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Emotion Regulation Difficulties in Anorexia Nervosa: Relationship to Self-Perceived Sensory Sensitivity

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Abstract

Changes in sensation (e.g., prickly skin) are crucial constituents of emotional experience, and the intensity of perceived changes has been linked to emotional intensity and dysregulation. The current study examined the relationship between sensory sensitivity and emotion regulation among adults with anorexia nervosa (AN), a disorder characterized by disturbance in the experience of the body. Twenty-one individuals with AN, 20 individuals with AN who were weight-restored, and 23 typical controls completed self-report measures of sensory sensitivity and emotion regulation. AN participants reported heightened sensory sensitivity and greater difficulty regulating emotions relative to controls. Self-perceived sensory sensitivity was associated with greater emotion dysregulation. Weight-restored AN participants reported greater ability to regulate emotions than their currently underweight counterparts, despite heightened sensitivity. Findings suggest that hypersensitivity may be a persisting feature in AN, and that weight restoration may involve improved ability to cope with sensation.

Keywords

emotion regulation; sensory sensitivity; sensation; anorexia nervosa; acceptance

Anorexia nervosa (AN) is a perplexing disorder in which the most basic biological needs are neglected or defied. The state of starvation characteristic of AN seems to be the preferred condition for individuals with AN, with devastating, life-threatening symptoms highly valued and experienced as ego-syntonic (Vandereycken, 2006). Why AN is coveted by those affected is not fully understood; however, corroborating evidence suggests that the illness, often emerging in adolescence or young adulthood, may help some individuals cope with the stress associated with increasingly complex social-emotional contexts (Schmidt & Treasure,

2006; Zucker et al., 2007). From this perspective, AN may be conceptualized as a maladaptive emotion regulation strategy which effectively attenuates psychological distress via attempts to directly manipulate the body (Harrison, Sullivan, Tchanturia, & Treasure, 2010; Merwin, et al., 2011).

Emotions are complex experiences that include somatic constituents (e.g., prickly skin, racing heart) (Damasio, 1995) and the intensity of these sensations usually corresponds with emotional intensity (Pollatos, Gramann, & Schandry, 2007). Since the earliest conceptualizations of AN (e.g., Bruch, 1962), there has been an attempt to understand how individuals affected by the illness experience bodily sensation (e.g., states of arousal, hunger/satiety) and how this may contribute to psychopathology. It is sometimes assumed that sensation is muted among individuals with AN and that hyposensitivity to somatic-affective cues contributes to symptom development by encouraging reliance on rules and other external cues to dictate food intake and other behaviors (Fassino, Pierò, Gramaglia, & Abbate-Daga, 2004; Pollatos et al., 2008). However, it is equally plausible that individuals with AN are *hypersensitive* to sensory experience but have difficulty tolerating or interpreting these sensations, and this translates to an inability to use these signals to guide behavior adaptively. If individuals vulnerable to AN are hypersensitive to somatic sensations (globally or in specific sensory domains), this might make the emotional upheaval of life transitions (e.g., adolescence) particularly aversive. Under such conditions, a symptom cluster that decreases the experience of visceral sensation (e.g., via biological adaptations that occur with starvation, or by directing attention away from these stimuli) may be highly reinforcing, especially in the absence of other strategies to down regulate this arousal healthfully.

Investigators most often study individuals with AN in the acutely ill (and thus starved) state. This state may mask heightened visceral sensitivity that predates illness onset. For example, in Pollatos et al (2008), individuals with AN demonstrated deficits in the ability to detect their own heartbeat (suggesting hyposensitivity), however, performance was significantly correlated with body-mass index (BMI). Although this might indicate greater hyposensitivity is associated with illness severity, an alternative explanation is that difficulty detecting one's heartbeat was a byproduct of degree of starvation and associated complications (e.g., bradycardia) which would weaken somatic signals. In fact, the existence of this BMI-visceral sensation relationship points to the potential utility of AN as strategy to dampen emotional experience via attenuating the somatic correlates. Studies have established that individuals with AN are nonaccepting of emotional responses and this is associated with dietary restraint (e.g., Merwin, Zucker, Lacy, & Elliott, 2010). However, it is unclear why emotions are rejected in this patient population and whether this is functionally related to AN symptomatology.

