Cogn Ther Res (2008) 32:370–385
DOI 10.1007/s10608-006-9085-y

ORIGINAL ARTICLE

The Role of Anxiety Sensitivity in Eating Pathology

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Published online: 15 February 2007

Abstract
Background In past research, anxiety sensitivity (AS) has been identified as a risk factor for anxiety, mood, and alcohol problems. Little work, however, has examined the relationship between AS and eating pathology. We predicted that individuals high in AS would have elevated rates of eating disorder symptoms as measured by the Eating Disorder Inventory (EDI).
Methods Participants in two studies—one undergraduate sample ($N = 88$) and one clinical sample ($N = 96$)—were assessed for anxiety sensitivity and eating disorder symptoms.
Results In both samples, AS was significantly related to EDI-Bulimia scores, controlling for depressive symptoms, trait anxiety symptoms, and impulsivity. In the clinical sample, AS was also significantly related to EDI-Drive for Thinness, controlling for the same covariates. A follow-up analysis suggested that the relationship between AS and EDI eating disorder symptoms was mediated by EDI-Interoceptive Awareness.
Limitations Both studies were cross-sectional, which prohibits causal interpretations. The follow-up mediational analysis must be interpreted with caution due to overlap between the measures of AS and interoceptive awareness. Because of a small sample size and significant comorbidity, the exploratory results analyzing diagnostic categories in Study 2 must be interpreted with caution.
Conclusions AS has a statistically significant relationship to certain eating disorder symptoms measured by the EDI. Future research should investigate whether high AS individuals utilize certain eating behaviors in an effort to regulate somatic symptoms of anxiety.

Keywords Anxiety sensitivity · Bulimia nervosa · Emotion regulation

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Introduction

Anxiety sensitivity (AS) is a traitlike cognitive characteristic that predisposes individuals to the development of anxiety problems (Taylor, 1999). It encompasses fears of physical, mental, and publicly observable experiences (Zinbarg, Barlow, & Brown, 1997), all of which are believed to amplify preexisting anxiety (Reiss, 1991). For example, if a person perceives bodily sensations associated with autonomic arousal as a sign of imminent personal harm, this individual is theorized to be high in AS and therefore to experience elevated levels of anxiety and be at increased risk for a panic attack. Individual differences in AS are hypothesized to emerge from the combined influences of genetic variation along with any number of experiences that ultimately lead to the acquisition of beliefs about the potentially aversive consequences of arousal and anxiety-related states (Reiss & Havercamp, 1996).

Research suggests a clear linkage between AS and panic vulnerability (Schmidt, Lerew, & Jackson, 1997). Because individuals with other types of psychopathology may similarly fear anxiety symptoms, researchers have also evaluated the association between this cognitive factor and other forms of psychopathology (Stewart, Karp, Pihl, & Peterson, 1997; Taylor, 1999). A careful review of this literature suggests that several additional disorders are linked with high levels of AS, including mood and alcohol use disorders (AUD).

The stable presence of high levels of AS is likely to cause a significant degree of stress. Along these lines, several theorists have posited that people suffering from high levels of AS attempt to regulate their emotions through behavioral means and thus find momentary relief from an otherwise constant barrage of stress and negative affect (Cooper, Russell, Skinner, & Windel, 1992; DeHaas, Calamari, Bair, & Martin, 2001; Lejuez, Paulson, Daughters, Bornovalova, & Zvolensky, 2006; Novak, Burgess, Clark, Zvolensky, & Brown, 2003; Stewart & Zeitlin, 1995; Zvolensky & Leen-Feldner, 2005). Much of the research on behavioral attempts to regulate emotions by individuals high in AS has focused on substance use and abuse. For example, among certain daily smokers with high AS, smoking serves important affect regulatory functions. These individuals, specifically, expect tobacco use to help alleviate aversive anxiety states and often are motivated to smoke for affect regulation purposes (Zvolensky et al., 2005). Similarly, AS is associated with high levels of coping motives for drinking (Cooper et al., 1992; DeHaas et al., 2001).

From a functional standpoint, these findings highlight the importance of considering the manner in which AS influences individuals' behavioral approaches to managing anxiety, as different motivations have exhibited different degrees of associations with problematic behaviors. Specifically, coping motives for alcohol consumption have been shown to predict future alcohol problems (Cooper, Russell, & George, 1988) and, as such, the drive to utilize behaviors to dampen negative affect serves as a significant risk factor for the development of pathology.

The degree to which AS, separately from trait anxiety, prompts individuals to engage in these types of problematic behaviors has been debated. However, AS has, in fact, been shown to be a distinct construct (McNally, 1999; Taylor, Koch, & Crockett, 1991), capable of serving as an anxiety accelerant (Rapee & Medoro, 1994; Schmidt, Lerew, & Jackson, 1999; Zvolensky & Leen-Feldner, 2005) that contributes to stronger negative reactions than those experienced by low AS individuals, regardless of trait anxiety level. In an attempt to further elucidate the relationship between AS and maladaptive

behaviors, some researchers have examined the role of tension reduction. Tension-reduction theory (Conger, 1956) explains certain maladaptive behaviors through their negatively reinforcing tendencies, positing that individuals habitually engage in maladaptive behaviors that serve to dampen negative affect. According to this theory, alcohol consumption in particular serves as a functional behavior for the alleviation of anxiety, as one of the major effects of depressants is to reduce somatic anxious sensations. This immediate relief from feared stimuli consequently negatively reinforces the maladaptive coping behavior (DeHaas, Calamari, & Blair, 2002).

