Journal of Obsessive-Compulsive and Related Disorders 21 (2019) 18–25

Contents lists available at ScienceDirect

Journal of Obsessive-Compulsive and Related Disorders

journal homepage: www.elsevier.com/locate/jocrd

# Anxiety sensitivity does not predict treatment outcome or treatment length in obsessive-compulsive disorder and related anxiety disorders

Caroline M.B. Kwee[a,*], Marcel A. van den Hout[a,b]

[a] Altrecht Academic Anxiety Centre, Altrecht Mental Health Centre, Nieuwe Houtenseweg 12, 3524 SH Utrecht, the Netherlands
[b] Department of Clinical Psychology, Utrecht University, Heidelberglaan 1, 3584 CS Utrecht, the Netherlands

ARTICLE INFO

*Keywords:*
Anxiety sensitivity
Treatment outcome
Obsessive-compulsive disorder
Anxiety disorders

ABSTRACT

The present study aimed to replicate the findings of Blakey, Abramowitz, Reuman, Leonard, and Riemann (2017) that higher anxiety sensitivity (AS) predicted worse treatment outcome in 187 patients with obsessive-compulsive disorder (OCD), treated with cognitive-behavioral therapy. We also tested whether this finding is observed in other anxiety (related) disorders and if AS would predict treatment length. Assuming that exposure assignments would be more difficult for high AS individuals, we hypothesized that higher AS would predict worse treatment outcome and longer treatment length. Controlling for the presence and severity of pretreatment symptoms, these hypotheses were tested in 110 OCD patients and in 285 patients with mixed anxiety disorders. We failed to replicate the earlier findings. Hierarchical linear regressions revealed that AS did not contribute to the prediction of treatment outcome or treatment length; neither in OCD or in the other disorders. Findings are critically discussed.

## 1. Introduction

In the European Union, anxiety disorders, obsessive-compulsive disorder (OCD), posttraumatic stress disorder (PTSD), and health anxiety[1] are the most frequently occurring mental disorders (12-month prevalence of around 14%; Wittchen et al., 2011). Cognitive-behavioral therapy (CBT) has been extensively studied and is generally successful in treating these disorders (for meta-analyses, see Butler, Chapman, Forman, & Beck, 2006; Cooper, Gregory, Walker, Lambe, & Salkovskis, 2017; Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012; Hofmann & Smits, 2008). However, approximately 40% of patients (Taylor, Abramowitz, & McKay, 2012) do not respond satisfactorily: That is, their symptoms do not decrease, or their symptoms still fall in the range of a dysfunctional population after treatment (Eddy, Dutra, Bradley, & Westen, 2004; Jacobson & Truax, 1991; Taylor et al., 2012). Identification of robust predictors of treatment outcome would be valuable. Pretreatment symptom severity and comorbidity, and relatives' attitude towards the patient have been the most consistently identified predictors (Taylor et al., 2012). Unfortunately, these factors are crude or not easy to modify. Anxiety sensitivity (AS) is defined as the tendency to respond fearfully to anxiety symptoms based on beliefs that these symptoms have adverse consequences like a heart attack (McNally, 1989), embarrassment, or mental illness (Reiss, Peterson, Gursky, & McNally, 1986). As such, it is an interesting candidate for predicting treatment success (or failure). Patients with panic disorder misattribute benign bodily sensations like dizziness or palpitations as predicting imminent catastrophes. The resulting state of apprehension intensifies bodily sensations. If this process continues, it can easily culminate in panic attacks (Clark, 1986; McNally, 2002). AS is thought to play a role in other anxiety disorders as well (Reiss & McNally, 1985). Empirical findings indicate that AS is positively associated with anxiety symptoms (Hong, 2010; Kemper, Lutz, Hock, Bähr, & Rüddel, 2012; Norton & Edwards, 2017; Olthuis, Watt, & Stewart, 2014; Rifkin, Beard, Hsu, Garner, & Björgvinsson, 2015; Schmidt, Mitchell, & Richey, 2008) and distinguishes patients with anxiety disorders, OCD (Olatunji & Wolitzky-Taylor, 2009; Reiss et al., 1986; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012), PTSD (Olatunji & Wolitzky-Taylor, 2009), or health anxiety (Wheaton et al., 2012) from healthy volunteers.

AS is typically measured with the Anxiety Sensitivity Index (ASI;

* Corresponding author at: Altrecht Academic Anxiety Centre, Altrecht Mental Health Centre, Nieuwe Houtenseweg 12, 3524 SH Utrecht, the Netherlands.
*E-mail address:* c.m.b.kwee@gmail.com (C.M.B. Kwee).

[1] The term 'health anxiety' refers to DSM-IV hypochondriasis and DSM-5 illness anxiety disorder, following, for example, Cooper et al. (2017). In DSM-5, hypochondriasis was replaced by illness anxiety disorder and somatic symptom disorder (Newby, Hobbs, Mahoney, Wong, & Andrews, 2017). Olatunji, Deacon, and Abramowitz (2009) argued that hypochondriasis has cognitive and behavioral mechanisms in common with anxiety disorders, so that it is appropriate to describe it as 'health anxiety'.

https://doi.org/10.1016/j.jocrd.2018.11.003
Received 25 June 2018; Received in revised form 3 October 2018; Accepted 8 November 2018
Available online 13 November 2018
2211-3649/ © 2018 Elsevier Inc. All rights reserved.

Peterson & Reiss, 1992) or its revisions (Olatunji & Wolitzky-Taylor, 2009): the Anxiety Sensitivity Index-Revised (ASI-R; Taylor & Cox, 1998a, 1998b) and the Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007). In contrast to the ASI and ASI-R, the ASI-3 has a stable three-factor structure (Taylor et al., 2007). In the ASI-3, beliefs about adverse consequences of experiencing anxiety are broken down to somatic concerns, cognitive concerns, and social concerns (Farris et al., 2015; Kemper et al., 2012; Reiss, 1991; Taylor et al., 2007; Wheaton et al., 2012). Cognitive and social concerns can be more reliably measured with the ASI-3 than with the ASI (Taylor et al., 2007).

The reasons why one may hypothesize that high scores on the ASI-3 predict low treatment efficacy are as follows: In CBT, repeated exposures to the cues that induce anxiety provide new learning experiences to the patient. This allows for correction of catastrophic misinterpretations (Craske, 2009), necessary to interrupt the vicious cycle that maintains anxiety symptoms (Olatunji et al., 2014). High AS, however, is thought to contribute to avoidance of situations and stimuli that provoke anxiety (Reiss, 1991) and to a reluctance to engage in exposures in therapy (Blakey et al., 2017; Boettcher, Brake, & Barlow, 2016). Not completing at least one exposure was associated with worse treatment outcomes in 1004 patients with panic disorder, generalized anxiety disorder, social anxiety disorder, or PTSD (Glenn et al., 2013). Practicing in between sessions with facing feared situations and refraining from avoidance behaviors is an integral part of CBT (Simpson et al., 2011). This, too, may be especially difficult for patients with high AS. Unfortunately, lower quantity and quality of "homework" compliance predicted worse treatment outcomes, controlling for baseline severity in panic disorder (Schmidt & Woolaway-Bickel, 2000) and in OCD (Simpson et al., 2011). Olatunji et al. (2014) theorize that lower homework compliance with exposure assignments might explain worse response to CBT for health anxiety as well. In sum, then, AS is thought to predict worse treatment outcome in OCD, anxiety disorders, health anxiety, and PTSD.