Emotion regulation models of psychiatric illness propose that heightened reactivity to emotional stimuli perpetuates the need for strategies to manage frequent and intense emotional arousal (e.g., Crowell, Beauchaine, & Linehan, 2009). Recently, this conceptualization has been applied to individuals with eating disorders (EDs) (Wildes, Ringham, & Marcus, 2010). However, evidence for this conceptualization remains tenuous and more work is needed. A recent review by Haynos and Fruzetti (2011) reported only one study that demonstrated slow return to baseline levels of arousal among individuals with AN. Further, the majority of supportive studies described actually assess increased attention and distress in response to pathology-relevant stimuli (e.g., food or body weight/shape stimuli) (e.g., Johansson, Ghaderi, & Andersson, 2005). These attention biases and psychological reactions are different from overall sensitivity to the changes in visceral organs that constitute emotional experience. Thus, while factors such as reactivity (per previous models) might increase the intensity or frequency of emotions, sensitivity to

visceral change could be responsible for the extent to which emotions are experienced as intolerable and perpetuate strategies to contain or attenuate these experiences.

The current study was an initial attempt to: 1) elucidate emotion regulatory difficulties among individuals with AN, specifically parsing the effects of undernourishment by including a weight-restored AN control group, and 2) assess whether perceived sensitivity to sensation contributes to difficulties regulating emotion, thereby motivating AN symptomatology. We hypothesized that, relative to control participants, reported deficits in emotion regulation would be greater among individuals with AN as well as those with a history of AN who were weight-restored (albeit to a lesser degree than among their undernourished counterparts). We expected similar findings for sensory sensitivity (AN-C sensitivity > WR sensitivity > TC sensitivity). We also tested the exploratory hypothesis that the relationship between perceived sensory sensitivity and emotion dysregulation would be moderated by group, suggesting a process unique to AN.

In the current study, we used self-report measures to assess constructs of interest. Although it would be ideal to study emotion regulation capacities and sensory sensitivity using laboratory-based paradigms or sampling behavior in the natural environment, a reasonable first step is to assess these variables using self-report measures with demonstrated validity. If reliable relationships are found, this would support additional more involved studies assessing whether these are objective differences in emotion regulation (e.g., actual deficits in emotional clarity) and/or sensation, or differences in how individuals with AN perceive their abilities and sensations.

This work extends studies that found individuals with AN differed from participants without a history of an ED in emotion regulatory capacities (Harrison, Sullivan, Tchanturia, & Treasure, 2009; Harrison et al., 2010) by including a weight-restored control group and examining the relationship between these deficits and perceived sensitivity to sensation.

Method

Participants

The sample was comprised of 59 adult females who agreed to participate in a study of social functioning in AN. Participants were recruited via flyers posted within a 50-mile radius of the hosting university and electronic advertisements posted to parent forums and web-sites devoted to EDs. Notices were sent to a mailing list of healthcare providers within a 60-minute traveling distance known to specialize in EDs and to all therapists in two university-based ED programs. The sample was limited to females. While ideal to recruit both genders, AN affects females at a much higher rate (Hoek & Hoeken, 2003) and including males for comparisons would require an unwieldy sample size.

Individuals who called with interest in the study completed a telephone screening. Final eligibility was determined by self-report measures of current and lifetime ED symptoms and a face-to-face structured diagnostic interview of ED symptoms conducted by a PhD level psychologist with expertise in the assessment of EDs. To be eligible, individuals had to meet diagnostic criteria for AN currently (AN-C) or had to have met criteria in the past but be weight-restored (WR) for a minimum of 6 months prior to study participation. To be eligible as a control participant (CN), individuals had to have no current or past ED symptoms. Diagnosis of AN was based on the Diagnostic and Statistical Manual for Mental Disorders IV (DSM-IV) (American Psychiatric Association, 1994) criteria. However, consistent with previous studies (Roberto, Steinglass, Mayer, Attia, & Walsh, 2008), the criterion of amenorrhea was relaxed for the current study. As such, the current AN group (AN-C) is

most accurately conceptualized is a combination of AN and AN-NOS, with the subtype that continues to menstruate.