Several other behaviors could potentially be capable of providing similar negatively reinforcing somatic and psychological effects. Because certain aspects of eating pathology have been shown to share the emotion regulatory function of substance abuse, it appears reasonable to examine the potential role AS might play in these disorders (Arnow, Kenardy, & Agras, 1995; Greeno, Wing, & Shiffman, 2000; Heatherton & Baumeister, 1991; Powell & Thelen, 1996). To date, little work has been done relating AS to eating pathology. This model of coping related motives for regulating emotions through behavioral means may be applicable, however, as the symptoms of eating pathology have shown in past research to be subject to risk factors that are also involved in other behaviors that have been directly linked to AS, such as substance abuse (Fischer, Anderson, & Smith, 2004). As such, we examined whether AS is significantly related to bulimia nervosa (BN) symptoms, thus suggesting that individuals with increased fear of somatic symptoms of anxiety may eat in an effort to reduce tension and then subsequently purge. Additionally, we examined the relationship between AS and the two other symptom-based measurements on the Eating Disorder Inventory (EDI; Garner, Olmstead, & Polivy, 1983)—Body Dissatisfaction and Drive for Thinness. These subscales were selected because they measure the primary symptoms associated with disordered eating, whereas the other EDI subscales (e.g., Interpersonal Distrust, Maturity Fears) measure risk factors for eating disordered behaviors. Significant relationships between AS and the targeted EDI subscales would provide preliminary support for the consideration of AS as a risk factor for disordered eating symptoms.

To fully understand any potential links found between AS and the symptoms of eating pathology measured on the EDI, it is imperative that other potentially relevant variables be considered as well. By expanding the analysis in this way, interpretive errors can be avoided and the degree to which AS itself explains any statistically significant relationships can be discerned. Another trait frequently implicated in the onset of certain aspects of eating pathology has been impulsivity (Benjamin & Wulfert, 2005; Colder & Chassin, 1997; Fischer et al., 2004; Kane, Loxton, Staiger, & Dawe, 2004; Steiger et al., 2001; Vitaro, Arseneault, & Tremblay, 1999). Because impulsivity has been found to play a significant role in the etiology and maintenance of many maladaptive behaviors, it is important to control for its effects in any analysis examining the potential role of other risk factors for eating pathology. Likewise, given the high degree of overlap between eating, mood, and anxiety symptoms (Brewerton et al., 1995; Viesselman & Roig, 1985; Walsh, Roose, Glassman, Gladis, & Sadik, 1985), it is important to control for the effects of mood and anxiety symptoms when attempting to isolate the link between AS and disordered eating symptoms.

Going even a step further, AS might not be the only variable related to individuals' interpretation of bodily cues that maintains a statistically and clinically significant relationship with the use of maladaptive eating behaviors. Another variable that might potentially play a role in this process and, as such, merits

consideration in any analysis relevant to the topic, is interoceptive awareness, which is defined as the ability to discern subtle visceral and emotional cues from one another. Brusch (1969) posited that an inability to distinguish between emotional cues as well as a tendency to confuse sensations of hunger and satiety would contribute to the development and maintenance of eating disordered behaviors. Fitzgibbon, Sanchez-Johnsen, and Martinovich (2003) reported that eating pathology increased in severity as individuals exhibited less interoceptive awareness. Furthermore, Van Strien, Engels, Van Leeuwe, and Snoek (2005) found that lack of interoceptive awareness partially mediated the relationship between negative affect and overeating. This particular finding indicates that the inability to understand affective and visceral experiences may explain the relationship between negative affect and one aspect of disordered eating, thus indicating that the coping behavior may be motivated in large part by a lack of understanding of current affective and physical states and not simply a desire to dampen negative affect. In other words, these individuals may have misinterpreted bodily or emotional sensations, potentially believing them to be signals for hunger. The question thus becomes whether any statistically significant relationships between AS and the symptoms of eating pathology are mediated by interoceptive awareness, indicating that a general inability to correctly interpret somatic and affective cues, rather than a dearth of ability specific to anxiety, explains the relationship.

