Distress tolerance across self-report, behavioral and psychophysiological domains in women with eating disorders and healthy controls

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Abstract

Background and objectives: The tendency to engage in impulsive behaviors when distressed is linked to engagement in disordered eating. The current study comprehensively examines emotional responding to a distress tolerance task by utilizing self-report and psychophysiological measures (respiratory sinus arrhythmia [RSA], skin conductance responses [SCRs] and tonic skin conductance levels [SCLs]). *Methods*: A treatment seeking sample with Bulimia Nervosa (BN), Binge Eating Disorder (BED), Anorexia Nervosa (AN) and healthy controls (HCs) completed ratings of self-reported emotions and psychophysiological measurements before and after completion of the Paced Auditory Serial Addition Task- Computerized (PASAT-C). *Results*: Overall, we found an effect of Time, such that all participants reported greater negative emotions, fewer positive emotions, lower RSA, more SCRs and higher tonic SCLs after completion of the PASAT-C relative to baseline. We did not find differences in performance on the PASAT-C between groups. We found an effect of Group, such that individuals with BN, BED and AN reported overall higher levels of negative emotions relative to HCs. Furthermore, we found an effect of Group for greater urges to binge eat and lower RSA values among BED, relative to individuals with BN, AN and HCs. *Conclusion*: This is the first study to examine emotional responding utilizing multiple methods among different eating disorder diagnoses. The finding that individuals with BN, BED and AN report overall greater self-reported negative emotions without concurrent deleterious effects on PASAT-C performance relative to HCs suggests that subjective distress, rather than behavioral distress, may be an important area for intervention.

Keywords: eating disorders; distress tolerance; psychophysiology; PASAT-C

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Eating disorders (EDs) are a serious public health concern, affecting up to 4.64% of adults (Le Grange, Swanson, Crow, & Merikangas, 2012). EDs have significant medical and psychosocial outcomes (Baiano et al., 2014) and disordered eating, in general, has been conceptualized as a transdiagnostic maladaptive response to difficulties with tolerating distress (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Anestis, Smith, Fink, & Joiner, 2009; Fischer, Smith, & Cyders, 2008; Haynos & Fruzzetti, 2011; Leyro, Zvolensky, & Bernstein, 2010).

A number of studies have demonstrated difficulties in tolerating distress across the spectrum of EDs (Anestis, Selby, Fink, & Joiner, 2007; Corstorphine, Mountford, Tomlinson, Waller, & Meyer, 2007; Peterson & Fischer, 2012). The tendency to engage in impulsive behaviors when distressed (negative urgency) is linked to engagement in disordered eating (Anestis, Smith, et al., 2009; Fischer et al., 2008; Stice, Akutagawa, Gaggar, & Agras, 2000). Disordered eating behaviors may temporarily reduce distress, thereby negatively reinforcing their use (Corstorphine et al., 2007). However, our understanding of distress tolerance in EDs is limited by empirical studies that typically utilize one or two methods of assessment (Anestis et al., 2007; Anestis, Smith, et al., 2009; Claes, Vandereycken, & Vertommen, 2005; Peterson & Fischer, 2012; Wenzel, Weinstock, Vander Wal, & Weaver, 2014; Wu et al., 2013), with few studies integrating multiple methods to assess the cognitive, behavioral, and psychophysiological components of emotional responses (Lang, Greenwald, Bradley, & Hamm, 1993). Multiple methods of assessment provide a more comprehensive understanding of affective response in EDs (Gross, 2013), and may further elucidate the transdiagnostic mechanisms of psychopathology.

While previous research consistently provides evidence in the form of self-reported difficulties in distress tolerance among individuals with EDs, there is a dearth of evidence regarding the psychophysiological and behavioral components of this relationship (Gross, 2013). The psychophysiological component of emotional response is comprised of parasympathetic and sympathetic nervous system activity. Respiratory sinus arrhythmia (RSA), which consists of heart rate variability (HRV) in conjunction with respiration, is a measure of parasympathetic nervous system activity. Low resting RSA and decreased RSA when emotionally aroused are associated with greater symptoms of psychopathology (Beauchaine, 2015). Neuroimaging studies show that there is a positive relationship between RSA and increased activation of the right pregenual anterior cingulate, right subgenual anterior cingulate and right rostral medial prefrontal cortex and the left sublenticular extended amygdala or ventral striatum (Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). These structures are implicated in emotion processing and the psychophysiological aspects of emotional responses (Thayer et al., 2012). Reduced resting RSA has been theorized as a risk factor for psychopathology (Crowell, Beauchaine, & Linehan, 2009), although systematic reviews and meta-analysis have suggested that higher resting HRV is associated with both bulimia nervosa (BN) (Peschel et al., 2016) and anorexia nervosa (AN) (Mazurak, Enck, Muth, Teufel, & Zipfel, 2011) compared to controls.