BMI determination—Maintenance of an unhealthy low-weight is a defining feature of AN and the DSM-IV provides a criterion for this determination of BMI less than 18.5. However, ideal body weight for a given individual is complex and most precisely defined by an individual's weight history along with other parameters that index physical recovery (Dei, Seravalli, Bruni, Balzi, & Pasqua, 2008; Misra et al., 2006). We determined ideal BMI and whether participants had current or past low BMI by combining information from a detailed weight history, structured diagnostic interview, and self-report measures. Medical record abstraction was also used when further clarification was needed. For some participants, this strategy resulted in deviations from the classic distinction of a BMI of 18.5 or less as underweight, but more accurately depicts the weight history and optimal functioning of a given individual. This approach is not only less arbitrary but also a better depiction of the inter-individual clinical complexity of AN.

Procedure

Participants completed a structured diagnostic interview and a self-report assessment battery. The Eating Disorder Examination (EDE) (Fairburn & Cooper, 1993) was used to quantify ED symptomatology, the Difficulties in Emotion Regulation Scale (DERS) (Gratz & Roemer, 2004) provided an index of emotion regulation difficulties, and the Sensory Profile (SP) (Brown, Tollefson, Dunn, Cromwell, & Fillion, 2001) characterized individuals' self-perceived sensitivity to sensation.

Participants also completed the Brief Symptom Inventory (BSI) to quantify anxiety and depression and self-reported psychotropic medications. Individuals with AN often have comorbid anxiety and mood disorders, either predating illness onset (Deep, Nagy, Weltzin, Rao, & Kaye, 1995) or occurring in the context of AN symptoms which impact cognitive, social and emotional functioning (Zucker et al., 2007). Although the current study sample size precludes controlling for comorbid psychiatric symptoms and psychotropic medications, we gathered this information for descriptive purposes (provided in Table 1) and consider the findings in light of this limitation.

Measures

Eating Disorder Examination (EDE; Fairburn & Cooper, 1993): The EDE is a widely implemented structured diagnostic interview of ED psychopathology. Behavioral and attitudinal features are assessed according to DSM-IV diagnostic criteria. While the interview largely focuses on symptoms over the past 28 days, behavioral diagnostic criteria (e.g., self-induced vomiting, driven exercise) are assessed for presence, frequency, and total days of occurrence over the past three months in accordance with the duration criteria specified in DSM-IV. Attitudinal items are rated according to a seven-point scale with higher scores reflecting more severe ED psychopathology. These items form four subscale scores including Restraint, Eating Concern, Shape Concern, and Weight Concern computed as the mean of constituent scale items. Discriminant validity, internal consistency, and concurrent validity are well documented for the EDE (Fairburn & Cooper, 1993).

Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994): The EDE-Q is a 41-item self-report version of the EDE. The EDE-Q measures ED symptomatology and yields the same diagnostic criteria and four subscales scores. Good convergence between the EDE and EDE-Q has been documented among community and clinical samples, though inconsistencies have been reported (e.g., Fairburn & Beglin, 1994; Mond, Hay, Rodgers, Owen, & Beumont, 2004). Both the interview and the self-report

version of the EDE were administered to insure we captured the breadth of eating pathology given the demand characteristics of a live interview.

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004): The DERS is a 41-item scale assesses how an individual responds to emotions. Capacities are differentiated, such as the ability and willingness to recognize and label affective experience, acceptance of emotional responses, ability to behave effectively when experiencing emotion and access to healthy ways to cope with heightened affect. The subscales, along with sample items, are provided below. The DERS has demonstrated good psychometric properties in college-aged samples. Scores on the DERS correspond as expected with behavioral tendencies and forms of psychopathology theoretically linked to deficits in emotion regulatory abilities (e.g., self-harm, Borderline Personality Disorder; Gratz & Roemer, 2004).

Nonacceptance of Emotional Responses: Sample item: *When I am upset, I feel guilty for feeling that way.*

Difficulties Engaging in Goal-Directed Behavior: Sample item: *When I am upset I have difficulty concentrating.*

Impulse Control Difficulties: Sample item: *When I am upset, I feel out of control.*

Lack of Emotional Awareness: Sample item: *I pay attention to how I feel* (Reverse scored).

