Published in final edited form as:
Behav Ther. 2013 September ; 44(3): 417–431. doi:10.1016/j.beth.2013.03.006.

Anxiety Sensitivity and Interoceptive Exposure: A Transdiagnostic Construct and Change Strategy

James F. Boswell, **Todd J. Farchione**, **Shannon Sauer-Zavala**, **Heather W. Murray**, **Meghan R. Fortune**, and **David H. Barlow**
Boston University

Abstract

Recent findings support the relevance of anxiety sensitivity (AS) and interoceptive exposure (IE) across emotional disorders. This study (a) evaluated levels of AS across different anxiety disorders, (b) examined change in AS over the course of transdiagnostic psychological intervention, and its relationship with outcome, and (c) described the implementation of IE to address AS with patients with different anxiety disorders. Participants (N = 54) were patients who received treatment with the Unified Protocol for Transdiagnostic Treatment of Emotional Disorders (UP) in two consecutive treatment trials. Participants completed a measure of AS at pre- and posttreatment, and multiple occasions during treatment. Symptom severity was assessed at pre- and posttreatment, and clinical information related to physical symptoms and IE were collected as part of routine clinical practice. Elevated AS was observed at pretreatment across diagnoses and decreases in AS were observed from pre- to posttreatment. Similar changes occurred across the diagnostic categories, notably coinciding with the introduction of IE. Change in AS was correlated with reduced symptom levels at posttreatment and 6-month follow-up. Patients with different anxiety disorders endorsed similar physical symptoms and practiced similar IE exercises with similar effects. Results provide preliminary support for the usefulness of IE as a treatment strategy across the spectrum of anxiety disorders, and additional support for the transdiagnostic relevance of AS.

Keywords

anxiety sensitivity; interoceptive exposure; transdiagnostic treatment; cognitive-behavioral therapy

A variety of constructs have been described as transdiagnostic processes that may be important to the development and maintenance of more than one mental disorder. Several transdiagnostic treatments exist and are postulated to address such processes (e.g., Barlow et al., 2011; Fairburn et al., 2009; Norton & Barrera, 2012). Specifically, these interventions are designed to target a range of disorders by treating common underlying factors hypothesized to contribute to their development and maintenance, rather than focusing primarily on disorder-specific symptoms (e.g., worry, panic attacks, binge eating). Furthermore, the increased focus on transdiagnostic interventions and processes has been driven by the realization that the abundance of increasingly specific treatment manuals, many of which have only minor and somewhat trivial variations in treatment procedures, has had the paradoxical effects of increased burden on practicing clinicians and significant strain

Address correspondence to: James F. Boswell, 648 Beacon St., 6th floor, Department of Psychology, Boston University, Boston, MA 02216; jboswell@bu.edu.

on transportability and dissemination (McHugh & Barlow, 2010). A primary advantage of transdiagnostic treatments is their potential for decreasing this burden on clinicians.

The Unified Protocol for Transdiagnostic Treatment of Emotional Disorders (UP; Barlow et al., 2011) represents one such transdiagnostic treatment approach. The UP was designed to address the heightened tendency to experience negative emotions as well as the tendency to react to such experiences with distress and anxiety (i.e., neuroticism, see Barlow, Sauer-Zavala, Carl, Ellard, & Bullis, in preparation). The UP is comprised of eight modules (five are considered "core") that include motivational enhancement (Module 1), psychoeducation regarding the function of emotions (Module 2), development of present-focused, nonjudgmental awareness (Module 3, core), cognitive flexibility (Module 4, core), attenuation of emotional and behavioral avoidance (Module 5, core), increased tolerance of physical sensations (Module 6, core), situational emotion exposures (Module 7, core) and, finally, relapse prevention (Module 8). There is growing evidence to support the UP's efficacy in reducing anxiety and mood symptoms (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010; Farchione et al., 2012), as well as addressing transdiagnostic processes such as trait anxiety/ neuroticism (Carl et al., under review), intolerance of uncertainty (Boswell, Thompson-Hollands, Farchione, & Barlow, 2013), and deficits in emotion regulation (Sauer-Zavala et al., 2012).

One core component of the UP that has yet to be evaluated as a transdiagnostic intervention strategy is the inclusion of a module to address intolerance of the physical sensations that signify an emotional state through the use of interoceptive exposure (IE). Traditionally, IE has been used as a cognitive-behavioral therapy (CBT) strategy to target sensitivity toward physical sensations of anxiety and fear seen in panic disorder (PD). IE involves repeatedly inducing the physical sensations associated with anxiety and fear (e.g., shortness of breath, heart palpitations, dizziness, muscle tension), with the goal of promoting increased tolerance and reducing distress associated with these symptoms (Craske & Barlow, 2007). Some common examples of IE exercises (see Meuret, Ritz, Wilhelm, & Roth, 2005) are included in Table 1. IE has been a core component of treatments that have been shown to effectively reduce panic attack frequency and the fear of physical sensations that occurs as a primary feature of PD (e.g., Barlow, Gorman, Shear, & Woods, 2000; Craske et al., 1997). IE is, thus, regarded as an essential component of empirically supported treatments for PD.

In the context of the UP, IE has the potential to be used as a *component* in the treatment of a range of emotional disorders, rather than continue to be narrowly applied to PD. For example, in support of this position, Wald and colleagues have noted that many patients with posttraumatic stress disorder (PTSD) are unable to experience the full benefit of trauma-related imaginal exposures due to an inability to tolerate the resulting increases in physiological arousal (Wald, 2008; Wald & Taylor, 2010; Wald, Taylor, Chiri, & Sica, 2010). They describe several case studies in which four sessions of IE were conducted with patients prior to trauma-related prolonged exposures that typically characterize PTSD treatment. Results showed significant reductions in PTSD symptoms and increases in trauma-related memories were evidenced following the IE sessions.

Heightened physiological arousal is a core component of many anxiety disorders (Barlow, 2002), suggesting that IE could play a beneficial adjunctive role in their treatment; furthermore, cued panic attacks are common in social phobia (SOC), obsessive-compulsive disorder (OCD), and generalized anxiety disorder (GAD) (Baillie & Rapee, 2005; Craske et al., 2010; Goodwin & Hamilton, 2001; Reed & Wittchen, 1998). Similar to the observations of Wald and colleagues, increased sensitivity to physiological cues of anxiety may interfere with imaginal or in vivo exposures for SOC, OCD, and GAD. Furthermore, many individuals with SOC report being concerned about the implications of appearing anxious

and are hypervigilant to changes in physiology (e.g., increased temperature, sweating, turning red; Hope, Heimberg, & Turk, 2010). Additionally, muscle tension is an important diagnostic feature of GAD (American Psychiatric Association, 2000) that contributes to the intensity of worry experienced (Borkovec, Grayson, & Cooper, 1978), and reduced reactivity to muscle tension could lead to fewer worry episodes. Despite its potential clinical utility, we were unable to find any published studies that examined the role of IE in these commonly diagnosed anxiety disorders (with the exception of PD and, more recently, PTSD), and this appears to be an important gap in the literature that is in need of investigation.

