

The Contribution of Experiential Avoidance and Anxiety Sensitivity in the Prediction of Health Anxiety

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Anxiety sensitivity (AS) refers to a fear of arousal-related body sensations based on beliefs that such sensations are dangerous. Experiential (emotional) avoidance (EA) involves an unwillingness to endure upsetting emotions, thoughts, memories, and other private experiences. As both of these constructs are thought to be predictive of health anxiety, the present study examined their relative contribution in the prediction of health anxiety symptoms. A large sample of nontreatment-seeking participants completed measures of AS, EA, and health anxiety. An analogue sample of participants with clinical levels of health anxiety endorsed more AS and EA relative to those with less health anxiety. Within the analogue sample, both AS and EA predicted health anxiety symptoms. However, whereas AS (specifically, the physical concerns domain) uniquely predicted health anxiety, EA did not contribute significantly over and above the contributions of AS. Results are also discussed in terms of the conceptualization and treatment of health anxiety.

Keywords: health anxiety; anxiety sensitivity; experiential avoidance; psychological flexibility

Given the importance of physical well-being it is not surprising that most people experience occasional health-focused thoughts and concerns (Freeston et al., 1994; Looper & Kirmayer, 2001). For people suffering from (or at risk of developing) serious medical conditions, health concerns are adaptive in that they motivate the individual to attend to bodily sensations and other cues to ensure that threatening signs and symptoms are dealt with in a timely fashion. In fact, as part of their self-care, at-risk patients may be instructed to monitor their body for possible symptoms. In other instances, intense health concerns (or health *anxiety*) develop in the absence of any objective cause for concern, such as when a person perceives him or herself to be seriously ill on the basis of a *misinterpretation* of ostensibly benign bodily signs or sensations (e.g., “my stomach cramp is actually a sign of a rare and serious gastrointestinal disorder”). At the clinical extreme, hypochondriasis (HC) involves a pattern of intense health anxiety that gives rise to preoccupation with a suspected medical problem, selective attention to illness-related stimuli (Owens, Asmundson, Hadjistavropoulos, & Owens, 2004), body vigilance (Abramowitz, Olatunji, & Deacon, 2007), and irresistible urges to seek medical advice and reassurance to the extent that it impairs psychosocial functioning (American Psychiatric Association, 2000).

Various theoretical models have been proposed for understanding hypochondriasis and health anxiety (Starcevic & Lipsitt, 2001). The most widely researched approach is that based on Beck's (1976) cognitive specificity theory, which states that emotional disorders arise from dysfunctional beliefs. For example, social phobia is thought to arise from exaggerated beliefs about being evaluated negatively (Clark & Wells, 1995), and depression, from overly negative beliefs about oneself, the world, and the future (Beck, Rush, Shaw, & Emery, 1979). In health anxiety, the dysfunctional beliefs are thought to focus on the dangerousness of unexpected or uncomfortable bodily sensations and perturbations (e.g., "Any pain means there is something seriously wrong with me"), the assumption that one is "at high risk" of disease, and a general distrust of modern medicine (e.g., Taylor & Asmundson, 2004). These types of beliefs are thought to lead the person to misinterpret innocuous bodily signs and sensations as indicating serious disease, giving rise to anxiety, uncertainty, and to excessive behaviors aimed at reducing this anxiety and providing reassurance of health status (e.g., looking up information on the internet, asking for assurances from medical personnel, etc.). A growing body of empirical evidence supports this cognitive-behavioral approach to health anxiety (e.g., Abramowitz & Braddock, 2008) and the efficacy of treatment strategies such as exposure therapy, cognitive restructuring, and psychoeducation, which largely aim to correct the faulty dysfunctional beliefs and interpretations (e.g., Taylor, Asmundson, & Coons, 2005).