Limited Access to Emotional Regulation Strategies: Sample item: *When I am upset, I believe that there is nothing I can do to make myself feel better.*

Lack of Emotional Clarity: Sample item: *I have difficulty making sense out of my feelings.*

Adult Sensory Profile (SP; Brown, et al., 2001): The SP is a widely used and validated measure of the subjective experience of sensation across multiple sensory domains. Individuals characterize their sensitivity (Sensory Sensitivity) and lack of sensation (Low Registration) in the visual, auditory, touch, taste, smell, vestibular, and kinesthetic domains. Behavioral responses to sensations in each domain are also assessed and individuals that seek sensation (Sensation Seeking) are differentiated from those who avoid sensation (Sensation Avoiding). Evidence of construct validity (Pfeiffer, Kinnealey, Reed, & Herzberg, 2005), reliability and discriminant validity of the SP has been established (Chen, Rodgers, & McConachie, 2009). Although the SP generates subscale scores that reflect Sensory Sensitivity, Low Registration, Sensation Seeking, and Sensation Avoidance collapsed across sensory domains (e.g., visual, auditory), only the Sensory Sensitivity subscale was used here given its relevance to the question at hand. Sample items for the Sensory Sensitivity subscale include: “I startle easily to unexpected or loud noises,” “I become frustrated when trying to find something in a crowded drawer or messy room,” and “I am afraid of heights” (Brown & Dunn, 2002).

Brief Symptom Inventory (BSI; Derogatis, 1993): The BSI is a shortened form of the revised version of the Symptom Checklist-90 (SCR-90; Derogatis, 1993), a self-report measure of that captures comorbid symptomatology in a continuous fashion. The BSI consists of 49-items that form nine symptom dimensions, including anxiety and depression. Participants indicate level of distress over the past seven days using a Likert scale ranging from “0 = Not at all” to “4 = Extremely.” Sample items include “Feeling lonely” and “Nausea or upset stomach.” The BSI has shown good internal consistency reliability for the

nine dimensions with alpha coefficients ranging from 0.71 to 0.85, and test-retest reliability coefficients ranging from 0.68 to 0.91 (Derogatis, 1993). Good convergent, construct, and predictive validity have been reported (Derogatis, 1993).

Data Analysis

Group descriptives were calculated and one-way analyses of variance (ANOVA) were used to validate group membership (e.g., differences in EDE scores). For all outcome variables of interest (e.g., DERS subscales), distributions were examined both visually and via measurements of skewness and kurtosis to ensure the appropriateness of parametric statistical methods. Due to inter-correlation between DERS subscales, we used a multivariate analysis of variance to test for differences in emotion regulatory capacities with group as the fixed factor (AN-C, WR, CN) and the DERS subscales as dependent variables. This omnibus F test was followed with post-hoc analyses as appropriate. Effect sizes were generated to ascertain the clinical relevance of the observed group differences.

Exploratory moderation analyses were conducted to determine if the relationship between difficulties with emotion regulation and self-perceived sensory sensitivity differed as a function of group. Two sets of regression analyses were estimated. In a first analysis evaluating main effects, DERS scores were regressed on sensory sensitivity and a three-level proxy variable denoting group membership using a 2 degrees of freedom Wald chi-square test to assess the overall significance of the group proxy. In the instance of a statistically significant finding for an overall group effect, pair-wise contrasts between the three possible condition combinations can be subsequently tested using closed (step-down) testing. To determine if the association between emotional dysregulation and perceived sensation sensitivity (tested as a main effect above) varied by group, the model was re-estimated in a second analysis following the addition of an interaction term crossing the proxy variable for group with the measure of sensation sensitivity. Models were estimated using standard linear regression procedures.

Prior to such analyses, independent regression models were run by group and leverage, standardized residuals, Dfbeta, and Dffit were examined. In instances where case diagnostics exceeded recommended levels, case values were examined for accuracy and models were run with and without offending cases to determine differences in the pattern of results. One case in the CN group exhibited undue leverage. The responses of this case were within normal limits and the pattern of results did not change with this case eliminated. Thus, the case was retained.

Results

Validation of Group Membership

AN-C participants had a mean BMI of 17.42 kg/m² ($SD = 1.41$), WR had a mean BMI of 21.07 kg/m² ($SD = 1.88$), and CN had a mean of 22.62 kg/m² ($SD = 3.84$). The mean BMI of the individuals who currently met criteria for AN was significantly lower than the other two groups, $F(2, 57) = 18.94$, $p < .001$ with post hoc comparisons significant at $p < .001$. There was not a significant difference between WR and CN, $p > .05$. Groups differed as expected on the EDE-Q Global Score, $F(2, 55) = 72.10$, $p < .001$, with post hoc comparisons indicating significant differences between all groups, AN-C ($M = 4.06 \pm .93$), WR ($M = 2.31 \pm 1.04$), and CN ($M = .84 \pm .68$), all p 's $< .001$. Tables 1-2 further characterize the sample.