IE is typically used, at least in part, to address the hypothesized transdiagnostic construct of anxiety sensitivity. Anxiety sensitivity has been conceptualized as a trait like dispositional predicate (Reiss, Peterson, Gursky, & McNally, 1986), as well as a vulnerability, that may lead individuals to associate panic attacks with interoceptive and exteroceptive conditioned stimuli (Mineka & Zinbarg, 2006). The literature suggests that individuals who report higher levels of anxiety sensitivity are at greater general risk for developing any anxiety disorder (Baillie & Rapee, 2005; McNally, 1996; Reiss, 1991). In a large prospective study, Schmidt, Zvolensky, and Maner (2008) recently demonstrated that anxiety sensitivity acts as a vulnerability factor in the pathogenesis of Axis I diagnoses. Furthermore, high levels of anxiety sensitivity have been demonstrated across anxiety disorder diagnoses, not just in PD (Naragon-Gainey, 2010). With the exception of specific phobia, Taylor, Koch, and McNally (1992) found significant differences in AS severity between “normal controls” and individuals who were diagnosed with heterogeneous anxiety disorders. A hallmark feature of anxiety disorders is increased reactivity to physiological sensations (Brown & Barlow, 2009), often resulting in maladaptive strategies to reduce such sensations. The treatment aims of IE, repeated provocation of somatic sensations across the anxiety disorders, are thought to increase tolerance of such sensations and accompanying emotional states as well as to decrease reliance on maladaptive coping strategies (i.e., avoidance, checking, overpreparing). Although IE may represent only one of several potentially useful strategies for facilitating these processes, its apparent success with PD indicates that this strategy may hold promise in the treatment of other anxiety disorders.

Several potential mechanisms have been postulated regarding the effects of IE on anxiety sensitivity. For example, contemporary learning theory suggests that IE leads to reduced anxiety through the extinction of conditioned associations between previously neutral physical sensations and frightening experiences, such as an unexpected panic attack (Bouton, 2002; Bouton, Mineka, & Barlow, 2001; for additional discussions on the subject of Pavlovian interoceptive conditioning see McNally, 1990, and Reiss, 1987). Inducing physical symptoms that do not escalate to panic attacks and observing that such physical symptoms are not harmful will eventually result in the extinction of the acquired fear response that has developed from this pairing. A potential cognitive mechanism of action for IE has also been posited (Beck & Shipherd, 1997; Clark, 1986), which suggests that through repeated exposure to feared sensations in the absence of feared consequences, new information is incorporated that disconfirms irrational beliefs about these sensations and their consequences (Salkovskis, Hackman, Wells, Gelder, & Clark, 2007). Furthermore, enhancing one’s ability to cope with anxiety-related sensations may lead to increased self-efficacy (Bandura, 1983). Finally, increased emotional acceptance through learning to tolerate sensations without efforts to change or control them has also been identified as a potential mechanism of IE (Hayes, 2002). Although there is some disagreement regarding the primary mechanism of IE, the ideas described above are not altogether incompatible. Each fosters the notion that IE promotes tolerance of arousal-related sensations and opportunities for new learning.

SPECIFIC AIMS AND HYPOTHESES

This paper had several aims, which we pursued by combining both quantitative and qualitative methods. Broadly, we were interested in providing preliminary evidence for the impact of the IE module of the UP in a sample of patients presenting with heterogeneous anxiety disorder diagnoses in two concurrent treatment trials.

First, in an attempt to replicate previous findings demonstrating that high levels of anxiety sensitivity are present in anxiety disorders other than PD (Naragon-Gainey, 2010; Taylor et al., 1992), we calculated and compared pretreatment levels of anxiety sensitivity in different anxiety disorders. Results from previous research have varied in regard to which specific anxiety disorder(s) exhibited the highest level of anxiety sensitivity relative to other disorders, further bringing into question the diagnostic specificity of this construct. As such, we hypothesized that each of the four primary diagnostic groups represented in the two trials (PD, SOC, GAD, and OCD) would display elevated yet comparable levels of anxiety sensitivity at pretreatment.

Second, we examined patterns of change in anxiety sensitivity over the course of treatment, and tested whether differences emerged in relation to the principal anxiety disorder diagnosis. We hypothesized that a significant decrease in anxiety sensitivity would be observed between pre- and posttreatment, and that such a decrease would not be specific to diagnosis. That is, individuals with different principal diagnoses would evidence similar temporal patterns of change. In addition, although largely exploratory, we predicted that the greatest magnitude of change in anxiety sensitivity levels would coincide with the introduction of the IE module (typically occurring between Sessions 7 and 10). We also predicted that change in anxiety sensitivity would be associated with lower levels of symptom severity at posttreatment and 6-month follow-up. To our knowledge, this is the first study to concurrently examine patterns of change in anxiety sensitivity in association with IE across different anxiety disorders in a single treatment. In addition to these quantitative aims, we were also interested in presenting qualitative, clinical data regarding the implementation of IE with patients presenting with each of the four principal diagnostic groups represented in the two trials (PD, SOC, GAD, and OCD), in order to demonstrate feasibility and treatment process.

Method

PARTICIPANTS

This study's sample included adults seeking treatment at an urban mental health center for anxiety and mood disorders, who were recruited to participate in one of two consecutive treatment outcome studies examining the efficacy of the UP. Inclusion/ exclusion criteria were consistent across the two studies. To be eligible, participants needed to be 18 years or older in age, be fluent in English, be able to attend all treatment sessions and assessments, and be able to provide informed consent. Participants also needed to present with a principal or co-principal (most interfering and severe) current anxiety disorder diagnosis: panic disorder with or without agoraphobia (PD), social phobia (SOC), obsessive-compulsive disorder (OCD), generalized anxiety disorder (GAD), posttraumatic stress disorder (PTSD), or anxiety disorder NOS. Individuals with a comorbid or co-principal unipolar depressive disorder were also eligible (e.g., major depressive disorder, dysthymic disorder, or depressive disorder NOS). Participants were excluded if they met criteria for a current substance use disorder and if they received a prior course of CBT within the past 5 years (see Ellard et al., 2010, and Farchione et al., 2012, for more detailed information regarding inclusion/exclusion criteria).

A portion of the primary outcome results from the earlier open trial were reported by Ellard et al. (2010; presented as "Study 2"),[1] and the primary outcome results for the more recent randomized controlled trial were reported in Farchione et al. (2012). We chose to combine cases from these two consecutive trials in order to increase the power of our analysis and generalizability of our results. The validity of this approach was supported by several factors. The studies were conducted in the same clinic and recruited from the same community population; the UP was administered in both trials by the same pool of therapists; studies utilized the same baseline assessment procedures. The only notable methodological difference was that in Farchione et al.'s trial, patients were initially randomized to either immediate treatment with the UP or a delayed treatment wait-list condition. As reported by Farchione et al., no pretreatment differences in demographic variables or severity were observed between the two conditions, and the magnitude of change on study variables during active treatment did not differ between the conditions. Thus, Farchione et al. reported both between-condition and combined, within-treatment results for the trial, and the combined sample was used in the current study. In order for a patient to be included in the present analysis, they needed to have completed a baseline diagnostic assessment, entered treatment (e.g., completed at least one session), and provided data on the relevant study variables at a minimum of one assessment point. A total of 18 (out of 18) patients met these criteria from the earlier open trial, and a total of 36 (out of 37) patients (combined sample) met these criteria from the more recent randomized controlled trial.