To date, four studies addressed AS as a potential predictor of treatment outcome (Blakey et al., 2017; Ino et al., 2017; Nowakowski, Rowa, Antony, & Mccabe, 2016; Wolitzky-Taylor, Arch, Rosenfield, & Craske, 2012). Blakey et al. tested whether AS predicted outcome of CBT in OCD patients. They studied $N = 187$ patients who received residential CBT for OCD. Assessments included the ASI, the Dimensional Obsessive-Compulsive Scale (DOCS; Abramowitz et al., 2010), a measure of OCD symptom severity, and the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996), a measure of depressive symptom severity. Pretreatment OCD and depression severity, established predictors of attenuated response to CBT for OCD, were controlled for. It was found that higher pretreatment AS predicted higher posttreatment OCD symptom severity. AS accounted for 5% of the variance in posttreatment OCD symptom severity, with a $p$-value of .049 and Cohen's $f^2 = 0.08$. The individual ASI subscales did not predict unique variance in posttreatment DOCS scores. The authors suggested that engaging in exposure assignments would be more difficult for high AS individuals and concluded that targeting general AS could enhance treatment response.

In a different study, it was tested whether AS dimensions would predict dimensions of general psychopathology after CBT for panic disorder (Ino et al., 2017). The sample consisted of $N = 118$ patients with a primary diagnosis of panic disorder with or without agoraphobia. Patients received 10 sessions of CBT for panic disorder in a group format. AS dimensions were measured with the ASI, dimensions of general psychopathology with the Symptom Checklist-90 Revised (SCL-90-R; Derogatis, 1975a), and panic disorder severity with the Panic Disorder Severity Scale (PDSS; Shear et al., 1997). Next to dimensions of AS, pretreatment dimensions of general psychopathology, pretreatment panic disorder severity, age and sex were entered as predictors. Higher pretreatment ASI social concerns predicted higher posttreatment scores on several SCL-90-R subscales, whereas higher pretreatment ASI cognitive concerns scores predicted lower scores and ASI somatic concerns was not a significant predictor. The authors concluded that it may be useful to direct more attention to AS social and cognitive concerns to improve psychiatric symptoms.

In a third study, Wolitzky-Taylor et al. (2012) evaluated potential moderators of treatment outcome of CBT and acceptance and commitment therapy (ACT) for patients that met the criteria for an anxiety disorder, OCD, or PTSD. $N = 49$ patients completed 12 sessions of CBT and were included in the analyses. Treatment outcome was operationalized as severity of general distressing experiences of anxiety and measured with the Mood and Anxiety Symptom Questionnaire-General Anxiety Subscale (MASQ-GA; Watson & Clark, 1991). Wolitzky-Taylor et al. (2012) tested whether higher pretreatment AS, measured with the ASI, would be associated with worse treatment outcome. They controlled for pretreatment MASQ-GA and explored the possibility of a non-linear association. It was found that $ASI^2$ interacted with treatment condition. High and low pretreatment ASI scores were associated with worse treatment outcome in CBT, compared to ASI scores near to the mean of the sample. The authors concluded that in high AS, anxiety-related concerns may be so fixed that they are difficult to modify with CBT. In low AS, these concerns may be so unimportant that they are not a relevant treatment target. Both would result in less improvement with CBT.

Rather than focusing on the prognostic value of pretreatment AS, Nowakowski et al. (2016) examined pre-to posttreatment change in AS as a predictor of treatment outcome in 108 social anxiety disorder and 88 panic disorder patients who received 12 sessions of group CBT. AS was measured with the ASI, social anxiety symptoms with the Social Phobia Inventory (SPIN; Connor et al., 2000), and panic symptoms with the PDSS (Shear et al., 1997). Depressive symptoms were assessed with the depression subscale of the short version of the Depression Anxiety Stress Scales (DASS-21; Lovibond & Lovibond, 1995). Nowakowski et al. hypothesized that decrease in ASI somatic concerns predicted treatment outcome in panic disorder beyond pretreatment panic symptoms and pre- to posttreatment decrease in depressive symptoms. It was hypothesized that decrease in ASI social concerns would have prognostic significance for treatment outcome in social anxiety disorder. As expected, the ASI somatic concerns dimension was relevant for the prediction of treatment outcome in panic disorder. Decrease in both ASI social and somatic concerns predicted treatment outcome in social anxiety disorder. The authors concluded that AS dimensions differentially influence anxiety disorders.

In sum, we discussed four studies which empirically tested whether AS would predict treatment outcome of CBT for OCD and anxiety (related) disorders. While these studies are suggestive, there is room for a critical replication: First, in all studies AS was measured with the ASI, which was criticized because of its unstable factor structure and questionable content validity of two of its dimensions (Taylor et al., 2007). Second, with 5% of the variance in posttreatment symptom severity ascribed to AS (Blakey et al., 2017), the conclusion that targeting AS could enhance treatment response is overstated. Third, not correcting for multiple testing, an inadequate sample size (Ino et al., 2017) and exploratory testing (Wolitzky-Taylor et al., 2012) may have led to chance findings that will not replicate to other samples. Fourth, Nowakowski et al. (2016) used ASI change scores in the prediction of treatment outcome, but did not control for pre- to posttreatment change in anxiety symptoms. It has been repeatedly demonstrated that AS and anxiety symptoms are related. A failure to tease apart these factors hampers the interpretation of the prognostic value of AS.

The first aim of the present study was to replicate the findings of Blakey et al. (2017) that higher AS predicts worse treatment outcome in OCD patients. We hypothesized that higher pretreatment AS would predict worse treatment outcome. Second, theoretically this effect should be observed for all anxiety (related) disorders treated with exposure therapies. Therefore, the second hypothesis was that AS would predict worse treatment outcome for patients with a primary anxiety disorder, health anxiety, or PTSD. Finally, there is the issue of treatment

length. As Keeley, Storch, Merlo, and Geffken (2008) noticed, treatment length is fixed in many controlled studies. Worse end-state functioning in patients with severe initial symptoms (de Haan et al., 1997; Keeley et al., 2008), comorbidity (Dreessen & Arntz, 1998), and high AS (Wolitzky-Taylor et al., 2012) may be due to this restriction. The variability of treatment length in the present sample allowed for studying AS as its predictor. Considering the difficulties with (learning from) exposure assignments that we expected for high AS individuals, our third hypothesis was that higher AS would predict longer treatment length.

These hypotheses were tested in patients with anxiety (related) disorders treated at an outpatient Anxiety clinic between 2007 and 2016. Patients received psychological treatment which included confrontations with feared stimuli and situations, supplemented with medication if clinically indicated. AS was measured with the ASI-3 (Taylor et al., 2007). Treatment outcome was measured with the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989) and the Brief Symptom Inventory (BSI; de Beurs & Zitman, 2006).

## 2. Method

### 2.1. Sample

Patients were referred between November 2007 and January 2016. After the intake interview, as part of the full intake procedure, primary[2] and comorbid diagnoses were established through the Dutch translation (van Groenestijn, Akkerhuis, Kupka, Schneider & Nolen, 1998) of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1996), conducted by trained interviewers. A power analysis conducted with G*Power (Faul, Erdfelder, Buchner, & Lang, 2009) indicated that for a power of 80% and Cohen's $f^2 = 0.08$ for the local effect of AS on treatment outcome (based on the study of Blakey et al., 2017) a sample of 107 patients is required. This requirement was met for patients with a primary OCD diagnosis ($n = 110$). Sample sizes of patients with a primary anxiety disorder, health anxiety, or PTSD, were too small to answer the research question for each disorder separately. Therefore, we considered these disorders together ($n = 285$).[3] To detect a small effect of pretreatment AS predicting treatment length, a sample size of 395 patients is required for a power of 80%. With treatment length known for 394 patients, this requirement was nearly met.