Group Differences in Emotion Regulation and Perceived Sensory Sensitivity

The multivariate test of group differences in DERS scores using Wilks' Lambda was statistically significant, $F(6, 57) = 273.89$, $p < .001$. Follow-up analyses indicated

significant differences between the AN-C and CN on all DERS subscales, with poorer emotion regulatory capacities among individuals with AN. Individuals with current AN also had significantly greater deficits on several subscales of the DERS compared to individuals with AN who were weight restored (p 's < .001). AN-C reported significantly greater nonacceptance of emotional responses, greater deficits in emotional awareness and clarity, and less access to strategies to regulate emotions. They did not differ from WR in the extent to which they endorsed feeling a loss of control when emotional or an inability to pursue goals when experiencing heightened affect. See Table 3.

Effect size estimates for differences in the Nonacceptance subscale were large between AN-C and CN (*Cohen's d* = 2.77) and AN-C and WR (*Cohen's d* = 1.98), and WR relative to CN (*Cohen's d* = 1.05). Effects were also large for the Lack of Clarity Subscale (AN-C vs. CN: *Cohen's d* = 2.37; AN-C vs. WR: *Cohen's d* = 1.44; WR vs. CN: *Cohen's d* = 1.21). Effect sizes estimates for between group differences in the remaining subscales were also substantial, with the greatest differences between AN-C and CN (Goals: *Cohen's d* = 1.26, Impulse: *Cohen's d* = 1.11, Awareness *Cohen's d* = 1.48 and Strategies: *Cohen's d* = 1.78). Effect sizes for AN-C relative to WR ranged from medium to large [Goals *Cohen's d* = 0.64, Impulse *Cohen's d* = 0.44, Awareness *Cohen's d* = 1.29 and Strategies *Cohen's d* = 1.24] as well as effect size estimates for WR relative to CN [Goals *Cohen's d* = 0.79, Impulse *Cohen's d* = 0.84, Awareness *Cohen's d* = 0.32 and Strategies *Cohen's d* = 0.66].

Cronbach's alpha for the Sensory Sensitivity subscale was .71. Results of a one-way analysis of variance with post hoc Tukey comparisons indicated AN-C participants reported greater perceived sensitivity to sensation ($M = 44.1 \pm 7.7$) than CN, $F(2, 57) = 9.27, p < .001, M_{diff} = 9.8, p = .001$. AN-C did not differ in self-reported sensory sensitivity from those with a history of AN who were weight restored ($M_{diff} = .38, ns$).

Relationship between Emotion Dysregulation and Perceived Sensory Sensitivity

Regression estimates for tests of main effects of the moderation analysis are presented in Table 4. Type 3 analysis for both group and sensory sensitivity were significant. In subsequent pair-wise contrasts between groups, the largest estimated difference occurred contrasting subjects with current AN and CN. The difference was also substantial, although diminished, when AN-C were contrasted against WR participants, and was at its smallest magnitude in comparisons between WR and CN with a p -value just over .05. The positive association between emotional dysregulation and perceived sensation sensitivity was also significant, indicating that greater perceived sensory sensitivity was associated with increasing difficulty regulating emotions.

To explore whether the association between emotional dysregulation and perceived sensory sensitivity varied differentially by group, the above model was re-estimated after adding an interaction term crossing group with the measure of sensory sensitivity. As in the main effects model (above) the association between emotional dysregulation and sensory sensitivity was linear and positive for both WR and CN. This was in contrast to AN-C participants who demonstrated a sustained level of emotional dysregulation regardless of the level of sensation. The interaction term testing for a differential association between groups failed to attain significance (Table 5).

Discussion

Results suggest that deficits in emotion regulation are linked to AN symptom expression, with individuals with AN demonstrating greater deficits in this domain than both their weight-restored counterparts and typical controls. Effect size estimates for all DERS subscales were medium to large; however, the largest between group effects were for non

acceptance of emotional experience followed by difficulties with clarity. This suggests that difficulty understanding and accepting emotions is related to AN symptoms. Examination of differences between AN-C and WR suggest individuals who are weight-restored feel a loss of control and inability to pursue goals when experiencing heightened affect, but do presumably have clarity regarding emotions and an ability to accept emotional responses and regulate more healthfully. This may be functionally related to symptom remediation; however, without longitudinal data, this is unknown.