A series of ANOVA and chi-square analyses were conducted to help determine the appropriateness of combining data collected in these separate UP trials. Comparisons indicated nonsignificant differences in demographic characteristics, such as age $F(1, 53) = 0.06$, $p = .80$, gender $\chi^2 (1) = 0.96$, $p = .33$, race $\chi^2 (3) = 6.35$, $p = .10$, and marital status $\chi^2 (3) = 4.83$, $p = .19$. Nonsignificant differences were also observed for pre-treatment principal diagnosis CSR $F(1, 53) = 1.31$, $p = .26$, pretreatment panic disorder severity scale-self report (PDSS-SR) $F(1, 53) = 0.42$, $p = .52$, pretreatment anxiety sensitivity index (ASI) Total score $F(1, 53) = 0.67$, $p = .42$, pretreatment ASI Physical score $F(1, 53) = 0.69$, $p = .41$, pretreatment ASI Cognitive score $F(1, 53) = 0.15$, $p = .70$, and pretreatment ASI Social score $F(1, 53) = 0.47$, $p = .50$. Similarly, no differences emerged between the trials in posttreatment principal diagnosis CSR, $F(1, 53) = 0.54$, $p = .47$, post-treatment PDSS-SR score $F(1, 53) = 0.71$, $p = .40$, or likelihood of completer status: $\chi^2 (1) = 0.99$, $p = .32$. In addition, completer status was unrelated to initial readiness to change score (measured by the University of Rhode Island Change Assessment, URICA; McConnaughy et al., 1983), pretreatment PDSS-SR score, pretreatment ASI Total score, or the above demographic variables.

Consequently, there was sufficient evidence to move forward with the combined data set. In this sample of 54 patients, the average age was 30.00 years ($SD = 9.05$, range = 18 to 52); 57.4% of the sample were women. The majority of the sample was White/Caucasian (90.7%), followed by Asian-American (3.7%), "other" (3.7%), and African-American (1.9%). The majority of the sample was single and never married (74.1%), followed by married (14.8%), cohabitating (7.4%), and widowed or divorced (3.7%). A total of 12 (23.6%) patients had a principal diagnosis of PD, 13 (23.6%) a principal diagnosis of SOC, 12 (21.8%) a principal diagnosis of OCD, 11 (20.0%) a principal diagnosis of GAD, and 6 (10.9%) an "other" principal diagnosis (PTSD [$n = 1$], anxiety disorder NOS [$n = 2$], or co-

[1]Ellard et al. (2010) report the results of two consecutive open trials examining the efficacy of the UP (presented as "Study 1" and "Study 2"). As described in their article, modifications were made to the UP procedures between the first and second study. The version of the UP that was examined in Study 2 was identical to the version that was investigated in Farchione et al.'s (2012) RCT. The present study included participants from Ellard et al.'s Study 2 and Farchione et al.'s RCT.

principal diagnoses [*n* = 3]). Most of the patients in these trials had at least one clinically significant comorbid disorder (mode = two clinical diagnoses, range = 1 to 5), including 18 patients with comorbid GAD, 18 patients with comorbid SOC, 14 patients with comorbid PD, 12 patients with comorbid OCD, and 12 patients with a comorbid depressive disorder.

MEASURES

Anxiety Disorders Interview Schedule for DSM-IV–Lifetime Version (ADIS-IV-L; DiNardo, Brown, & Barlow, 1994)—Baseline diagnoses were assessed with the ADIS-IV-L. This semistructured, diagnostic clinical interview focuses on *DSM-IV* diagnoses of anxiety disorders, mood disorders, and their accompanying mood states, somatoform disorders, and substance and alcohol use. Principal (most interfering and severe) and additional diagnoses are assigned a clinical severity rating (CSR) on a scale from 0 (*no symptoms*) to 8 (*extremely severe symptoms*), with a rating of 4 or above (*definitely disturbing/disabling*) marking the clinical threshold for *DSM-IV* diagnostic criteria. The condition with the highest rated CSR is considered principal. The pre- and posttreatment CSRs were used in the analysis examining treatment outcome. The ADIS-IV-L has consistently demonstrated good to excellent interrater reliability for the anxiety and mood disorders (Brown, DiNardo, Lehman, & Campbell, 2001). Although ongoing diagnostic reliability data were not ascertained during the conduct of the trials of interest, all ADIS interviewers were trained to a high level of reliability (≥.80) and underwent a rigorous certification process involving many different cases (see Brown et al., 2001) with the ADIS-IV-L developer prior to participation as a study assessor. In addition, study staff held weekly meetings during which all initial diagnostic interviews conducted that week were discussed in the presence of senior clinicians, and in the instance of diagnostic disagreement the sources of these differences were reviewed and a consensus diagnosis was reached.

Anxiety Sensitivity Index (ASI; Reiss et al., 1986; Peterson & Reiss, 1987)—This 16-item self-report measure is commonly used to assess anxiety sensitivity, which refers to beliefs about the dangerousness of anxious symptoms, as well as the resulting fear of these symptoms (Reiss & McNally, 1985). Individuals rate the level of agreement for each item on a 5-point Likert-type scale. Higher scores represent greater anxiety sensitivity. The ASI has three subscales: ASI Physical (distress regarding somatic symptoms of anxiety), ASI Cognitive (distress regarding the presence of anxious cognitions), and ASI Social (distress regarding the social consequences of being anxious). The ASI and its subscales have demonstrated good internal consistency and convergent validity (Peterson & Reiss, 1987; Vujanovic, Arrindell, Bernstein, Norton, & Zvolensky, 2007). The internal consistency of the pretreatment ASI Total was α = .89 in the earlier trial and .84 in the more recent trial. In both trials, patients completed the ASI at baseline (pretreatment), after Session 4, after Session 8, after Session 12, and at posttreatment.

Panic Disorder Severity Scale–Self-Report Version (PDSS-SR; Houck, Speigel, Shear, & Rucci, 2002)—We used this measure primarily as a control variable to aid in the comparability of the trial samples and assist in the missing value analysis described below. The PDSS-SR is a 7-item measure that assesses panic severity, including frequency of panic attacks and interference on functioning. A total score is calculated that ranges from 0 to 28, with higher scores indicating greater symptom severity and impairment. This scale has demonstrated good psychometric properties (Houck et al., 2002; Shear et al., 2001). The internal consistency of the pretreatment PDSS-SR was α = .91 in the earlier trial and .92 in the more recent trial. In both trials, all patients completed the PDSS-SR baseline and at posttreatment.

Clinical Measures—A core component of CBT is the use of monitoring and recording forms to gather clinically useful data for use both within and between sessions. Two forms, in particular, are utilized in the UP module that focuses on increasing awareness and tolerance of physical sensations and the use of IE exercises.

Symptom Induction Test Form—This clinical worksheet is completed during the session(s) in which symptom-induction exercises are conducted. After each symptom-induction exercise is conducted for the first time, the therapist gathers and records the following information from the patient: (a) a list of physical symptoms experienced during the exercises, (b) ratings of symptom intensity, using a 1–8 scale (where 1 indicates not at all intense and 8 indicates extremely intense), (c) ratings of subjective distress experienced during the exercise, using a 1–8 scale (where 1 indicates not at all intense and 8 indicates extremely intense), and (d) ratings of the degree of similarity in the symptoms experienced during the exercise to those experienced in a typical episode of strong anxiety or panic, using a 1–8 scale (where 1 indicates no similarity to what they normally experience and 8 indicates that the symptoms were identical in nature and intensity).

Symptom Induction Practice Form—This form is completed by the patient during independent practice with the symptom-induction (i.e., interoceptive exposure) exercises that are assigned as part of the between-session homework. Based on the results of the symptom induction exercises, the patient and therapist agree upon which exercises (e.g., narrow straw breathing) the patient will practice independently over the next week. Patients are instructed to complete an IE several times during the week. For each IE exercise, the patient records the date and number of trials of each exercise completed. Similar to the Symptom Induction Test Form, the patient then rates the intensity, distress, and similarity of the sensations elicited during the exercises on the Symptom Induction Practice Form.