Experimental work examining RSA reactivity to stressors among individuals with EDs has produced mixed findings. Using the Stroop task to induce cognitive stress, researchers found that non-ED obese women flexibly recover from stress exposure, as measured by RSA. This is relative to women with BN, who showed poorer recovery from stress exposure, and obese women with Binge Eating Disorder (BED), who did not exhibit changes in RSA levels and appeared to be insensitive to stress exposure (Messerli-Bürgy, Engesser, Lemmenmeier, Steptoe,

& Laederach-Hofmann, 2010). However, in another study that compared obese women with and without BED, obese women with BED demonstrated poorer cardiovascular recovery after induced mental stress, relative to non-BED obese women (Friederich et al., 2006); although there were no baseline differences in RSA. Additional research suggests that decreased RSA after mental stress may not simply be due to weight status, but rather due to engagement in clinically significant levels of binge eating (Udo et al., 2014). Taken together, these findings suggest that while RSA is linked to brain structures that aid processing of emotions and psychophysiological modulation of emotional responses, the level of parasympathetic dominance in individuals with EDs relative to controls is unknown.

Skin conductance (SC), which indexes the time it takes for a current to pass through the skin, is a measure of sympathetic nervous system response (Boucsein et al., 2012), and higher skin conductance responses (SCRs) are associated with greater emotional arousal (Kreibig, 2010; Lang et al., 1993). Previous research suggests that women with BN do not exhibit different patterns of skin conductance levels (SCL) at baseline or during lab stressors compared to women who report restrained eating or healthy controls (Tuschen-Caffier & Vögele, 1999). Furthermore, SCL are similar between women with BN and BED in response to personalized interpersonal, achievement or intrapersonal stressors (Hilbert, Vögele, Tuschen-Caffier, & Hartmann, 2011).

Overall, the evidence base for the psychophysiological component of emotional response across the range of EDs has been mixed, limited by small sample sizes, and there is a lack of research utilizing the full spectrum of EDs. The current study extends past research (Leehr et al., 2015; Naumann, Tuschen-Caffier, Voderholzer, Caffier, & Svaldi, 2015; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012; Svaldi, Tuschen-Caffier, Lackner, Zimmermann, & Naumann, 2012) by examining distress tolerance across all ED diagnoses and healthy control participants with a multi-modal assessment of affective response, including self-report, behavioral, and psychophysiological assessment, with the latter assessing both the parasympathetic and sympathetic nervous system. This study induced distress by using the Paced Auditory Serial Addition Task-Computerized (PASAT-C) (Lejuez, Kahler, & Brown, 2003) and indexed emotional response by using self-reported emotions, psychophysiological measures of arousal, and latency to termination as a behavioral measure of distress tolerance (Feldner, Leen-Feldner, Zvolensky, & Lejuez, 2006; Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006). This is an important step forward, which allows for a transdiagnostic examination of EDs, in line with recommendations from the Research Domain Criteria (Insel et al., 2010).

We predicted a main effect of time, such that self-reported negative emotions would be greater, positive emotions would be lower, and urges to binge eat would be greater after the PASAT-C, in comparison to baseline and at recovery. Furthermore, we expected that diagnostic group would moderate this effect. Specifically, we expected that healthy controls would demonstrate fewer negative emotions, more positive emotions and lower urges to binge eat during the PASAT-C, compared to individuals with BN, BED or AN. As the PASAT-C has been shown to induce distress in clinical samples (Gratz et al., 2006; Sauer & Baer, 2012), we expected that individuals with BN, BED and AN would have poorer performance on the PASAT-C. Specifically, individuals with BN, BED and AN would be more likely to prematurely terminate the PASAT-C and exhibit shorter latency to terminate the PASAT-C than healthy controls. Given the proposed role of difficulties tolerating distress in EDs (Anestis, Peterson, et al., 2009; Anestis et al., 2007; Peterson & Fischer, 2012), we expected that RSA values would be lower and SCRs and tonic SCL values would be higher during the PASAT-C in ED groups compared to HC, relative to baseline and recovery.

