiner, Kumpula, & Orcutt, 2009). Researchers have suggested that AS may amplify the emotional reaction to trauma (Taylor, 2004) and longitudinal studies do show that those with high AS are more likely to develop PTSD symptoms after a trauma (Keogh, Ayers, & Francis, 2002). However, researchers have also postulated that high AS might arise from trauma exposure (Taylor, 2004) or that there may be a reciprocal relationship between AS and PTSD symptoms after a trauma exposure (Marshall, Miles, & Stewart, 2010).

High levels of AS also exist among those with SP (Norton, Cox, Hewitt, & McLeod, 1997). This is likely due to the fear that a display of observable anxiety symptoms might lead to negative public evaluation (Cox, Borger, & Enns, 1999). In accordance, research has shown that lower AS levels predict recovery from SP (Vriends et al., 2007). High AS has also been found among those with GAD (Deacon & Abramowitz, 2006; Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004) and non-clinical worriers (Viana & Rabian, 2008). High AS may be connected to GAD by way of fears of cognitive dyscontrol (Rector, Szacun-Shimizu, & Leybman, 2007). Researchers suggest that concerns of cognitive dyscontrol reported by high AS individuals (e.g., “When I cannot keep my mind on a task, I worry that I might be going crazy”) are consistent with cognitive appraisals of worry characteristic of GAD. In other words, the cognitive concerns

element of AS may tap the hallmark meta-cognitive belief of those with GAD that repetitive thought/worry is uncontrollable and dangerous (Rector et al., 2007; Wells, 2005).

People with depression also report higher AS levels as compared to healthy controls, and depressed people's AS levels are on par with those seen in anxiety disorders (Otto et al., 1995). Moreover, high AS predicts depression five weeks later (Schmidt et al., 1997). Several studies have shown that the cognitive concerns lower order factor of AS (which captures a fear of losing control), rather than the physical or social concerns factors, predicts depressive symptoms in clinical (Cox, Enns, & Taylor, 2001) and non-clinical samples (Deacon, Abramowitz, Woods, & Tolin, 2003). In fact, AS cognitive concerns have been argued to be a "depression-specific form of anxiety sensitivity" (Taylor, Koch, Woody, & McLean, 1996, p. 478). Recently, AS cognitive concerns have also been associated with suicidal ideation and attempts among a clinical sample with mixed diagnoses (Capron et al., 2012).

In light of the relation between AS and anxiety and depression, it seems useful to explore interventions that target AS; such interventions may have transdiagnostic implications. Research shows that CBT-oriented interventions which include psychoeducation and cognitive restructuring and/or focus on interoceptive exposure to arousal-related body sensations can reduce high AS (Keough & Schmidt, 2012; Smits, Berry, Tart, & Powers, 2008; Watt, Stewart, Lefaivre, & Uman, 2006). A number of these interventions (e.g., Keough & Schmidt, 2012; Watt et al., 2006) are brief in nature (i.e., one to three sessions), in part because they were designed to be implemented among non-clinical populations with high AS. AS is also a mediator of anxiety and depression treatment outcome (Arch, Wolitzky-Taylor, Eifert, & Craske, 2012; Otto et al., 1995; Smits, Powers, Cho, & Telch, 2004) including Barlow's Unified Protocol (Sauer-Zavala et al., 2012).

Given the promise of an AS-focused intervention for reducing anxiety and depression, our objective was to test the efficacy of a CBT intervention designed to reduce high AS. In addition, we aimed to deliver the treatment in a way that would increase its accessibility. According to a national survey, only 11% of those with a current anxiety disorder received some form of treatment in the previous year (Ohayon, Shapiro, & Kennedy, 2000). Obstacles to treatment can include time constraints and other responsibilities (e.g., work, childcare), transportation difficulties, a lack of qualified clinicians or available services, fear of stigma, physical or mental health conditions limiting travel, and long waiting lists (Collins, Westra, Dozois, & Burns, 2004; Mojtabai et al., 2011). Many of these barriers are particularly relevant for rural communities, which tend to have fewer services and qualified clinicians, and longer commutes to services (Hauenstein et al., 2006). These obstacles can discourage individuals from seeking treatment, increase the severity of psychopathology, and create a negative relationship between treatment seekers and service organizations (McGrath & Cunningham, 2005).

A distance delivery approach to treatment is one way to increase access to services while still delivering empirically-supported treatment. Distance-based treatment involves using remote communication technologies (e.g., telephone, email, videoconferencing) to connect therapist and client, in place of face-to-face meetings. This communication is supplemented by the provision of materials to the client by mail, book, and/or the Internet. Distance delivery can facilitate treatment access for those with difficulties getting to services and increase patient confidentiality by allowing individuals to engage in treatment from the privacy of their home. Recent systematic reviews of distance-based treatment suggest that distance-based CBT is more effective than a waiting list control and as effective as face-to-face CBT in treating anxiety and depression (Andrews, Cuijpers, Craske, McEvoy, & Titov, 2010; Bee et al., 2008; Spek et al., 2007).

Study Aims and Hypotheses

The present study had two objectives. First, we aimed to test the efficacy of a telephone-delivered CBT intervention for AS in reducing AS among a community sample of treatment-seeking individuals with high AS. Second, we explored the transdiagnostic implications of this intervention by examining changes in panic, generalized anxiety, posttraumatic stress, SP, and depression symptoms pre- to post-treatment. In particular, to test if the AS-focused intervention was achieving its beneficial therapeutic effects by way of reducing AS, we investigated the mediating role of AS in any anxiety or depressive symptom reduction. We compared the theoretical mechanism of changes in AS as a treatment mediator with the more general theoretical mechanism of changes in neuroticism as a mediator.

Traditional transdiagnostic treatments (e.g., Johnston, Titov, Andrews, Spence, & Dear, 2011; McEvoy & Nathan, 2010; Norton & Barrera, 2012; Titov, Andrews, Johnston, Robinson, & Spence, 2010; Titov et al., 2011) typically involve participants with various anxiety disorders and/or depression following a standard package of treatment modules under the premise that different anxiety and depressive disorders are characterized by similar patterns of thinking and behaving (e.g., maladaptive cognitions and avoidance), and as such can be treated using similar strategies (e.g., cognitive restructuring and exposure). In contrast, instead of targeting shared patterns of thinking and behaving, the present treatment targets an underlying risk factor (i.e., AS) that might contribute to each disorder by leading to these shared patterns of thinking and behaving. In this way, the present treatment is more similar to the transdiagnostic treatment developed by Barlow and colleagues (2011) who targeted neuroticism as an underlying risk factor for a variety of emotional disorders.

The current treatment was developed from an evidence-based brief CBT protocol for reducing AS (Watt et al., 2006). This, in addition to meta-analytic evidence for the amenability of AS to CBT treatment (Smits et al., 2008), meant we anticipated that the current treatment would reduce AS. Moreover, because AS has been shown to mediate anxiety and depression treatment outcome (Smits et al., 2004), we hypothesized that the present AS-focused treatment would lead to decreases in anxiety and depressive symptoms and that changes in AS would mediate these symptom reductions.

Method

The present clinical trial was prospectively registered on the protocol registration system ClinicalTrials.gov with the identifier NCT01194765.

Participants

We recruited participants through notices in newspapers and posters in health, education, and community centres (February to December, 2011) advertising a research study for those with fears of anxiety-related sensations. To be eligible to participate, individuals had to be ≥18 years of age, have access to a telephone, and meet criteria for high AS, as reflected by a score of ≥23 on the Anxiety Sensitivity Index – 3 (ASI-3; Taylor et al., 2007), which is one standard deviation above the non-clinical population mean (*M*=12.8, *SD*=10.6; Reiss, Peterson, Taylor, Schmidt, & Weems, 2008). Individuals also completed Health Canada's Physical Activity Readiness Questionnaire (PARQ; Shephard, Cox, & Simper, 1981) to screen for any contraindications to physical activity (e.g., hypertension, cardiac disease) that would prohibit them from participating in the exercise (i.e., interoceptive exposure) component of treatment. If the PARQ raised concerns with an individual's suitability for physical exercise, he/she was required to secure a note from his/her doctor indicating his/her readiness for exercise to be eligible.

In addition, individuals could not be engaged in other current psychotherapy. They were permitted to be using a pharmacological intervention as long as their medication type and dosage had been stable for the three months prior to treatment and remained so during the study. Finally, individuals were screened for psychosis using the Psychotic Screening Module of the Structured Clinical Interview for DSM-IV-TR (SCID; First, Spitzer, Gibbon, & Williams, 2002), and current suicidal ideation with an item from the Beck Depression Inventory – II (Beck, Steer, & Brown, 1996). The treatment under investigation did not address these mental health concerns and thus individuals with current psychosis and/or suicidal ideation were excluded.

Overall, 182 individuals expressed interest in participating. Of those, 109 qualified for participation and 80 consented, completed pre-treatment assessment procedures, and were randomized to a treatment condition (see Figure 1). Characteristics of the final study sample (N = 80) can be found in Table 1 (and Supplemental Table 1). According to the baseline SCID, 33 participants (41%) presented with one current Axis I diagnosis, 23 (29%) had a primary and comorbid condition, 11 (14%) qualified for two or more comorbidities, and 13 (16%) did not qualify for a DSM-IV diagnosis.

Procedure

Those who qualified for participation provided verbal informed consent over the telephone and signed a written informed consent form via mail. Participants were then randomized using an online random number generator (www.randomization.com) to either the CBT treatment condition (CBT) or to a waiting list control (WLC), but were not informed of their random assignment until after completing the pre-treatment assessment. Participants completed a self-report questionnaire by mail and a telephone-administered SCID (First et al., 2002) assessment by an interviewer blind to treatment condition. Interviewers were clinical

psychology PhD students trained in SCID administration and supervised by registered psychologists. Upon completion of the telephone interview and the self-report questionnaire, participants began either the CBT or WLC condition.

All participants completed assessment measures eight and 12 weeks later to coincide with completion of the telephone therapy sessions and the interoceptive exposure component of treatment, respectively, for those in the CBT condition. Those in the CBT condition also completed the same measures at 20 weeks. Twelve weeks after the start of both the CBT and WLC conditions, participants also completed a telephone-administered SCID (First et al., 2002) conducted by interviewers blind to participants' treatment condition. Participants were compensated with $10 gift cards to a local grocery or book store for completing questionnaires at each time point. All study procedures were approved by the relevant Research Ethics Board.