Most patients (63%) identified as female. On average, patients were 34.3 ($SD = 10.3$) years old. The majority (75%) had received previous treatment. Within this group, more than half (at least 53%) had received CBT. The remainder received other psychological and/or pharmacological treatments. Comorbidity rates are provided in Table 1. Patients who filled in the Y-BOCS reported pretreatment symptoms ($M = 21.51$, $SD = 8.06$) in the moderate to severe range (van Balkom, de Beurs, Hovens, & van Vliet, 2004). Pretreatment BSI scores ($M = 1.34$, $SD = 0.68$) indicated clinically significant general symptom severity (de Beurs & Zitman, 2006). Pretreatment ASI-3 scores ($M = 24.06$, $SD = 13.59$) were clinically significant as well (Taylor et al., 2007; Wheaton et al., 2012). A paired samples $t$-test revealed that posttreatment Y-BOCS scores ($M = 13.52$, $SD = 8.39$) were significantly lower than pretreatment scores, $t(104) = 11.78$, $p < .001$, $d = 0.99$, a large effect. Posttreatment BSI scores ($M = 0.90$, $SD = 0.68$) were significantly lower than pretreatment scores, $t(394) = 15.33$, $p < .001$, $d = 0.64$, a medium to large effect. Likewise, posttreatment ASI-3 scores ($M = 16.59$, $SD = 12.52$) were significantly lower than pretreatment scores, $t(345) = 11.55$, $p < .001$, $d = 0.54$, a medium effect. Pre- and posttreatment BSI and ASI scores, split by primary disorder, are presented in Table 2.

The complete sample used in the present study ($n = 395$) was compared to patients who were excluded because of missing values in BSI and/or ASI ($n = 832$) with respect to comorbidity, leaving therapy prematurely, pretreatment general symptom severity and pretreatment AS. A Chi square test revealed that there was no significant association between missingness and having one or more comorbid disorders, $\chi^2(1) = 0.48$, $p = .49$. There was, however, a significant association between missingness and leaving therapy prematurely, $\chi^2(1) = 80.70$, $p < .001$. The odds of leaving treatment prematurely was 46.06 times higher for patients with missing values than for patients without missing values. Because of the issue of non-normality and different group sizes, the non-parametric Kruskal-Wallis test was used to compare patients with and without missing values on pretreatment general symptom severity and pretreatment AS. Patients with missing values had higher pretreatment BSI scores ($M = 1.56$, $SD = 0.78$) than patients with no missing values ($M = 1.34$, $SD = 0.68$), $H(1) = 19.23$, $p < .001$, $\varepsilon^2 = 0.02$, a small effect. Patients with and patients without missing values did not differ in pretreatment ASI-3 scores, $H(1) = 1.31$, $p = .25$.

### 2.2. Measures

After referral patients received per e-mail a link to generic measures of psychopathology and daily functioning, including the BSI and the ASI-3. Disorder specific measures such as the Y-BOCS were sent to patients depending on primary diagnosis. The questionnaires were filled in on patients' personal computers outside of the clinic. After termination of treatment, patients filled in the measures again.

#### 2.2.1. Obsessive-compulsive symptom severity

Obsessive compulsive symptom severity in the past week was measured with a self-report version of the Y-BOCS (Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989), which consists of 10 items. Ratings were provided on a 5-point Likert scale with a range from 0 ("none") to 4 ("extreme"). From 2012 onward, a more fine-grained 11-point scale was utilized for the items measuring time spent on obsessions and compulsions. For the present study these answers were recoded to the original 5-point scale. The instrument is sensitive to measure change associated with treatment (van Oppen, Emmelkamp, van Balkom, & van Dyck, 1995). Interrater-reliability, convergent and divergent validity were demonstrated in a Dutch clinical sample (Arrindell, de Vlaming, Eisenhardt, van Berkum, & Kwee, 2002). Internal consistencies in the current sample were good; $\alpha = .88$ for the obsessions subscale, $\alpha = .91$ for the compulsions subscale, and $\alpha = .89$ for the total scale.

#### 2.2.2. General symptom severity

General symptom severity was measured with the Dutch translation (de Beurs & Zitman, 2006) of the BSI (Derogatis, 1975b), a 53-item shortened version of the SCL-90-R (Derogatis, 1975a). The BSI measures 9 dimensions of psychopathology: anxiety, agoraphobia, depression, somatization, cognitive-performance deficits, interpersonal sensitivity, hostility, paranoid ideation, and psychoticism. For each item, patients were asked how much they were bothered by that problem during the past week. Answers were provided on a 5-point Likert scale ranging from 0 ("not at all") to 4 ("very much"). The BSI was found to be as sensitive to measure improvement with therapy as disorder specific measures (van der Mheen, ter Mors, van den Hout, & Cath, 2018). De Beurs and Zitman demonstrated concurrent, convergent, and divergent validity, an acceptable to good test-retest reliability, and acceptable to good internal consistencies. The psychoticism subscale demonstrated questionable internal consistency in the current sample ($\alpha = .68$). Internal consistency was acceptable to good for the other

[2] Primary in severity or relevance to treatment, as judged by the interviewer.

[3] For the sake of brevity, we refer to anxiety disorders, health anxiety, and PTSD as mixed anxiety disorders throughout the remainder of the text.

**Table 1**
Psychiatric comorbidity rates.

| | | | Comorbid disorder | | | | |
|---|---|---|---|---|---|---|---|
| Primary disorder | Anxiety | PTSD | HA | OCD | Mood | Other | ≥ 1 |
| OCD ($n$ = 110) | 46 (42%) | 6 (5%) | 7 (6%) | NA | 51 (46%) | 23 (21%) | 77 (70%) |
| SAD ($n$ = 89) | 19 (21%) | 3 (3%) | 5 (6%) | 7 (8%) | 66 (74%) | 7 (8%) | 72 (81%) |
| PD ($n$ = 82) | 32 (39%) | 3 (4%) | 4 (5%) | 5 (6%) | 45 (55%) | 5 (6%) | 59 (72%) |
| GAD ($n$ = 44) | 23 (52%) | 1 (2%) | 5 (11%) | 6 (14%) | 26 (59%) | 1 (2%) | 33 (75%) |
| PTSD ($n$ = 40) | 20 (50%) | 0 | 0 | 0 | 28 (70%) | 4 (10%) | 34 (85%) |
| HA ($n$ = 17) | 11 (65%) | 0 | NA | 1 (6%) | 7 (41%) | 1 (6%) | 12 (71%) |
| SP ($n$ = 13) | 6 (46%) | 0 | 1 (8%) | 1 (8%) | 4 (31%) | 1 (8%) | 9 (69%) |
| Total ($n$ = 395) | 157 (40%) | 13 (3%) | 22 (6%) | 20 (5%) | 227 (57%) | 42 (11%) | 296 (75%) |

*Note.* OCD = obsessive-compulsive disorder; SAD = social anxiety disorder; PD = panic disorder; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; HA = health anxiety; SP = specific phobia.