Having considered several potential risk factors for eating pathology, we predict that a significant relationship between AS and the three eating disorder symptom subscales (EDI-Bulimia, Body Dissatisfaction, and Drive for Thinness) will emerge in both an undergraduate sample (Study 1) and a clinical sample (Study 2), even when controlling for the effects of depressive symptoms, anxiety symptoms, and impulsivity. Due to the potential affect regulatory function of bulimic behaviors, we believe that the relationship between ASI and EDI-Bulimia will be the most consistent of these findings, as such behaviors might become more readily reinforced due to their potential ability to provide immediate momentary relief from the feared sensations that define AS. In other words, while we believe that many individuals with elevated levels of AS will develop a poor body image and a desire to attain an unrealistic ideal of thinness due to their catastrophic interpretations of normal bodily sensations, a far more common result will be for individuals high in AS to react quickly to their discomfort through behavioral means (e.g. bulimic behaviors) and thus maintain a short term focus on the immediate alleviation of unwanted physical sensations. Additionally, because the hallmark feature of AS is a fear of somatic sensations, it seems reasonable to believe a behavior capable of directly addressing those sensations would have a stronger statistical relationship with AS than would a cognitive symptom of eating pathology. Furthermore, in a follow-up analysis, we expect EDI-Interoceptive Awareness to mediate statistically significant relationships between AS and ED symptoms as measured by the EDI, again controlling for important covariates. In order to evaluate the relationship between AS and eating disorder symptoms more comprehensively, we utilized a sample with generally low levels of psychopathology (e.g. undergraduate research participants), as well as a sample with relatively higher levels of psychopathology (e.g., a clinical sample. Moreover, the use of a second sample allowed us to attempt to replicate any findings from the first sample, thus increasing our confidence in the generalizability of our results.

Study 1

Method

Participants and procedure

Participants in Study 1 were enrolled in an undergraduate introductory psychology class at a large Southeastern university and received course credit for their participation. All 88 participants (77.5% female) signed an informed consent form agreeing to participate in a study on emotions and personality, completed a battery of questionnaires, and were fully debriefed following the study. All procedures were approved by the university's Institutional Review Board. The ethnic composition of the sample was 75.3% Caucasian, 7.9% African-American, 10.1% Hispanic, 2.2% Asian-American, 1.1% Arab or Middle Eastern, 1.1% other, and 2.2% coded as missing data. The participants' ages ranged from 17 to 53 (M = 19.41, SD = 3.82).

Measures

Predictor variables. The Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gurskey, & McNally, 1986) was utilized to assess anxiety sensitivity. The ASI is a 16-item self-report measure and includes items such as ''When I cannot keep my mind on a task, I worry that I might be going crazy.'' Each item on the questionnaire is rated on a five-point scale ranging from 0 (very little) to 4 (very much). The ASI is widely used, and is a valid and reliable measure of anxiety sensitivity (Peterson & Plehn, 1999). The coefficient alpha in the present study was .88.

Dependent variables. The EDI (Garner et al., 1983) consists of 64 questions about eating attitudes and behaviors. The participants were asked to rate symptoms on a scale from 1 (never) to 6 (always). The EDI yields eight subscale scores: Drive for Thinness, Perfectionism, Bulimia, Body Dissatisfaction, Ineffectiveness, Interpersonal Distrust, Interoceptive Awareness, and Maturity Fears. Bulimic symptoms were measured using the Bulimia subscale of the EDI, which includes items such as, ''I stuff myself with food.'' Higher scores on this subscale reflect more severe levels of bulimic symptoms. The alpha coefficient of the Bulimia subscale was .89 for the current sample. Eighty-four of the eighty-eight participants completed the Bulimia subscale without any missing items and were included in the data analysis. The Drive for Thinness and Body Dissatisfaction subscales of the EDI were also used, and their alpha coefficients were .93 and .91, respectively. The Drive for Thinness subscale taps an individual's desire to become thinner and includes items such as, ''I think about dieting,'', while the Body Dissatisfaction subscale includes items such as, ''I think that my stomach is too big.'' Higher scores were indicative of higher levels of the construct being tapped in each subscale.

Control variables. Anxiety symptoms were measured with the Beck Anxiety Inventory (BAI; Beck & Steer, 1993), which consists of 21 cognitive and physiological symptoms related to anxiety. Participants were instructed to indicate the degree to which they were affected by certain anxiety symptoms (e.g., nervous, shaky, faint) ''during the past two weeks'' from 0 to 3 (0 = not at all, 1 = mildly, 2 = moderately, 3 = severely). The alpha coefficient for the BAI in this sample was .92.

Depression symptoms were assessed with the Beck Depression Inventory – Second Version (BDI-II; Beck, Steer, & Garbin, 1998). The BDI-II is a self-report questionnaire that consists of 21 items. The respondent is asked to select one statement from a group of four statements that reflects his/her current level of depression symptom severity (scored from 0 to 3). For example, one item uses the statement ''I do not feel sad,'' as the least severe (and would be rated as a 0), while the statement that is most severe (and would be rated as 3) is, ''I am so sad or unhappy that I can't stand it.'' The BDI has been shown to be reliable and valid (see Beck et al., 1998). In the current sample, the alpha coefficient was .90.

The Urgency, (lack of) Premeditation, (lack of) Perseverance, Sensation Seeking Impulsive Behavior Scale (UPPS; Whiteside & Lynam, 2001) is a 45-item self-report measure that yields four subscales: Urgency, (lack of) Premeditation, (lack of) Perseverance, and Sensation Seeking. Whiteside and Lynam (2001) proposed that the construct of impulsivity could be broken down into four distinct pathways to impulsive behavior. The four subscales of the UPPS are designed to represent each of the pathways. The Urgency subscale is designed to measure one's tendency to act rashly in the face of distress, perhaps to assuage distress in the short-term, regardless of long-term negative consequences. Thus, the Urgency subscale was utilized in Study 1, because of its relevance to the prediction that individuals with AS would engage in disordered eating behaviors in an attempt to alleviate distress associated with AS. The Urgency subscale includes 12 items such as ''When I feel bad, I will often do things I later regret to make myself feel better now.'' Each item on the questionnaire is rated on a 5-point scale ranging from 1 (not true of me) to 5 (very true of me). The UPPS is widely used, and is a valid and reliable measure of impulsivity (e.g., Claes, Vandereycken, & Vertommen, 2005). The coefficient alpha for the Urgency subscale of the UPSS in this sample was .89.