Telephone-delivered CBT. Participants in the CBT condition received telephone-delivered CBT for high AS. Research suggests a general receptiveness to receiving psychiatric services via telephone (Grubaugh, Cain, Elhai, Patrick, & Frueh, 2008). Treatment was designed to target high AS in general, as opposed to any of its specific components in particular (i.e., physical, cognitive, or social concerns). The CBT protocol was developed from a brief empirically validated CBT intervention for high AS (Watt et al., 2006), published as a self-help resource by Watt and Stewart (2008). While the current treatment included many of the same components evident in brief AS treatments (e.g., Keough & Schmidt, 2012; Watt et al., 2006), we expanded the protocol from its original, brief form, into an eight session intervention. We adopted this change because we anticipated that the large majority of our high AS, treatment-seeking participants would have clinically-significant anxiety problems, in contrast to the non-

clinical samples who undertook the brief interventions. We anticipated that the presence of these notable mental health problems would require a more comprehensive treatment approach.

Participants were mailed Watt and Stewart's (2008) self-help book at the outset of treatment and it served as a treatment manual. Participants were assigned weekly reading and homework exercises and a therapist guided participants through the treatment by providing individualized support and feedback in weekly 50 minute telephone sessions (for a total therapist-client contact time of 400 minutes). When scheduled telephone contact with a participant was unsuccessful (after three attempts within the first 15 minutes of the scheduled session), research assistants re-contacted the participant by phone and email to reschedule the session at the earliest possible date. Completion of treatment components for each week was indexed by participant self-report.

The intervention was divided into an eight week program consisting of four modules. The first module included psychoeducation about AS, anxiety symptoms so feared by individuals with high AS, and accurate information about the meaning of anxiety-related sensations, as well as how AS is related to mental health problems (weeks 1-2). The second module focused on cognitive restructuring, particularly with respect to the main thinking errors characteristic of those with high AS – catastrophizing the meaning of anxiety sensations and overestimating the probability of negative outcomes of these sensations (weeks 3-4). The third module introduced interoceptive exposure (weeks 5-6); participants were asked to run/brisk walk three times per week for 10 minutes through the remainder of treatment and for four weeks after the telephone therapy sessions had concluded. This particular interoceptive exposure exercise is specifically intended to expose participants to feared physical sensations similar to those experienced when anxious. Prior research strongly supports the efficacy of physical exercise as an intervention for

the reduction of high AS (Broman-Fulks, Berman, Rabian, & Webster, 2004; Broman-Fulks & Storey, 2008; Smits et al., 2008). Participants completed weekly interoceptive exposure tracking sheets and were provided with a Polaris heart rate monitor to ensure they were raising their heart rate sufficiently to mimic body arousal sensations during the exposure.[1] Finally, the fourth module focused on relapse prevention (week 7) and how individuals could extend treatment gains (week 8). Therapists were six registered psychologists and six senior clinical psychology PhD students trained in the CBT protocol by one of the authors. Students were supervised by registered psychologists during weekly one hour group supervision sessions.

Waiting list control (WLC). Participants assigned to the WLC condition did not receive any intervention. They received a check-in phone call from research personnel after four weeks designed to encourage their continued engagement in the research study. WLC participants were invited to switch over to the CBT condition after the 12 week assessment.

Materials

Structured Clinical Interview for DSM-IV-TR (SCID; First et al., 2002). We administered all modules of the SCID, a structured diagnostic interview, to assess for DSM-IV diagnoses. Reliability estimates tend to be >.60 across disorders (Zanarini et al., 2000). The SCID has been administered successfully over the telephone (Furmark et al., 2009).

Anxiety Sensitivity Index – 3 (ASI-3; Taylor et al., 2007). The ASI-3 is an 18-item self-report measure that indexes AS, or the amount of fear an individual experiences with respect to anxiety-related body sensations. It was developed from the original 16-item ASI (Peterson & Reiss, 1992) to better assess the three core components of AS. Participants indicate the extent to which they agree or disagree with each item (e.g., "It scares me when my heart beats rapidly") on

[1] Unfortunately, many participants had difficulty using the heart rate monitors, finding they did not reliably register their heart rate and/or provided very unrealistic numbers. Because of the questionable nature of this data it was not used in our analyses; future studies could use a better physiological index of exercise intensity.

a 5-point Likert scale (0 = *very little* to 4 = *very much*). Items are summed for a total score. The ASI-3 can also be separated into three 6-item lower order subscales measuring fear of physical sensations (physical concerns), fear of psychological sensations (cognitive concerns), and fear of social consequences of anxiety sensations (social concerns). The ASI-3 has good internal reliability and criterion validity (Taylor et al., 2007). Twelve week test-retest reliability for the WLC in the present study was $r = .74$.

We used the ASI-3 to assess participants' 'recovery' status post-treatment. Participants whose post-treatment ASI-3 scores were closer to the normal, vs. dysfunctional, population mean and who evidenced reliable change according to Jacobson and Truax's (1991) formula were considered to be 'recovered' at post-treatment.

NEO Five Factor Inventory – Neuroticism Subscale (NEOFFI-N; Costa & McCrae, 1992). To test the specificity of the present treatment and the mediating role of AS relative to another common underlying contributor to mental health problems, levels of neuroticism were measured using the Neuroticism subscale of the NEOFFI. On this subscale, participants are asked to indicate the extent to which they agree or disagree (*Strongly Agree* to *Strongly Disagree*) with each of 12 statements (e.g., "I often feel inferior to others"). The full NEOFFI has been well-validated (Costa & McCrae, 1992).

Panic Attack Questionnaire – IV (PAQ-IV; Norton, Zvolensky, Bonn-Miller, Cox, & Norton, 2008). We used portions of the PAQ-IV, a measure of the specific features of PD, asking participants to report the degree to which they experienced 14 panic symptoms (e.g., sweating) on a 5-point Likert scale (0 = *doesn't occur* to 4 = *very severe*); summing these items provided a PAQ-IV total score. While the PAQ-IV is relatively new, previous versions of the PAQ have been empirically validated and the PAQ-IV has strong concurrent validity (Norton et al., 2008).

Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is a 16-item self-report questionnaire that assesses an individual's general tendency to worry excessively – a core trait of GAD. Participants are asked to indicate on a 5-point Likert scale (1 = *not at all typical* to 5 = *very typical*) how typical of them is a statement (e.g., "I worry all the time"). Items are summed for a total score. The PSWQ has good internal consistency and test-retest reliability (Molina & Borkovec, 1994) and validity in relation to GAD (Brown, Antony, & Barlow, 1992).

Life Stressor Checklist – Revised (LSC-R; Wolfe & Kimmerling, 1997) and the **Modified PTSD Symptom Scale** (MPSS; Falsetti, Resnick, Resick, & Kilpatrick, 1993). On the LSC-R, participants were asked to indicate which, if any, of a list of traumatic events had happened to them. Those who indicated that they had experienced a traumatic event completed the MPSS, which assesses how often participants experience a series of PTSD symptoms (e.g., "Have you had repeated or intrusive upsetting thoughts or recollections of the event?") using a 5-point Likert scale (0 = *not at all* to 3 = *5 or more times per week*) as well as how severe these symptoms were (1 = *not at all distressing* to 5 = *extremely distressing*). An overall score was calculated by summing these two subscales. Only participants endorsing a DSM-IV criterion A qualifying traumatic event were included in analyses with the MPSS. Those who reported experiencing more than one traumatic event completed the MPSS in relation to the event that was currently affecting them to the greatest degree. The MPSS has shown good internal consistency and concurrent validity (Falsetti et al., 1993).

Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987). SP symptoms were measured using the LSAS. The LSAS presents 16 social or performance situations (e.g., "Calling someone you don't know very well") for which participants rate their fear (0 = *none* to 3 = *severe*) and the

degree to which they avoid each situation (0 = *never* to 3 = *usually*). We created an overall SP score by summing these two subscales. The LSAS has strong internal reliability ($\alpha = 0.95$) and good convergent and discriminant validity (Baker, Heinrichs, Kim, & Hofmann, 2002).

Depression Anxiety Stress Scales – 21 Depression Subcale (DASS-21 Depression; Lovibond & Lovibond, 1995*).* Current, non-specific symptoms of depression were evaluated using the DASS-21 Depression subscale. Individuals indicate the extent to which a particular negative emotional state (e.g., "I felt that I had nothing to look forward to") has applied to them over the past week on a 4-point scale (0 = *did not apply to me* to 3 = *applied to me very much or most of the time*). Depression subscale scores are calculated by summing across the subscale items and multiplying by two to compare it to DASS-42 norms. The DASS-21 has good internal consistency and concurrent validity (Antony, Bieling, Cox, Enns, & Swinson, 1998).

Sheehan Disability Scale (SDS; Leon, Shear, Portera, & Klerman, 1992). Using an 11-point scale, participants rated their functional disability, or the extent to which their mental health symptoms disrupted their functioning in three domains: Work/school, family life/responsibilities, and social life. The SDS has been shown to be reliable and have satisfactory construct and criterion validity (Leon et al., 1992).

Treatment Satisfaction. To assess satisfaction with treatment, participants answered a series of open-ended questions about their experience (e.g., "What was the most helpful part of the treatment?"). Participants rated how satisfied they were with the treatment on a 10-point scale (10 = *very satisfied*) and whether they would recommend the treatment to a friend. Participants also answered an open-ended question as to any concerns with the present treatment.

Working Alliance Inventory – Short Form Revised (WAI-SR; Hatcher & Gillaspy, 2006). The WAI-SR is a 12-item self-report measure of therapeutic alliance comprised of three

subscales: (a) Agreement on the goals of therapy (e.g., "My therapist and I are working towards mutually agreed upon goals"), (b) agreement on the tasks of therapy (e.g., "My therapist and I agree on what it is important for me to work on"), and (c) the therapist-client bond (e.g., "I feel that my therapist appreciates me"). Participants responded using a 5-point Likert scale (1 = *seldom* to 5 = *always*). Overall and subscale scores are calculated by summing the appropriate items. The WAI-SR has good internal consistency and convergent and discriminant validity (Hatcher & Gillaspy, 2006).