PROCEDURE

Patients were recruited to participate in two consecutive treatment outcome studies examining the efficacy of the UP. All participants completed a baseline diagnostic interview, in addition to self-report measures of anxiety and related symptoms and impairment. Additional assessments were conducted after Sessions 4, 8, 12, and at posttreatment. All participants received individual, weekly psychotherapy (UP), with each session lasting approximately 60 minutes. In both trials, patients could receive a maximum of 18 therapy sessions ($M = 17$ in the earlier study and $M = 15$ in the more recent study). As noted, the UP modules are designed to be applied flexibly. Most patients in this sample were introduced to the IE module between Sessions 7 and 10. This module involves psychoeducation regarding the nature of somatic symptoms of anxiety, the interaction between physical symptoms, thoughts, and behaviors, and the importance of reducing avoidance and promoting greater tolerance of uncomfortable physical sensations. In addition to psychoeducation and providing a rationale, the primary intervention of this module is the use of IE both within and between sessions. As noted, an in-session assessment is first conducted using the Symptom Induction Test Form, and relevant exercises are assigned as homework in subsequent weeks using the Symptom Induction Practice Form.

In the interest of increasing the clinical relevance of this information and providing additional support for the feasibility of utilizing IE in problem areas where it might seem less intuitive to do so, we selected four example cases to present in more detail. Specifically, we selected a case to represent each of the principal diagnoses with the greatest representation (GAD, SOC, PDA, and OCD) in this sample. These cases were selected primarily for illustrative purposes, rather than completely at random. For example, given that the homework forms and clinical notes pertaining to a case were collected first and

foremost as clinical information, rather than for research, some homework forms were not retained by the therapist and other forms that were collected and retained did not include clearly written information. Thus, in addition to choosing a case from each of these four diagnostic groups, we selected cases where both the Symptom Induction Test and Symptom Induction Practice forms were available and legible, and the session in which the IE module was first introduced was clearly noted. In an effort to maintain confidentiality, the patients' names have been changed and their identifying information has been altered. All study procedures were reviewed and approved by a university-based institutional review board (IRB), and all participating patients provided verbal and written consent for their de-identified data to be used for research and publication purposes.

Results

MISSING DATA

As is commonly the case in treatment research involving repeated assessments, missing data were present for some cases. In both of the trials of interest in this study, the ASI was administered at pretreatment, after Sessions 4, 8, and 12, and at posttreatment. A missing value analysis was conducted to examine the nature of the missing data (Little & Rubin, 2002) using SPSS 20 software. In addition to observed ASI scores, several variables were included in this analysis, in order to assist in the detection of patterns and determine if the data were Missing Completely at Random(MCAR), Missing at Random (MAR), or Not Missing at Random (NMAR): Pre and posttreatment severity on the PDSS-SR and principal diagnosis CSR, the trial from where the data for each case were derived, treatment condition (immediate or wait-list/delayed), gender, race, and completer status. No single item on the ASI at each assessment point evidenced more than 5% missing-ness. As is typical for longitudinal data, descriptive data and a visual inspection of the patterns indicated more missing cases at later time points. Two individuals (3.7%) were missing baseline ASI data; 4 individuals (7.4%) were missing Session 4 ASI data; 5 individuals (9.3%) were missing Session 8 ASI data; 10 individuals (18.5%) were missing Session 12 ASI data; 8 individuals (14.8%) were missing ASI posttreatment data. Nevertheless, Little's MCAR test was nonsignificant, χ^2 (50) =57.25, p = .22, indicating that the data were likely MAR or MCAR.

In order to account for these missing data and increase the power and efficiency of our inferential analyses, we estimated missing data using the Expectation Maximization (EM) algorithm (Dempster, Laird, & Rubin, 1977; Do & Batzoglou, 2008; Schafer, 1997) in SPSS. Along with multiple imputation, maximum-likelihood-based approaches are commonly used for estimating data that are MAR. Often used to address incomplete repeated measures variables, the EM algorithm is a likelihood based method (Enders, 2013) involving two iterative steps that use all available data as covariates to generate maximum-likelihood-based statistics: (1) posterior probabilities are computed for each missing data value based on all existing data (i.e., missing variables are regressed on the observed variables for each case), resulting in a set of possible values, and (2) maximum likelihood estimation is used to generate new parameter estimates (Enders, 2001). Using this approach, we first estimated missing ASI data at the item level (which was, as noted, less than 5% for any item at any time point); specifically, the final iteration of the resulting covariance matrix was used to estimate item-level missing data points. We then once again applied the EM algorithm at the case/wave level; however, rather than imputing values from the final iteration, this time we used the resulting covariance matrix alone to estimate missing data in subsequent inferential analyses (e.g., ANOVA; Enders, 2001). Although alternative approaches exist, likelihood-based approaches, such as EM, have been shown to be more reliable and robust than missing data replacement strategies such as list-wise or pair-wise deletion, mean substitution, or regression substitution (Enders, 2001, 2013; Schafer, 1997). In addition, multiple imputation and maximum-likelihood approaches have been shown to

yield comparable results when applied at the wave level in longitudinal research (Enders, 2013).

LEVELS OF PRETREATMENT ANXIETY SENSITIVITY

Compared to normative, nonclinical samples (see Naragon-Gainey, 2010; Reiss et al., 2008: $M = 18.7$, $SD = 9.11$), moderate-to-high levels of anxiety sensitivity were observed at pretreatment across principal diagnostic categories ($M = 30.0$, $SD = 12.6$; see Table 2). A power analysis indicated that, despite combining samples from the two trials, an ANOVA examining between-diagnosis differences would be underpowered (< .80 with 5 levels, approximately 12 participants per level, medium effect size, *alpha* = .05). As such, we report Hedge's g effect sizes for the differences between the principal diagnosis groups in Table 3 as a method for comparing initial ASI scores. The "other" diagnostic group (comprised of PTSD, anxiety disorder NOS, and co-principal diagnoses [only one of which was PD]) evidenced the highest mean ASI Total score, followed by SOC, PD, OCD, and GAD. The largest observed difference was between the "other" and GAD groups (Hedge's $g = .75$). Conversely, PD and SOC and OCD and GAD, respectively, evidenced comparably high levels of anxiety sensitivity. Similar patterns emerged for the ASI subscales scores at pretreatment (see Table 3), with the "other" group demonstrating the highest mean scores, followed by SOC (ASI Cognitive and ASI Social), PD (ASI Physical), and the GAD group demonstrating the lowest mean scores (with the exception of the ASI Cognitive subscale where OCD evidenced the lowest score).

PATTERNS OF CHANGE IN ANXIETY SENSITIVITY DURING TREATMENT

In order to assess changes in anxiety sensitivity over the course of treatment, a series of repeated measures ANOVAs were conducted using the ASI data collected for each participant at pretreatment, after Sessions 4, 8, and 12, and at posttreatment. Repeated measures ANOVA allows for the examination of within-subject influences of time (both linear and curvilinear) on anxiety sensitivity, while accounting for the dependency that frequently exists in repeated measures data. Repeated measures analysis of the ASI Total scores revealed a significant decrease in anxiety sensitivity from pre- to posttreatment, Wilks' $\lambda = 0.45$, $F(4, 46) = 13.96$, $p < .01$ $\eta_p^2 = .55$ (see Figure 1). Significant linear, $F(1, 49) = 36.72$, $p < .01$, $\eta_p^2 = .43$, and curvilinear/quadratic, $F(1, 49) = 9.97$, $p < .01$, $\eta_p^2 = .17$, effects for time were observed, indicating that the majority of change in anxiety sensitivity occurred between Session 8 and posttreatment. This interpretation was further supported by multiple comparison tests (see Table 4) demonstrating less change in ASI Total scores from pretreatment to Session 4, and a greater magnitude of change in anxiety sensitivity occurring after Session 8, temporally coinciding with the introduction of interoceptive and emotion exposures.