Perceived sensory sensitivity was explored as an individual difference variable that might increase the extent to which emotions are experienced as intolerable. Results indicated that, as a whole, self-perceived sensory sensitivity was associated with greater emotion dysregulation. When examined by group, a positive linear relationship between sensitivity and dysregulation was apparent among WR and CN, suggesting this may be the more normative relationship between these variables. This relationship was not apparent among AN-C participants who were exceedingly high on both measures. Despite this, there was a lack of a significant group by sensory sensitivity interaction. Post hoc power analyses indicated that failure to establish moderation might be due to insufficient sample size. AN-C and WR participants self-reported hypersensitivity to sensation. This suggests that recovery from AN may necessitate learning to cope with sensitivity to sensation as an individual difference or as a residual factor from a previously ill state. However, further investigation is needed to discern heightened sensory sensitivity as a risk variable.

WR individuals were more similar to CN than AN-C on the DERS subscales that assess awareness, clarity and acceptance of emotion, and belief that they have strategies to effectively regulate affect (i.e., effect sizes were smaller between WR and CN relative to WR and AN-C). However, WR individuals are more similar to AN-C than CN on DERS subscales focused on the ability to behave effectively when aroused. Thus, while individuals weight-restored from AN continue to associate emotional experiencing with a lack of control and report difficulties engaging in effective behavior when upset, they are more aware and accepting of emotional experience than their AN-C counterparts. Improvements in the ability to detect and label emotion, and accept emotional responses may therefore decrease the need for avoidance-based emotion regulation strategies, such as dietary restraint, even in the presence of these other difficulties and individual differences in perceived sensory sensitivity.

Limitations and Future Directions

AN research that utilizes acutely ill populations poses challenges as it is unclear whether sensitivities or deficits are state phenomena secondary to starvation or trait features that predate onset and increase illness vulnerability. Cross-sectional research that controls for undernourishment improves upon this, but cannot answer this question unequivocally as it is equally likely that deficits in emotional clarity among WR individuals is associated with remaining symptomatology as it is that difficulties with emotions exist pre-morbidly and are exacerbated in the acutely ill state.

Anxiety and depression, along with psychotropic medications to treat these comorbid conditions, were common among AN-C participants and to a lesser degree, WR participants. Sample size precluded analyses taking these variables into account. Thus the impact of anxiety, depression or psychopharmacology on perceived sensory sensitivity, ability to regulate emotions, or the relationship between these factors cannot be determined. Given that affective comorbidity is normative among individuals with AN (currently ill or recovered), future research should recruit larger samples to control for these variables and/or examine their relative impact. A larger sample would also address insufficient power to detect moderation and should include men to increase the generalizability of the findings.

In the current study, sensory sensitivity was assessed by self-report. This is a significant limitation in that we are unable to discriminate *perception of heightened sensitivity* from *actual* increased sensitivity of the visceral organs. The latter may only be determined by laboratory-based assessment that examines threshold for detection and other relevant parameters. Emotion regulation was also assessed via self-report and reflects self description of capacities rather than actual skills in this domain.

Despite limitations, greater perceived sensitivity to sensation and less access to skills to regulate these experiences in a healthy way was associated with symptomatology and is thus a target for intervention. Findings suggest treatments which address *how one reacts* to difficult thoughts, feelings, and body sensations (rather than changing the private events themselves) might be useful in decreasing AN symptomatology. Therapeutic approaches that emphasize observation and acceptance of somatic-affective experience and facilitate effective action in the presence of difficult thoughts and feelings (Baer, Fischer, & Huss, 2006; Merwin & Wilson, 2009; Wildes & Marcus, 2011) might be particularly well-matched to AN if individual differences in sensitivity are found to exist pre-morbidly and are trait-like and thus less amendable to change.

Study results indicate the need for experimental paradigms to determine whether sensory sensitivity among individuals with AN is objective or a subjective experience (i.e., whether there is indication of lower thresholds for detection of change in visceral sensation), and quantify perturbations in sensation experience, should they exist. More research is also needed to assess the directionality of relationships identified here and to test the effectiveness of the treatment strategies suggested by this work.