Proposed mediator variable for follow-up analysis. Interoceptive Awareness was assessed with the EDI. The current study utilized the Interoceptive Awareness subscale to assess the proposed mediator. This subscale includes items such as, ''I get frightened when my feelings are too strong,'' and ''I can clearly identify what emotion I am feeling (reversed).'' Higher scores reflect lower levels of interoceptive awareness. The Interoceptive Awareness subscale had an alpha coefficient of .86 in Study 1.

Results

Means and standard deviations for predictors and dependent variables, as well as their intercorrelations, are provided in Table 1.

ASI scores predicting EDI scores. Regression analyses were conducted to predict EDI scores from ASI scores, when controlling for BDI, BAI, and Urgency scores, in the non-clinical sample. In separate analyses, EDI-Bulimia, EDI-Drive for Thinness, and EDI-Body Dissatisfaction scores were used as the dependent variables. For each analysis containing a different dependent variable, the following regression analysis was conducted: Step 1: entry of BAI, BDI, and Urgency scores to control for levels of anxious symptoms, depressive symptoms, and impulsivity, respectively. Step 2: entry of ASI score. The results indicated that when controlling for BAI, BDI, and Urgency scores, ASI scores significantly predicted EDI-Bulimia scores (pr = .20, t = 2.23;

Table 1 Descriptive data and intercorrelations all variables

Measure	1	2	3	4	5	6	7	8
1. BAI	–	.62**	.40**	.34**	.31**	.26*	.27*	.45**
2. BDI	.65**	–	.32**	.23*	.21	.35**	.28**	.47**
3. ASI	.64**	.63**	–	.32**	.21	.39**	.26*	.54**
4. UPSS/IBS	.32**	.41**	.33*	–	.41**	.52**	.35**	.57**
5. EDI-DT	.27**	.48**	.43**	.25*	–	.54**	.76**	.57**
6. EDI-Bul	.29**	.43**	.46**	.14	.70**	–	.55**	.75**
7. EDI-BD	.16	.48**	.63**	.10	.72**	.55**	–	.53**
8. EDI-IA	.66	.70**	.72**	.39**	.55**	.58**	.51**	–
M (Study 1)	11.20	7.45	22.51	30.62	22.29	15.26	30.82	22.61
M (Study 2)	13.78	14.32	19.83	45.38	18.44	13.84	30.91	26.08
SD (Study 1)	9.62	6.80	11.85	9.09	8.23	6.88	11.07	7.85
SD (Study 2)	11.50	10.71	11.75	13.75	9.27	6.45	16.34	10.25

Note: Correlations above the diagonal represent intercorrelations between all measures among Study 1 (nonclinical) participants; correlations below the diagonal represent intercorrelations between all measures among Study 2 (clinical) participants. BAI: Beck Anxiety Inventory (Beck & Steer, 1993); higher scores indicate greater levels of anxiety. BDI: Beck Depression Inventory – Second Version (Beck et al., 1998); higher scores indicate greater levels of depressive severity. ASI: Anxiety Sensitivity Index (Reiss et al., 1986); higher scores indicate more severe levels of anxiety sensitivity. UPSS-Urg: The UPPS Impulsivity Scale – Urgency Subscale (Whiteside & Lynam, 2001); higher scores indicate greater levels of urgency (used in Study 1). IBS: Impulsive Behavior Scale (Rosotto et al., 1998); higher scores indicate greater levels of impulsivity (used in Study 2). EDI-IA: a subscale on the Eating Disorder Inventory, Interoceptive Awareness (Garner, 1983); higher scores indicate lower levels of interoceptive awareness. EDI-Bul: a subscale on the Eating Disorder Inventory, Bulimia (Garner, 1983); higher scores indicate higher levels of bulimic symptomatology. EDI-BD: a subscale on the Eating Disorder Inventory, Body Dissatisfaction (Garner, 1983); higher scores indicate higher levels of body dissatisfaction. EDI-DT: a subscale on the Eating Disorder Inventory, Drive for Thinness (Garner, 1983); higher scores indicate higher levels of drive for thinness. *M*: Mean. *SD*: Standard Deviation.* $P < .05$; ** $P < .01$

$P < .05$). However, when controlling for the same covariates, ASI scores did not significantly predict EDI-Body Dissatisfaction scores ($pr = .09$, $t = 0.88$; P = ns) or EDI-Drive for Thinness scores ($pr = .10$, $t = 0.14$; P = ns).[1]