A second repeated measures ANOVA was then conducted. Despite a power analysis indicating an inadequate within-diagnostic group sample size to reliably detect between-group statistical differences (< .80 with 5 levels, approximately 12 participants per level, medium effect size, *alpha* = .05), we elected to run this model with the between-subject principal diagnosis factor in order to generate an effect size estimate. The results indicated that diagnostic group exerted a relatively small effect (linear, $\eta_p^2 = .01$; quadratic, $\eta_p^2 = .06$), providing preliminary support for comparable trajectories of change in anxiety sensitivity over the course of treatment. Means for each diagnosis category are plotted in Figure 2.

These analyses were then repeated for each of the ASI subscales (Physical, Cognitive, and Social). For the sake of brevity, these results will merely be summarized. Similar to the model for the ASI Total scores, scores on the Physical, Wilks' $\lambda = 0.35$, $F(4, 46) = 21.07$, p

< .01, η_p^2=.65, Cognitive, Wilks' λ = 0.60, F(4, 46) = 7.66, p < .01, η_p^2=.40, and Social, Wilks' λ = 0.64, F(4, 46) = 6.41, p < .01, η_p^2=.36, subscales decreased significantly over time. Each of the subscales demonstrated a significant negative linear trend across diagnoses; however, the ASI Physical subscale also demonstrated a significant quadratic trend, F(1, 49) = 37.70, p < .01, η_p^2=.40, indicating that the majority of the change in physical concerns related to anxiety occurred between Session 8 and posttreatment. This interpretation was further supported by the results from multiple comparison tests (see Table 5), also indicating that a greater magnitude of change occurred after Session 8. Effect sizes for the between-subjects factor (diagnostic group) ranged from η_p^2=.01 (Physical subscale) to η_p^2=.03 (Cognitive and Social subscales), again providing preliminary support for comparable trajectories of change over the course of treatment.

CHANGE IN ANXIETY SENSITIVITY AND OUTCOME

A multiple regression analysis was conducted to examine the association between changes in anxiety sensitivity and principal diagnosis CSRs at posttreatment. Baseline principal diagnosis CSRs were entered simultaneously in the model with pre-post residualized change scores on the ASI. Results indicated that when controlling for initial severity, greater reductions in anxiety sensitivity were associated with lower levels of clinical severity at posttreatment (ASI change β = .64, SE = 0.02, t = 6.13, p < .01, CI_B = 0.07 : 0.13, pr = .65).

Six-month ASI follow-up data were collected from 37 participants (68.5%). As noted, ASI Total scores significantly decreased between pre- (M = 30.0, SD = 12.6) and posttreatment (M = 19.61, SD = 12.1, Hedge's g = −0.77). This reduction in ASI Total scores was not only maintained but decreased slightly at 6-month follow-up (n = 37, M = 17.62, SD = 10.87, Hedge's g = −0.17). A multiple regression analysis was conducted to examine the association between changes in anxiety sensitivity during treatment and principal diagnosis CSRs at 6-month follow-up. Baseline principal diagnosis CSRs were entered simultaneously in the model with pre-post residualized change scores on the ASI. Results indicated that when controlling for initial severity, greater reductions in anxiety sensitivity during treatment were associated with lower levels of clinical severity at 6-month follow-up (n = 37, ASI change β = .44, SE = 0.02, t = 3.17, p < .01, CI_B = 0.03 : 0.12, pr = .46).

CLINICAL DATA AND EXAMPLES

The gathering of clinical data is an important feature of CBT, including the UP. In line with this, the implementation of IE involves conducting a systematic symptom assessment/induction for each exercise. For each initial IE symptom assessment/induction, the therapist collects a list of experienced symptoms, as well as ratings of physical symptom intensity, subjective distress, and level of similarity to typical experiences of anxiety and panic. Patients are then asked to provide similar ratings when engaging in repeated IE homework between sessions. We present the following cases to illustrate similarities and differences among principal diagnostic categories, as well as feasibility of implementation.

Patricia: Principal PD With Agoraphobia—Patricia (age = 36) was assigned a principal diagnosis of PD with agoraphobia (CSR = 4) at pretreatment. No other disorders were noted at a clinically significant level. Patricia had a total score of 33 on the ASI at pretreatment, indicating moderate to high levels of anxiety sensitivity. Her initial symptom induction tests occurred in Session 10, followed by additional exercises in Session 11. The IE exercises that provoked the most intense physical sensations and the greatest degree of distress (scales ranging from 1–8) were hyperventilation (intensity = 8, distress = 8) and spinning while seated in a chair (intensity = 7, distress = 7). Both exercises were identified

as being highly similar to naturally occurring symptoms (rating = 8 for both), as was running in place (rating = 6). All three exercises were assigned for homework. Her recordings on the Symptom Induction Practice form indicated reductions in subjective distress across all three of the assigned IE exercises. As more IEs were conducted over the course of several weeks, fewer and fewer trials were needed to achieve mild levels of distress (ratings of 2 and 3 out of 8). Patricia's ASI total score decreased only slightly to 30 during the first half of treatment. However, during the second half of treatment, her ASI Total score dropped to 18 by Session 12 (following the interoceptive exposures) and finally to 17 by posttreatment.

William: Principal GAD—William (age = 53) was assigned a principal diagnosis of GAD (CSR = 5) at pretreatment, as well as concurrent diagnoses of major depressive disorder (MDD; CSR = 4), specific phobia (blood-injection-injury, CSR = 2), and specific phobia (driving, CSR = 2). William had a total score of 15 on the ASI at pretreatment, indicating more mild levels of anxiety sensitivity, yet similar to Patricia (above), his ASI score actually increased to 25 at the Session 4 assessment. His symptom induction tests occurred at Session 10. William reported high levels of physical intensity (rating of 7) and subjective distress (rating of 7) during the hyperventilation exercise, which he felt was quite similar to his naturally occurring symptoms (similarity rating of 8; scale range 1–8). In addition to a hyperventilation exercise, William completed a narrow straw breathing exercise (intensity = 3, distress = 4, and similarity = 2) and a spinning exercise (intensity = 5, distress = 3, and similarity = 4). The information gathered from the symptom induction tests indicated that physical symptoms related to hyperventilation were most relevant and distressing to William. As such, he and his therapist agreed that he would complete the hyperventilation exercise repeatedly, for 60 seconds at a time, each day over the subsequent week. William completed a series of hyperventilation IEs on 5 days between Sessions 10 and 11. Ratings on his Symptom Induction Practice Form indicated a minor reduction in the intensity of physical sensations, yet a more substantial decrease in subjective distress, both within the course of each daily practice and progressively over the course of the 5 days. William evidenced an ASI total score of 6 after Session 12, and a total score of 3 at posttreatment, indicating a substantial decrease in anxiety sensitivity in the latter half of treatment.