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Table 1
Demographic Information by Mean (SD) or Frequency (Percentage)

	AN-C (<i>n</i> = 20)	WR (<i>n</i> = 15)	CN (<i>n</i> = 24)	Total (<i>n</i> = 59)
Age	26.40 (8.74)	28.60 (10.75)	26.25 (9.24)	26.9 (9.57)
Years of Education	15.10 (2.49)	16.64 (3.69)	15.63 (3.19)	15.69 (3.10)
Race/Ethnicity				
White/Caucasian	20 (100%)	13 (86.7%)	14 (58.3%)	47 (79.7%)
Black/African American	0 (0%)	1 (6.7%)	5 (20.8%)	6 (10.2%)
Asian	0 (0%)	1 (6.7%)	1 (4.2%)	2 (3.4%)
Hispanic/Latino	0 (0%)	0 (0%)	1 (4.2%)	1 (1.7%)
Mixed Race	0 (0%)	0 (0%)	1 (4.2%)	1 (1.7%)
Other/Not Classified	0 (0%)	0 (0%)	2 (8.4%)	2 (3.4%)
Current Relationship Status				
Married	2 (10.0%)	4 (26.7%)	4 (16.7%)	10 (16.9%)
Divorced	1 (5.0%)	0 (0%)	0 (0%)	1 (1.7%)
Engaged	0 (0%)	1 (6.7%)	0 (0%)	1 (1.7%)
Single, partnered	4 (20%)	4 (26.7%)	11 (45.8%)	19 (32.2%)
Single, not partnered	13 (65.0%)	6 (40%)	9 (37.5%)	28 (47.5%)
Comorbid Psychiatric Symptoms and Medications				
Anxiety	1.84 (.92)	1.29 (1.12)	.37 (.57)	1.22 (1.10)
Depression	2.30 (.82)	1.16 (.96)	.36 (.39)	1.10 (1.06)
Antidepressants	10 (50.0%)	5 (33.3%)	0 (0%)	15 (25.4%)
Atypical Antipsychotics	5 (25.0%)	1 (6.7%)	0 (0%)	6 (10.2%)
Anticonvulsants	3 (15.0%)	0 (0%)	0 (0%)	3 (5.1%)
Mood stabilizers	2 (10.0%)	0 (0%)	0 (0%)	2 (3.4%)
Anxiolytics	2 (10.0%)	0 (0%)	0 (0%)	2 (3.4%)

Note. Groups were not significantly different on age or years of education. % (Percentage of each group as indicated). AN-C = current anorexia nervosa or AN NOS; WR = AN by history and weight-restored 6 months; CN = Control participants; individuals without a current or past eating disorder. Anxiety = Mean score for the Anxiety subscale of the Brief Symptom Inventory. Depression = Mean score for the Depression subscale of the Brief Symptom Inventory. For comparison purposes, norms derived for adult females indicated a Depression subscale $M = 0.36$ ($SD = 0.56$) for female nonpatients and $M = 1.90$ ($SD = 1.05$) for female psychiatric outpatients. Anxiety means were $M = 0.44$ ($SD = 0.54$) and $M = 1.82$ ($SD = 1.02$) for female nonpatients and female psychiatric outpatients respectively.

Table 2
Associated Eating Disorder Psychopathology by Group

	AN-C (<i>n</i> = 20)	WR (<i>n</i> = 15)	CN (<i>n</i> = 24)
Age of Eating Disorder Onset	17.65 (4.44)	15.33 (4.91)	
Presence of Lifetime Threshold Symptoms			
Lifetime Binge Eating	40.0%	60.0%	
Lifetime Purging	45.0%	26.7%	
Lifetime Driven Exercise	80.0%	73.3%	
Lifetime Laxative Use	50.0%	33.3%	
Weight History			
Lowest BMI	14.96 (1.55) ^{a †}	14.86 (1.78) ^{a †}	20.40 (2.12) ^b
Current BMI	17.48 (1.35) ^a	21.16 (1.92) ^{b †}	22.60 (3.77) ^{b †}
Current Self-Reported Eating Disorder Attitudes			
Restraint	3.93 (1.58) ^{a †, †}	2.36 (1.20) ^{b †, *}	.88 (.91) ^{c †, *}
Weight Concern	4.39 (1.07) ^{a †, †}	2.87 (1.18) ^{b †, *}	.90 (.87) ^{c †, *}
Shape Concern	4.75 (1.00) ^{a †, †}	2.91 (1.17) ^{b †, †}	1.25 (.94) ^{c †, †}
Eating Concern	3.44 (1.26) ^{a †, †}	1.30 (1.14) ^{b †, †}	.24 (.32) ^{c †, †}
Global Score	4.06 (.93) ^{a †, †}	2.31 (1.04) ^{b †, †}	.84 (.68) ^{c †, †}