Within this framework, anxiety sensitivity (AS) is a set of dysfunctional beliefs about the bodily sensations associated with anxious arousal (e.g., "When my heart beats very fast, I fear I'm having a heart attack"; Reiss & McNally, 1985). Although AS is a psychological risk factor for the development of panic disorder (e.g., Schmidt, Lerew, & Jackson, 1997), there is growing evidence that it is also associated with problems such as chronic pain (e.g., Asmundson & Norton, 1995; Asmundson & Taylor, 1996), tinnitus (Andresson & Vretblad, 2000), and health anxiety in both clinical and nonclinical samples (e.g., Abramowitz, Deacon, & Valentiner, 2007; Abramowitz, Olatunji, et al., 2007; Otto, Demopulos, McLean, Pollack, & Fava, 1998). In fact, AS was found to mediate the relationship between childhood learning experiences and the development of health anxiety among young adults (Watt & Stewart, 2000). AS therefore appears to act as a cognitive diathesis in health-focused preoccupation and anxiety, increasing a person's vulnerability to misinterpreting various harmless bodily signs and sensations (arousal- and nonarousal-related) as highly threatening.

Yet although strongly predictive, research indicates that AS does not completely explain all of the variability in health anxiety symptoms (e.g., Deacon & Abramowitz, 2008; Abramowitz, Olatunji, & Deacon, 2007; Watt & Stewart, 2000). Thus, it is important to consider additional factors that might also be predictive of health anxiety. Acceptance and Commitment Therapy (ACT; Hayes, Luoma, Bond, Masuda, & Lillis, 2006), has been introduced as a psychological treatment that addresses putative shortcomings of extant cognitive-behavioral approaches (Hayes, 2004). The specific foci of ACT include *psychological flexibility* and *experiential avoidance*, which are inversely related constructs thought to play a key role in a number of psychological disorders (e.g., Zvolensky & Forsyth, 2002). *Psychological flexibility* refers to the ability to observe one's own internal experiences (e.g., thoughts, feelings, images, physiological sensations) on a moment-to-moment basis, in an open and nonjudgmental manner, even when they are unpleasant or upsetting. In contrast, *experiential avoidance* (EA) is defined as the inability to maintain contact with unpleasant or upsetting internal experiences and thereby attempt to alter, avoid, suppress, or otherwise control them (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). A growing body of research suggests that these constructs are associated with depression, substance abuse, anxiety, panic, obsessions and compulsions, trichotillomania, posttraumatic stress symptoms, and reduced quality of life (e.g., Chawla & Ostafin, 2007; Hayes et al., 2004; Orsillo & Roemer, 2005).

From an EA perspective, problems with anxiety are thought to result when the individual tries to control or resist negative internal experiences. For example, OCD is thought to result

from attempts to control or resist normally occurring negative intrusive thoughts (*obsessions*, e.g., unwanted images of harming a loved one), and generalized anxiety disorder, from a similar tendency to try to control worries and other negative cognitions such as anticipation of dangerous outcomes (e.g., Hayes et al., 1996). Although to date there is no empirical research on EA in health anxiety, some authors have posited that excessive EA is involved in hypochondriasis (Hayes & Strosahl, 2004). Specifically, it is thought that when an individual attempts to control or resist unexpected bodily sensations, frightening health-related thoughts, and feelings of anxiety (e.g., by using strategies such as behavioral avoidance, reassurance-seeking, and other ritualistic behaviors), the vigilance needed to control or resist these experiences paradoxically reconnects them with (and even *amplifies*) the very negative experiences they seek to avoid. The end result is a self-sustaining vicious cycle.

To summarize, whereas the AS approach to health anxiety specifies ways in which the individual *misinterprets* unexplained bodily sensations, the EA approach focuses on psychological flexibility, or how the individual *tolerates* and *accepts* bodily stimuli and the associated anxious affect. The former approach is associated with cognitive-behavioral therapy (CBT) in which dysfunctional beliefs are challenged and modified using techniques such as cognitive restructuring and exposure therapy. The latter formulation is associated with ACT, which aims (among other things) to help individuals develop greater psychological flexibility (i.e., accept and endure negative experiences such as anxiety and unexplained bodily sensations), rather than resort to resistance, avoidance, and escape strategies (Eifert & Forsyth, 2005). Given that no previous research has examined the role of EA in health anxiety, nor the relative contribution of AS and EA in explaining health anxiety symptoms, the aim of the present study was to investigate these issues. On the basis of the research and theoretical models described earlier, we hypothesized that both AS and EA would be associated with more severe health anxiety symptoms, and that both EA and AS would emerge as unique predictors of health anxiety.

