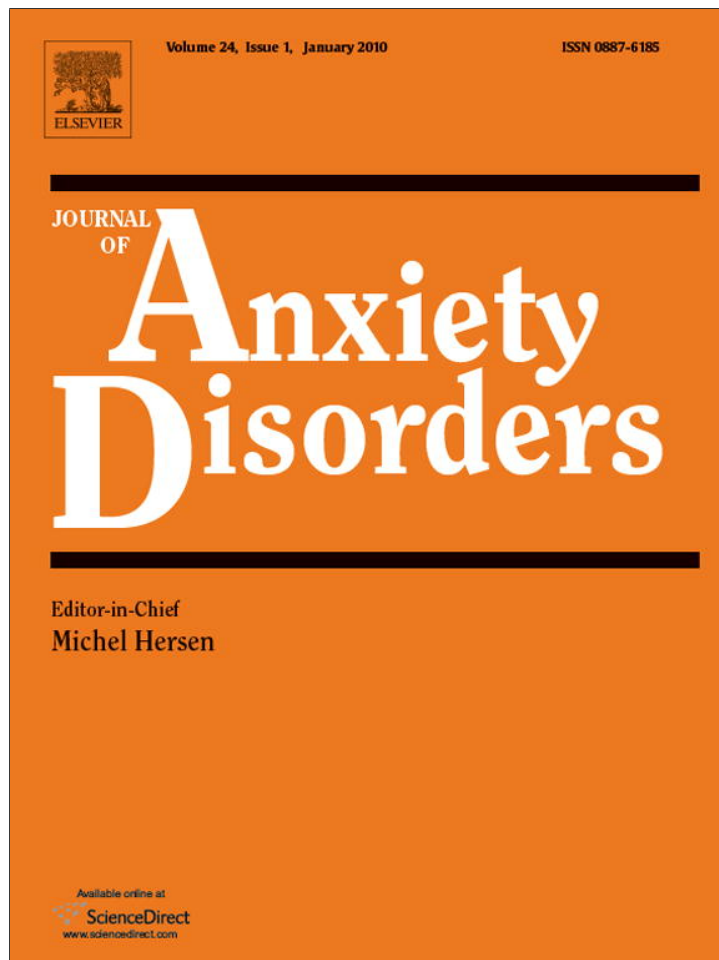


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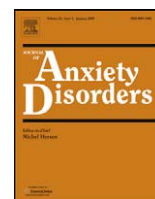
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Predicting anxiety: The role of experiential avoidance and anxiety sensitivity

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ABSTRACT

Anxiety sensitivity (AS), the tendency to fear arousal-related body sensations based on beliefs that they are harmful, is a strong psychological risk factor for development of anxiety psychopathology; however, in most studies AS explains only a portion of the variability in anxiety symptoms. Recent theoretical and research work has suggested that experiential avoidance (EA), unwillingness to endure unpleasant internal experiences (e.g., thoughts, emotions, memories), is related to anxiety disorders. The current study examined independent contributions of EA and AS in the prediction of anxiety symptoms in a sample of 42 adults with DSM-IV anxiety disorders. Participants completed measures of AS, EA, anxiety, and depression. Correlational analyses indicated associations between AS, EA, and anxiety, yet more conservative regression analyses indicated that the Physical Concerns dimension of AS predicted anxiety symptom severity independently of EA. Theoretical and treatment implications of the results are discussed.

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One widely researched conceptual framework for understanding anxiety disorders is the cognitive-behavioral approach, which proposes that emotional disorders arise from dysfunctional beliefs (Clark, 1999). For example, social phobia is thought to arise from exaggerated beliefs about being evaluated negatively (e.g., "People will think I am stupid if I make a mistake"; Clark & Wells, 1995). Panic and agoraphobia are thought to arise from catastrophic beliefs about the dangerousness of internal (arousal-related) sensations (e.g., "When my heart beats rapidly, I believe I'm having a heart attack"; Clark, 1986). These dysfunctional cognitions lead to catastrophic misinterpretations of danger cues, giving rise to situational anxiety and fear. Escape and avoidance behavior performed in response to feared negative outcomes paradoxically maintain the anxiety disorder by preventing the disconfirmation of dysfunctional cognitions (Clark, 1999). A large body of research provides support for the cognitive-behavioral approach and for cognitive-behavioral therapy (CBT), which aims to correct dysfunctional beliefs and eliminate maladaptive escape and avoidance behavior (e.g., Barlow, 2002).