**Table 2**
Pretreatment and posttreatment BSI and ASI-3 scores.

| | Pretreatment BSI | Posttreatment BSI | Pretreatment ASI-3 | Posttreatment ASI-3 |
|---|---|---|---|---|
| Primary disorder | *M* (*SD*) | *M* (*SD*) | *M* (*SD*) | *M* (*SD*) |
| OCD ($n$ = 110) | 1.30 (0.68) | 0.93 (0.68)* | 21.48 (13.93) | 16.46 (11.68)* |
| SAD ($n$ = 89) | 1.41 (0.55) | 0.89 (0.57)* | 24.38 (20.99) | 16.53 (11.13)* |
| Panic disorder ($n$ = 82) | 1.31 (0.65) | 0.91 (0.65)* | 27.43 (14.14) | 18.90 (13.81)* |
| GAD ($n$ = 44) | 1.27 (0.78) | 0.84 (0.79)* | 24.82 (13.65) | 14.93 (13.48)* |
| PTSD ($n$ = 40) | 1.63 (0.83) | 0.98 (0.84)* | 23.03 (16.17) | 13.91 (13.64)* |
| Health anxiety ($n$ = 17) | 1.17 (0.69) | 0.89 (0.85)NS | 28.65 (11.97) | 20.69 (14.15)NS |
| Specific phobia ($n$ = 13) | 0.91 (0.63) | 0.55 (0.47)NS | 17.15 (11.27) | 11.58 (8.98) NS |
| Total sample ($n$ = 395) | 1.34 (0.68) | 0.90 (0.68)* | 24.06 (13.59) | 16.59 (12.52)* |

*Note.* BSI = Brief Symptom Inventory; ASI-3 = Anxiety Sensitivity Index-3; OCD = obsessive-compulsive disorder; SAD = social anxiety disorder; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder.
* $p < .001$; NS = not significant at $\alpha = 0.003$ (Bonferroni corrected for multiple testing).

**Table 3**
Results from the hierarchical linear regression predicting posttreatment Y-BOCS scores from pretreatment ASI-3 scores, controlling for pretreatment Y-BOCS and BSI scores.

| | | ***B* (95% CI)** | ***SEB*** | ***β*** | ***p*** |
|---|---|---|---|---|---|
| Block 1 | Constant | − 1.69 [− 4.45, 1.20] | 1.61 | | .29 |
| $\Delta R^2 = .43$ | Pretreatment Y-BOCS | 0.60 [0.42, 0.78] | 0.09 | 0.58 | .001 |
| $p < .001$ | Pretreatment BSI | 1.80 [− 0.48, 4.23] | 1.15 | 0.15 | .12 |
| Block 2 | Constant | − 1.49 [− 4.34, 1.50] | 1.66 | | .39 |
| $\Delta R^2 < .001$ | Pretreatment Y-BOCS | 0.59 [0.41, 0.76] | 0.09 | 0.57 | .001 |
| $p = .77$ | Pretreatment BSI | 2.06 [− 0.72, 4.82] | 1.31 | 0.17 | .11 |
| | Pretreatment ASI − 3 | − 0.02 [− 0.14, 0.10] | 0.06 | − 0.03 | .78 |

*Note.* Y-BOCS = Yale-Brown Obsessive-Compulsive Scale; BSI = Brief Symptom Inventory; ASI-3 = Anxiety Sensitivity Index-3. 95% bias-corrected and accelerated confidence intervals reported in parentheses. Confidence intervals and standard errors based on 1000 bootstrap samples.

subscales ($\alpha$ = .79 to .88) and excellent for the BSI total score ($\alpha$ = .96).

#### 2.2.3. Anxiety sensitivity

AS was measured with the ASI-3 (Taylor et al., 2007), a self-report measure with three 6-item subscales: somatic concerns, social concerns, and cognitive concerns. Patients indicated how much they agreed with each statement on a 5-point scale (0 = very little, 4 = very much). Taylor et al. (2007) constructed the ASI-3 by selecting items from the ASI-Revised (Taylor & Cox, 1998a, 1998b) that correspond to only one of the three domains to emphasize content validity. The average total ASI-3 score was 10.7 for $n$ = 536 healthy individuals in the Netherlands (Taylor et al., 2007) and around 29 for patients with anxiety (related) disorders (in samples from the United States and Canada; Taylor et al., 2007; Wheaton et al., 2012). Taylor et al. (2007) demonstrated factorial validity of the ASI-3 in 6 replication samples, as well as convergent, discriminant, and criterion related validity. Internal consistencies of the scales were acceptable to good. The ASI-3 demonstrated good internal consistency in the current sample ($\alpha$ = .91 for the somatic subscale, $\alpha$ = .81. for the social subscale, $\alpha$ = .88 for the cognitive subscale, and $\alpha$ = .90 for the total score).

### 2.3. Treatment

Patients received treatment at the Academic Anxiety outpatient clinic of Altrecht Mental Health Centre in the Netherlands that treats patients suffering from severe anxiety disorders, health anxiety, obsessive-compulsive spectrum disorders, and PTSD. These patients have either not profited from earlier state of the art pharmacological and/or psychological treatment, or the risk of a clinically unfavorable prognosis after treatment in a non-academic clinic appears high. Patients received time-limited CBT with an average of 21.0 treatment sessions (*SD* = 13.6), consisting of mainly exposure therapy. In addition to discouraging safety behaviors, this real-life contact with situations or objects to learn that feared catastrophes are not occurring and/ or that

anxiety can be tolerated was a crucial common element in the treatments patients received. PTSD was mainly treated with eye movement desensitization and reprocessing (EMDR). In EMDR, patients need to focus on a disturbing image or memory as well as on emotions and cognitive elements connected with it whilst making eye movements. EMDR is at least as effective as CBT for treating PTSD (Bisson & Andrew, 2008; Chen, Zhang, Hu, & Liang, 2015).

Care was mainly delivered face-to-face, with the option of blended e-health. The choice for individual and/ or group treatment was made by taking in consideration patient preferences concerning therapy in individual or group format, the clinician's judgement and pragmatic reasons such as waitlists and the patient's availability. In the case of comorbid depression or partial or no (anticipated) recovery with only psychological treatment, treatment was supplemented with medication for approximately 60% of patients, mainly in the form of selective serotonin reuptake inhibitors (SSRIs). Treatment focused on the primary disorder had a positive impact on comorbid symptoms in some patients. In that case, not all diagnosed problems needed to be addressed. Other patients were left with significant (comorbid) psychopathology. For these patients either a different treatment focus was chosen, or referral to another clinic took place.

### 2.4. Data analyses

Data were analyzed with IBM SPSS Statistics Data Editor version 24. Hierarchical linear regressions were run at an alpha level of .05 to answer the research questions. 105 patients had pre- and posttreatment Y-BOCS data available. For these patients, posttreatment Y-BOCS served as the dependent variable with pretreatment Y-BOCS entered as predictor in the first block. Separate analyses, in OCD as well as in mixed anxiety disorders, were run with posttreatment BSI as dependent variable. Pretreatment BSI was entered in the first block to consider the potential effect of pretreatment presence and severity of symptoms on treatment outcome (Olatunji, Cisler, & Tolin, 2010; Taylor et al., 2012). Pretreatment ASI-3 was entered in the second block. Number of treatment sessions served as dependent variable in the prediction of treatment length. Item content of the social concerns dimension overlaps with the diagnostic criteria of social anxiety disorder (American Psychiatric Association (APA), 1994, 2013). Likewise, the somatic and cognitive concerns dimensions of AS are inextricably linked to the symptomatology of panic disorder (APA, 1994, 2013; Clark, 1986). Despite the unstandardized nature of treatment, we feel safe to assume that AS concerns were addressed in panic disorder treatments. Therefore, the prognostic value of AS in these patients may be obscured. To test this possibility, we conducted exploratory analyses excluding patients with a primary panic disorder or social anxiety disorder.

## 3. Results

Data were first screened for linearity, univariate and multivariate outliers, outliers in the prediction, influential cases, independence and normality of error terms, homoscedasticity, and multicollinearity.

### 3.1. Prediction of treatment outcome in OCD

We hypothesized that pretreatment AS would predict posttreatment OCD symptom severity and general symptom severity, controlling for pretreatment general symptom severity. Given the concern of heteroscedasticity of residuals, confidence intervals for the regression coefficients were bootstrapped. Results are displayed in Tables 3 and 4. Pretreatment ASI-3 scores did not lead to a significant improvement in the prediction of posttreatment Y-BOCS scores ($\Delta R^2 < .001$, $p = .77$) or BSI scores ($\Delta R^2 = .002$, $p = .51$).