METHOD

Participants

Six hundred and thirty-six self-selected undergraduates (67% women) enrolled in Introductory Psychology courses at a large university in the southeastern United States completed a computer-administered online questionnaire packet that included the short version of the Health Anxiety Inventory (SHAI; Salkovskis, Rimes, Warwick, & Clark, 2002) along with the other study measures that are described later. The participants had a mean age of 19.91 years ($SD = 2.24$). In order to investigate health anxiety in an analogue clinical population we divided the sample on the basis of their SHAI scores using an empirically derived cut point (SHAI total ≥ 18 ; Rode, Salkovskis, Dowd, & Hanna, 2006). This yielded two groups, the first constituting highly health anxious individuals [High-HA], $n = 124$, and the second constituting low health anxious individuals [Low-HA], $n = 512$.

Demographic characteristics of the two groups are presented in Table 1. As can be seen, there were no differences in age, gender composition, or ethnic diversity across the two groups. The mean SHAI score for the High-HA group (22.77 ; $SD = 5.87$) was similar to that reported for a group of clinically anxious individuals (18.5 ; $SD = 7.3$; Salkovskis et al., 2002), yet below that of an exclusively hypochondriacal sample ($M = 37.9$; $SD = 6.8$; Salkovskis et al., 2002). Likewise, the mean for the Low-HA group was similar to that reported for groups of healthy individuals ($M = 12.2$; $SD = 6.2$; Salkovskis et al., 2002) and students ($M = 12.6$; $SD = 5.0$; Salkovskis et al., 2002).

We tested our hypotheses using data from the High-HA group. Thus, an important issue concerns whether the study of analogue samples is relevant to understanding health anxiety per se. Empirical research indicates that health anxiety exists on a continuum, with the differences

TABLE 1. DEMOGRAPHIC AND CLINICAL CHARACTERISTICS FOR THE HIGH-HA ($N = 124$) AND LOW-HA ($N = 512$) GROUPS

Variable	Low-HA	High-HA	Test of the Difference
Demographic characteristics			
Mean age (<i>SD</i>)	19.95 (2.43)	19.79 (1.09)	$t = 0.68, p > .10$
No. of female (%)	339 (66.2)	85 (69.1)	$\chi^2 = 0.38, p > .30$
Racial/ethnic background			$\chi^2 = 4.87, p > .30$
No. of White (%)	385 (75.2)	86 (69.4)	
No. of African American (%)	63 (12.3)	16 (12.9)	
No. of Asian (%)	31 (6.1)	7 (5.6)	
No. of Latino (%)	16 (3.1)	8 (6.5)	
No. of Others (%)	17 (3.3)	7 (5.6)	
Clinical characteristics			
SHAI	9.99 (4.14)	22.77 (5.87)	$t = -28.17, p < .001$
CES-D	16.39 (8.35)	22.16 (9.85)	$t = -6.66, p < .001$
ASI-3 physical concerns	2.95 (3.34)	7.89 (5.04)	$t = -13.14, p < .001$
ASI-3 social concerns	7.13 (4.57)	10.85 (4.28)	$t = -8.21, p < .001$
ASI-3 cognitive concerns	1.93 (3.36)	6.38 (5.26)	$t = -12.81, p < .001$
AAQ-II	51.82 (8.44)	42.34 (9.16)	$t = 11.04, p < .001$

Note. SHAI = Short Health Anxiety Inventory; CES-D = Center for Epidemiologic Studies Depression Scale; ASI-3 = Anxiety Sensitivity Index-3; AAQ-II = Acceptance and Action Questionnaire-II.

between health concerns in the general population and those among individuals with clinical health anxiety (which occur across a range of disorders; e.g., Abramowitz, Olatunji, & Deacon, 2007) being quantitative rather than qualitative (Barsky, Wyshak, & Klerman, 1986). In addition, the clinical cutoff score on the SHAI reliably identified individuals meeting full *DSM-IV* criteria for hypochondriasis and has previously been used in research to identify individuals with significant health anxiety (Rode et al., 2006).