Data Analytic Plan

To check if randomization resulted in balanced groups, differences between the CBT and WLC conditions at baseline were assessed using analysis of variance (ANOVA) and chi-squares (χ^2) in SPSS 20.0. Hypotheses were tested using multilevel modelling with HLM 7.0 software (Scientific Software International, Inc., Lincolnwood, IL). A two-level model was specified with repeated measures (level-1) nested within people (level-2). Multilevel models have numerous advantages when compared to ANOVA: (a) They handle missing data using a maximum likelihood approach, which provides more statistical power, and relatively unbiased parameter estimates when compared to listwise deletion and single imputation methods (Graham, 2009); (b) they account for the non-independence of observations associated with repeated measurement, reducing the risk of Type I error; and (c) they can accommodate unequal time periods between assessment periods (Gueorguieva & Krystal, 2004).

We estimated separate models for each of the outcome variables using restricted maximum likelihood estimation. At level 1, time was entered as a predictor: Time 1 (baseline) was coded as 0, time 2 (8 weeks) as 2, and time 3 (12 weeks) as 3 to represent the unequal amounts of time between measurement occasions. Because there were three measurement

occasions, we tested a linear growth curve with random slopes and random intercepts. We also explored the possibility of quadratic growth curves using fixed slopes and random intercepts (at least four measurement occasions are required for random slopes in a quadratic growth curve; Mroczek & Griffin, 2007). To aid in interpretation of quadratic effects, we used orthogonal polynomial contrasts coding for time (linear = -1, 0, 1; quadratic = 1, -2, 1) when testing for quadratic effects so the linear coefficient would represent overall pre-to-post change, and the quadratic term would represent the degree of acceleration or deceleration. At level 2, treatment group (WLC = 0 and CBT = 1) was included as a predictor. The time*group interaction was tested by including a cross-level effect between time at level 1 and group at level 2. Thus, the equations for linear random-slopes analyses were:

Level-1 Model

$$OUTCOME_{ti} = \pi_{0i} + \pi_{1i}*(TIME_{ti}) + e_{ti}$$

Level-2 Model

$$\pi_{0i} = \beta_{00} + \beta_{01}*(GROUP_i) + r_{0i}$$
$$\pi_{1i} = \beta_{10} + \beta_{11}*(GROUP_i) + r_{1i}$$

And the equations for quadratic fixed-slopes analyses were:

Level-1 Model

$$OUTCOME_{ti} = \pi_{0i} + \pi_{1i}*(TIME_{ti}) + \pi_{2i}*(QUADTIME_{ti}) + e_{ti}$$

Level-2 Model

$$\pi_{0i} = \beta_{00} + \beta_{01}*(GROUP_i) + r_{0i}$$
$$\pi_{1i} = \beta_{10} + \beta_{11}*(GROUP_i)$$
$$\pi_{2i} = \beta_{20} + \beta_{21}*(GROUP_i)$$

When choosing which analysis to report, we reported quadratic fixed-slopes models if the quadratic*group interaction was significant; otherwise, we reported linear random-slopes models. If the treatment was successful, we would expect a significant linear time x group cross-level interaction. When cross-level interactions were significant, we probed the interaction using a simple slopes approach (Preacher, Curran, & Bauer, 2006). We used the formula for Cohen's d

($d_{GMA\text{-}raw}$) adapted for use in growth-curve models by Feingold (2009) as a measure of effect size. When quadratic equations were modeled, both the linear and quadratic coefficients were used to calculate pre-post change. $d_{GMA\text{-}raw}$ represents the difference in pre-post change in outcome variables between two conditions. Like Cohen's d, a value of .30 can be considered small, .50 medium, and .80 or higher large. To test mediated moderation, significance of indirect effects was calculated using a Monte Carlo Method (Preacher & Selig, 2012). This analysis tested whether the time*group interaction had an indirect effect on outcomes through AS, when controlling for Neuroticism. Both AS and Neuroticism were entered as Level 1, time-varying covariates in this mediated moderation model. We measured clinical significance by: (1) Examining changes in functional disability in a multilevel model, (2) examining the number of SCID diagnoses per participant as an outcome variable in a multilevel model, (3) using the Jacobson and Truax (1991) approach for assessing clinically significant change for AS, and (4) testing whether gains in the CBT group were maintained from the 12 to 20 week follow-up.

Results

Pre-Treatment Differences on Demographic Variables

The two groups did not differ significantly on sex, $\chi^2(1) = 0.67$, $p = 0.41$, age, $F(1,79) = .02$, $p = 0.88$, and use of psychotropic medication, $\chi^2(1) = 1.92$, $p = 0.17$.

Participant Dropout and Treatment Completion

Of the 80 participants randomized, 69% completed the post-treatment assessment and 74% completed the 12 week assessment. In the CBT condition, 30/40 participants completed at least six of the eight sessions and were considered "completers" as these six sessions covered the core treatment content (the last two focused on relapse prevention). Of these 30 participants, 15 (50%) returned all 7 weekly interoceptive exposure exercise logs to the study investigators. An

additional two participants returned five weeks worth of interoceptive exposure exercise logs, while a further three participants returned one week of logs. In addition, of these 30 participants, five did not return post-treatment measures and two surveys were lost in the mail. Reasons for drop out from the CBT condition were: No time for treatment (*n*=4), moved out of the area of licensed practice (*n*=2), no reason provided (*n*=3), and treatment was a bad fit (*n*=1). There was not a significant difference (χ^2=3.52) between the number of participants who qualified as "completers" in the treatment condition (n=30 or 75%, see above) and the number of participants who completed the waiting list condition (n=32 or 80%; see Figure 1).

Normality and Descriptive Statistics

We assessed normality of variables using a visual inspection of the shape of the distribution and an interpretation of the SPSS skew statistic using a threshold of ±1.00 as indicative of a departure from normality (Meyers, Gamst, & Guarino, 2006). The MPSS data was very positively skewed so we $\log_{10}$ transformed MPSS scores before analysis. Table 2 presents mean scores for each outcome variable across all three time points. Mean ASI-3 scores at pre-treatment were as high as those found in individuals with PD (*M* = 32.6, *SD* = 14.3) and SP (*M* = 31.4, *SD* = 11.9; Reiss et al., 2008). Mean LSAS scores were higher than those found in non-anxious controls (*M* = 13.68, *SD* = 9.91), but somewhat less than clinical samples (*M* = 74.41, *SD* = 20.40), falling in the "moderate to marked SP" range (Heimberg & Holaway, 2007). PSWQ mean scores were slightly less than those in clinical samples (*M* = 68.1, *SD* = 7.33; Fresco, Mennin, Heimberg, & Turk, 2003). Symptom severity endorsement at an item level on the PAQ resembled that of clinical panickers (e.g., chest pain or discomfort *M* = 1.64, *SD* = 1.23 in our sample and *M* = 1.57, *SD* not reported, in a clinical sample; Norton et al., 2008). The mean score on the MPSS did not reach the clinical cut-off for community (≥46) samples (Falsetti et al.,

1993). Mean DASS-21 Depression scores were lower than among individuals with MDD (*M* = 29.96, *SD* = 9.18) but still within the "moderate severity" range and higher than among individuals with PD (*M* = 12.75, *SD* = 10.15) or SP (*M* = 13.19, *SD* = 9.28; Antony et al., 1998). Finally, mean scores on the NEOFFI-N subscale were substantially higher than those found amongst non-clinical populations (*M* =19.5, *SD* = 8.6; Egan, Deary, & Auston, 2000).

Table 2 also presents correlations between study variables at pre-treatment. The ASI-3 was correlated with PAQ, LSAS, and DASS-21 Depression scores, but unexpectedly not with PSWQ or MPSS scores. There was an expected amount of intercorrelation among the anxiety measures. DASS-21 Depression scores were correlated with each of the other measures. Neuroticism scores were significantly correlated with PSWQ, MPSS, LSAS, and DASS-21 Depression scores but not with PAQ or ASI-3 scores.

Multilevel Models

The coefficients for cross-level time*group interaction effects, the simple slopes separated by treatment group, and effect size ($d_{GMA\text{-}raw}$) values are presented in Table 3. Both random-slopes linear and fixed-slopes quadratic models were tested; we reported results for fixed-slopes quadratic models only if the cross-level interaction between treatment group and the quadratic polynomial contrasts was significant. When significant cross-level interactions were found, data were plotted graphically to aid interpretation (see Figures 2-4).

Anxiety sensitivity. The change in ASI-3 scores over time was best described as quadratic. A quadratic model with fixed slopes and random intercepts revealed a significant quadratic time*group interaction (see Figure 2). While the WLC condition showed a smaller but significant linear decrease in AS over time, the CBT condition showed a significant quadratic change, with a linear reduction in AS sharper than that of the WLC group during the first eight

weeks of treatment that was maintained in the subsequent four weeks. We also examined separate models for each of the AS subscales (Table 3). Overall, the pattern of results for subscales was generally similar to results found with the full ASI-3; however, the effect sizes for the physical and social concerns subscales were moderate-large and only small-moderate for cognitive concerns.

Neuroticism. Both the intervention and the control group experienced slight decreases in Neuroticism over time. However, there was a significant quadratic interaction (see Figure 3d). The control participants experienced a very slight linear decline over time. In contrast, the CBT participants had a quadratic pattern, with a sharper decline from baseline to 8-weeks compared to control; however, by 12-weeks they were roughly equivalent to the control group. Overall then, the groups did not differ in the amount of linear pre-post change in Neuroticism, though the pattern of change over time did vary across groups.

Panic symptoms. There was a significant linear time*group interaction (see Figure 3a) predicting PAQ panic symptoms. This relation was not better explained by a quadratic relation and the effect size for group differences in linear pre-post change over time was medium-large ($d_{GMA\text{-}raw}$ = .74) in favour of the CBT condition.

Social phobia symptoms. There was a significant linear time*group interaction predicting SP symptoms (see Figure 3b). This relation was not better explained by a quadratic relation and the effect size for group differences in linear pre-post change over time was small ($d_{GMA\text{-}raw}$ = .34) in favour of the CBT condition.