Methods

Participants

The sample consisted of 107 treatment-seeking females with an ED and 26 healthy controls with no history of psychiatric disorders from the community. Of the participants with an ED, 34.26% of the sample was diagnosed with BN (*n*=37), 50.90% with BED (*n*=55), and 14.02% with AN (*n*=15). The sample identified predominantly as non-Hispanic (91.7%) and were 63.2% Caucasian, 13.5% African-American, 4.5% Asian, and 10.5% of mixed race. The mean age of participants was 35.15 years (*SD* = 11.48), and the mean body mass index (BMI) was 29.47 kg/m^2 (*SD* = 9.52).

Procedure

The present study was reviewed and approved by the university institutional review board and took place over two separate testing sessions. At the first session, eligibility screening and informed consent was completed, followed by an assessment of psychiatric and ED symptoms by Masters-level clinicians using the measures described below. Diagnoses were confirmed at a weekly best-estimate meeting with a licensed clinical psychologist (Klein, Ouimette, Kelly, Ferro, & Riso, 1994; Kosten & Rounsaville, 1992). Height and weight were measured to calculate BMI. During the second session, participants completed the laboratory procedures for the PASAT-C, described below. Diagnostic and psychophysiological assessments occurred prior to treatment for individuals with EDs.

Measures

Psychiatric symptoms. The Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders – IV- Text Revision (DSM – IV – TR) Axis I Disorders (SCID – I) (First, Spitzer, Gibbon, & Williams, 2002) was used to assess for psychiatric symptoms.

Eating disorder behaviors. The Eating Disorder Examination 16.0 (EDE) (Fairburn, Cooper, & O'Connor, 2008) is an investigator-based interview that assesses DSM– IV-TR (American Psychiatric Association, 2000) ED symptoms. The EDE – 16.0 was used to diagnosis AN, BN and BED and generates subscales that tap into ED behaviors and cognitions: Eating Concern, Shape Concern, Weight Concern, Dietary Restraint, as well as a Global score. In the current study, the EDE total score demonstrated an internal consistency of α = .940.

Current subjective emotional state. An abbreviated Positive and Negative Affect State (PANAS) (Watson, Clark, & Tellegen, 1988) measured current subjective emotional state at baseline, after the PASAT-C, and during recovery from the PASAT-C (see Figure 1). Self-report negative and positive affective adjectives that assessed anxiety, fear, frustration, happiness, sadness, tension, and urges to binge eat were scored on a 100-point Likert scale. Higher scores indicated greater intensity of the response. In the present study, we created a composite for negative emotions using an average of scores from anxiety, fear, frustration, sadness and tension. Urge to binge eat and happiness were assessed separately. In the present study, internal consistency of the negative emotion composite score at each time point ranged from α = .84 to .90.

Psychophysiological measures. Psychophysiological measures included respiratory sinus arrhythmia (RSA) as a measure of parasympathetic activity (Grossman & Taylor, 2007; Thayer et al., 2012) and skin conductance responses (SCRs) and tonic skin conductance levels (tonic SCL) as measures of sympathetic activity (Wolfram Boucsein, 2011). Electrocardiogram (ECG) data were collected from two Biopac Ag-AgCl spot electrodes placed on the bottom of the palm of the non-dominant hand, using a modified Lead II configuration (Boucsein, 2011). We derived RSA by using a band pass filter on the ECG signal and spectral analysis to extract the high-frequency component (> .15 hz) of heart rate variability. Skin conductance was recorded from two electrodes attached to the palm of the non-dominant hand. Increases in SCRs have been associated with negative affect following exposure to emotional stimuli, even when cardiac measures did not change significantly (Salters-Pedneault, Gentes, & Roemer, 2007) (Boucsein et al., 2012). Tonic SCL referred to the level of skin conductance per data collection period.

Laboratory Procedures.

Emotion induction. The Paced Auditory Serial Addition Task-Computerized (PASAT-C) was used to induce a negative mood. The PASAT-C is a computer-based task that requires the participant to add a visually presented digit to the previous visually presented digit. Explosion sounds followed incorrect answers or when the participant failed to respond quickly enough. The PASAT-C was presented for a maximum of twelve minutes, consisting of four levels of equal time intervals. With each successive level of the PASAT-C, the latency between trials was decreased and negative feedback of "*go faster*", "*do better*", and "*go faster, do better*" was added. By the fourth level (PASAT-C Level 4), there was a one second latency of trials with negative feedback. After completion of Level 2, participants were given the option to quit the task. Performance on the PASAT-C was operationalized as termination of the task when given the option and latency to quit. The PASAT-C has been shown to reliably induce negative affect (Daughters, Lejuez, Kahler, Strong, & Brown, 2005; Feldner et al., 2006; Holdwick & Wingenfeld, 1999; Schloss & Haaga, 2011) and short-term anxiety, frustration, and irritability (Gratz et al., 2006; Lejuez et al., 2003).