Within the cognitive-behavioral framework, anxiety sensitivity (AS) is a set of trait-like dysfunctional beliefs about harmful consequences of anxious arousal (Reiss & McNally, 1985).

Consistent evidence demonstrates that AS is a strong psychological risk factor for the development of anxiety psychopathology (e.g., Schmidt, Lerew, & Jackson, 1997; Schmidt, Buckner, & Keough, 2007; Zvolensky, Schmidt, Bernstein, & Keough, 2006). Studies (e.g., Taylor et al., 2007) have identified three dimensions of AS, including (a) Physical Concerns (e.g., "When I feel pain in my chest, I worry that I'm going to have a heart attack"), (b) Social Concerns (e.g., "I worry that other people will notice my anxiety," and (c) Cognitive Concerns (e.g., "It scares me when I am unable to keep my mind on a task"). According to cognitive-behavioral theory, AS is a cognitive diathesis such that individuals high in AS will become anxious in the presence of a feared stimulus not only due to their specific fear of the stimulus itself, but also because of their fear of the physiologic sensations associated with anxiety. For example, someone with a snake phobia may fear confronting a snake because of the threat of being poisoned, but also because of the discomfort associated with feeling very anxious in the snake's presence.

Although strongly predictive of anxiety psychopathology, the construct of AS does not completely explain the variability in anxiety symptoms (Schmidt et al., 1997). Thus, it is worth attending to theoretical proposals that offer unique perspectives on psychological factors that might also be predictive of anxiety disorders. One approach that has garnered attention recently is *experiential* (emotional) *avoidance* (EA), a key component in acceptance and commitment therapy (ACT). EA involves an unwillingness to endure upsetting emotions, thoughts, memories, and other private experiences. Such unwillingness is thought to

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lead to maladaptive efforts to resist, escape, and avoid such experiences (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Authors have posited that EA underlies and plays an important role in various psychological disorders, including anxiety disorders (e.g., Hayes et al., 1996; Orsillo & Roemer, 2005).

In the EA framework, anxiety disorders are conceptualized as problems in which the individual uses ineffective strategies in an attempt to suppress negative affect (e.g., fear, worry), uncomfortable physiologic sensations (e.g., racing heart, breathlessness), unwanted thoughts (e.g., obsessional intrusions, memories of traumatic events), and other negative cognitions (e.g., anticipation of dangerous outcomes; Hayes et al., 1996). Thus, the “EA model” of anxiety disorders departs from the cognitive-behavioral model, in that EA is focused not on one’s interpretation of events or surroundings, but rather how he or she tolerates anxious affect in the presence of fear cues. ACT incorporates EA by helping anxious patients accept and endure the negative experiences described above (i.e., develop greater “psychological flexibility”), rather than resort to avoidance and escape strategies (Eifert & Forsyth, 2005). Several studies have shown efficacy of ACT in the treatment of anxiety disorders (Powers, Vording, & Emmelkamp, 2009), lending support to validity of EA as a model for conceptualizing the psychopathology and treatment of anxiety.