Posttraumatic stress symptoms. PTSD symptoms were predicted by significant linear and quadratic time*group interactions (see Figure 3c), and the effect size for group differences in linear pre-post change over time was small ($d_{GMA\text{-}raw}$ = .39). PTSD symptoms in the CBT group

decreased steadily over time, with a sharper decrease from baseline to 8-weeks, than from 8-weeks to 12-weeks. In contrast, there was no significant change in the WLC group's PTSD symptoms over time.

Generalized anxiety symptoms. While there was an overall main effect of time, showing that generalized anxiety significantly decreased over time for both the CBT and WLC conditions, no significant linear or quadratic time*group interactions emerged. This suggests generalized anxiety did not improve due to treatment.

Depression. There was an overall main effect of time on depression symptoms revealing a decrease in depressive symptoms over time in both the CBT and WLC groups. However, no significant linear or quadratic time*group interactions emerged, suggesting that the intervention did not reduce symptoms of depression to a greater extent than the passing of time.

Clinical Significance

There was a significant linear time*group interaction in predicting functional disability (see Figure 4a). There was a significant linear reduction in functional disability over time for both the CBT and WLC groups; however, the effect size for group differences in linear pre-post change over time was large ($d_{GMA\text{-}raw}$ = .85) in favour of the CBT condition.

There was a significant linear time*group interaction in predicting number of log transformed SCID diagnoses (β = -0.17, t_{78} = -3.15, p < .01; see Figure 4b). Simple slopes showed a significant linear reduction in number of diagnoses over time for both groups with the CBT group showing a steeper rate of change (β = -0.26, t_{39} = -7.20, p < .001) than the WLC (β = -0.10, t_{39} = -2.68, p < .05). The effect size for group differences in linear pre-post change over time was large ($d_{GMA\text{-}raw}$ = .85).

We also identified the proportion of participants in each condition whose post-treatment ASI-3 scores were closer to the normal population mean than to the dysfunctional population mean and who evidenced reliable change according to Jacobson and Truax's (1991) formula. Of the participants in the CBT group who completed the ASI-3 at the 12 week assessment, 45.8% met criteria for recovery (i.e., evidence of reliable change and ASI-3 score closer to normal population mean), another 16.7% showed clinically significant improvement (i.e., evidence of reliable change), 33.3% were unchanged, and 4.2% had deteriorated. In contrast, of the participants in the WLC group who completed the ASI-3 at the 12 week assessment, 17.6% were recovered, another 8.8% were improved, 73.5% were unchanged, and none had deteriorated.

Finally, participants in the CBT group also completed a follow-up questionnaire at 20 weeks. In this analysis, we were primarily interested in changes from 12 to 20 weeks. To analyze this data, we created a dataset that included only participants in the CBT group with values for outcomes at all four timepoints (baseline, 8 weeks, 12 weeks, and 20 weeks). Time was modeled as categorical using a dummy-coded time variable with week 20 as the reference group. Only the dummy-coded time variable was entered in as a predictor of outcomes. Effect size was calculated by dividing the mean difference in outcomes from 12 to 20 weeks by the baseline standard deviation (Feingold, 2009). Findings showed no difference between 12 week and 20 week follow-up scores for the CBT group for each of AS (t_{117} = -0.10, p = 0.92, d = 0.02), neuroticism (t_{117} = 1.47, p = 0.14, d = 0.15), panic (t_{117} = 0.71, p = 0.48, d = 0.18), SP (t_{118} = 1.11, p = 0.27, d = 0.17), posttraumatic stress (t_{118} = 1.77, p = 0.08, d = 0.23), generalized anxiety (t_{117} = 1.25, p = 0.74, d = 0.17), and depressive symptoms (t_{117} = 0.33, p = 0.74, d = 0.05), or functional disability (t_{120} = 0.23, p = 0.82, d = 0.05).

Mediation Analyses

Given our hypotheses about the role of AS reductions in decreasing mental health symptoms, we also tested for mediated moderation. Specifically, we investigated whether the linear time*group and quadratic time*group interactions predict reduced AS, which in turn predicts decreased mental health symptoms. The indirect effect is defined by the product of the a-path (predictor to mediator) and the b-path (mediator to outcome). If the indirect effect is statistically significant, mediation has occurred. The statistical significance of the indirect effect was calculated using a Monte Carlo method with 20,000 resamples (Preacher & Selig, 2012). The effect size of the indirect effect was calculated by taking a ratio of the indirect effect to the direct effect, ab / (ab + c') (Preacher & Kelly, 2011). The a-paths were calculated for both linear and quadratic interaction terms. A model with time (linear and quadratic), condition, and time*condition (linear and quadratic) interaction terms as predictors was evaluated, with the time*condition interaction terms producing the estimates for the a-paths. When examining AS as a predictor of outcomes for the b-paths, AS was entered as a level 1 time-varying covariate. Table 4 displays all relevant paths, a 95% CI for the indirect effect, and an effect size for the indirect effect.[2] Moreover, to show that this mechanism is specific to AS and not subsumed by changes in Neuroticism, we entered Neuroticism in as a level 1 time-varying covariate in these mediation analyses. Finally, we only tested indirect effects when the time*group interaction had a significant total effect on outcome variables in Table 3, before controlling for Neuroticism (i.e., PAQ, LSAS, MPSS, & SDS).

[2] In some analyses, the c'-path and ab-path can have opposite signs. This is known as inconsistent mediation (see Mackinnon, Fairchild, & Fritz, 2007). In this case, the mediator is acting as a suppressor variable, and the direct effect (c-path) can sometimes be larger than the total effect (c'-path) contrary to typical mediation models. In these cases, we took the absolute value of c' before calculating effect size to avoid proportions greater than 1.0.

The quadratic relationship for AS remained significant once controlling for Neuroticism (a-paths in Table 4). AS was also a significant predictor of all outcomes over-and above Neuroticism (b-paths in Table 4). Once controlling for Neuroticism, the linear time*group interaction still had a total effect on panic symptoms and functional disability; however, the linear time*group interaction no longer predicted social phobia nor post-traumatic stress symptoms (c-paths in Table 4)[3].

The indirect effects for the linear time*group interaction show that the treatment leads to more pre-post change in AS, which in turn leads to changes in specific outcomes. Table 4 shows that the linear indirect effects were significant for panic symptoms, social phobia symptoms, and functional disability, but not post-traumatic stress symptoms. The indirect effects for the quadratic time*group interaction show that the treatment results in substantial changes in AS between pre-treatment and 8 weeks and a smaller decrease between 8 and 12 weeks while the control group has a more steady smaller linear decrease; in turn this leads to changes in outcome variables. Table 4 shows that the quadratic indirect effects were significant for panic symptoms and functional disability, but not for social phobia or post-traumatic stress symptoms.

Treatment Satisfaction and Therapeutic Alliance

All participants in the CBT condition reported that they would recommend the treatment to a friend. The mean treatment satisfaction was 8.9/10 (*SD* = 1.1). Participants' responses on the WAI-SR suggest a high degree of therapeutic alliance. The mean score on the WAI-SR was 55.48 (*SD* = 3.39) with mean scores of 18.50 (*SD* = 1.56) on the goal subscale, 19.05 (*SD* = 1.40) on the bond subscale, and 17.79 (*SD* = 1.44) on the task subscale (the maximum score for each subscale is 20). When asked what was most helpful about the treatment, participants equally

[3] In modern testing of mediation in the indirect effects framework, the total effect (c-path) does not need to be significant. As such, we can go on to test these relations further.

endorsed each of: The phone therapy sessions, the personalized support, learning strategies such as examining the evidence, the physical exercise, and the combination of reading and phone therapy. All but one participant found the treatment very convenient to engage in, particularly because they did not have to leave their home and because therapists offered flexible scheduling. Only a few concerns were reported: Wanted more sessions (*n*=7), would have preferred face-to-face treatment or to see therapist's picture (*n*=4), parts of the sessions were not relevant to the individual (*n*=2), difficulty scheduling sessions (*n*=2), and parts of the treatment manual were dry, confusing, and/or too theory-based (*n*=7).

Discussion

The present study investigated the efficacy of a telephone-based CBT intervention for high AS among a community-recruited treatment-seeking sample. The primary aim was to test the intervention's efficacy in reducing high AS as compared to a WLC, while a second objective was to investigate reductions in high AS-associated mental health symptoms as a result of the treatment and to test the role of AS as a treatment mediator. Results showed that the treatment was successful in reducing AS when compared to a WLC. In line with prior research showing the susceptibility of AS to CBT (Smits et al., 2008), AS decreased over the first 8 weeks of treatment, and these gains were maintained at 12 week and 20 week follow-ups.

In addition to the treatment effects on AS, we also found reductions in some, but not all, of the associated mental health symptom measures. Participants in the CBT group showed significantly greater reductions in panic, SP, and posttraumatic stress symptoms than participants in the WLC condition, and these gains were maintained at 12 and 20 week follow-ups. These findings were expected given previously established associations between high AS and a variety of anxiety disorders suggesting that AS might contribute to their development and maintenance

(Olatunji & Wolitzky-Taylor, 2009). Notably, the treatment resulted in the largest effect sizes for panic symptoms and only small effects with respect to SP and posttraumatic stress symptoms. These findings may reflect the trend in research showing that AS is most strongly and consistently related to panic as compared to other specific disorders (Olatunji & Wolitzky-Taylor, 2009).

Unexpectedly, depressive and generalized anxiety symptoms did not reliably decrease as a result of treatment. This may be because the intervention only led to small magnitude reductions in AS cognitive concerns. Research suggests that the AS-depression association may be specifically due to the link between AS cognitive concerns and depressive symptoms (Cox et al., 2001; Deacon et al., 2003) to the extent that AS cognitive concerns might be a "depression-specific form of anxiety sensitivity" (Taylor et al., 1996, p. 478). Similarly, the connection between AS and generalized anxiety is likely through AS cognitive concerns, possibly due to the common fear of uncontrollable psychological symptoms (e.g., worry, difficulty concentrating; Rector et al., 2007; Rodriguez et al., 2004). Thus, we may not have seen a change in depressive or generalized anxiety symptoms due to a mismatch between the content of the intervention and the nature of the relation between AS and both depression and generalized anxiety. It may be that the interoceptive exposure component of the intervention (i.e., physical exercise) did not sufficiently target AS cognitive concerns but was more relevant to AS physical concerns. A second possible reason for the lack of treatment effect on worry pertains to the lack of correlation between worry and AS at pre-treatment. While previous studies have documented a connection between AS and the PSWQ with the original ASI (Keough, Riccardi, Timpano, Mitchell, & Schmidt, 2010), we did not find a correlation between the PSWQ and ASI-3 at baseline, which

may help explain the limited change in participants' generalized anxiety with an AS focused treatment.