Procedure. Once sensors were attached, participants were asked to sit quietly without moving for a 5-minute baseline period. Following this, participants were instructed to begin the PASAT-C. Upon completion of the PASAT-C, participants were asked to sit quietly for an additional 5-minutes without moving for a recovery period. Psychophysiological measures were collected during true baseline, PASAT-C, and the recovery period. Please see Figure 1 for a schematic of the procedure.

Statistical Treatment

Chi-square tests were conducted to assess for group differences in all demographic variables and medical and psychiatric comorbidities, with the exception of age, which was assessed using an ANOVA. Variables related to presence of medication use (e.g., stimulant medication, anxiolytics) and medical co-morbidities were included as covariates in the analyses if there were significant differences between ED groups (BN, BED, AN) due to their effects on psychophysiological responses (Grossman, Stemmler, & Meinhardt, 1990; Masi, Hawkley, Rickett, & Cacioppo, 2007). As it was expected that HCs would exhibit significantly less medical co-morbidities and medication use relative to the ED groups (Hudson, Hiripi, Pope, & Kessler, 2007; Johnson, Spitzer, & Williams, 2001; Kessler et al., 2013; Padierna, Quintana, Arostegui, Gonzalez, & Horcajo, 2000), medical co-morbidities and medication use were only included as covariates if there were significant differences between BN, BED and AN.

For analyses involving self-reported emotions and psychophysiological measures, independent variables were Time (baseline, PASAT-C, recovery) and Group (BN, BED, AN and HC). Six repeated measures Time x Group ANOVAs were conducted with the self-reported negative emotion composite score, happiness, urge to binge eat, RSA, SCRs and Tonic SCL as dependent variables. Planned simple effects analyses were conducted to probe significant interactions. To examine group differences in PASAT-C completion vs. non-completion, a chi square test was conducted. To examine group differences in PASAT-C latency to quit, a univariate ANOVA was conducted. Partial η^2 was used to report effect sizes for repeated measures ANOVAs and ANOVAs, with the following cut-off conventions: small (.01), medium (.06) and large (.14) (Cohen, 1988).

Results

Preliminary Analyses

Please see Table 1 for a summary of sample demographic information, including medical and psychiatric co-morbidities. There were no significant group differences on minority status (p = .190), never-married status (p = .075), full-time employment or student status (p = .782), or completion of 4-year college (p = .095). There were significant differences between groups on age (p < .001) and annual income below $25,000 ($p$ = .001), such that individuals with BED were older relative to BN and HCs and were less likely to earn an annual income below $25,000.

As expected, there were significant group differences on presence of a lifetime mood disorder (p < .001), anxiety disorder (p < .001), medical co-morbidities (p = .006) and medication use (p = .002), which were driven by the lack of psychiatric and medical co-morbidities among HCs. There were no significant differences between BN, BED and AN on presence of a lifetime mood disorder, medical co-morbidities or medication use, but AN were significantly more likely to have an anxiety disorder relative to BN (p < .05).

Table 2 describes the clinical characteristics and severity of ED symptoms of the sample. Consistent with past research (Dingemans & van Furth, 2012; Hudson et al., 2007), there were significant differences in BMI (F [4, 127] = 26.32, p < .001), such that HCs had significantly lower BMI relative to the BED group and significantly higher BMI relative to the AN group (ps < .009), but did not differ significantly from the BN group. Scores from the EDE Eating Concern subscale, Weight Concern subscale, Shape Concern subscale or Global scores did not differ significantly between the BN, BED or AN groups (ps > .230) but were significantly elevated relative to HCs (Fs [4, 127] = 26.04 to 40.99, *p*s < .001). AN and BN groups did not differ significantly on the EDE Dietary Restraint subscale (p = .474), but the BED group reported significantly lower dietary restraint relative to AN and BN and significantly greater dietary restraint relative to HCs (F [4, 127] = 18.62, *p*s < .001). Consistent with diagnostic criteria, there were significant differences in frequency of binge eating episodes (F [4, 127] = 10.06, $p < .001$) and frequency of compensatory behaviors (F [4, 127] = 18.40, $p < .001$). Specifically, BED and BN reported significantly greater frequency of binge eating episodes than AN (*p*s < .008) and BN and AN reported significantly greater frequency of compensatory behaviors (*p*s < .001) than BED. BED and BN did not differ significantly on frequency of binge eating episodes (p = .154), nor did BN and AN on frequency of compensatory behaviors (p = .837). The majority of participants with BN (N = 31, 83.78%) and approximately half of the participants with AN (N = 8, 57%) engaged in at least one compensatory behavior per week.