Both AS and EA relate to emotional difficulties, yet there are distinctions between the two concepts. For example, whereas EA concerns negative private experiences in general, AS is specifically concerned with arousal-related body sensations. Also, whereas AS is described as a set of trait-like dysfunctional beliefs (Taylor, 1999), EA is conceptualized as a psychological process (Hayes et al., 1996). Some authors (Hayes, Luoma, Bond, Masuda, & Lillis, 2006) suggest that EA/ACT represents a new approach for conceptualizing and treating problems such as anxiety disorders. Others, however, have challenged this notion, arguing that the EA/ACT model does not improve upon the cognitive-behavioral framework (Hofmann & Asmundson, 2008). Given lack of empirical data on this issue, we designed the present study to examine the relationship between EA and AS, and the independent and relative contributions of these constructs in the prediction of clinically significant anxiety symptoms. As AS involves the catastrophic misinterpretation of anxiety symptoms and EA involves the tendency to avoid such internal experiences, we hypothesized that these two variables would be positively correlated. On the basis of clinical observation and previous research, we also predicted that both EA and AS would be associated with anxiety symptoms. Given the absence of research on the relative contributions of EA and AS in the prediction of anxiety symptoms, we undertook an exploratory approach to examine this question.

1. Method

1.1. Participants

Participants included 42 adults (22 women, 20 men) with a mean age of 27.21 years ($SD = 13.06$; range = 18–63 years). Of the sample, 91% identified as Caucasian. All 42 participants received the Anxiety Disorders Interview Schedule (ADIS; Di Nardo, Brown, & Barlow, 1994) at one of the following sites: the Anxiety and Stress Disorders Clinic at the University of North Carolina (Chapel Hill, NC) and the OCD and Related Disorders Program at Alexian Brothers Behavioral Health Hospitals (Hoffman Estates, IL). To be included in the current sample, participants must have received a primary diagnosis of an anxiety disorder. Primary diagnoses within this group included OCD ($n = 12$, 29%), panic disorder with or without agoraphobia ($n = 6$, 14%), social phobia ($n = 10$, 24%), specific phobia ($n = 1$, 2%), GAD ($n = 5$, 12%), and other anxiety (e.g., PTSD, Anxiety NOS; $n = 8$, 19%).

1.2. Procedure

All individuals presenting for evaluation and treatment at the two sites completed a packet of self-report questionnaires that included the measures described below. Each participant was then given a diagnostic interview by a trained assessor. After completing the initial interview and formulating a diagnosis, the first interviewer presented the assessment data to a more expert clinician (i.e., the director or senior clinician at each site), who subsequently met and reviewed the assessment data with the patient. Although formal inter-rater reliability checks were not conducted, only patients for whom both interviewers agreed on diagnostic status were included in the study (i.e., 100% inter-rater agreement).

1.3. Measures

The following measures were included in the present study:–*Acceptance and Action Questionnaire-II* (AAQ-II; Bond et al., 2007). The AAQ-II is a 10-item revision of the original 9-item AAQ (Hayes, 2000). The scale assesses EA, also known as “psychological flexibility,” which is a core construct of the ACT model of psychopathology (Hayes et al., 2006). The AAQ-II has been shown to have good psychometric properties and good convergent, discriminant, and incremental validity. Factor analytic findings suggest the AAQ-II is a unidimensional measure (Bond et al., 2007). Higher scores on the AAQ-II indicate greater psychological flexibility (less pathology).

Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988). The BAI is a self-report instrument that assesses 21 common symptoms of clinical anxiety (e.g., sweating, fear of losing control). Respondents indicate the degree to which they have recently been bothered by each symptom during the past week. The BAI was designed to assess anxiety symptoms independently from depression symptoms, which it does successfully (Beck et al., 1988). It has excellent internal consistency in anxiety disorder samples (range of Cronbach’s alpha = .85–.93; Beck et al., 1988).

Although it continues to be widely used as a measure of anxiety symptoms in general (e.g., Abramowitz, Khandker, Nelson, Rygwall, & Deacon, 2006; Schmidt et al., 1997), some authors have noted that many items on the BAI assess physiologic correlates of anxiety, and argue that this measure is confounded with, or actually measures, panic attack symptoms rather than anxiety in general (Cox, Cohen, Dorenfeld, & Swinson, 1996). Other authors (Steer & Beck, 1996) point out that panic symptoms are present across the anxiety disorders (American Psychiatric Association, 2000), and that 11 of the 21 BAI items are reflective of symptoms of generalized anxiety disorder (GAD). As the BAI was used as the main dependent measure in the present study, we considered the implications of this conceptual issue in our data analyses and conclusions.