Follow-up analysis

Interoceptive awareness as a mediator between anxiety sensitivity and eating disordered symptoms. To test the hypothesis that EDI-Interoceptive Awareness scores would serve as a mediator of any significant relationship between ASI and an EDI subscale when BDI, BAI, and Urgency scores were considered as covariates, mediational analyses were conducted. In this sample, the only EDI subscale that was significantly predicted by ASI scores was EDI-Bulimia. According to Baron and Kenny (1986), four criteria must be met to indicate the presence of a significant mediating variable: The predictor variable (ASI) must significantly predict the mediator variable (EDI-interoceptive awareness; $\beta = .20$, $t = 3.46$, $P < .01$), β^2 the mediating variable must predict the dependent variable (EDI-Bulimia; $\beta = .60$, $t = 7.06$, $P < .001$), the predictor variable

[1] When no covariates were used in the regression analyses, ASI scores significantly predicted EDI-bulimia ($P < .001$) and EDI-body dissatisfaction ($P < .05$) scores. However, ASI scores only significantly predicted EDI-drive for thinness scores at a trend level ($P = .06$).

[2] refers to the unstandardized beta weight.

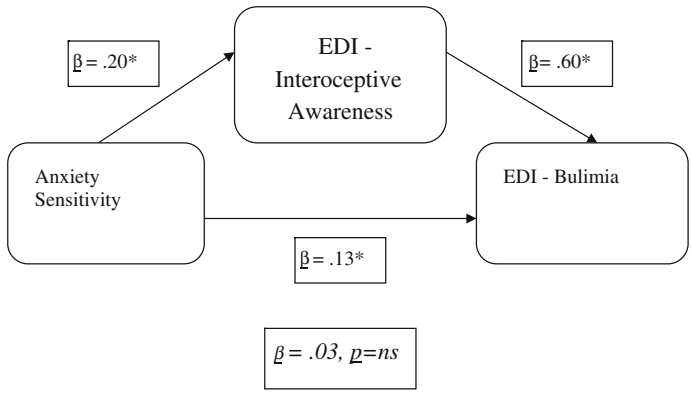

Fig. 1 EDI-Interoceptive Awareness scores mediate the relationship between Anxiety. Sensitivity Index scores and EDI-Bulimia scores in the non-clinical sample. *Notes*: * indicates $p < .05$. EDI stands for Eating Disorder Inventory. Unstandardized beta weight when mediator variable is included in the model is presented in *italics*

must predict the dependent variable ($\beta = .13$, $t = 2.23$, $P < .05$), and the relationship between the predictor variable and the dependent variable must become non-significant when the mediator variable is added to the mode (ASI's new values: $\beta = .03$, $t = 0.52$, P = ns). Thus, our results were consistent with all four criteria. Please see Fig. 1.

Furthermore, a direct significance test of the mediated pathway was conducted as suggested by Sobel (1982). This test was significant ($z = 4.68$, $P < .001$), suggesting that EDI-Interoceptive Awareness scores mediated the relationship between ASI scores and EDI-Bulimia scores. However, the correlation between the ASI and the EDI-Interoceptive Awareness subscale in this sample was quite high (.54) and, as such, the results from this particular analysis should be interpreted with caution.[3]

Study 2

In an attempt to establish convergent validity for our findings in Study 1 and, additionally, to examine whether those findings can be replicated in a sample featuring more consistently elevated levels of psychopathology, we performed the same study utilizing a clinical sample. Should the results prove concordant with those reported above, this would justify increased confidence in their generalizability across samples and offer preliminary evidence that the relationship between AS and eating disorder symptoms extends beyond minor symptomatology to include clinical symptom levels.

[3] Due to concerns about the high correlation between AS and EDI-Interoceptive Awareness, an alternative approach towards the mediational analysis was also utilized. Items on both the ASI and the EDI-Interoceptive Awareness scale that rationally appeared to overlap across constructs were deleted from each measure (items 33, 40, 47, and 64 on the EDI-IA; items 5 and 16 on the ASI). The correlation between the two scales decreased from .54 to .44. After this step, the mediational analyses were run again. The results were identical to those found with the original versions of the scale, with interoceptive awareness mediating the relationship between AS and eating disordered symptoms as measured by the EDI. This increases our confidence that this mediational relationship is worthy of further exploration in future studies, but nonetheless, caution should be used in interpreting the results.

Methods

Participants and procedures

Study 2 involved the analysis of data that were collected from adults who applied for psychological services at an outpatient mental health clinic between May 2004 and April 2006. The outpatient clinic is affiliated with a university psychology department, and treats clients with a variety of clinical disorders. The only exclusion criteria for services at the clinic are that individuals with psychotic- or bipolar-spectrum disorders must be stabilized on medications before treatment at the clinic, and those representing an imminent danger to self or others are referred to inpatient hospitalization. Upon applying for services, all clients were informed of the research and training nature of the clinic. All participants signed a consent form to participate in ongoing research, and all procedures were approved by the university's Institutional Review Board.

The sample was comprised of 96 clients; 54.2% were women. The average age of participants in the sample was 25.74 (*SD* = 9.48; range = 18–54). Regarding ethnicity, 78.1% of the participants were White, 8.3% were Black, 8.3% were Hispanic, 1.1% were Asian/Pacific Islander, 2.1% were American Indian/Alaskan Native, and 2.1% chose not to identify their ethnicity.