Procedure

Participation in this study was available to all undergraduate students enrolled in Introductory Psychology classes at the study site. These classes include a research participation requirement, and all participants received course credit for their participation in the study. The study was reviewed and approved by the University IRB.

After signing up for the experiment via an Internet-based software program, participants provided consent to participate and were directed to a secure project website where they completed the study measures. All data were collected using Qualtrics, an online web survey development tool. The design of the internet version of the study questionnaires was based on empirically derived suggestions for how to develop computer questionnaires (e.g., Hewson, Yule, Laurent, & Vogel, 2003). Results from a number of studies indicate that the administration of anxiety-related assessment measures using Internet-based and paper-and-pencil formats yield highly comparable results (e.g., Coles, Cook, & Blake, 2006).

Upon accessing the secure project website, participants were presented with a set of instructions for completing the self-report instruments. Participants were informed that all responses were confidential and that no personal identifying information would be included in the computer-generated dataset other than the date and time they completed the study. At the end of the last questionnaire, a debriefing statement was presented.

Measures

The following measures were included in the present study.

Acceptance and Action Questionnaire-II (AAQ-II). The AAQ-II is a 10-item revision of the original 9-item AAQ (Bond et al., 2007). The scale assesses psychological flexibility, also known as experiential avoidance, which is the 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. Higher scores on the AAQ-II indicate greater psychological flexibility (less pathology).

Center for Epidemiological Studies–Depression Scale (CES-D). The CES-D consists of 20 items developed as a global measure to assess psychological distress or well-being in general community samples (Radloff, 1977). Participants are asked to rate how often they have felt (or behaved) in certain ways (e.g., “I felt sad”; “My sleep was restless”) over the past week from 0 (*rarely*) to 3 (*most of the time*). Items are summed (4 are reverse-scored) to obtain a total score ranging from 0 to 60. Scores of 16 or greater indicate the possibility of clinical depression.

Short Health Anxiety Inventory (SHAI). The SHAI is a self-report measure that contains 18 items assessing health anxiety independently of physical health status (Salkovskis et al., 2002). Items measure worry about health, awareness of bodily sensations or changes, and feared consequences of having an illness using a multiple choice format from 0 (*no symptoms*) to 3 (*severe*). The SHAI has demonstrated good reliability and validity as a measure of health anxiety in clinical and nonclinical samples (Abramowitz, Deacon, & Valentiner, 2007; Salkovskis et al., 2002).

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 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).

Data Analytic Strategy

We first conducted between-group comparisons to examine differences between the High-HA and Low-HA groups' mean scores on the study measures. Second, using only the High-HA group's data, we first computed correlation coefficients to examine zero-order relationships among general distress (CES-D), anxiety sensitivity (ASI-3), EA (AAQ-II), and health anxiety (SHAI). Third, we computed partial correlations to examine whether anxiety sensitivity and EA predict health anxiety after controlling for each other. Fourth, two multiple regression analyses were performed with the SHAI as the dependent variable. In both regressions, the CES-D was entered in the first step to control the overlap between general distress, health anxiety symptoms, and the predictor variables. In Step 2 of the first regression, the ASI-3 subscales were entered to control for AS. The AAQ-II was then entered in Step 3. In the second regression analysis, 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.