Anxiety Sensitivity Index-3 (ASI-3). The ASI-3 (Taylor et al., 2007) is an 18-item version of the original ASI (Reiss, Peterson, Gursky, & McNally, 1986) that measures beliefs about the feared consequences of symptoms associated with anxious arousal (e.g., “It scares me when I become short of breath”). Respondents indicate their agreement with each item from “very little” (coded as 0) to “very much” (coded as 4). Total scores range from 0 to 72. The ASI-3 contains three empirically established subscales relating to fears of social concerns (e.g., It is important for me not to appear nervous), fears of physical symptoms (e.g., It scares me when my heart beats rapidly), and fears of cognitive dyscontrol (e.g., It scares me when I am unable to keep my mind on a task). The measure possesses excellent psychometric properties (Taylor et al., 2007).

Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report scale that assesses the severity of affective, cognitive, motivational, vegetative, and psychomotor

components of depression. Higher total scores reflect greater depression severity. The BDI-II has excellent reliability and validity as a measure of general distress, and is widely used in clinical research (Beck et al., 1996).

2. Data analytic strategy

As our aim was to examine the relationship between the predictor variables (EA and AS) and the symptoms of general anxiety (as opposed to disorder-specific signs and symptoms; e.g., obsessions, compulsions, avoidance, panic attacks), we combined patients with different anxiety diagnoses into a single group for our analyses. To test our hypotheses, we first computed correlation coefficients to examine zero-order relationships among EA (AAQ-II), AS and its three dimensions (ASI-3 and its subscales), anxiety (BAI), and depressive symptoms (BDI-II). Second, we computed partial correlations to examine whether the AS dimensions and EA are associated with anxiety symptoms after controlling for each other. Third, we performed two hierarchical regression analyses with the BAI as the dependent variable. In both of these analyses, the BDI-II was entered in the first step to control for the overlap between depressive and anxiety symptoms, and the predictor variables. In Step 2 of the first regression, the ASI-3 subscales were entered to control for AS as specified in the cognitive-behavioral model. We entered the ASI-3 subscales separately in order to understand how the various components of anxiety sensitivity predict anxiety symptoms. In Step 3 of this first analysis, the AAQ-II was entered. In the second regression, Steps 2 and 3 were reversed so that the AAQ-II was entered in Step 2 to control for psychological flexibility, and the ASI-3 subscales were entered together in Step 3.

3. Results

3.1. Sample characteristics

Table 1 presents the group means and standard deviations on the study measures. As would be expected in a group of individuals diagnosed with anxiety disorders, mean scores were indicative of significant levels of anxious psychopathology.

3.2. Zero-order correlations

To examine the relationships among study variables, we computed zero-order correlations. These results are presented in Table 2. The AAQ-II is unique among the study measures in that higher scores indicate less psychopathology. Therefore, its correlations with other study measures are negative. As expected, the ASI-3 total score and the Physical and Cognitive Concerns subscales were significantly correlated with the AAQ-II. Additionally, the AAQ-II and all ASI-3 subscales were significantly associated with the BAI. The AAQ-II and ASI-3 Cognitive Concerns subscale were correlated with the BDI-II, whereas the ASI-3 Social and Physical Concerns were not.

Table 1
Group means and standard deviations on study measures.

Measure	Mean (SD)
ASI-3 total	25.38 (14.61)
Social concerns	9.33 (5.29)
Physical concerns	7.52 (5.61)
Cognitive concerns	8.52 (6.01)
AAQ-II	38.14 (11.42)
BAI	22.33 (13.45)
BDI-II	19.29 (9.64)

Note: ASI-3: Anxiety Sensitivity Index; AAQ-II: Acceptance and Action Questionnaire; BAI: Beck Anxiety Inventory; BDI-II: Beck Depression Inventory.

Table 2
Zero-order correlations among study measures.