Mediated moderation analyses revealed that reductions in AS accounted for changes in panic, SP, and PTSD symptoms resulting from treatment. In other words, the treatment reduced AS over time, which in turn led to decreases in panic, SP, and PTSD symptoms. Again, this finding is in line with evidence that AS might mediate the outcome of disorder-specific anxiety treatments (Arch et al., 2012; Smits et al., 2004) and transdiagnostic protocols (Sauer-Zavala et al., 2012). Importantly, given the treatment did not have direct effects on generalized anxiety or depressive symptoms, we did not explore any mediation findings for these symptoms. Our findings suggest that AS reductions might account for treatment-related symptom reductions for some (e.g., panic, social phobia) but not all (e.g., generalized anxiety, depressive) anxiety treatments, and only some outcomes of transdiagnostic protocols. Alternatively, as suggested above, it may be that modifications to the treatment under investigation, for instance to the exposure component, may result in this AS-focused intervention having broader disorder-specific effects in future studies. It will be necessary for future studies to address the question of the mediating role of AS in a more rigorous manner. Thoroughly examining mediation would require repeated and frequent assessment of the mediator (AS) and outcome variables (mental health symptom measures) at multiple points (e.g., weekly) throughout treatment. Such an approach would allow for a more nuanced understanding of the changes over time in and AS and mental health symptoms. Moreover, it would allow for a time-lagged analysis of changes in AS relative to changes in outcome variables to establish the temporal relation between the two, a requirement of mediation.

Importantly, our findings also provide some evidence of the present treatment's specific relevance to AS. In comparison to neuroticism, a related personality construct that has been targeted in similar transdiagnostic interventions (Barlow et al., 2011), the present treatment showed more robust effects on AS. When neuroticism was included as a covariate in our analyses, the quadratic relations for AS remained significant. When neuroticism was added as a treatment mediator, AS remained a significant predictor of panic and SP, but not posttraumatic stress, treatment outcomes over and above neuroticism. In addition, our AS-targeted treatment showed at least as large an effect on panic symptoms as previous neuroticism-targeted interventions have shown on a self-report measure of anxiety known to tap into some panic symptoms (i.e., the Beck Anxiety Inventory; Farchione et al., 2012). Taken together, this demonstrates the value of conceptualizing AS as an important underlying contributor or shared risk factor for mental health problems and considering the value of exploring AS-targeted treatments rather than solely focusing on neuroticism-targeted interventions.

As one measure of clinical significance, results showed a greater reduction in the number of SCID diagnoses for participants in the CBT group from pre- to post-treatment than those in the WLC group. This finding suggests that symptom reductions resulting from treatment were sufficient to lead to diagnostic remission. In addition to mental health symptoms, we also considered whether participants experienced an improvement in functional disability as a result of treatment as another measure of clinical significance. Participants in the CBT group endorsed a significantly greater reduction in symptom interference in their life than those in the WLC. This suggests that symptom reductions stemming from treatment had real-life implications.

Taken together, the present findings provide support for the use of a multi-component CBT intervention including psychoeducation, cognitive restructuring, and interoceptive exposure

to reduce AS, and partial support for the efficacy of targeting AS as a transdiagnostic approach to treating some AS-relevant anxiety disorders (i.e., panic, social phobia, and posttraumatic stress). Like Barlow et al.'s (2011) Unified Protocol, by targeting AS as an underlying risk factor the present study resulted in symptom reductions across the AS-relevant anxiety disorders of panic, social phobia and PTSD. Thus, this treatment appears promising in helping a broad array of clients with various anxiety disorders. By targeting AS we may have a better ability to treat comorbid conditions that share AS as a common risk or maintenance factor. It remains an empirical question as to whether our expanded eight session treatment format was necessary, or if a briefer one to three session protocol (as implemented in prior brief AS-targeted CBT interventions; Keough & Schmidt, 2012; Watt et al., 2006) would have been sufficient with the present largely clinical sample. It is also important to note that the present sample was selected on the basis of high AS; the present study was intended to test the transdiagnostic relevance of AS-focused CBT for those with high AS. Nevertheless, future research could investigate if the treatment would have yielded similar efficacy if participants were selected on the basis of anxiety or mood disorder diagnosis irrespective of AS level. Such an investigation would allow for a more comprehensive understanding of the relevance of AS (or lack thereof) to emotional disorders.

One important consideration in the interpretation of the present findings is the comparison of the current treatment to other transdiagnostic interventions. Despite differing from the majority of transdiagnostic treatments that target patterns of thinking and behaving across disorders, our intervention approach similarly led to transdiagnostic symptom reduction by targeting an underlying mental health risk factor (i.e., AS). Our results also align with the treatment outcome of other transdiagnostic treatments that have targeted underlying mental

health risk factors (e.g., Barlow et al., 2011). As such, despite differences in approach, the empirical literature, including our findings, suggests that both types of transdiagnostic interventions are effective and practical approaches to treatment delivery.

In addition to the promise of transdiagnostic interventions, the present study adds to evidence of the efficacy of telephone service delivery (Hecker, Losee, Roberson-Nay, & Maki, 2004; Lovell et al., 2006). Despite the lack of face-to-face sessions, telephone-based interventions can still include all of the key components of CBT including psychoeducation, cognitive restructuring, and exposure. Qualitative feedback collected from study participants about the telephone-based treatment was overwhelmingly positive. The majority of participants extolled its convenience and their comfort with communicating with their therapist via telephone. Participants reported finding the phone therapy sessions helpful for their personalized support and for learning useful strategies to target anxiety. Therapeutic alliance scores were also high, in line with other telephone-based treatment studies (Lingley-Pottie & McGrath, 2006), supporting the idea that an alliance based on mutual agreement of goals and tasks and a positive bond can exist between therapist and client via this treatment delivery medium.

The present study has limitations. First, analyses relied predominantly on self-report symptom measures; participants may have under- or over-reported the severity of their symptoms. Second, due to limited resources, the telephone-administered SCIDs were not reviewed by a second, independent assessor; this may have introduced some degree of interviewer bias into the data. The standardized nature of the SCID does, however, attempt to control for interviewer bias and interviewers were blind to participant treatment group. We also did not record telephone sessions to check therapist adherence to the treatment protocol; this limitation is particularly relevant as 12 different clinicians provided treatment services. All

therapists were trained in the protocol and received weekly supervision in which adherence was discussed. Future steps with this protocol should include more rigorous fidelity checks. Third, we did not include any measure of participants' treatment adherence or compliance beyond anecdotal report from study therapists and participant return of interoceptive exposure exercise logs. Fifty percent of treatment completers returned exercise logs recording all of the required interoceptive exposures, while approximately 30% of treatment completers did not return any of these logs. Unfortunately, anecdotal report from therapists indicated that some participants completed the exercises but did not return the exercise logs, so return of logs is not an exact index of compliance with the interoceptive exposure component of treatment. Nevertheless, these numbers do indicate less than optimal compliance with the interoceptive exposure component of treatment, suggesting that the effects of treatment may be even larger than detected, if all participants had engaged in all the prescribed interoceptive exposures. More rigorous treatment adherence and compliance assessment methods should be included in future studies.

Fourth, participants' appreciation of the convenience of treatment was due in part to the flexible hours offered by therapists, including evenings and weekends. This may not accurately reflect the practicalities of real-world clinical practice and might have inflated participants' positive feedback of the intervention. Fifth, participants' prior experience with physical exercise may have affected the efficacy of the interoceptive exposure. However, the actual intensity of participants' self-reported physical exercise is difficult to gauge[1] and, as research suggests that high AS individuals tend to avoid exercise (Sabourin, Hilchey, Lefaivre, Watt, & Stewart, 2011), it is possible that reported exercise was not of sufficient intensity to illicit salient bodily arousal sensations. Moreover, the equivalent number of exercisers between groups (35% in the control

group vs. 40% in the treatment group) at pre-treatment makes it less likely that this participant factor would have differentially affected the treatment and control conditions.

A sixth limitation is the amount of missing data. Our assessment return rates were 69% at eight weeks and 74% at 12 weeks. This is a significant limitation as a large amount of data remains unaccounted for, and the relation between missing data and response to treatment remains unknown. Importantly, these rates do not exactly reflect treatment completion rates; several participants completed six to eight treatment sessions without completing post-treatment assessment measures, and there was a nation-wide postal strike during the study, both of which lowered our rate of return. As such, the low rates of return are not a direct reflection of the palatability or relevance of the intervention. We addressed missing data using a maximum likelihood approach in HLM. Nevertheless, better methods to increase (a) treatment retention and (b) assessment return rates are needed. We might have improved return rates by offering the option of completing the measures online for convenience, or by shortening the questionnaire. To improve treatment retention we might consider reducing the amount of homework participants were assigned as several participants cited lack of time as a reason for withdrawal. The amount of missing data must be considered when interpreting study findings.

Despite these limitations, the present study highlights the promise of treating AS. Future studies should explore the mechanisms of action of this intervention, as Sauer-Zavala and colleagues (2012) did for Barlow et al.'s Unified Protocol (2011), and dismantle the components of this multi-component intervention to determine its active ingredients. As a part of this endeavour, future studies should better index participants' engagement in exercise before, during, and after the treatment program. Better indexing participants' engagement in exercise will help elucidate whether the exposure or non-exposure components of treatment are largely

responsible for the treatment's effect. It might also be helpful to use the present post-hoc findings regarding treatment effects on specific AS components to improve the current intervention in the future to make it more truly transdiagnostic. Reformulating and broadening the exposure component of the treatment may allow it to impact AS cognitive concerns more strongly, as it does AS physical and social concerns. This might mean adding exposure activities or tailoring exposure to be specific to an individual's elevated AS subscale(s). For instance, hyperventilation alone or with spiral staring or a strobe light may be useful in inducing depersonalization or derealization, both feared symptoms in those with elevated AS cognitive concerns (Lickel, Nelson, Lickel, & Deacon, 2008). Strongly addressing all AS components, may extend the treatment's impact to generalized anxiety and depressive symptoms. Finally, it will be important for future research to better investigate the longer-term outcomes of this treatment and its implications for AS levels over time.