Main Analyses

Hypothesis 1: Self-reported emotions. Overall, there was a main effect of Time, such that participants reported higher negative emotions, $F_{(2, 107)} = 47.61$, $p < .001$, $\eta^2 = .31$, and lower happiness, $F_{(2, 107)} = 39.10$, $p < .001$, $\eta^2 = .27$, after completion of the PASAT-C, in comparison to baseline and recovery (*ps* < .05). There was a main effect of Group for negative emotions, $F_{(3, 107)} = 12.09$, $p < .001$, $\eta^2 = .25$, but not happiness (p = .067). Healthy control participants reported lower negative emotions (*ps* < .001) in comparison to individuals with BN, BED or AN. There were no Time x Group interactions (*ps* = .123 - .265) for negative emotions or happiness.

For urges to engage in binge eating, there was a main effect of Group, $F_{(3, 107)} = 16.51$, $p < .001$, $\eta^2 = .32$, such that individuals with BED demonstrated significantly greater urges to binge eat compared to BN, AN and HCs (*ps* < .036), individuals with BN demonstrated significantly greater urges to binge eat compared to HCs ($p < .001$), and individuals with AN did not differ significantly from BN or HC (*p*s > .061). There was no effect of Time ($p = .561$) or an interaction between Time x Group ($p = .933$), but individuals with AN exhibited a trend for decreased urge to binge eat after completion of the PASAT-C. See Table 3 for a summary of findings.

Hypothesis 2: PASAT-C performance: latency to quit and termination. A univariate ANOVA suggested that there were no differences between groups on PASAT-C latency to quit, $F_{(3,129)} = 1.39$, $p = .250$, $\eta^2 = .03$. A chi square test indicated that there were no differences between groups on PASAT-C completion versus non-completion, $\chi^2_{(3)} = 3.85$, $p = .278$. See Table 4 for means and standard deviations.

Hypothesis 3: Psychophysiological measures. There was a significant effect of Time for RSA values, $F_{(2,129)} = 12.28$, $p < .001$, $\eta^2 = .09$, such that all participants exhibited lowered RSA during the PASAT-C, in comparison to baseline ($p = .048$) and recovery ($p < .001$). There was a significant effect of Group, $F_{(3,129)} = 6.15$, $p = .001$, $\eta^2 = .13$, such that individuals with BED exhibited overall lowered RSA levels in comparison to individuals with BN or HCs ($ps \leq .006$) but not AN ($p = .281$). There was no Time x Group interaction for RSA values.

There was a significant effect of Time for Tonic SCL values, $F_{(2,110)} = 53.06$, $p < .001$, $\eta^2 = .33$, and SCR values, $F_{(2,110)} = 33.44$, $p < .001$, $\eta^2 = .24$, such that all participants exhibited greater Tonic SCL and SCR values during the PASAT-C in comparison to baseline ($ps < .001$), but not recovery (*p*s =.441 - .756). There was no effect of Group or Time x Group interaction for Tonic SCL or SCR values. See Table 5 for means and standard deviations.

Discussion

The current study extends the current literature on distress tolerance in EDs by integrating self-report, behavioral and psychophysiological measures (RSA, SCRs and tonic SCLs) of emotion responding to a distress tolerance task (Daughters et al., 2005; Feldner et al., 2006; Holdwick & Wingenfeld, 1999; Schloss & Haaga, 2011). Although there was a lack of group differences on PASAT-C performance (latency to quit and termination of task), findings were consistent across self-report and psychophysiological measures. Specifically, self-reported negative emotions increased, positive emotions decreased, RSA values decreased and Tonic SCL and SCR values increased significantly after completion of the PASAT-C relative to the baseline. This finding of synchrony between self-reported emotions and psychophysiological measures in response to tolerating distress is consistent with past research using multi-modal assessments of emotion responding (Gross, 2013; Lang et al., 1993). Notably, despite similar psychophysiological responding (RSA, SCRs and tonic SCL) to the PASAT-C and similar performance on the PASAT-C across all groups, individuals with BN, BED and AN reported greater overall negative emotions and lower overall happiness compared to HCs. The discrepancy between psychophysiological responding and behavioral performance with self-report of emotional experience suggests that individuals with EDs experience disproportionately greater subjective distress relative to HCs when presented with the same stimuli.