Variable	ASI-3 social	ASI-3 physical	ASI-3 cognitive	AAQ-II	BAI	BDI-II
ASI-3 total	.87**	.83**	.89**	-.48**	.52**	.18
Social		.57**	.70**	-.28	.35*	.13
Physical			.59**	-.33*	.53**	-.02
Cognitive				-.61**	.46**	.33*
AAQ-II					-.43**	-.65**
BAI						.25

ASI-3: Anxiety Sensitivity Index; AAQ-II: Acceptance and Action Questionnaire; BAI: Beck Anxiety Inventory; BDI-II: Beck Depression Inventory.

* $p < .05$.
** $p < .01$.

As discussed above, many items on the BAI assess the physical manifestations of anxiety, which overlap with the symptoms of panic attacks (e.g., sweating, trembling, heart palpitations; Cox et al., 1996). Given the strong association between AS and panic symptoms, we considered whether the relationship between the BAI and ASI-3 was stronger than that between the AAQ-II and BAI. If this were the case, it could explain the findings reported in the sections below. Inspection of Table 2, however, revealed that both the ASI-3 and AAQ-II were only moderately correlated with the BAI, reflecting their measurement of separate constructs: the BAI measuring physical manifestations of anxiety and the ASI-3 measuring beliefs about autonomic arousal. Moreover, a post hoc significance test of the difference in correlation magnitudes indicated that the zero-order correlation between the BAI and ASI-3 was not significantly stronger ($t = 0.66$; $p > .05$) than that between the BAI and the AAQ-II (albeit the two were in different directions because of how the measures are scored).

3.3. Partial correlations

To further examine relationships between the predictors (AS and EA) and anxiety symptoms, we computed a series of partial correlations to test the association between ASI-3 subscales and the BAI while controlling for AAQ-II scores, and conversely, the association between the AAQ-II and the BAI while controlling for each ASI-3 subscale. Table 3 displays the results of these analyses, which indicate that even after controlling for AAQ-II scores, the ASI-3 Physical Concerns subscale remained significantly correlated with the BAI. Additionally, the AAQ-II remained significantly correlated with the BAI after controlling for the Social and Physical subscales, but was not significantly correlated with the BAI after controlling for the Cognitive subscale.

3.4. Regression analyses

Results of the hierarchical regression analyses were as follows: In step one of both regression analyses, the BDI-II explained a small and nonsignificant portion of the variance in BAI scores ($R^2 = .06$,

Table 3
Partial correlations between predictor variables and dependent measures.

Predictor variable	Controlling for	Partial correlation with BAI
ASI-3 social	AAQ-II	.26
ASI-3 physical	AAQ-II	.46**
ASI-3 cognitive	AAQ-II	.27
AAQ-II	ASI-3 Social	-.36*
AAQ-II	ASI-3 Physical	-.32*
AAQ-II	ASI-3 Cognitive	-.22

ASI-3: Anxiety Sensitivity Index; AAQ-II: Acceptance and Action Questionnaire; BAI: Beck Anxiety Inventory.

* $p < .05$.
** $p < .01$.

Table 4
Stepwise regressions predicting BAI scores.

Variable	R ²	Beta	t	p
Equation 1				
Step 1	.06			<.01
BDI-II		.25	1.64	n.s.
Step 2	.36			
ASI-3 cognitive		.14	.68	n.s.
ASI-3 physical		.49	2.80	<.01
ASI-3 social		-.06	-.31	n.s.
Step 3	.37			
AAQ-II		-.17	-0.75	n.s.
Equation 2				
Step 1	.06			<.01
BDI-II		.25	1.64	n.s.
Step 2	.18			
AAQ-II		-.47	-2.46	<.05
Step 3	.37			
ASI-3 cognitive		.06	.24	n.s.
ASI-3 physical		.46	2.55	<.01
ASI-3 social		-.02	-.09	n.s.

ASI-3: Anxiety Sensitivity Index; AAQ-II: Acceptance and Action Questionnaire; BAI: Beck Anxiety Inventory; BDI-II: Beck Depression Inventory.