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Table 1. *Participant Characteristics*

Characteristic	Total Sample *N*=80	Waiting List *n*=40	Treatment *n*=40
Age at Pre-Treatment	*M*=36.3 (11.3)	*M*=36.5 (10.4)	*M*=36.2 (12.2)
Range	18-65 yrs	20-58 yrs	18-65 yrs
Sex	78.8% women	72.5% women	85% women
Taking Medication	37.5%	37.5%	37.5%
SSRI	17.6%	20.0%	15.0%
Benzodiazepine	12.6%	10.0%	15.0%
Other	14.3%	12.5%	15.0%
Marital Status			
Single	27.5%	35.0%	20.0%
Married/Common-Law	37.5%	27.5%	47.5%
Divorced/Separated/Widowed	13.8%	15.0%	12.5%
<6 month relationship	3.8%	7.5%	0.0%
>6 month relationship	17.5%	15.0%	20.0%
Education Level			
Some High School/High School/GED	13.8%	12.5%	20.0%
Trade School or Community College	17.6%	20%	15.0%
Some University/University	66.3%	67.5%	65.0%
Income*			
<$10,000	10.0%	12.5%	7.5%
$10,001 - $35,000	26.3%	17.5%	35.0%
$35,001 - $60,000	22.5%	27.5%	17.5%
$60,001 - $85,000	10.0%	10.0%	10.0%
> $85,001	26.3%	27.5%	25.0%
Ethnicity^			
Native Canadian	2.5%	2.5%	2.5%
Black or African Canadian	1.3%	2.5%	0.0%
Caucasian or Euro Canadian	76.3%	75.0%	77.5%
Asian or Asian Canadian	2.5%	5.0%	0.0%
Mixed	7.5%	2.5%	12.5%
Vigorous Exercise ≥ Once/Week	37.5%	35%	40%

Note. SSRI = selective serotonin reuptake inhibitor. *5% not reported. ^10% not reported.

Table 2.

Observed Means, Standard Deviations, and Correlations for Study Measures

Measure	Group	Pre *M* (*SD*)	8 Week *M* (*SD*)	12 Week *M* (*SD*)	1	2	3	4	5	6	7.
1. ASI-3	WLC	36.83 (13.67)	31.31 (13.71)	28.56 (13.16)	.90	.34**	.05	.20	.41***	.29**	.14
	CBT	39.93 (13.50)	23.57 (13.44)	24.54 (14.71)							
2. PAQ	WLC	17.85 (13.62)	10.42 (14.62)	8.09 (12.61)		.88	.35**	.25*	.16	.25*	.19
	CBT	27.60 (12.18)	10.35 (12.90)	9.32 (13.98)							
3. PSWQ	WLC	61.68 (9.32)	58.74 (9.50)	58.21 (12.12)			.90	.21	.22	.30**	.60***
	CBT	62.50 (11.27)	54.91 (11.88)	53.48 (9.35)							
4. MPSS^	WLC	27.20 (31.67)	27.03 (28.80)	22.66 (28.32)				.97	.11	.38**	.23*
	CBT	38.42 (34.64)	21.00 (25.28)	24.04 (31.19)							
5. LSAS	WLC	66.97 (28.52)	60.23 (26.23)	56.25 (30.08)					.96	.36**	.44***
	CBT	62.95 (30.01)	46.09 (29.89)	45.40 (31.83)							
6. DASS-Dep	WLC	16.10 (12.63)	12.63 (11.53)	12.65 (12.39)						.93	.61***
	CBT	19.35 (12.33)	9.91 (11.00)	11.44 (11.41)							
7. Neuroticism	WLC	30.58 (6.33)	29.47 (7.13)	27.29 (8.44)							.81
	CBT	31.90 (7.45)	25.48 (6.56)	27.08 (8.51)							

Note. Correlations between study variables were calculated using pre-treatment values. Cronbach alpha's are listed along the diagonal – they were calculated by averaging the pre-treatment, 8 week, and 12 week alpha's for each variable. WLC = waiting list control; CBT = cognitive behaviour therapy group; ASI-3 = Anxiety Sensitivity Index – 3; PAQ = Panic Attack Questionnaire; PSWQ = Penn State Worry Questionnaire; MPSS = Modified PTSD Symptom Scale; LSAS = Liebowitz Social Anxiety Scale; DASS-Dep = Depression, Anxiety, Stress Scales – Depression subscale; SDS = Sheehan Disability Scale. ^MPSS mean scores are before log transformation, however correlations were calculated using log transformed scores. *p<.05, **p<.01, ***p<.001.

Table 3.

Hierarchical Linear Modeling Results

	Emotional Disorder Symptom Measures										
	ASI-3	ASI-P	ASI-C	ASI-S	PAQ	LSAS	MPSS	PSWQ	DASS-Dep	SDS	N
						Time*group interaction effect					
Linear B	-4.35	-1.76	-1.22	-1.44	-3.39	-3.31	-6.02	-1.59	-1.34	-1.93	-0.90
Linear t_{df}	-2.98_{109}**	-3.04_{109}**	-1.87_{109}	-2.42_{109}*	-3.23_{78}**	-2.06_{77}*	-2.16_{99}*	-1.60_{78}	-1.38_{78}	-3.28_{75}**	-1.07_{110}
Quadratic B	2.57	1.03	0.85	0.69	------	------	3.62	------	------	------	1.43
Quadratic t_{df}	2.88_{109}**	2.94_{109}**	2.15*	2.23†	------	------	2.14_{99}*	------	------	------	2.79_{110}**
						Intervention group only: time simple effect					
Linear B	-7.82	-2.58	-2.46	-2.86	-6.56	-6.36	-8.40	------	------	-2.81	-2.44
Linear t_{df}	-5.80_{45}**	-5.37_{45}**	-3.87_{45}**	-5.76_{45}**	-7.23_{39}**	-4.41_{38}**	-3.93_{40}**	------	------	-5.10_{36}**	-3.18_{46}**
Quadratic B	2.89	1.07	0.96	0.82	------	------	3.26	------	------	------	1.42
Quadratic t_{df}	3.47_{45}**	3.64_{45}**	2.44_{45}*	2.68_{45}*	------	------	2.52_{40}*	------	------	------	2.95_{46}**
						Control group only: time simple effect					
Linear B	-3.39	-0.82	-1.11	-3.39	-3.15	-2.99	-2.38	------	------	-1.00	-1.55
Linear t_{df}	-4.51_{64}***	-2.36_{64}*	-3.76_{64}**	-4.51_{64}***	-4.89_{39}**	-3.52_{39}**	-1.33_{59}	------	------	-3.08_{39}**	-3.57_{64}**
Quadratic B	0.29	0.03	0.11	0.30	------	------	-0.37	------	------	------	0.04
Quadratic t_{df}	0.65_{64}	0.12_{64}	0.64_{64}	0.65_{64}	------	------	-0.34_{59}	------	------	------	0.14_{64}
						Effect Size					
$d_{GMA\text{-}raw}$	-0.77	-0.70	-0.41	-0.63	-0.74	-0.34	-0.39	------	------	-0.85	-.29

Note. When quadratic models are not reported, the quadratic slope with fixed slopes and random intercepts was not significant and so the linear model with random slopes and random intercept is reported instead. In linear models, time is coded 0, 2, 3 to account for unequal time lags. In quadratic models, orthogonal polynomial contrasts (linear = -1, 0, 1; quadratic = 1, -2, 1) are used for coding time to aid interpretability. ASI-3 = Anxiety Sensitivity Index – 3; ASI-P = Anxiety Sensitivity – Physical Concerns; ASI-C = Anxiety Sensitivity – Cognitive Concerns; ASI-S = Anxiety Sensitivity – Social Concerns; PAQ = Panic Attack Questionnaire; PSWQ = Penn State Worry Questionnaire; MPSS = Modified PTSD Symptom Scale ($\log_{10}$ transformed); LSAS = Liebowitz Social Anxiety Scale; DASS-Dep = Depression, Anxiety, Stress Scales – Depression subscale; SDS = Sheehan Disability Scale; N = Neo Five Factor Neuroticism Subscale. $d_{GMA\text{-}raw}$ can be interpreted as a Cohen's d for the difference in the linear pre-post change in outcomes between the intervention and the control group (Feingold, 2009). Effect sizes not reported when neither the linear*group nor the quadratic*group interactions were significant. †p=.06 *p<.05, **p<.01.

Table 4.

*Unstandardized Indirect Effects of the treatment*time Interaction on Outcomes through Anxiety Sensitivity controlling for Neuroticism*

Predictor	**Mediator**	**Outcome**	**a-path**	**b-path**	**c-path**	**c'-path**	**95% CI ab**	**P_M**
linear*group	ASI-3	PAQ	-3.72**	0.27**	-4.36*	-3.84*	[-2.15, -0.15]*	0.21
quad*group	ASI-3	PAQ	1.70*	0.27**	0.60	0.00	[0.09, 2.93]*	1.00
linear*group	ASI-3	LSAS	-3.72**	0.69**	-2.89	-0.26	[-5.31, -0.44]*	0.91
quad*group	ASI-3	LSAS	1.70*	0.69**	0.85	-0.28	[-0.07, 1.28]	0.81
linear*group	ASI-3	MPSS	-3.72**	0.01*	-0.07	-0.05	[-0.06, .0005]	0.32
quad*group	ASI-3	MPSS	1.70*	0.01*	0.02	0.01	[-.0002, 0.03]	0.49
linear*group	ASI-3	SDS	-3.72**	0.10**	-2.48**	-2.09**	[-0.86, -0.05]*	0.16
quad*group	ASI-3	SDS	1.70*	0.10**	-0.21	-0.38	[0.02, 0.40]*	0.31

Note. ASI-3 = Anxiety Sensitivity Index – 3; PAQ = Panic Attack Questionnaire; LSAS = Liebowitz Social Anxiety Scale; SDS = Sheehan Disability Scale. The a-path is the path from predictor to mediator. The b-path is the path from the mediator to the outcome. The c-path is total effect, and is the path from predictor to the outcome before controlling for the mediator. The c'-path is the path from predictor to outcome after controlling for the mediator. The 95% confidence interval of ab was calculated using a Monte Carlo method with 20,000 resamples. P_M is a measure of effect size that represents a ratio of the indirect effect to the direct effect, and was calculated by ab / (ab + c'). For inconsistent mediation models (ab and c' have reversed signs), we took the absolute value of c' before calculating effect sizes. The mediator (ASI-3) was entered as a Level 1 time-varying covariate. Neuroticism was entered in as a Level 1 time-varying covariate in all analyses described in Table 4. *SE* = Standard Error *p<.05 **p<.01.