**Table 4**
Results from the hierarchical linear regression predicting posttreatment BSI scores from pretreatment ASI-3 scores, controlling for pretreatment BSI scores, in OCD patients.

| | | *B* (95% CI) | *SEB* | *β* | *p* |
|---|---|---|---|---|---|
| Block 1 | Constant | 0.05 [− 0.11, 0.22] | 0.08 | | .55 |
| $\Delta R^2 = .47$ | Pretreatment BSI | 0.68 [0.53, 0.81] | 0.07 | 0.69 | .001 |
| $p < .001$ | | | | | |
| Block 2 | Constant | 0.06 [− 0.09, 0.22] | 0.08 | | .44 |
| $\Delta R^2 = .002$ | Pretreatment BSI | 0.72 [0.49, 0.96] | 0.11 | 0.73 | .001 |
| $p = .51$ | Pretreatment ASI-3 | − 0.003 [− 0.01, 0.01] | 0.01 | − 0.06 | .57 |

*Note.* BSI = Brief Symptom Inventory; ASI-3 = Anxiety Sensitivity Index-3. 95% bias-corrected and accelerated confidence intervals reported in parentheses. Confidence intervals and standard errors based on 1000 bootstrap samples.

**Table 5**
Results from the hierarchical linear regression predicting posttreatment BSI scores from pretreatment ASI-3 scores, controlling for pretreatment BSI scores, in mixed anxiety disorders.

| | | *B* (95% CI) | *SEB* | *β* | *p* |
|---|---|---|---|---|---|
| Block 1 | Constant | 0.02 [− 0.11, 0.14] | 0.07 | | .83 |
| $\Delta R^2 = .41$ | Pretreatment BSI | 0.64 [0.53, 0.76] | 0.06 | 0.64 | .001 |
| $p < .001$ | | | | | |
| Block 2 | Constant | − 0.01[− 0.15, 0.13] | 0.07 | | .91 |
| $\Delta R^2 = .002$ | Pretreatment BSI | 0.61 [0.48, 0.74] | 0.07 | 0.61 | .001 |
| $p = .37$ | Pretreatment ASI-3 | 0.003 [− 0.003,0.01] | 0.003 | 0.05 | .38 |

*Note.* BSI = Brief Symptom Inventory; ASI-3 = Anxiety Sensitivity Index-3. 95% bias-corrected and accelerated confidence intervals reported in parentheses. Confidence intervals and standard errors based on 1000 bootstrap samples.

### 3.2. Prediction of treatment outcome in mixed anxiety disorders

There were univariate outliers in posttreatment BSI scores in our subsample of mixed anxiety disorders, identified by z-scores of 3.34, 3.39, and 3.42. There were, however, no cases with undue influence on the model (Cooks distance < 1), so we decided to retain these cases. Given the concern of heteroscedasticity, confidence intervals for the regression coefficients were bootstrapped. The results from the regression are shown in Table 5. Pretreatment ASI-3 scores did not contribute significantly to the prediction of posttreatment BSI scores in patients with mixed anxiety disorders, $\Delta R^2 = .002$, $p = .37$. The results of the exploratory analysis excluding patients with a primary panic disorder or social anxiety disorder resembled those from the primary analysis. Pretreatment ASI-3 scores did not contribute significantly to the prediction of posttreatment BSI scores, $\Delta R^2 = .003$, $p = .42$.[4]

### 3.3. Prediction of treatment length in OCD and mixed anxiety disorders

Inspection of z-scores revealed that there were three univariate outliers in number of treatment sessions, with values 3.95, 4.17, and 6.89. No cases with undue influence on the model were found (Cook's distance < 1), so the outliers were retained. There were 12 cases with standardized residual > 2.58, which suggests the model is a poor fit to the data. Given the concern of non-normality of residuals, confidence intervals for the regression coefficients were bootstrapped. Results are shown in Table 6. Pretreatment ASI-3 scores did not contribute

[4] Additional analyses were performed with BSI subscales anxiety, agoraphobia, and somatization. These subscales were thought to be more closely related to AS than BSI total score. Neither in OCD, mixed anxiety disorders, or mixed anxiety disorders excluding panic disorder and social anxiety disorder did ASI-3 scores predict treatment outcome as measured with these subscales.

**Table 6**
Results from the hierarchical linear regression predicting treatment length from pretreatment ASI-3 scores, controlling for pretreatment BSI scores, in OCD and mixed anxiety disorders.

| | | *B* (95% CI) | *SEB* | *β* | *p* |
|---|---|---|---|---|---|
| Block 1 | Constant | 17.62 [15.15, 20.35] | 1.31 | | .001 |
| $\Delta R^2 = .02$ | Pretreatment BSI | 2.55 [0.85, 4.18] | 0.89 | 0.13 | .01 |
| $p = .01$ | | | | | |
| Block 2 | Constant | 18.02 [15.44, 20.93] | 1.34 | | .001 |
| $\Delta R^2 = .002$ | Pretreatment BSI | 3.28 [0.77, 5.72] | 1.27 | 0.16 | .01 |
| $p = .39$ | Pretreatment ASI-3 | − 0.06 [− 0.18, 0.06] | 0.06 | − 0.06 | .35 |

*Note.* BSI = Brief Symptom Inventory; ASI-3 = Anxiety Sensitivity Index-3. 95% bias-corrected and accelerated confidence intervals reported in parentheses. Confidence intervals and standard errors based on 1000 bootstrap samples.

significantly to the prediction of posttreatment BSI scores, $\Delta R^2 = .002$, $p = .39$. To explore whether excluding patients with a primary panic disorder or social anxiety disorder would lead to different results, we conducted the regression again excluding these patients. Pretreatment ASI-3 scores did not contribute significantly to prediction of posttreatment BSI scores, $\Delta R^2 = .003$, $p = .38$.

## 4. Discussion

The hypotheses that AS would predict treatment outcome and treatment length in OCD, anxiety disorders, health anxiety, or PTSD were not supported by the data. These null findings contradict earlier studies, in which AS predicted treatment outcome in CBT for OCD (Blakey et al., 2017), for panic disorder (Ino et al., 2017), for panic disorder and social anxiety disorder (Nowakowski et al., 2016), and for OCD, anxiety disorders, and PTSD (Wolitzky-Taylor et al., 2012). A first explanation for the absence of a prognostic value of ASI-3 scores may be that, over the course of treatment, clinicians may have reduced patients' concerns about anxiety symptoms. AS can be effectively targeted by cognitive-behavioral techniques (Boswell et al., 2013; Schmidt, Norr, Allan, Raines, & Capron, 2017; Smits, Berry, Tart, & Powers, 2008). For example, by talking about their anxiety symptoms to a therapist, the patient can experience that he/she is not negatively evaluated because of them. Despite the limitation of unstandardized treatment content, we assumed that AS was an issue at least in panic disorder treatments. However, excluding these patients from the analyses did not change our results. In fact, in our sample ASI-3 scores decreased from pretreatment to posttreatment across diagnoses. Unfortunately, it is unclear whether this decrease in AS preceded change in symptom severity.

A second possible explanation points to differences in clinical characteristics. For example, the sample of Blakey et al. (2017) received residential treatment, whereas the sample in the present study received outpatient care. In the latter, patients' progress with CBT is even more dependent on patients doing their "homework" without the direct support of a therapist. In turn, clinicians in an outpatient clinic may be more alert on beliefs that interfere with engaging in exposure assignments, challenging these beliefs in time. Third, the reason that we failed to replicate earlier findings might be statistical issues in the studies that reported them. Without controlling for change in anxiety symptoms, the prognostic value of pre- to posttreatment change in AS (Nowakowski et al., 2016) remains uncertain. The large number of variables in the analyses of Wolitzky-Taylor et al. (2012) and Ino et al. (2017) in combination with their modest sample sizes could result in overfitting. That is, the results may reflect noise in the sample at hand and may not replicate in other samples (e.g., Babyak, 2004). Not certain what to expect, Wolitzky-Taylor et al. (2012) explored the prognostic value of both AS and a quadratic AS term. They noticed that their inclusion of several predictors, with and without a theoretical basis, could have led to Type I error. In our data there was neither a linear nor a non-linear association between AS and treatment outcome.