RESULTS

Group Comparisons

The bottom portion of Table 1 displays the group mean scores on each of the study measures. As expected, between-group *t*-tests (see the far right column of Table 1) revealed that the High-HA group had higher scores than the Low-HA group on the ASI-3 subscales and the AAQ-II. The High-HA group also showed significantly higher CES-D scores relative to the Low-HA group. A subsequent analysis of covariance revealed that the group differences in ASI-3 and AAQ-II scores remained significant after controlling for differences in general distress (CES-D scores; all *ps* < .001).

Zero-Order Correlations

All of the remaining analyses we report were conducted using only data from the analogue sample of participants scoring above the empirically validated clinical cutoff on the SHAI (i.e., the High-HA group). The distributions for all variables were within acceptable limits of skewness and kurtosis (i.e., within 2 standard errors). Intercorrelations (*rs*) among the ASI-3 subscales ranged from .37 (Social with Cognitive) to .58 (Physical with Cognitive), suggesting it was appropriate to consider these subscales as measuring separate constructs. Thus, the correlational and regression analyses reported later were conducted at the subscale level for the ASI-3.

Correlations (*rs*) between the AAQ-II and ASI-3 subscales were as follows: ASI-3 Physical = $-.27$; ASI-3 Social = $-.47$; and ASI-3 Cognitive = $-.33$. All of these moderately strong relationships were statistically significant (*ps* < .001).

Correlations between the predictor (theoretical) variables (i.e., AAQ-II and ASI-3 subscales) and symptom measures (i.e., CES-D and SHAI) are displayed in Table 2. As can be seen, after applying a Bonferroni corrected alpha of .006 to correct for multiple tests, the SHAI was significantly associated with both the ASI-3 and AAQ-II. Correlations ranged from weak to moderate in magnitude. The CES-D was significantly correlated with the AAQ-II but not the ASI-3 subscales.

Partial Correlations

To examine independence of relationships between the predictor variables and HA symptoms we computed a series of partial correlations in which the ASI-3 subscales were used to predict SHAI

TABLE 2. ZERO-ORDER CORRELATIONS BETWEEN THEORETICAL VARIABLES AND SYMPTOM VARIABLES FOR THE HIGH-HA GROUP (*N* = 124)

Clinical Variables	Cognitive Variables			AAQ-II
	ASI-3 Physical	ASI-3 Social	ASI-3 Cognitive	
SHAI	.45*	.32*	.25*	-.28*
CES-D	.09	.21	.28	-.39*

Note. SHAI = Short Health Anxiety Inventory; CES-D = Center for Epidemiologic Studies Depression Scale; ASI-3 = Anxiety Sensitivity Index-3; AAQ-II = Acceptance and Action Questionnaire-II.

**p* < .006 (Bonferroni corrected alpha).

scores while controlling for AAQ-II scores, and the AAQ-II was used to predict SHAI scores while controlling for the ASI-3 subscales. A Bonferroni corrected alpha of .008 was applied to correct for multiple tests. Table 3 displays the results of these analyses, which indicate that after controlling for the AAQ-II, the ASI-3 Physical subscale, but not the Social and Cognitive Concerns subscales, was a significant predictor of SHAI scores. Controlling for the ASI-3 subscales resulted in a reduction in the magnitude of correlations between the AAQ and the SHAI, and this relationship failed to remain statistically significant after controlling for each ASI-3 subscale.

Regression Analyses

Results of the regression analyses outlined previously are presented here and in Table 4, which shows the summary statistics for each variable in the final step of each regression equation. In Step 1 of both equations, the CES-D explained a small, nonsignificant portion of the variance (2%) in SHAI scores ($R^2 = .02, p > .05$). In Step 2 of the first equation, adding the ASI-3 subscales explained an additional 20% of the variance (R^2 change = .20, $p < .01$). In Step 3, however, adding the AAQ did not account for any significant additional variance (R^2 change = .02, $p > .05$). In Step 2 of the second equation, the AAQ explained significant additional variance (6%) over and above the CES-D (R^2 change = .06, $p < .01$). In Step 3, addition of the ASI-3 explained an additional 16% of the variance (R^2 change = .16, $p < .01$). The final model explained nearly a quarter of the variance in SHAI scores and was statistically significant ($R^2 = .24, p < .01$). As Table 4 shows, the ASI-3 Physical concerns subscale emerged as the sole significant unique predictor of SHAI scores.