Although HCs reported significantly fewer negative emotions and greater happiness relative to all other groups, individuals with BED were differentiated from other ED groups and HCs by exhibiting significantly lower RSA values without concomitant differences from other groups in Tonic SCL or SCR values. Research has demonstrated an association between BMI and RSA and suggests that cardiovascular autonomic function changes as a function of weight loss (Karason, Mølgaard, Wikstrand, & Sjöström, 1999; Rissanen, Franssila‐Kallunki, &

Rissanen, 2001). BMI was not controlled for in the current study, as 92% of participants diagnosed with BED were overweight or obese, likely due to the cumulative effects of objective binge episodes on weight gain over time (Hudson et al., 2010). Therefore, participants with BED are "doubly diagnosed" with BED and obesity, making it difficult to dissociate diagnostic and weight status. Future studies are needed to dissociate diagnosis and weight status.

Contrary to expectations, we did not find a Time (baseline, PASAT-C and recovery) by Group interaction in self-reported urge to binge eat. However, there were Group differences in urge to binge eat, such that individuals with BED and BN reported overall greater urges to binge eat across all time-points in comparison to individuals with AN or HC. Perhaps individuals who engage in habitual binge eating experience consistent urges to engage in binge eating, but only act on this urge when they experience negative affect (Hilbert & Tuschen‐Caffier, 2007) or are under stress. As the current study only assessed urges to binge eat, future research may seek to explore factors that influence whether one will or will not act upon urges to binge eat when experiencing negative affect (Haedt-Matt & Keel, 2011; Hilbert & Tuschen‐Caffier, 2007).

The PASAT-C achieved the desired result of increasing distress as evidenced by increased negative emotions, decreased positive emotions, decreased RSA values and increased SCRs and tonic SCLs across the sample, without differences in performance. This suggests that regardless of one's baseline level of negative emotions or diagnostic group, the PASAT-C was distress inducing, which is consistent with past research utilizing samples with clinically significant psychopathology (Gratz et al., 2006). The success of the PASAT-C to induce distress may have produced a ceiling effect on performance, such that all participants were equally sensitive to its effects regardless of diagnosis. However, the lack of significant group differences in performance is counter to another study that found significantly poorer PASAT-C performance among undergraduate students who endorsed binge eating behaviors relative to controls (Eichen, Chen, Boutelle, & McCloskey, 2017). The difference in findings may be due to sampling differences between the two studies. The current study recruited treatment-seeking patients with a range of EDs; the other study recruited non-treatment seeking college students.

Future Directions

The inclusion of all ED diagnostic groups can be considered a strength and a limitation of the current study. Comparisons were possible between ED diagnoses and HCs, but were underpowered to detect significant effects for individuals with AN relative to other groups. For example, individuals with AN exhibited a trend for decreased urges to binge eat after completion of the PASAT-C in comparison to baseline, which is in contrast to individuals with BED and BN who exhibited similar urges to binge eat at baseline and after the PASAT-C. This may reflect an increased desire for control over eating when experiencing negative affect among individuals with AN (Fairburn, Shafran, & Cooper, 1999). A larger sample of individuals with AN may clarify the urge to binge eat in response to stress among different ED diagnoses. Furthermore, as the PASAT-C generated similar levels of negative affect across the entire sample, future research could utilize emotion inductions that may be more salient to EDs, such as cues involving body shape/weight concerns or food. For example, participants could complete behavioral approach/avoidance tasks that involve examining the body in a mirror or observing images of high-caloric food. In terms of behavioral assessments, future research could utilize distress tolerance measures that more closely map onto eating disorder concerns, such as sampling foods.

Finally, the current study’s group of individuals with BED primarily consisted of individuals who are also overweight. Although individuals with BED are at a higher risk for obesity (e.g., de Zwaan, 2001), the medical condition of obesity is not synonymous with the psychological condition of BED (e.g., Klatzkin, Gaffney, Cyrus, Bigus, & Brownley, 2015). Future studies may seek to examine whether a similar pattern of findings are found for individuals with BED who are of normal weight, or for overweight and normal-weight individuals without EDs.