Lillian: Principal OCD—Lillian (age =24) was assigned a principal diagnosis of OCD (CSR = 7) at pretreatment, as well as concurrent diagnoses of PDA (CSR = 7) and GAD (CSR = 5). She had an ASI Total score of 33 at pretreatment. Her symptom induction test exercises were first introduced at Session 7. Several exercises were conducted, including hyperventilation, narrow straw breathing, and spinning. However, only the hyperventilation exercise produced symptoms that were rated as being more than moderately intense (rating = 5) and distressing (rating = 5), as well as similar to naturally occurring symptoms (rating = 7). Consequently, she and the therapist agreed that she would complete hyperventilation IEs for homework. She conducted a series of these IEs on three occasions between Sessions 7 and 8. Lillian's distress ratings remained consistently moderate across trials in the first two series of IE exercises (despite completing 10 consecutive trials on the second occasion). However, by the end of the third set of IE trials (also involving 10 consecutive trials), the final distress rating was in the mild range (rating = 2). Interestingly, reductions in symptom intensity were also reported across the three series of IEs (final trial rating = 2). Lillian's ASI Total score reached its peak (total = 37) at the Session 4 assessment, and her total score at posttreatment was 30.

Walter: Principal SOC—Walter (age = 41) was assigned a principal diagnosis of SOC (CSR = 5) at pretreatment, as well as concurrent diagnoses of MDD (CSR = 4) and OCD(CSR = 4). His ASI Total score at pretreatment was 35. Walter's symptom induction test exercises were first introduced in Session 7, and included hyperventilation, narrow straw

breathing, spinning in a chair, jumping jacks (to increase heart rate), and placing his head between his legs and then lifting it quickly (to produce feelings of lightheadedness). With the exception of straw breathing, all of these exercises were rated as being at least moderately similar to his naturally occurring symptoms (≥4 out of 8), with the sensations elicited from jumping jacks being identified as most similar (rating = 7). Despite a lower level of perceived similarity (rating = 3), Walter rated straw breathing as evoking the most physically intense (rating = 6) and distressing (rating = 6) symptoms, relative to the other exercises. Several exercises were assigned for homework; specifically, lifting his head between his legs, hyperventilation, and narrow straw breathing. A series of IE trials were conducted on 5 days over the course of the next week. The Symptom Induction Practice Form indicated that all three IE exercises elicited at least a moderate level of physical intensity (≥4 out of 8). However, only narrow straw breathing and head between legs produced more than moderate levels of subjective distress (≥4 out of 8). Slight reductions in distress were noted across IE trials, and by the end of the week, none of the IE exercises evoked more than mild levels of distress (≤2 out of 8). Walter's ASI Total scores decreased in a more linear and gradual fashion than the above examples. His posttreatment ASI total score was 20.

Discussion

Despite traditionally being associated with panic disorder (PD), both conceptually and empirically, the extant research using both clinical and nonclinical samples has shown that anxiety sensitivity is implicated in the development and maintenance of a broad range of emotional disorders (Naragon-Gainey, 2010; Schmidt et al., 2008). This implies that anxiety sensitivity may be an important transdiagnostic target of treatment, and that reductions in anxiety sensitivity may represent an important process of change across the spectrum of emotional disorders. Historically, IE has been utilized as a strategy of intervention to target anxiety sensitivity in CBT treatments for panic disorder (e.g., Antony, Ledly, Liss, & Swinson, 2006; Craske & Barlow, 2007; Schmidt & Trakowski, 2004). However, accumulating basic (see Barlow, 2002) and applied (e.g., Wald, 2008) research supports the relevance and impact of IE for addressing anxiety sensitivity and promoting change across a range of disorders. Nevertheless, little is known about the temporal patterns of anxiety sensitivity in treatment across heterogeneous disorders, and we are unaware of previous research examining such patterns in a single, transdiagnostic treatment. Furthermore, we believe that the implementation of IE in clinical practice with diverse anxiety disorders deserves more attention. Consequently, this study aimed to replicate and extend previous research on anxiety sensitivity and IE in psychotherapy by examining this factor and strategy of intervention in heterogeneous anxiety disorders, as well as provide preliminary evidence for the impact of the IE module of the UP.

Our first aim was to examine levels of anxiety sensitivity at pretreatment across the four principal diagnostic groups represented in two UP trials: PD, SOC, GAD, and OCD. Patients evidenced moderate-to- high levels of anxiety sensitivity at pretreatment, with all diagnostic groups demonstrating higher levels of anxiety sensitivity compared to previously reported nonclinical samples. Consistent with Naragon-Gainey (2010), individuals with a principal PD diagnosis did not evidence the most severe anxiety sensitivity scores in this sample. Interestingly, the "other" group consistently demonstrated the highest scores, and individuals with SOC demonstrated comparably high levels of anxiety sensitivity to that of individuals with PD. Given that individuals with social phobia often report the belief that others are acutely aware of their anxiety (i.e., others will notice immediately if my face turns red), it is not surprising that these individuals endorsed increased sensitivity to their own emotional arousal (Heimberg & Becker, 2002; Hope et al., 2010).

Results from our second study aim showed a significant decrease in anxiety sensitivity over the course of treatment, with the greatest degree of change occurring between Session 8 and posttreatment (largely coinciding with the presentation of the IE module). Temporal patterns of change in anxiety sensitivity over the course of treatment appeared to be similar across the diagnostic groups. The same pattern of results emerged for the specific subscales of the ASI, indicating similar decreases in physical, cognitive, and social concerns over the course of treatment, across diagnoses, and that most of this change occurred in the second half of treatment. Finally, greater reductions in anxiety sensitivity were associated with lower levels of clinical severity at posttreatment and 6-month follow-up.

These results provide additional support for the transdiagnostic relevance of anxiety sensitivity across the spectrum of anxiety disorders, and can be incorporated into the existing literature questioning the diagnostic specificity of anxiety sensitivity in PD. Reductions in this trait can be reliably observed in transdiagnostic CBT, and such reductions are predictive of outcome, providing preliminary empirical support for anxiety sensitivity as a transdiagnostic change factor. Furthermore, most of the change observed in anxiety sensitivity took place in the second half of treatment, along with or after the introduction of the IE module, providing preliminary, indirect evidence for the specific effects of IE on anxiety sensitivity.

When considered alongside existing research in this area, these results also suggest that clinicians should be mindful of their patients' level of anxiety sensitivity across a range of presenting problem areas. Although individual differences will emerge in its presence and relevance patient-to-patient, interventions aimed at decreasing anxiety sensitivity (which are unlikely to be limited to IE) may facilitate better outcomes. Targeting change in this construct may be particularly appealing because it does not imply that the experience of anxiety or fear is "bad" or should be completely eliminated. Sustained mindful awareness of strong physical sensations in the context of emotional experiences, in the absence of avoidance and negative consequences, may allow for the extinction of anxiety to intense emotions to occur. However, the precise mechanism(s) by which IE leads to changes in anxiety sensitivity remain less well understood. Although different psychological theories have been postulated (e.g., conditioning, cognitive), researchers have also demonstrated changes in anxiety sensitivity in PD with pharmacotherapy (see Simon et al., 2004), potentially indicating equifinality.

Additionally, although the pattern of change in anxiety sensitivity was best characterized as linear, some significant curvilinear patterns were found. Relative to the total amount of change in anxiety sensitivity, little change seemed to occur between pretreatment and Session 4. In fact, there was indication (including two of the cases described above) that anxiety sensitivity actually increased for many patients early on in treatment. As described above, the initial modules of the UP are focused on increasing awareness of emotions, learning to observe emotions in their context, and learning to observe and relate to strong emotions in a new way. For patients who have spent much of their time trying desperately to control and suppress their emotional experience, it is not surprising that increased awareness and emotional approach would lead, at least initially, to increased sensitivity and distress. Such patterns are commonly observed when emotional processing is facilitated (Foa & Kozak, 1986), and clinicians should monitor their patients closely and prepare them for this possibility at the beginning of treatment.