It can of course not be ruled out that our study is a false non-replication. Insufficient power and bias in the replication effort (Ioannidis, 2012) increase the chance of making this Type II error. Our power calculations suggest that our analyses were adequately powered. That said, by relying on a single effect size value reported by Blakey et al. (2017) and disregarding sampling variability, we could have overestimated the obtained power (Maxwell, Lau, & Howard, 2015). Complete case analysis could have led to biased results. Our conclusions may only apply to a specific subgroup; patients who were included in the analyses were characterized by lower pretreatment general symptom severity than patients who were excluded because of missing values. Nevertheless, the size of this effect was small; in both groups scores were mostly in the clinical range. In addition, groups did not differ in having one or more comorbid disorders. Patients who were not included in the analyses were more likely to leave therapy prematurely but did not differ in AS, our variable of interest.

Our null findings cast doubt on the credibility of earlier scientific results. Of course, multiple replication studies are needed to conclude that an effect is non-existent (Maxwell et al., 2015). But note that for the ASI-3 to be clinically helpful in predicting treatment results, the effects should not only be statistically significant but also clinically meaningful. In the study of Blakey et al. (2017) AS predicted 5% of the variance in treatment outcome scores. In the study of Wolitzky-Taylor et al. (2012), the best and the worse treatment outcome that could be ascribed to variability in AS both fell within 1 *SD* from average experiences of anxiety in the general population (Schalet, Cook, Choi, & Cella, 2014). In the study of Ino et al. (2017), 1 *SD* increase in ASI subscale scores predicted from |0.11| to |0.32| *SD* variation in posttreatment psychiatric and psychosomatic symptom severity. The clinical relevance of this finding remains obscure. From these figures it seems unlikely that the ASI will come to serve as a useful clinical tool in the prediction of therapy success or failure.

Future research on treatment outcome with CBT may perhaps not focus only on patient characteristics. For example, Wolitzky-Taylor et al. (2018) focused on barriers to delivering and receiving exposure-based CBT in anxiety disorders and PTSD at the level of the patient, the clinician, the organization, and the service system, from the perspectives of patients, providers, and clinic administrators. Shafran et al. (2009) and Waller (2009) pointed to the role of clinicians in the suboptimal delivery of CBT. For example, clinician's adherence to the CBT protocol interacted with panic disorder patients' motivation to change to predict treatment outcome ($N = 205$; Huppert, Barlow, Gorman, Shear, & Woods, 2006). Predicting treatment effects may be improved by including variables other than patient features.

## Acknowledgement

We are very grateful to Mathilde Huisman-van Dijk, who provided the data and supervised the statistical analyses.

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

## References

Abramowitz, J. S., Deacon, B. J., Olatunji, B. O., Wheaton, M. G., Berman, N. C., Losardo, D., ... Hale, L. R. (2010). Assessment of obsessive-compulsive symptom dimensions: Development and evaluation of the dimensional obsessive-compulsive scale. *Psychological Assessment, 22*, 180–198. https://doi.org/10.1037/a0018260.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders* ((4th ed.)). Washington DC: American Psychiatric Association.

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* ((5th ed.)). Washington DC: American Psychiatric Association.

Arrindell, W. A., de Vlaming, I. H., Eisenhardt, B. M., van Berkum, D. E., & Kwee, M. G. T. (2002). Cross-cultural validity of the Yale–Brown Obsessive Compulsive Scale. *Journal of Behavior Therapy and Experimental Psychiatry, 33*, 159–176. https://doi.