The current study is one of the first to examine emotional responding across individuals with EDs and HCs in response to a task that induces distress. It provides a step towards enhancing our understanding of the similarities and differences in emotional responding across ED diagnoses using multiple measures of emotional responding. The finding that EDs are associated with overall greater subjective distress without deleterious effects on the PASAT-C performance relative to HCs has treatment implications for the function of disordered eating to decrease distress (Corstorphine et al., 2007). Individuals with EDs may benefit from treatments that focus on fostering greater acceptance of one's emotional experiences, such as Dialectical Behavior Therapy (Chen et al., 2015; Safer, Robinson, & Jo, 2010) or Acceptance and Commitment Therapy (Berman, Boutelle, & Crow, 2009; A. Juarascio et al., 2013; A. S. Juarascio, Forman, & Herbert, 2010). Finally, our finding that individuals with BED exhibit overall reduced levels of RSA responding and experience consistent urges to binge eat over time may be relevant for understanding how urges to binge eat interacts with the actual binge eating behavior and provides potential targets for treatment interventions.

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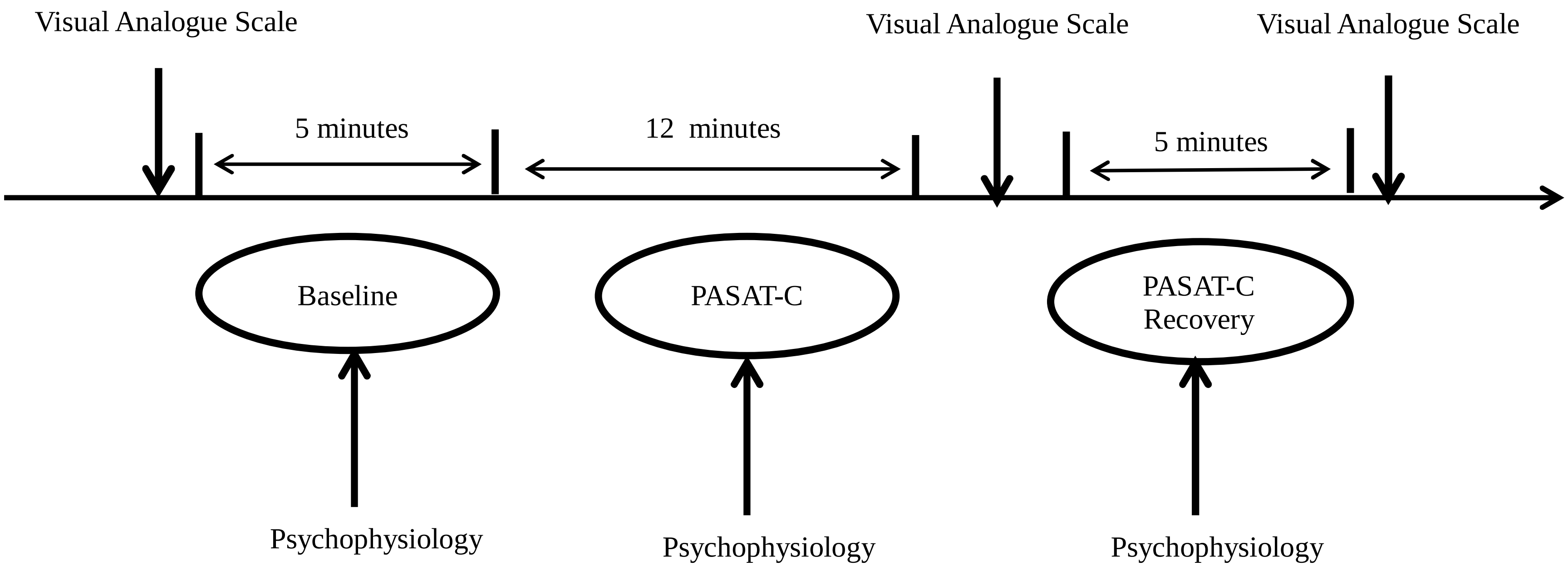
Figure 1

Schematic of the procedure and time periods assessed.



Visual Analogue Scale: self-reported negative emotions, positive emotions, urges to binge eat

Psychophysiology: respiratory sinus arrhythmia, skin conductance response, tonic skin conductance levels

Table 1

Demographic information and clinical characteristics of participants at baseline.