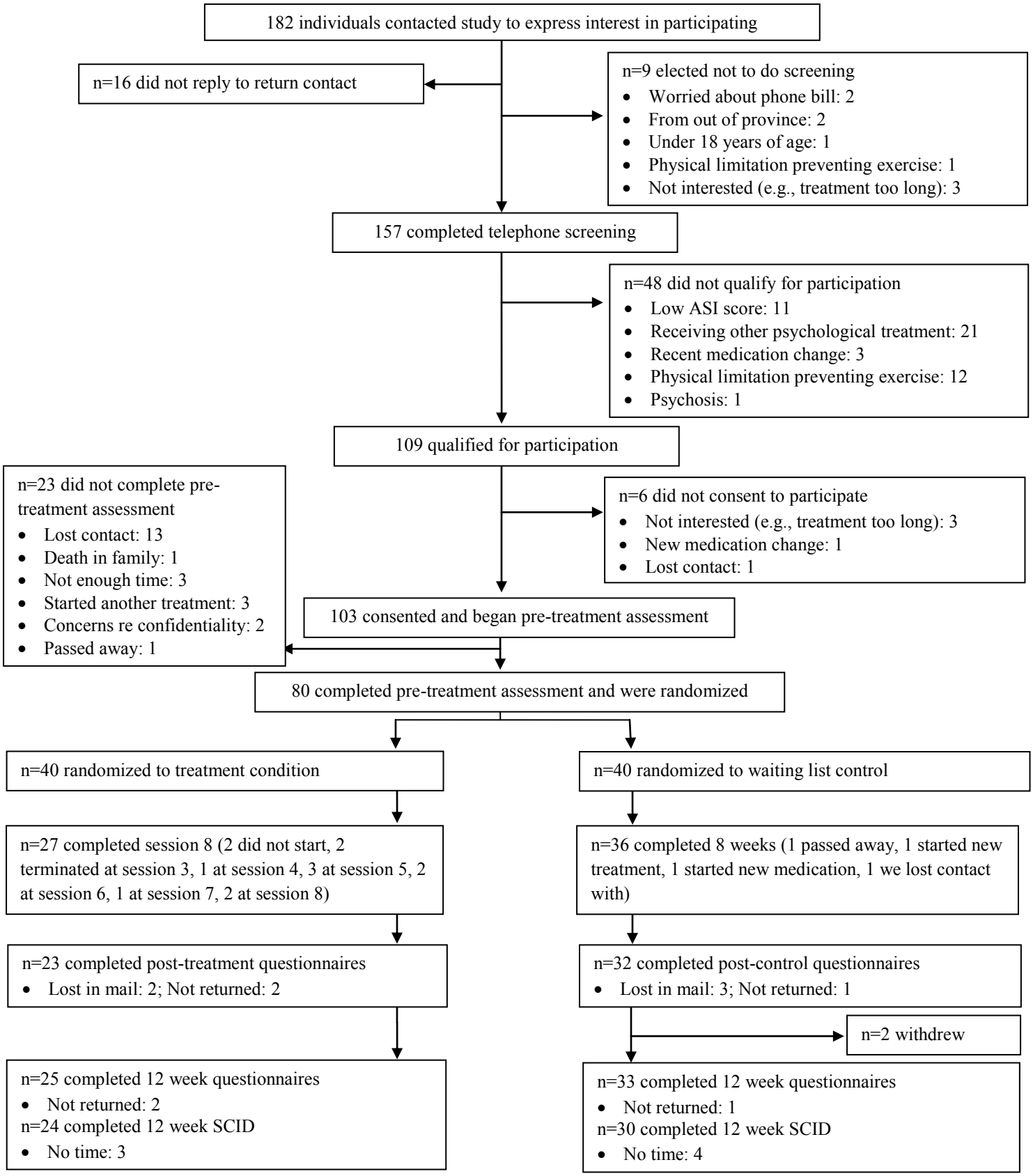

Figure 1. PRISMA diagram of participant flow through the randomized controlled trial.

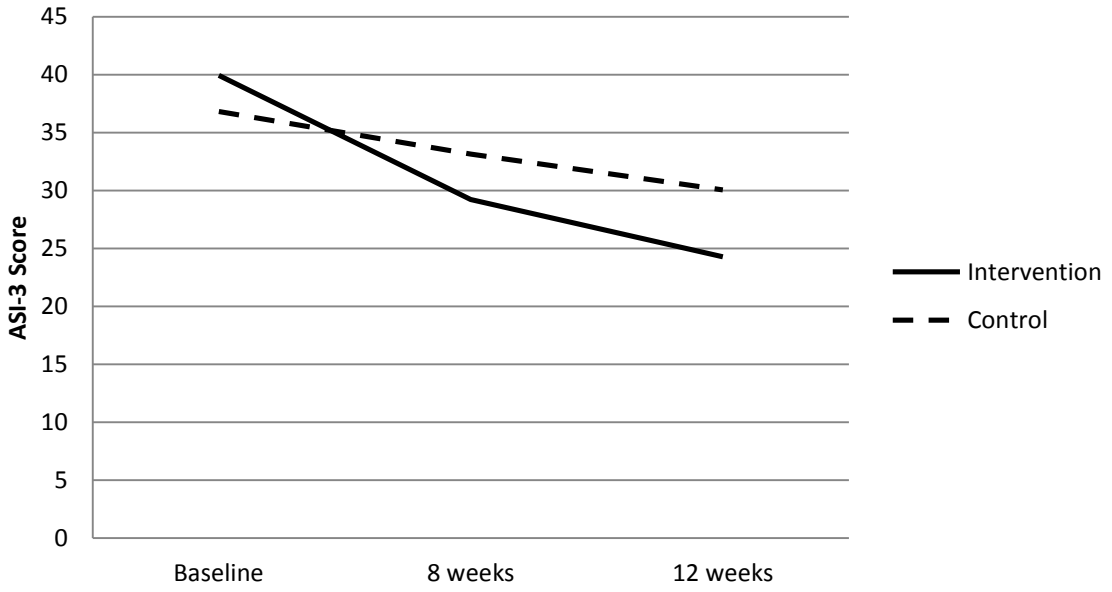

Figure 2. Group*time interaction for ASI-3.

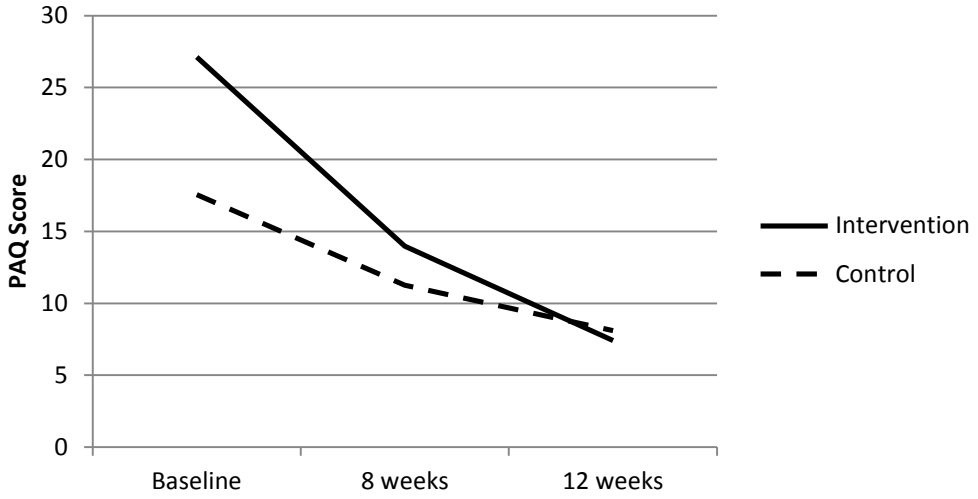

Figure 3a. Group*time interaction for PAQ

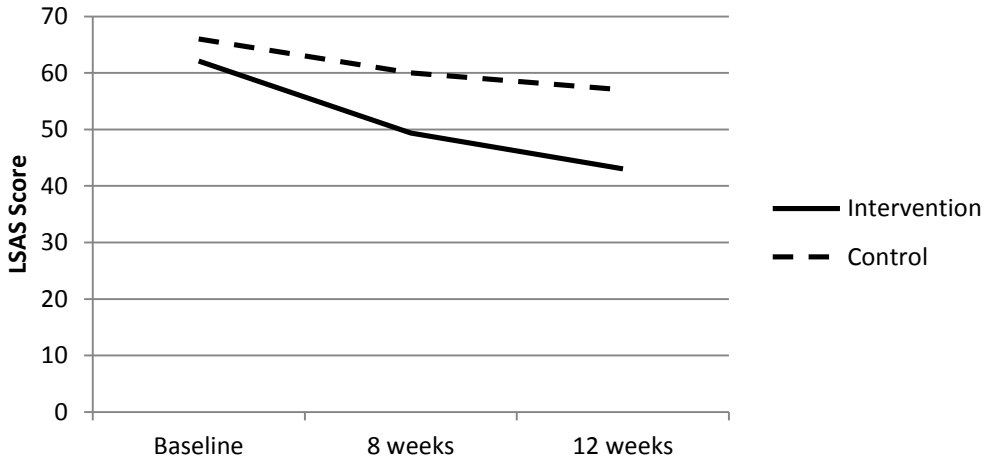

Figure 3b. Group*time interaction for LSAS.