DISCUSSION

The aims of this study were to investigate relationships between AS, EA, and health anxiety symptoms; and to examine the independent and relative contributions of AS and EA in predicting health anxiety. AS is a well-established cognitive theoretical factor in the prediction of anxiety symptoms such as panic, posttraumatic stress, and as focused on in the present study, health anxiety (e.g., Taylor, 1999). Interventions such as cognitive restructuring and exposure therapy (interoceptive and situational exposure) that directly target AS-related beliefs are associated with reductions in anxiety symptoms, including health-related anxiety (Taylor et al., 2005). To date, neither EA nor psychological flexibility have been studied as specific factors in health anxiety. The present study therefore represents the first to empirically examine the relationship between EA and health anxiety.

TABLE 3. PARTIAL CORRELATIONS BETWEEN THEORETICAL VARIABLES AND HEALTH ANXIETY

Predictor Variable	Predicting	Controlling for	Partial Correlation
ASI-3 physical	SHAI	AAQ-II	.41*
ASI-3 social	SHAI	AAQ-II	.23
ASI-3 cognitive	SHAI	AAQ-II	.18
AAQ-II	SHAI	ASI-3 physical	-.19
AAQ-II	SHAI	ASI-3 social	-.16
AAQ-II	SHAI	ASI-3 cognitive	-.22

Note. SHAI = Short Health Anxiety Inventory; ASI-3 = Anxiety Sensitivity Index-3; AAQ-II = Acceptance and Action Questionnaire-II.

* $p < .008$ (Bonferroni corrected).

TABLE 4. SUMMARY STATISTICS FOR THE FINAL STEP OF REGRESSION EQUATIONS PREDICTING HEALTH ANXIETY (SHAI SCORES)

Variable	R^2	Beta	t	p
Final model	.24			<.01
CES-D		.049	0.55	<i>ns</i>
AAQ-II		-.153	-1.53	<i>ns</i>
ASI-3 physical		.414	3.78	<.01
ASI-3 social		.036	0.34	<i>ns</i>
ASI-3 cognitive		-.048	-0.47	<i>ns</i>

Note. SHAI = Short Health Anxiety Inventory; CES-D = Center for Epidemiologic Studies Depression Scale; ASI-3 = Anxiety Sensitivity Index—3; AAQ-II = Acceptance and Action Questionnaire-II.

As expected, the High-HA group evidenced greater levels of AS and EA relative to the Low-HA group. Analyses of covariance confirmed that these between group differences could not be accounted for by general levels of psychological distress. Moreover, both AS and EA were correlated with health anxiety. Thus, elevated levels of AS and EA appear at least broadly related to health anxiety symptoms. These findings are consistent with those from previous research demonstrating that the AS domains are associated with health anxiety (e.g., Cox, Borger, & Enns, 1999; Deacon & Abramowitz, 2008), as opposed to being specifically related to the symptoms of panic disorder. Our findings are also consistent with the hypothesis that problems with anxiety are associated with reduced psychological flexibility and the tendency to try to escape or avoid unpleasant psychological and physical (bodily) experiences.

As predicted, we found that EA and AS were correlated with one another. This is not surprising given that both constructs relate to negative experiences associated with health anxiety: AS is thought to underlie the misinterpretation of certain innocuous body sensations as dangerous and threatening, which leads to fear, avoidance, and the use of safety behaviors to reduce anxiety and the perceived risk of feared consequences. Similarly, EA represents attempts to avoid or suppress emotions, thoughts, and other private experiences such as feared bodily sensations and anxiety responses.