Measures

Predictor variables. The predictor variable (ASI) was assessed through the same measures in Study 2 as they were in Study 1.

Dependent variables. The dependent variables (EDI Bulimia, Drive for Thinness, and Body Dissatisfaction) in the second sample were measured in the same manner as they were in Sample 1.

Control variables. The control variables (trait anxiety, depression, and impulsivity) in the second sample were identical to those utilized in Sample 1 with the exception of the impulsivity construct, which was measured with the Impulsive Behavior Scale (IBS; Rosotto, Yager, & Rorty, 1998) instead of the UPPS-Urgency subscale. The IBS is a 25-item measure that asks participants to indicate how many times they have engaged in particular impulsive behaviors (e.g., self-mutiliation, shoplifting, promiscuous sex). Participants are asked to indicate how many times they have engaged in each of the impulsive behaviors on a 5-point scale (1 = never; 2 = once; 3 = on occasion; 4 = sometimes; 5 = regularly). The coefficient alpha for this sample was .88.

Proposed mediator variable for follow-up analysis. This variable (EDI interoceptive awareness) was measured in Study 2 with the same instrument that was utilized in Study 1.

Data analytic strategy

Multiple regression analyses were used to determine whether AS scores significantly predicted EDI-Bulimia, Body Dissatisfaction, and Drive for Thinness scores when controlling for trait anxiety, depressive symptoms, and impulsivity. To further examine any significant relationships between AS and eating disorder symptoms as measured by the EDI, follow-up mediational analyses were conducted to determine whether EDI-Interoceptive Awareness scores mediated the relationship between AS and eating disordered symptomatology.

Results

Means and standard deviations for predictors and dependent variables, as well as their intercorrelations, are provided in Table 1.

ASI scores predicting EDI scores. Regression analyses essentially identical to those utilized in Study 1 were conducted to predict EDI scores from ASI scores, when controlling for BDI, BAI, and IBS scores in the clinical sample, the only difference being the use of the IBS in place of the UPPS. The results indicated that when controlling for BAI, BDI, and IBS scores, ASI scores significantly predicted EDI-Bulimia scores (pr = .25, t = 3.15; P < .01) and EDI-Drive for Thinness scores (pr = .18, t = 2.25; P < .05). However, ASI scores did not significantly predict EDI-Body Dissatisfaction scores (pr = .08, t = 0.96; P = ns).[4]

Follow-up analysis

Interoceptive awareness as a mediator between anxiety sensitivity and eating disordered symptoms. To test the hypothesis that EDI-Interoceptive Awareness scores would serve as a mediator of any significant relationship between ASI and an EDI subscale in the clinical sample when BAI, BDI, and IBS scores were included as covariates, mediational analyses were again conducted. In this sample, both EDI-Bulimia and EDI-Drive for Thinness were significantly predicted by ASI scores. The mediational analysis for EDI-Bulimia scores was conducted first. Our results indicated that Baron and Kenny's (1986) four criteria were met, such that ASI scores significantly predicted EDI-Interoceptive Awareness scores (β = .29, t = 4.34, P < .001), EDI-Interoceptive Awareness scores predicted EDI-Bulimia scores (β = .38, t = 5.62, P < .001), ASI scores predicted EDI-Bulimia scores (β = .18, t = 3.15, P < .01), and the relationship between ASI and EDI-Bulimia became non-significant when EDI-Interoceptive Awareness was added to the model (ASI's new values: β = .08, t = 1.44, P = ns). Furthermore, a direct significance test of the mediated pathway was conducted as suggested by Sobel (1982). This test, which was significant (z = 4.70, P < .001), suggests that EDI-Interoceptive Awareness scores mediated the relationship between ASI scores and EDI-Bulimia scores (see Fig. 2). The correlation between the ASI and the EDI-Interoceptive Awareness was quite high in this sample (.67) and, as such, the results should be interpreted with caution. Please see Fig. 2.

Next, the potential mediational relationship of EDI-Interoceptive Awareness was examined when EDI-Drive for Thinness served as the DV. Our results indicated that Baron and Kenny's (1986) four criteria were met, such that ASI scores significantly predicted EDI-interoceptive awareness scores (β = .29, t = 4.34, P < .001), EDI-Interoceptive Awareness scores predicted EDI-Drive for Thinness scores, (β = .46, t = 4.47, P < .001), ASI scores predicted EDI-Drive for Thinness scores (β = .19, t = 2.25, P < .05), and the relationship between ASI and EDI-Drive for Thinness became non-significant when EDI-Interoceptive Awareness was added to the model (ASI's new scores: β = .06, t = 0.77, P = ns). Furthermore, a direct significance test of the mediated pathway was conducted as suggested by Sobel (1982). This test was significant (z = 4.54, P < .001), suggesting that EDI-Interoceptive Awareness scores mediated the relationship between

[4] When ASI scores were used to predict EDI-bulimia, EDI-interoceptive awareness, EDI-drive for thinness, and EDI-body dissatisfaction scores with no covariates, all of the analyses were significant (i.e., ASI significantly predicted each EDI subscale score; all p's < .001).