$p > .05$). In the first equation with the ASI-3 subscales entered in Step 2 and the AAQ-II entered in Step 3, adding the ASI-3 subscales explained significant additional variance in BAI scores ($\Delta R^2 = .30$, $p < .01$). In Step 3, adding the AAQ-II accounted for no additional variance in BAI scores ($\Delta R^2 = .01$, $p > .05$). In the second equation with the order of entry reversed, adding the AAQ-II in Step 2 explained significant additional variance in BAI scores ($\Delta R^2 = .13$, $p < .05$). However, In Step 3, adding the ASI-3 subscales made a further significant contribution ($\Delta R^2 = .18$, $p < .05$). As can be seen in Table 4, the final model with all variables entered, accounted for 37% of the variance in BAI scores. Additionally, in the first regression (Equation 1 in Table 4), where ASI-3 subscales were entered in Step 3, the ASI-3 Physical Concerns subscale emerged as the only significant, unique predictor.

4. Discussion

The aims of this study were to investigate relationships between AS and EA, and to examine the independent and relative contributions of these variables in explaining anxiety symptoms. AS is a well-established psychological risk factor for anxiety psychopathology (e.g., Schmidt et al., 1997, 2007; Zvolensky et al., 2006). EA has also received empirical support as a factor in theoretical models of anxiety (e.g., Eifert & Heffner, 2003; Hayes et al., 1996; Levitt, Brown, Orsillo, & Barlow, 2004). The present study, however, represents the first empirical evaluation of the relative contribution of these constructs in predicting anxiety symptoms in a clinical sample. Identifying the relative predictive value of these two constructs may help to highlight the most empirically consistent approach for understanding and treating anxiety problems.

As expected, zero-order correlations indicated that EA and AS are associated with one another. This is not surprising given that both constructs relate to negative experiences with internal events. Additionally, both of these constructs were correlated with anxiety symptoms, which is consistent with both the cognitive-behavioral and the ACT approaches, respectively. From the cognitive-behavioral perspective, AS underlies the catastrophic misinterpretation of arousal-related body sensations, which is thought to lead to fear, avoidance, and safety behaviors. From the ACT perspective, EA leads to attempts to suppress emotions, thoughts, and other private experiences, and this psychological inflexibility is believed to underlie emotional distress (e.g., anxiety disorders).

In our partial correlation analyses, the Physical Concerns dimension of AS, but not the Social or Cognitive dimensions, was significantly associated with anxiety symptoms even after partialling out the effects of EA. In addition, EA remained significantly associated with anxiety symptoms even after controlling for the Social and Physical Concerns, but not the Cognitive Concerns dimension of AS. Although the correlational findings reported above support associations between AS, EA, and anxiety symptoms, regression analyses were conducted to determine whether AS and EA would emerge as unique predictors of anxiety.

Our regression analyses indicated that after controlling for depressive symptoms (Step 1), the AS dimensions and EA predicted anxiety symptoms in Step 2. However, results of the final steps of the regressions suggest that irrespective of the level of EA, the Physical Concerns dimension of AS was significantly associated with anxiety symptoms. The converse, however, did not apply as EA did not account for additional variance beyond the AS dimensions. This suggests that the relationship between EA and anxiety may be a by-product of the variance shared with AS. Specifically, the belief that physical harm will result from experiencing anxious arousal independently predicted anxiety symptoms over and above the tendency to find particular private experiences (e.g., bodily sensations, emotions, thoughts, memories) intolerable (i.e., EA).

Hayes et al. (1996) proposed that many forms of psychopathology, including anxiety disorders, “are usefully viewed as unhealthy methods of experiential avoidance” (p. 1154). Our correlational findings are consistent with this view and indicate a moderate relationship between EA and anxiety. However, our regression findings that EA failed to uniquely contribute to the prediction of anxiety reveal that the variance in anxiety symptoms accounted for by EA overlaps with that accounted for by AS (particularly the Physical Concerns dimension), which explains anxiety symptoms over and above the explanatory power of EA. These findings suggest that specific dysfunctional beliefs about internal experiences provide a more empirically valid basis for understanding anxious psychopathology relative to the general unwillingness to endure such private events (i.e., EA). It may be that the concept of EA is too general to independently explain anxiety symptoms, especially in comparison to cognitive-behavioral models.