Although this study was unable to demonstrate a causal relationship between the IE module and reductions in anxiety sensitivity (or reductions in anxiety sensitivity and symptom change), the observed patterns and associations between the IE module and changes on the ASI in the second half of treatment are noteworthy. As illustrated in the cases presented

above, the process of implementing within and between-session IEs can be essentially the same across principal problem areas. The induction tests and homework that a patient with GAD completes will look remarkably similar to those that are completed by a patient with PD. We find conducting the symptom induction practice exercises particularly important because of the inter-individual variability observed in the particular anxiety symptoms reported and which symptoms/feelings are experienced as most distressing. Because these induction tests set the stage for between-session practice (where most of the learning is likely to take place), it is important to have a clear understanding of the experiences that are most relevant to the individual patient. In our experience, most patients are all too familiar with the physical symptoms of anxiety, and, in comparison, tend to have more difficulty with identifying specific cognitions in anxious experiences. The rationale for conducting IEs in the UP is consistent across the principal problem areas, as well as with the overall treatment rationale, and is typically understood and accepted with little difficulty. Of course, few patients will "like" going through the symptom inductions, yet they seem ultimately to ascertain the benefits.

Several study limitations should be noted when interpreting these findings. First, although combining data from two similar trials increased the sample size and power, the specific diagnostic groups were still relatively small (between and 11 and 13 individuals in each), which compromised the use of ANOVA to address some study questions and led us to focus more on effect sizes. Second, the overall sample had a high degree of ethnic homogeneity, which limits generalizability. Third, it is important to note that the UP is multicomponent treatment. We cannot conclude that the IE exercises were causally related to reductions in anxiety sensitivity. It is possible that material learned from earlier modules had delayed effects and/or, perhaps more likely, worked cumulatively with the IE module. Similar issues preclude making any causal claims about the relationship between anxiety sensitivity and treatment outcome. Alternative research designs may help to address these limitations in future research. For example, dismantling studies could help to isolate the direct and specific effects of IEs on anxiety sensitivity (and outcome). Additionally, more intensive measurement of constructs prior to, during, and after the introduction of IEs would facilitate the examination of mediation hypotheses, which would provide more direct evidence for anxiety sensitivity being a mechanism of change, and IE as a strategy to facilitate this change. Fourth, although we presented this information largely for illustrative purposes (as opposed to a rigorous qualitative analysis), the cases that we presented were not randomly selected, primarily due to a lack of consistent clinic data for participants. A wealth of clinically useful data are collected by practitioners on a daily basis. This is certainly the case for treatment studies, over and above the information collected during predetermined research assessments. This study served as a reminder that important information can be gleaned from routinely collected clinical data, and we should not lose sight of this when conducting controlled research that includes more systematic evaluation.

Anxiety sensitivity appears to be a transdiagnostic construct of clinical relevance, and IE continues to be an important strategy of intervention in CBT. Consistent with other emerging research in this area, our results indicate that IE is not just for panic anymore. Interoceptive exposure can be a useful intervention for patients with and without panic, and it can be relatively seamlessly integrated into most CBT treatments in a manner that is consistent with what has been previously specified for panic disorder (e.g., Craske & Barlow, 2007). Future research, including studies utilizing the designs suggested above, will provide more information about the nature of anxiety sensitivity and the unique effects and mechanisms of IE in various emotional disorders.

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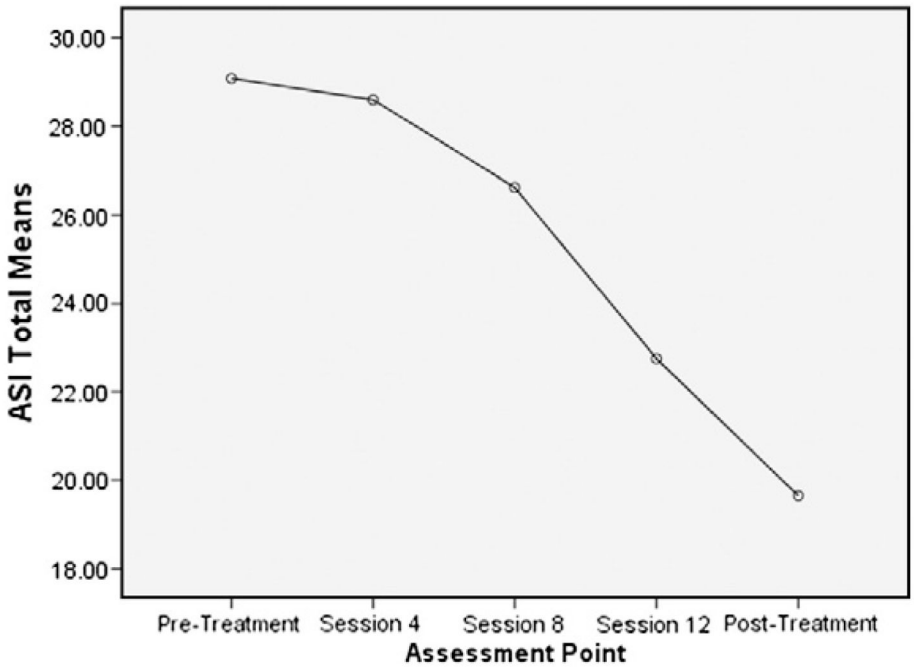

FIGURE 1.
Mean Anxiety Sensitivity Index (ASI) total scores over the course of treatment.

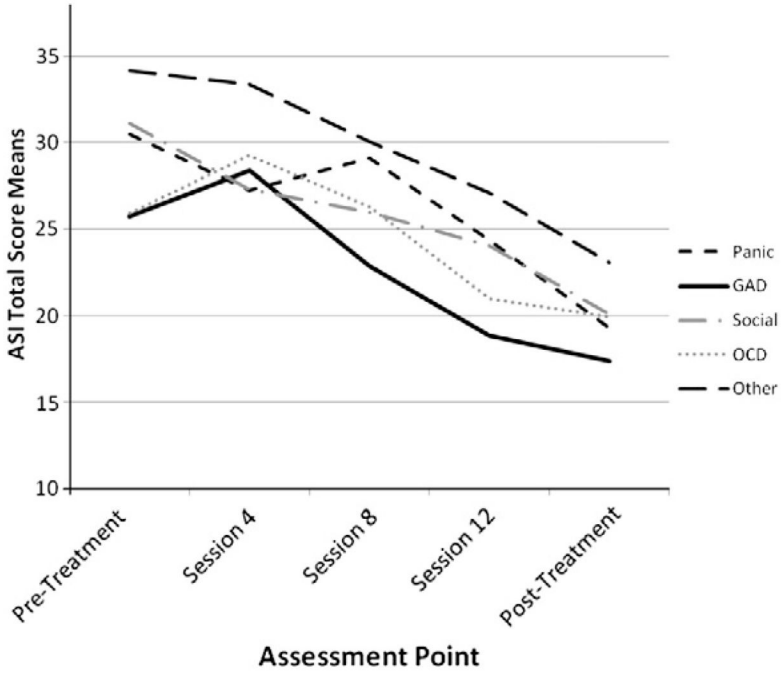

FIGURE 2.
Mean Anxiety Sensitivity Index (ASI) total scores for each principal diagnosis over the course of treatment. GAD = generalized anxiety disorder; Panic = panic disorder with agoraphobia; Social = social phobia; OCD = obsessive-compulsive disorder; Other = posttraumatic stress disorder and anxiety disorder NOS.