Note. Mean (standard deviation). % (Percentage of each group as indicated). Different superscripts connote differences between groups. BMI = body mass index. AN-C = current anorexia nervosa or AN NOS; WR = AN by history and weight-restored 6 months; CN = Control participants; individuals without a current or past eating disorder.

* = $p < .01$.

† = $p < .001$

Table 3
Mean (SD) for DERS Total and DERS Subscales by Diagnosis

	AN-C (n = 20)	WR (n = 15)	CN (n = 24)
DERS Total	114.60 (21.12) ^a	82.33 (13.32) ^b	4.50 (17.05) ^c
Nonacceptance	23.00 (5.25) ^a	14.07 (3.63) ^b	10.04 (4.04) ^c
Goals	18.65 (4.91) ^a	15.93 (3.49) ^{a,b}	12.79 (4.41) ^b
Impulse	13.75 (6.01) ^a	11.47 (4.05) ^{a,b}	8.54 (2.86) ^b
Awareness	19.00 (5.70) ^a	12.87 (3.52) ^b	11.63 (4.14) ^b
Strategies	23.85 (6.53) ^a	16.67 (4.92) ^b	13.29 (5.27) ^b
Clarity	16.35 (4.15) ^a	11.33 (2.64) ^b	8.21 (2.52) ^c

Note: AN-C = current anorexia nervosa or AN NOS; WR = AN by history and weight-restored 6 months; CN = Control participants; individuals without a current or past eating disorder. Means in the same row with different subscripts differ at $p < 0.01$ as determined by means of Tukey post-hoc tests. Higher scores indicate greater difficulty in that domain (i.e., Higher scores on the Clarity subscale indicates greater deficits in clarity of emotional experience, or a lack of emotional clarity). Nonacceptance = nonacceptance of emotional responses. Goals = Difficulties engaging in goal-directed behavior when experiencing heightened affect. Impulse = Impulse control difficulties, or feeling out of control when emotional. Awareness = Lack of emotional awareness. Strategies = Limited access to emotion regulation strategies. Clarity = Lack of emotional clarity.

Table 4
Regression Estimates for Tests of Main Effects of the Moderation Analysis

Parameter	Est	SE	-95CI	+95CI	Wald X ²	Pr>X ²
Intercept	43.51	10.62	22.69	64.32	16.79	<.0001
AN-C	43.67	5.71	32.47	54.87	58.41	<.0001
WR	11.64	6.06	-0.23	23.51	3.69	0.054
CN	0.00	0.00	0.00	0.00	.	.
Sensory Sensitivity	0.62	0.29	0.06	1.18	4.79	0.029

Type 3 Analysis			
Source	Df	X ²	Pr>X ²
Group	2	43.44	<.0001
Sensory Sensitivity	1	4.60	0.03

Note: AN-C = current anorexia nervosa or AN NOS; WR = AN by history and weight-restored 6 months; CN = Control participants; individuals without a current or past eating disorder.

Table 5
Interaction Model

Parameter	Est	SE	−95CI	+95CI	Wald X ²	Pr>X ²
Intercept	3.59	0.18	3.23	3.95	389.61	<.0001
AN-C	1.13	0.31	0.53	1.73	13.76	<.001
WR	0.29	0.33	−0.35	0.94	0.78	0.378
CN	0.00	0.00	0.00	0.00	.	.
Sensory Sensitivity	0.02	0.01	0.01	0.03	9.90	0.002

Type 3 Analysis

Source	Df	X ²	Pr>X ²
Group	2	12.43	0.00
Sensory Sensitivity	1	7.70	0.01
Group X Sensory Sensitivity	2	4.51	0.11

Note: AN-C = Individuals with current anorexia nervosa. CN = Control participants; individuals without a current or past eating disorder. WR = Individuals with a past history of anorexia nervosa who were weight-restored 6 months.