org/10.1016/S0005-7916(02)00047-2.
Babyak, M. A. (2004). What you see may not be what you get: A brief, nontechnical introduction to overfitting in regression-type models. *Psychosomatic Medicine, 66*, 411–421.
Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II.* San Antonio, TX: Psychological Corporation.
Bisson, J., & Andrew, M. (2008). Psychological treatment of posttraumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews, 3*, 1–82. https://doi.org/10.1002/14651858.CD003388.pub3.
Blakey, S. M., Abramowitz, J. S., Reuman, L., Leonard, R. C., & Riemann, B. C. (2017). Anxiety sensitivity as a predictor of outcome in the treatment of obsessive-compulsive disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 57*, 113–117. https://doi.org/10.1016/j.jbtep.2017.05.003.
Boettcher, H., Brake, C. A., & Barlow, D. H. (2016). Origins and outlook of interoceptive exposure. *Journal of Behavior Therapy and Experimental Psychiatry, 53*, 41–51. https://doi.org/10.1016/j.jbtep.2015.10.009.
Boswell, J. F., Farchione, T. J., Sauer-Zavala, S., Murray, H. W., Fortune, M. R., & Barlow, D. H. (2013). Anxiety sensitivity and interoceptive exposure: A transdiagnosticconstruct and change strategy. *Behavior Therapy, 44*, 417–431. https://doi.org/10.1016/j.beth.2013.03.006.
Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review, 26*, 17–31. https://doi.org/10.1016/j.cpr.2005.07.003.
Chen, L., Zhang, G., Hu, M., & Liang, X. (2015). Eye movement desensitization and reprocessing versus cognitive-behavioral therapy for adult posttraumatic stress disorder: Systematic review and meta-analysis. *Journal of Nervous and Mental Disease, 203*, 443–451. https://doi.org/10.1097/NMD.0000000000000306.
Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy, 24*, 461–470. https://doi.org/10.1016/0005-7967(86)90011-2.
Connor, K. M., Davidson, J. R. T., Churchill, E. L., Sherwood, A., Weisler, R. H., & Foa, E. (2000). Psychometric properties of the Social Phobia Inventory: New self-rating scale. *British Journal of Psychiatry, 176*, 379–386. https://doi.org/10.1192/bjp.176.4.379.
Cooper, K., Gregory, J. D., Walker, I., Lambe, S., & Salkovskis, P. M. (2017). Cognitive behaviour therapy for health anxiety: A systematic review and meta-analysis. *Behavioural and Cognitive Psychotherapy, 45*, 110–123. https://doi.org/10.1017/S1352465816000527.
Craske, M. G. (2009). *Cognitive-behavioral therapy.* Washington, DC: American Psychiatric Association.
de Beurs, E., & Zitman, F. G. (2006). De Brief Symptom Inventory (BSI): De betrouwbaarheid en validiteit van een handzaam alternatief voor de SCL-90. *Maandblad Geestelijke Volksgezondheid, 61*, 120–141.
de Haan, E., van Oppen, P., van Balkom, A. J. L. M., Spinhoven, P., Hoogduin, K. A. L., & van Dyck, R. (1997). Prediction of outcome and early vs. late improvement in OCD patients treated with cognitive behaviour therapy and pharmacotherapy. *Acta Psychiatrica Scandinavica, 96*, 354–361. https://doi.org/10.1111/j.1600-0447.1997.tb09929.x.
Derogatis, L. R. (1975aa). *The Symptom Checklist-90-R.* Baltimore, MD: Clinical Psychometric Research.
Derogatis, L. R. (1975bb). *The Brief Symptom Inventory.* Baltimore, MD: Clinical Psychometric Research.
Dreessen, L., & Arntz, A. (1998). The impact of personality disorders on treatment outcome of anxiety disorders: Best-evidence synthesis. *Behaviour Research and Therapy, 36*, 483–504. https://doi.org/10.1016/S0005-7967(98)00026-6.
Eddy, K. T., Dutra, L., Bradley, R., & Westen, D. (2004). A multidimensional meta-analysis of psychotherapy and pharmacotherapy for obsessive-compulsive disorder. *Clinical Psychology Review, 24*, 1011–1030. https://doi.org/10.1016/j.cpr.2004.08.004.
Farris, S. G., DiBello, A. M., Allan, N. P., Hogan, J., Schmidt, N. B., & Zvolensky, M. J. (2015). Evaluation of the Anxiety Sensitivity Index-3 among treatment-seeking smokers. *Psychological Assessment, 27*, 1123–1128. https://doi.org/10.1037/pas0000112.
Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods, 41*, 1149–1160. https://doi.org/10.3758/BRM.41.4.1149.
First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *User's guide for the Structured Clinical Interview for the DSM-IV Axis I disorders - research version.* New York: Biometrics Research Department, New York State Psychiatric Institute.
Glenn, D., Golinelli, D., Rose, R. D., Roy-Byrne, P., Stein, M. B., Sullivan, G., ... Craske, M. G. (2013). Who gets the most out of cognitive-behavioral therapy for anxiety disorders?: The role of treatment dose and patient engagement. *Journal of Consulting and Clinical Psychology, 81*, 639–649. https://doi.org/10.1037/a0033403.
Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Delgado, P., Heninger, G. R., & Charney, D. S. (1989). The Yale-Brown Obsessive Compulsive Scale: II. Validity. *Archives of General Psychiatry, 46*, 1012–1016. https://doi.org/10.1001/archpsyc.1989.01810110054008.
Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleishmann, R. L., Hill, C. L., ... Charney, D. S. (1989). The Yale-Brown Obsessive Compulsive Scale: I. Development, use, and reliability. *Archives of General Psychiatry, 46*, 1006–1011.
Hofmann, S. G., Asnaani, A., Vonk, I. J. J., Sawyer, A. T., & Fang, A. (2012). The efficacy of cognitive behavioral therapy: A review of meta-analyses. *Cognitive Therapy and Research, 36*, 427–440. https://doi.org/10.1007/s10608-012-9476-1.
Hofmann, S. G., & Smits, J. A. J. (2008). Cognitive-behavioral therapy for adult anxiety disorders: A meta-analysis of randomized placebo-controlled trials. *Journal of Clinical Psychiatry, 69*, 621–632.
Hong, R. Y. (2010). Neuroticism, anxiety sensitivity thoughts, and anxietysymptomatology: Insights from an experience-sampling approach. *Cognitive Therapy and Research, 24*, 254–262. https://doi.org/10.1007/s10608-009-9231-4.
Huppert, J. D., Barlow, D. H., Gorman, J. M., Shear, M. K., & Woods, S. W. (2006). The interaction of motivation and therapist adherence predicts outcome in cognitive behavioral therapy for panic disorder: Preliminary findings. *Cognitive and Behavioral Practice, 13*, 198–204. https://doi.org/10.1016/j.cbpra.2005.10.001.
Ino, K., Ogawa, S., Kondo, M., Imai, R., Ii, T., Furukawa, T. A., & Akechi, T. (2017). Anxiety sensitivity as a predictor of broad dimensions of psychopathology after cognitive behavioral therapy for panic disorder. *Neuropsychiatric Disease and Treatment, 13*, 1835–1840. https://doi.org/10.2147/NDT.S121360.
Ioannidis, J. P. A. (2012). Why science is not necessarily self-correcting. *Perspectives on Psychological Science, 7*, 645–654. https://doi.org/10.1177/1745691612464056.
Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59*, 12–19. https://doi.org/10.1037/0022-006X.59.1.12.
Keeley, M. L., Storch, E. A., Merlo, L. J., & Geffken, G. R. (2008). Clinical predictors of response to cognitive-behavioral therapy for obsessive–compulsive disorder. *Clinical Psychology Review, 28*, 118–130. https://doi.org/10.1016/j.cpr.2007.04.00.
Kemper, C. J., Lutz, J., Hock, M., Bähr, T., & Rüddel, H. (2012). Construct validity of the Anxiety Sensitivity Index-3 in clinical samples. *Assessment, 19*, 89–100. https://doi.org/10.1177/1073191111429389.
Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy, 33*, 335–343. https://doi.org/10.1016/0005-7967(94)00075-U.
Maxwell, S. E., Lau, M. Y., & Howard, G. S. (2015). Is psychology suffering from a replication crisis? What does a "failure to replicate" really mean? *AmericanPsychologist, 70*, 487–498. https://doi.org/10.1037/a0039400.
McNally, R. J. (1989). Is anxiety sensitivity distinguishable from trait anxiety? Reply to Lilienfeld, Jacob, and Turner (1989). *Journal of Abnormal Psychology, 98*, 193–194. https://doi.org/10.1037/0021-843X.98.2.193.
McNally, R. J. (2002). Anxiety sensitivity and panic disorder. *Biological Psychiatry, 25*, 938–946. https://doi.org/10.1016/S0006-3223(02)01475-0.
Newby, J. L., Hobbs, M. J., Mahoney, A. E. J., Wong, S., & Andrews, G. (2017). DSM-5 illness anxiety disorder and somatic symptom disorder: Comorbidity, correlates, and overlap with DSM-IV hypochondriasis. *Journal of Psychometric Research, 101*, 31–37. https://doi.org/10.1016/j.jpsychores.2017.07.010.
Norton, P. J., & Edwards, K. S. (2017). Anxiety sensitivity or interoceptive sensitivity: An analysis of feared bodily sensations. *European Journal of Psychological Assessment, 33*, 30–37. https://doi.org/10.1027/1015-5759/a000269.
Nowakowski, M. E., Rowa, K., Antony, M. M., & Mccabe, R. (2016). Changes in anxiety sensitivity following group cognitive-behavior therapy for social anxiety disorder and panic disorder. *Cognitive Therapy and Research, 40*, 468–478. https://doi.org/10.1007/s10608-015-9750-0.
Olatunji, B. O., Cisler, J. M., & Tolin, D. F. (2010). A meta-analysis of the influence of comorbidity on treatment outcome in the anxietydisorders. *Clinical Psychology Review, 30*, 642–654. https://doi.org/10.1016/j.cpr.2010.04.008.
Olatunji, B. O., Deacon, B. J., & Abramowitz, J. S. (2009). Is hypochondriasis an anxiety disorder? *The British Journal of Psychiatry, 194*, 481–482. https://doi.org/10.1192/bjp.bp.108.061085.
Olatunji, B. O., Kauffman, B. Y., Meltzer, S., Davis, M. L., Smits, J. A. J., & Powers, M. B. (2014). Cognitive-behavioral therapy for hypochondriasis/health anxiety: A meta-analysis of treatment outcome and moderators. *Behaviour Research and Therapy, 58*, 65–74. https://doi.org/10.1016/j.brat.2014.05.002.
Olatunji, B. O., & Wolitzky-Taylor, K. B. (2009). Anxiety sensitivity and the anxietydisorders: A meta-analytic review and synthesis. *Psychological Bulletin, 135*, 974999. https://doi.org/10.1037/a0017428.
Olthuis, J. V., Watt, M. C., & Stewart, S. H. (2014). Anxiety Sensitivity Index (ASI-3) subscales predict unique variance in anxiety and depressive symptoms. *Journal of Anxiety Disorders, 28*, 115–124. https://doi.org/10.1016/j.janxdis.2013.04.009.
Peterson, R. A., & Reiss, S. (1992). *Anxiety Sensitivity Index revised manual.* Worthington, OH: IDS Publishing.
Reiss, S. (1991). Expectancy model of fear, anxiety, and panic. *Clinical Psychology Review, 11*, 141–153. https://doi.org/10.1016/0272-7358(91)90092-9.
Reiss, S., & McNally, R. (1985). Expectancy model of fear. In S. Reiss, & R. R. Bootzin (Eds.). *Theoretical issues in behavior therapy* (pp. 107–121). New York: Academic Press.
Reiss, S., Peterson, R. A., Gursky, D. M., & McNally, R. J. (1986). Anxiety sensitivity,anxiety frequency, and the prediction of fearfulness. *Behaviour Research and Therapy, 24*, l–8. https://doi.org/10.1016/0005-7967(86)90143-9.
Rifkin, L. S., Beard, C., Hsu, K. J., Garner, L., & Björgvinsson, T. (2015). Psychometric properties of the anxiety sensitivity index-3 in an acute and heterogeneous treatment sample. *Journal of Anxiety Disorders, 36*, 99–102. https://doi.org/10.1016/j.janxdis.2015.09.010.
Schalet, B. D., Cook, K. F., Choi, S. W., & Cella, D. (2014). Establishing a common metric for self-reported anxiety: Linking the MASQ, PANAS, and GAD-7 to PROMIS Anxiety. *Journal of Anxiety Disorders, 28*, 88–96. https://doi.org/10.1016/j.janxdis.2013.11.006.
Schmidt, N. B., Mitchell, M. A., & Richey, J. A. (2008). Anxiety sensitivity as an incrementalpredictor of later anxiety symptoms and syndromes. *Comprehensive Psychiatry, 49*, 407–412. https://doi.org/10.1016/j.comppsych.2007.12.004.
Schmidt, N. B., Norr, A. M., Allan, N. P., Raines, A. M., & Capron, D. W. (2017). Arandomized clinical trial targeting anxiety sensitivity for patients with suicidalideation. *Journal of Consulting and Clinical Psychology, 85*, 596–610. https://doi.org/10.1037/ccp0000195.
Schmidt, N. B., & Woolaway-Bickel, K. (2000). The effects of treatment compliance on outcome in cognitive-behavioral therapy for panic disorder quality versus quantity. *Journal of Consulting and Clinical Psychology, 68*, 13–18. https://doi.org/10.1037/0022-006X.68.1.13.