Table 1

Examples of Interoceptive Exposure (IE) Exercises and Related Symptoms

Exercise	Symptom(s)
Running in place	Increase heart rate, temperature, sweating
Spinning	Dizziness, nausea
Overbreathing/hyperventilation	Lightheadedness, blurred vision, numbing/tingling
Narrow straw breathing	Shortness of breath
Tense body	Muscle tension, fatigue

Table 2

ASI Means and Standard Deviations at Pretreatment by Principal Diagnosis

	Pre-Treatment ASI Total	Pre-Treatment ASI Physical	Pre-Treatment ASI Cognitive	Pre-Treatment ASI Social
GAD n = 11				
Mean	25.77	12.85	5.87	7.06
SD	9.99	6.48	4.50	2.79
Min.	13.00	6.00	1.00	3.00
Max.	46.00	26.00	15.00	12.00
Panic n = 12				
Mean	30.57	15.72	6.47	8.38
SD	13.21	7.23	5.27	3.20
Min.	9.00	4.00	0.00	3.56
Max.	50.00	27.00	15.00	14.00
Social n = 13				
Mean	31.08	13.23	7.23	10.62
SD	14.33	9.12	4.09	3.33
Min.	4.00	0.00	0.00	4.00
Max.	57.00	30.00	15.00	15.00
OCD n = 12				
Mean	25.92	14.00	4.75	7.17
SD	13.14	7.99	5.15	2.29
Min.	4.00	0.00	0.00	4.00
Max.	48.00	24.00	14.00	12.00
Other n = 6				
Mean	34.17	17.67	7.67	8.83
SD	12.30	6.65	4.50	4.07
Min.	19.00	10.00	2.00	5.00
Max.	51.00	26.00	14.00	16.00

Note. ASI = Anxiety Sensitivity Index; GAD = generalized anxiety disorder; Panic = panic disorder with agoraphobia; Social = social phobia; OCD = obsessive-compulsive disorder; Other = posttraumatic stress disorder and anxiety disorder NOS.

Table 3

Effect Sizes (Hedge's *g*) of the Difference in ASI Scores Between Principal Diagnostic Groups

	ASI Total			
	PDA	**GAD**	**SOC**	**OCD**
PDA	–			
GAD	−.41 [−.79: −.03]	–	–	
SOC	.04 [−.34:.41]	−.43 [−.81: −.05]	–	
OCD	−.35 [−.73:.03]	−.01 [−.39:.36]	.38 [−.01:.76]	–
Other	.28 [−.10:.66]	−.75 [−1.14: −.36]	−.23 [−.61:.15]	−.65 [−1.04: −.26]
	ASI Physical			
	PDA	GAD	SOC	OCD
PDA	–			
GAD	.42 [.04:.80]	–	–	
SOC	.30 [−.08:.68]	−.05 [−.43:.33]	–	
OCD	.23 [−.15:.60]	−.16 [−.54:.22]	−.09 [−.47:.29]	–
Other	−.28 [−.66:.10]	−.73 [−1.12: −.34]	−.56 [−.94: −.17]	−.50 [−.88: −.12]
	ASI Cognitive			
	PDA	GAD	SOC	OCD
PDA	–			
GAD	.12 [−.26:.50]	–	–	
SOC	−.16 [−.54:.22]	−.32 [−.70:.06]	–	
OCD	.33 [−.05:.71]	.24 [−.15:.61]	.53 [.15:.92]	–
Other	−.25 [−.62:.13]	−.40 [−.78: −.02]	−.10 [−.48:.28]	−.60 [−.99: −.22]
	ASI Social			
	PDA	GAD	SOC	OCD
PDA	–			
GAD	.44 [.06:.83]	–	–	
SOC	−.69 [−1.07: −.30]	−1.16 [−1.57: −.75]	–	
OCD	.44 [.05:.82]	−.04 [−.42:.33]	1.21 [.80:1.62]	–
Other	−.12 [−.50:.26]	−.51 [−.87: −.12]	.48 [.10:.86]	−.50 [−.89: −.12]

Note. ASI = Anxiety Sensitivity Index; GAD = generalized anxiety disorder; Panic = panic disorder with agoraphobia; Social = social phobia; OCD = obsessive-compulsive disorder; Other = posttraumatic stress disorder and anxiety disorder NOS.

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Table 4

Multiple Comparisons of ASI Total Scores at Different Time Points

Time 1	Time 2	M_{diff}	SE	Lower CI	Upper CI
Pre-Treatment	Week 4	0.48	1.22	−1.97	2.93
	Week 8	2.46*	1.00	0.45	4.47
	Week 12	6.33**	1.50	3.32	9.34
	Post	9.43**	1.56	6.30	12.57
Week 4	Pre	−0.48	1.22	−2.93	1.97
	Week 8	1.98*	0.97	0.04	3.92
	Week 12	5.85**	1.33	3.18	8.51
	Post	8.95**	1.21	6.53	11.38
Week 8	Pre	−2.46*	1.00	−4.47	−0.45
	Week 4	−1.98*	0.97	−3.92	−0.04
	Week 12	3.87**	0.93	2.01	5.72
	Post	6.97**	0.94	5.10	8.85
Week 12	Pre	−6.33**	1.50	−9.34	−3.32
	Week 4	−5.85**	1.33	−8.51	−3.18
	Week 8	−3.87**	0.93	−5.72	−2.01
	Post	3.10**	0.89	1.31	4.89
Post-Treatment	Pre	−9.43**	1.56	−12.67	−6.30
	Week 4	−8.95**	1.21	−11.38	−6.53
	Week 8	−6.97**	0.94	−8.85	−5.10
	Week 12	−3.10**	0.89	−4.89	−1.31

Note.

*$p < .05$,

**$p < .01$. $N = 54$.

Table 5

Multiple Comparisons of ASI Physical Scores at Different Time Points

Time 1	Time 2	M_{diff}	SE	Lower CI	Upper CI
Pre-Treatment	Week 4	−0.75	0.71	−2.17	0.67
	Week 8	0.10	0.55	−1.00	1.19
	Week 12	2.71**	0.86	0.98	4.44
	Post	4.62**	0.87	2.87	6.37
Week 4	Pre	0.75	0.71	−0.67	2.17
	Week 8	0.84	0.58	−0.31	2.00
	Week 12	3.46**	0.74	1.97	4.95
	Post	5.37**	0.65	4.06	6.67
Week 8	Pre	−0.10	0.55	−1.19	1.00
	Week 4	−0.84	0.58	−2.00	0.31
	Week 12	2.62**	0.60	1.42	3.81
	Post	4.53**	0.58	3.37	5.68
Week 12	Pre	−2.71**	0.86	−4.44	−0.98
	Week 4	−3.46**	0.74	−4.95	−1.97
	Week 8	−2.62**	0.60	−3.81	−1.42
	Post	1.91**	0.52	0.86	2.96
Post-Treatment	Pre	−4.62**	0.87	−6.37	−2.87
	Week 4	−5.37**	0.65	−6.67	−4.06
	Week 8	−4.53**	0.58	−5.68	−3.37
	Week 12	−1.91**	0.52	−2.96	−0.86

Note.

* $p < .05$,

** $p < .01$. $N = 54$.