Our partial correlational analyses revealed that the physical concerns domain of AS, but not social or cognitive concerns, predicted health anxiety even after controlling for EA. On the other hand, EA did not remain a significant predictor of health anxiety after controlling for each individual AS domain. Similarly, the results of our regression analyses were only partially supportive of our hypothesis that both EA and AS would uniquely predict health anxiety. Specifically, although we found that both EA and the physical concerns domain of AS predicted anxiety symptoms after accounting for general distress (Step 2), the results from Step 3 of the regression models indicated that independent of the level of psychological flexibility (i.e., EA), AS physical concerns remained a significant predictor of health anxiety symptoms; yet the converse did not apply. That is, EA did not account for additional variance above and beyond that accounted for by the physical concerns dimension of AS. This suggests that the relationship between EA and health anxiety may be a by-product of the variance shared with beliefs and misinterpretations about the dangerousness of physical sensations. Such beliefs independently predicted health anxiety symptoms over and above the unwillingness to endure or tolerate private experiences (i.e., EA).

Hayes et al. (1996) asserted that various forms of psychopathology, including clinical anxiety, can be conceptualized as “unhealthy methods of experiential avoidance” (p. 1154). Our

correlational findings are consistent with this view and indicate a moderate relationship between EA and health anxiety. Our partial correlation and regression analyses, however, provide evidence that the variance in health anxiety symptoms that is explained by EA overlaps with that which is explained by the physical concerns dimension of AS, which explains health anxiety symptoms over and above the explanatory power of EA. It may be that relative to the unwillingness to endure private events and experiences (i.e., EA), specific dysfunctional *beliefs* about internal experiences provide a more empirically valid basis for understanding the psychopathology of health anxiety. Perhaps the concept of EA is too general to independently explain health anxiety symptoms, especially in comparison to explanations based on Beck's (1976) cognitive model.

To the extent that our nontreatment seeking, analogue sample can be used to draw inferences about individuals experiencing clinically significant health anxiety symptoms, our findings have implications for the treatment of health anxiety and hypochondriasis. Traditional cognitive-behavioral therapy (CBT) aims to directly modify beliefs and assumptions, such as those present in the domains of AS, that are associated with problems such as health anxiety (e.g., Abramowitz & Braddock, 2008; Taylor & Asmundson, 2004). ACT, which is considered a part of CBT, purports to add to traditional CBT approaches by targeting EA and other aspects of psychological flexibility (Hayes et al., 2006). The specificity with which the physical concerns dimension of AS predicts health anxiety, however, suggests that these AS-related concerns tap into the pathological process underlying health anxiety more than does EA, and would therefore seem to be a more effective and efficient target for therapeutic change relative to EA. Given the lack of data directly comparing treatment by cognitive restructuring and exposure-based therapy to ACT for health anxiety, and the conceptual overlap between AS and EA, definitive conclusions regarding the preferred treatment approaches require further study.

A number of limitations of the present research should be noted. First, our data were obtained from a nontreatment-seeking sample; albeit these individuals displayed high levels of health anxiety and previous research suggests that results obtained from highly anxious unscreened samples are generalizable to clinical individuals (Burns, Formea, Keortge, & Sternberger, 1995). Second, the correlational nature of this study precludes conclusions about cause and effect relationships. Although our findings are *consistent* with the notion that AS-related beliefs about physical signs and sensations and EA give rise to health anxiety, an alternative explanation is that health anxiety symptoms lead to the acquisition of AS beliefs and the tendency to avoid unpleasant internal experiences. Moreover, it is possible that one or more third variables account for the relationships we report here. Third, the broad scope of the EA concept represents a limitation. The AAQ-II is a general measure of EA that cuts across emotional valence domains and does not discriminate between specific avoidance strategies. Chawla and Ostafin (2007) have argued that the distinction between EA and related constructs such as thought suppression and avoidance coping are not entirely clear. We would encourage authors to develop more specific (i.e., disorder-specific) measures of EA and psychological flexibility to more appropriately test the hypotheses addressed in the present study.

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