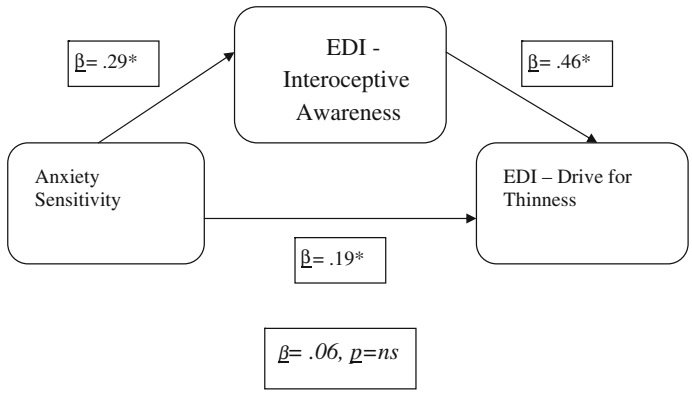

Fig. 2 EDI-Interoceptive Awareness scores mediate the relationship between Anxiety. Sensitivity Index scores and EDI-Drive for Thinness scores in the clinical sample. *Notes*: * indicates $P < .05$. EDI stands for Eating Disorder Inventory. Unstandardized beta weight when mediator variable is included in the model is presented in *italics*

ASI scores and EDI-Drive for Thinness scores[5,6] (see Fig. 3). Because of the high correlation between the ASI and the EDI-Interoceptive Awareness subscale in this sample, these results must also be interpreted with caution. Please see Fig. 3.

Discussion

In both samples, we found a statistically significant relationship between AS and bulimic symptoms as measured by the EDI. These results were robust even when controlling for depressive symptoms, anxiety symptoms, and impulsivity. This constitutes a fairly stringent test of our hypothesis, as the consistently significant relationships between these covariates and the outcome variable are certain to account for a substantial amount of variance. Additionally, some debate exists as to whether or not the BAI, which we used to measure trait anxiety, also overlaps with certain aspects of anxiety sensitivity, thus indicating that our use of the BAI as a covariate may indeed represent a stringent approach to this analysis. Our results indicate that individuals with particularly potent fears of the somatic manifestations of anxiety may be at an increased risk for exhibiting some of the problematic eating behaviors and beliefs measured by the EDI.

[5] Due to the low number of patients with eating disorder diagnoses ($n = 5$) and the large amount of comorbidity within this sample, analyses examining the role of anxiety sensitivity in diagnosis were essentially impossible. However, the patients included in this sample exhibited elevated eating disorder symptoms as indicated by mean scores on the EDI subscales (bulimia, drive for thinness, and body dissatisfaction). In a purely exploratory regression analysis, we found that ED diagnosis was, in fact, significantly related to AS scores. Future studies with larger samples of eating disorder diagnoses would help to further elucidate the nature of our findings.

[6] Due to concerns about the high correlation between AS and EDI-Interoceptive Awareness, the same alternative approach towards the mediational analysis utilized in Study 1 was also utilized in Study 2 and the correlation between the two scales was thus decreased from .67 to .65. Given this small decrease (i.e. the observation that the correlation did not significantly decrease in magnitude), subsequent mediational analyses were not conducted.

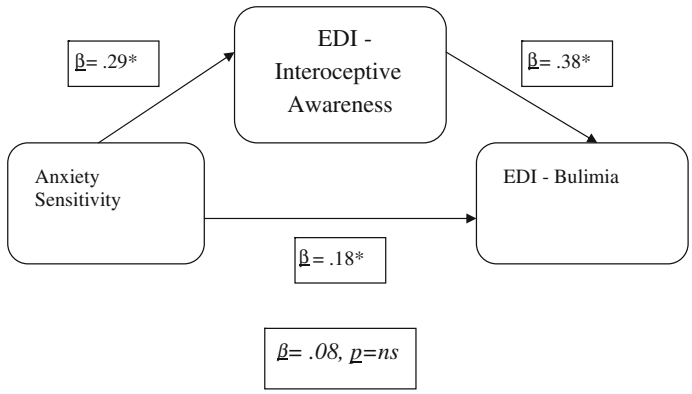

Fig. 3 EDI-Interoceptive Awareness scores mediate the relationship between Anxiety. Sensitivity Index scores and EDI-Bulimia scores in the clinical sample. *Notes*: * indicates $P < .01$. EDI stands for Eating Disorder Inventory. Unstandardized beta weight when mediator variable is included in the model is presented in *italics*

Traditionally, individuals with bulimia and individuals exhibiting subthreshold bulimic behavioral tendencies have been considered impulsive if for no other reason than the fact that the long term consequences of these behaviors outweigh their short term reinforcing values (Claes et al., 2005; Engel et al., 2005; Fischer, Smith, & Anderson, 2003). However, the significant positive relationship found here between AS and EDI-bulimia scores, even when controlling for impulsivity may point to an additional subset of patients with bulimia or eating disorder not otherwise specified (EDNOS) whose distinct symptom pattern requires additional clinical and research attention. Individuals affected by this particular aspect of the phenomenology of BN or EDNOS may react to a specific form of discomfort—somatic sensations related to anxiety—rather than general distress. Unlike their simply impulsive counterparts, these individuals may not be prone to this type of behavior in response to other forms of negative affect or distress.