Our findings may have implications for treatment of anxiety disorders. CBT aims to modify beliefs assumed to underlie symptoms of anxiety, whereas ACT aims to help individuals develop psychological flexibility and accept inner experiences, such as thoughts, memories, feelings, etc. The finding that AS, and particularly the Physical Concerns dimension, predicts anxiety symptoms independently of EA suggests that relative to EA, dysfunctional beliefs about the harmfulness of arousal-related body sensations better tap into the pathological process underlying anxiety symptoms and may therefore be a better target for therapeutic change. Thus, our present results suggest that with respect to the treatment of anxiety symptoms, it may be more effective to modify beliefs about the dangerousness of body sensations using CBT techniques such as psychoeducation, cognitive restructuring, and interoceptive exposure (e.g., Craske & Barlow, 2008), rather than engaging in ACT techniques which facilitate cognitive flexibility; for example, cognitive diffusion which aims to “deliteralize” language associated with anxiety. Naturally, this conclusion requires additional empirical evaluation via direct comparisons between the two treatment approaches. We encourage researchers to conduct experiments testing the relative efficacy of the mechanisms of change proposed by CBT and ACT for anxiety disorders.

A number of limitations of this study should be considered. First, because all data in the present study were collected using self-report measures, relationships among variables might be

artificially inflated. Second, the cross-sectional nature of this study precludes conclusions about cause and effect relationships. Although our findings are consistent with the notion that AS gives rise to anxiety symptoms, an alternative explanation is that anxiety symptoms lead to the acquisition of AS. It is also possible that one or more third variables account for anxiety symptoms and AS. Third, the broad scope of the EA concept represents a limitation. The AAQ-II is a general measure of experiential avoidance that cuts across emotional valence domains and does not discriminate between specific avoidance strategies. Chawla and Ostafin (2007) have argued that the distinction between experiential avoidance and related constructs such as avoidance coping are not entirely clear. Fourth, the sample was relatively small and predominately Caucasian. Increasing the sample size and including a more diverse sample would strengthen the power of the regression analyses and yield more generalizable results, respectively.

As mentioned previously, the BAI contains a number of items that assess the physical manifestations of anxiety (e.g., sweating, trembling, heart palpitations) and some authors have suggested that this measure actually assesses panic symptoms as opposed to anxiety in general (Cox et al., 1996). To the extent that this is the case, our findings might relate to the prediction of panic symptoms in addition to, or rather than, general anxiety. Additional studies using other measures of anxiety such as the State Trait Anxiety Inventory (Spielberger, 1983) or Depression, Anxiety, and Stress Scale (Henry & Crawford, 2005) should be undertaken when replicating the present findings. A related consideration is that if the BAI is actually a measure of panic disorder, which has a strong relationship to AS, this association might explain the BAI's unique relationship with the AS (particularly the ASI-3 Physical Concerns subscale), compared to the AAQ-II. However, as our zero-order correlations show (Table 2), the AS was only moderately correlated with the BAI, reflecting their measurement of different constructs: the BAI measuring physical manifestations of anxiety and the ASI-3 measuring beliefs about autonomic arousal. Moreover, the zero-order correlation between the BAI and ASI-3 was not significantly stronger than that between the BAI and the AAQ-II.

Finally, only 37% of the variance in BAI scores was accounted for by the BDI-II, AAQ-II, and ASI-3 in our final regression model, leaving open the possibility of additional factors in the prediction of anxiety symptoms. Future research should examine the prediction of more specific signs and symptoms of anxiety disorders (i.e., worry, panic attacks, compulsive rituals) to further investigate the relative contribution of EA and dysfunctional beliefs. Moreover, because dysfunctional beliefs, EA, and anxiety symptoms are found on a continuum in the general population, studies of both clinical and nonclinical populations can be helpful in better understanding the relationships between these constructs.

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