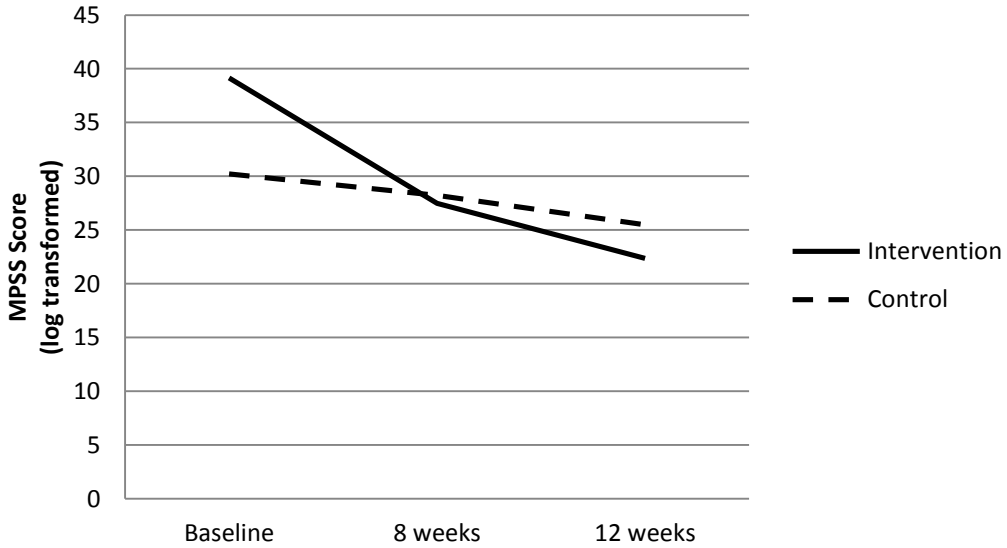

Figure 3c. Group*time interaction for MPSS.

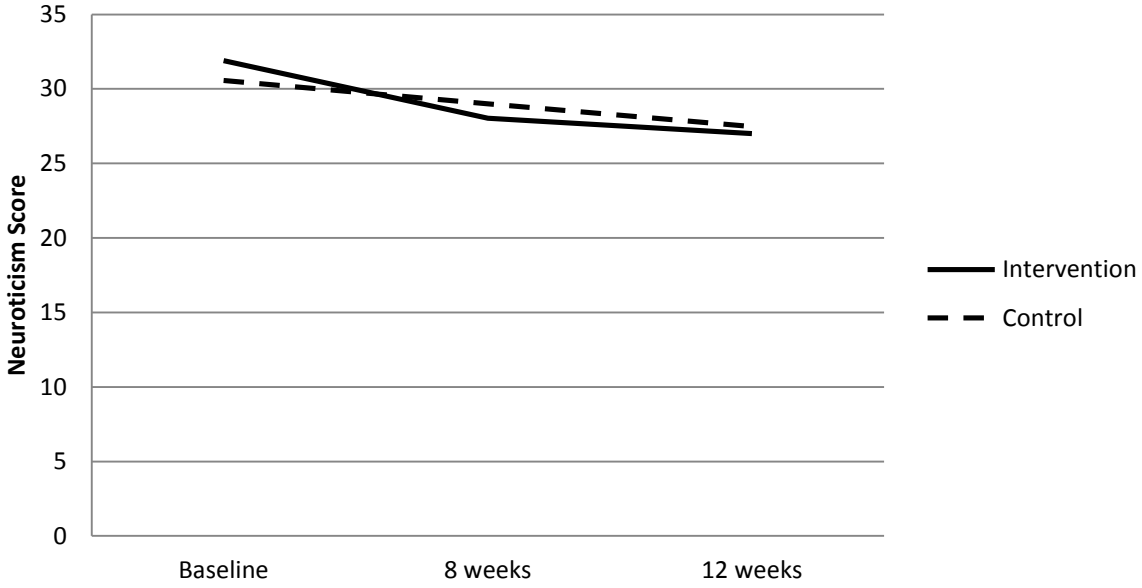

Figure 3d. Group*time interaction for Neuroticism.

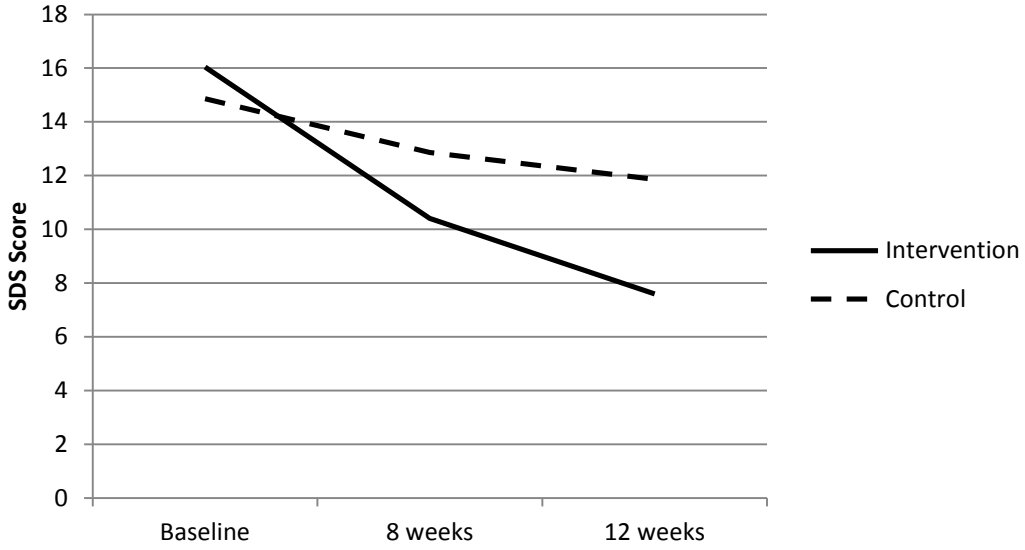

Figure 4a. Clinical significance: Group*time interaction for SDS.

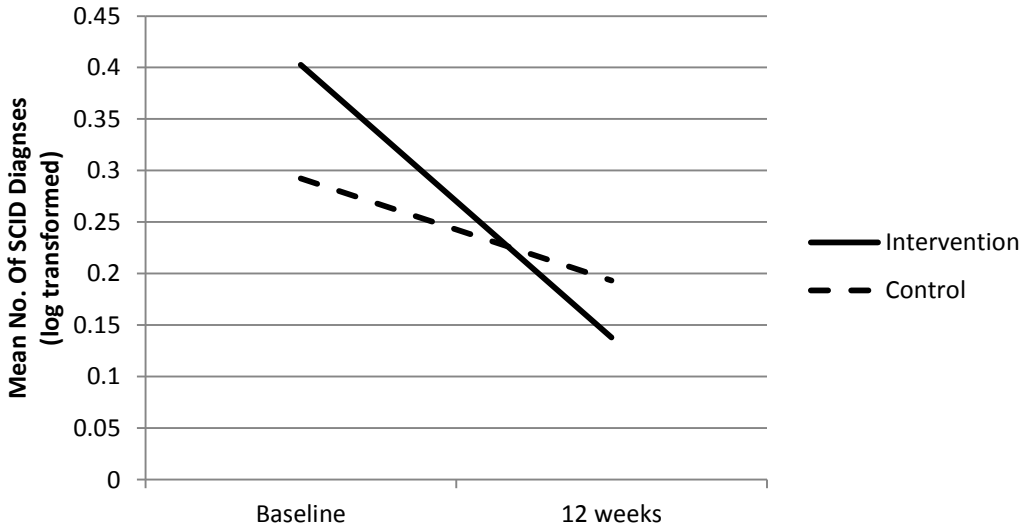

Figure 4b. Clinical significance: Group*time interaction for number of SCID diagnoses.

Supplemental Table 1. *Participants' Current Primary and Comorbid DSM-IV Diagnoses*

Diagnosis	Total Sample *N*=80	Waiting List *n*=40	Treatment *n*=40
Anxiety Disorders			
PD	17.5%	15.0%	20.0%
PD with Agoraphobia	12.5%	12.5%	12.5%
Agoraphobia	6.3%	5.0%	7.5%
Social Phobia	26.3%	25.0%	27.5%
Specific Phobia	3.8%	0.0%	7.5%
GAD	25.0%	15.0%	35.0%
PTSD	5.0%	0.0%	10.0%
OCD	5.0%	2.5%	7.5%
ADNOS	8.8%	7.5%	10.0%
Mood Disorders			
MDD	11.3%	7.5%	15.0%
Dysthymia	5.0%	7.5%	2.5%
Bipolar Disorder	1.3%	0.0%	2.5%
Cyclothymia	1.3%	2.5%	0.0%
Substance Use Disorders	3.8%	0.0%	7.5%
Somatoform Disorders			
Hypochondriasis	3.8%	5.0%	2.5%
Pain Disorder	1.3%	2.5%	0.0%
Eating Disorders	1.3%	0.0%	2.5%
Adjustment Disorder	5.0%	5.0%	5.0%
No Diagnosis	16.3%	22.5%	10.0%
Partial/Full Remission^			
Anxiety Disorder	16.3%/7.5%	5.0%/7.5%	27.5%/7.5%
Mood Disorder	15.0%/17.5%	10.0%/17.5%	20.0%/17.5%
Substance Use Disorder	2.5%/23.8%	2.5%/25.0%	2.5%/22.5%
Eating Disorder	3.8%/2.5%	5.0%/2.5%	2.5%/2.5%

Note. PD = Panic Disorder; GAD = Generalized Anxiety Disorder; PTSD = Posttraumatic Stress Disorder; OCD = Obsessive-Compulsive Disorder; ADNOS = Anxiety Disorder Not Otherwise Specified; MDD = Major Depressive Disorder. ^Data presented here reflect the percentage of participants reporting a history of a DSM-IV disorder at pre-treatment (i.e., if they met diagnostic criteria in their lifetime but did not meet criteria at pre-treatment).