Shafran, R., Clark, D. M., Fairburn, C. G., Arntz, A., Barlow, D. H., Ehlers, A., ... Wilson, G. T. (2009). Mind the gap: Improving the dissemination of CBT. *Behaviour Research and Therapy, 47*, 902–909. https://doi.org/10.1016/j.brat.2009.07.003.

Shear, M. K., Brown, T. A., Barlow, D. H., Money, R., Sholomskas, D. E., Woods, S. W., ... Papp, L. A. (1997). Multicenter collaborative panic disorder severity scale. *American Journal of Psychiatry, 154*, 1571–1575. https://doi.org/10.1176/ajp.154.11.1571.

Simpson, H. B., Maher, M. J., Wang, Y., Bao, Y., Foa, E. B., & Franklin, M. (2011). Patient adherence predicts outcome from cognitive-behavioral therapy in obsessive-compulsive disorder. *Journal of Consulting and Clinical Psychology, 79*, 247–252. https://doi.org/10.1037/a0022659.

Smits, J. A. J., Berry, A. C., Tart, C. D., & Powers, M. B. (2008). The efficacy of cognitive-behavioral interventions for reducing anxiety sensitivity: A meta-analytic review. *Behaviour Research and Therapy, 46*, 1047–1054. https://doi.org/10.1016/j.brat.2008.06.010.

Taylor, S., Abramowitz, J., & McKay, D. (2012). Non-adherence and non-response in the treatment of anxiety disorders. *Journal of Anxiety Disorders, 26*, 583–589. https://doi.org/10.1016/j.janxdis.2012.02.010.

Taylor, S., & Cox, B. J. (1998aa). An expanded Anxiety Sensitivity Index: Evidence for a hierarchic structure in a clinical sample. *Journal of Anxiety Disorders, 12*, 463–484. https://doi.org/10.1016/S0887-6185(98)00028-0.

Taylor, S., & Cox, B. J. (1998bb). Anxiety sensitivity: Multiple dimensions and hierarchic structure. *Behaviour Research and Therapy, 36*, 37–51. https://doi.org/10.1016/S0005-7967(97)00071-5.

Taylor, S., Zvolensky, M. J., Cox, B. J., Deacon, B., Heimberg, R. G., Ledley, D. R., ... Bouvard, M. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the Anxiety Sensitivity Index—3. *Psychological Assessment, 19*, 176–188. https://doi.org/10.1037/10403590.19.2.176.

van Balkom, A. J. L. M., de Beurs, E., Hovens, J. E. J. M., & van Vliet, I. M. (2004). Meetinstrumenten bij angststoornissen. *Tijdschrift voor Psychiatrie, 46*, 687–692.

van der Mheen, M., ter Mors, L. M., van den Hout, M. A., & Cath, D. C. (2018). Routine outcome monitoring bij de behandeling van angststoornissen: Diagnosespecifieke versus generieke meetinstrumenten. *Tijdschrift voor Psychiatrie, 60*, 11–19.

van Groenestijn, M. A. C., Akkerhuis, G. W., Kupka, R. W., Schneider, N., & Nolen, W. A. (1998). *SCID-I: Gestructureerd klinisch interview voor de vaststelling van DSM-IV as-I stoornissen.* Lisse: Swets en Zeitlinger.

van Oppen, P., Emmelkamp, P. M. G., van Balkom, A. J. L. M., & van Dyck, R. (1995). The sensitivity to change of measures for obsessive-compulsive disorder. *Journal of Anxiety Disorders, 9*, 241–248. https://doi.org/10.1016/0887-6185(95)00005-9.

Waller, G. (2009). Evidence-based treatment and therapist drift. *Behaviour Research and Therapy, 47*, 119–127. https://doi.org/10.1016/j.brat.2008.10.018.

Watson, D., & Clark, L. A. (1991). *The Mood and Anxiety Symptom Questionnaire*. Unpublished manuscript, University of Iowa, Department of Psychology, Iowa City.

Wheaton, M. G., Deacon, B., McGrath, P. B., Berman, N. C., & Abramowitz, J. S. (2012). Dimensions of anxiety sensitivity in the anxiety disorders: Evaluation of the ASI-3. *Journal of Anxiety Disorders, 26*, 401–408. https://doi.org/10.1016/j.janxdis.2012.01.002.

Wittchen, H. U., Jacobi, F., Rehm, J., Gustavsson, A., Svensson, M., Jönsson, B., ... Steinhausen, H.-C. (2011). The size and burden of mental disorders and other disorders of the brain in Europe 2010. *European Neuropsychopharmacology, 21*, 655–679. https://doi.org/10.1016/j.euroneuro.2011.07.018.

Wolitzky-Taylor, K. B., Arch, J. A. J., Rosenfield, D., & Craske, M. G. (2012). Moderators and non-specific predictors of treatment outcome for anxiety disorders: A comparison of cognitive behavioral therapy to acceptance and commitment therapy. *Journal of Consulting and Clinical Psychology, 80*, 786–799. https://doi.org/10.1037/a0029418.

Wolitzky-Taylor, K. B., Fenwick, K., Lengnick-Hall, R., Grossman, J., Bearman, S. K., Joanna Arch, J., ... Chung, B. (2018). A preliminary exploration of the barriers to delivering (and receiving) exposure-based cognitive behavioral therapy for anxiety disorders in adult community mental health settings. *Community Mental Health Journal,* 1–13. https://doi.org/10.1007/s10597-018-0252-x.