The significant relationship between AS and EDI-Drive for Thinness scores in the clinical sample further supplements the support for our hypothesis that AS plays an important role in the development and maintenance of eating disorder symptoms. It appears that individuals high in AS also experience increased levels of drive for thinness, indicating that their fear of somatic anxious symptoms may affect the degree to which they feel compelled to achieve an exaggerated ideal of thinness. As such, these results seem to paint the picture of individuals overwhelmed and confused by their own internal experiences who, in their attempts to stabilize their affective and physiological states, are at increased risk for developing a tendency to utilize maladaptive behaviors to cope with what they believe is an unacceptable immediate environment. This relationship could best be conceptualized as a reaction indicative of these individuals misinterpreting what might enable them to diminish the somatic sensations they tend to fear. In other words, if these individuals are misinterpreting their physiological cues of satiety and viewing them as aversive, their drive for thinness might be viewed as a desire to simply avoid the cause of an undesired outcome.

Also of interest is the fact that AS was not significantly related to drive for thinness in the undergraduate sample (Study 1). One interpretation of this is simply chance occurrence—a measurement artifact could have caused the relationship to be misrep-

resented in one or both of the samples. Additionally, a third variable could be playing a pivotal role in altering the relationship across the two samples. For instance, the more prominent presence of psychopathology in general in the clinical sample might be influencing the relationship between AS and EDI-Drive for Thinness. The clinical sample, in fact, had higher mean scores on the BDI (M = 14.32, SD = 10.78) and the BAI (M = 13.78, SD = 11.50) than did the nonclinical sample (BDI–M = 7.45, SD = 6.80; BAI–M = 11.20, SD = 9.62). If heightened pathology at least partially explains these divergent results, then it might be that AS is related to drive for thinness only in certain cases, perhaps indicating that an additional variable related to heightened pathology moderates the relationship between the two variables of interest.

The absence of a significant relationship between AS and body dissatisfaction in both samples is also an important consideration. This indicates that the effects of AS on problematic eating symptoms is limited to particular aspects of the phenomenon. More specifically, this indicates that AS has little or no effect on an individual's ability to be content with his or her body type. This makes intuitive sense in that AS has more to do with interpretations of somatic cues rather than cognitions regarding the aesthetic value of body type.

Our finding that EDI-Interoceptive Awareness may mediate the relationships between AS and both EDI-Bulimia and EDI-Drive for Thinness scores could potentially provide a theoretical framework within which to conceptualize the relationship between risk factor and maladaptive behavioral outcome. Individuals lacking in interoceptive awareness often endorse an inability to differentiate between hunger and satiety and, additionally, an inability to distinguish between subtle emotional cues and their physical correlates. As such, while the natural physical reaction to discomfort is a suppressed appetite due to resulting inhibition of gastric contractions and heightened blood sugar levels (Van Strien et al., 2005), these individuals might react in the opposite manner, misinterpreting bodily cues and seeking comfort in food.

These findings thus appear to indicate that the relationship between AS and the symptoms of eating pathology can be traced to a generally stunted ability to understand bodily and affective cues rather than a specific reaction to the physical cues of anxiety. Individuals with elevated AS who exhibit symptoms of eating pathology may thus fall within a broader category of individuals who, due to an inability to properly interpret somatic cues, engage in maladaptive behavior approaches towards alleviating what they believe is an undesirable and potentially harmful physical sensation. Further research utilizing measures capable of precisely discerning the nature of both AS and interoceptive awareness would be highly valuable, as it would clarify whether or not EDI-Interoceptive Awareness truly serves as a mediator in this relationship.

Both of these studies were cross sectional, which prohibits drawing firm conclusions regarding causation in the reported relationships. Also, both studies utilized the EDI on both men and women. While some recent studies have presented evidence that measures of eating disorder symptoms maintain the same factor structure and are reliable across genders (Boerner, Spillane, Anderson, & Smith, 1994), none have addressed the EDI in particular, which means our results must be considered in light of that potential confound. Future studies that utilize multiple methods for measuring the symptoms of interest, preferably including behavioral manipulations, would increase the degree to which the results could be confidently generalized across samples. Additionally, longitudinal measures would enable more reliable conclusions regarding causality.

Finally, the sample sizes in both studies was fairly small and, as such, it is unclear to what degree the results are generalizable.

Overall, these studies reveal a consistent and strong relationship between anxiety sensitivity and eating disorder symptoms, particularly those measured by the EDI-Bulimia subscale. As such, AS needs to be considered as a potential risk factor for the development of eating disorders in addition to its already accepted status as a potential precursor of substance abuse and anxiety disorders. These findings may have implications for treatment, as individuals who endorse high scores in AS could be taught alternative, adaptive approaches to diminishing the fear response they encounter in response to somatic anxious sensations. As such, preventative care could be given to a previously unknown high risk population. Future work examining the potential therapeutic effects of treating AS symptoms in BN or EDNOS patients exhibiting elevated AS would help determine the plausibility of administering such alternate treatment approaches.

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