Variable	Bulimia Nervosa *N* = 37		Binge Eating Disorder *N* = 55		Anorexia Nervosa *N* = 15		Healthy Controls *N* = 26	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Age in years	29.81	9.77	41.65	9.49	35.47	11.5	28.81	10.59
	N	%	*N*	%	*N*	%	*N*	%
Minority	11	29.70	23	41.80	4	28.60	11	42.30
Single	22	59.50	24	43.60	9	60.00	15	62.50
Employed or Full-Time Student	33	89.20	51	92.70	13	92.90	25	96.20
Completed 4-year college	23	62.20	43	78.20	11	78.60	14	53.80
Low Income (< 25 000)	20	54.10	16	29.10	8	57.10	17	65.40
Mood Disorder	27	73.00	35	63.60	7	50.00	0	0
Anxiety Disorder	12	32.40	20	36.40	9	64.30	0	0
Medical Comorbidity	22	59.50	26	47.30	7	50.00	4	15.40
Medications	18	48.60	28	50.90	6	42.90	2	7.70

Table 2

Demographic information and severity of eating disorder symptoms of participants at baseline.

	Bulimia Nervosa		Binge Eating Disorder		Anorexia Nervosa		Healthy Controls	
	N = 37		*N* = 55		*N* = 15		*N* = 26	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Body Mass Index	25.45	5.88	36.42	9.47	18.75	3.55	26.68	4.61
EDE Global	3.22	1.04	3.12	0.9	3.05	1.83	0.39	0.52
EDE Eating Concern	2.76	1.15	2.82	1.42	3.05	2.05	0.04	0.08
EDE Dietary Restraint	3.09	1.33	2.19	1.26	3.17	2.04	0.48	0.89
EDE Weight Concern	3.42	1.41	3.59	1.07	2.71	1.92	0.48	0.67
EDE Shape Concern	3.62	1.35	3.9	1.25	3.26	1.87	0.55	0.79
Objective Binge Episodes (3 months)	6.29	6.04	4.58	4.11	1.13	2.44	0	0
Compensatory Behaviors Episodes (3 months)	8.37	8.41	0.32	1.13	3.47	4.79	0	0

Table 3

Self-reported negative emotions, happiness and urges to binge eat at baseline, during the Paced Auditory Serial Addition Task – Computerized (PASAT-C), and during recovery from PASAT-C among women with Bulimia Nervosa, Binge Eating Disorder, Anorexia Nervosa and Healthy Controls.

	True Baseline *M* (*SD*)	PASAT-C *M* (*SD*)	Recovery *M* (*SD*)
	Negative Emotions		
Bulimia Nervosa	38.90 (*22.83*)	58.38 (*20.22*)	35.90 (*20.33*)
Binge Eating Disorder	39.40 (*22.82*)	57.24 (*20.20*)	30.55 (*18.25*)
Anorexia Nervosa	48.99 (*22.51*)	60.80 (*26.58*)	47.53 (*21.66*)
Healthy Control	8.91 (*13.18*)	37.07 (*24.47*)	15.40 (*16.38*)
	Happiness		
Bulimia Nervosa	43.98 (*18.74*)	21.03 (*21.58*)	29.50 (*23.21*)
Binge Eating Disorder	40.85 (*18.05*)	23.52 (*25.07*)	29.20 (*21.58*)
Anorexia Nervosa	37.56 (*25.16*)	22.47 (*26.08*)	17.68 (*24.51*)
Healthy Control	59.41 (*19.40*)	28.51 (*28.71*)	39.42 (*28.14*)
	Urge to Binge		
Bulimia Nervosa	33.15 (*28.48*)	31.84 (*36.86*)	34.77 (*32.55*)
Binge Eating Disorder	46.87 (*27.10*)	42.41 (*36.48*)	44.21 (*32.50*)
Anorexia Nervosa	21.70 (*29.03*)	11.42 (*25.86*)	20.41 (*33.67*)
Healthy Control	.77 (*1.97*)	2.98 (*11.41*)	.34 (*.71*)

Table 4

Completion time and latency to quit on the Paced Auditory Serial Addition Task – Computerized (PASAT- C) among women with Bulimia Nervosa, Binge Eating Disorder, Anorexia Nervosa and Healthy Controls.

	PASAT-C Completion N (%)	PASAT-C Latency to Quit M (*SD*)
Bulimia Nervosa	23 (62.2%)	643.84 (*128.05*)
Binge Eating Disorder	38 (69.1%)	683.84 (*89.32*)
Anorexia Nervosa	11 (73.3%)	682.00 (*100.69*)
Healthy Control	22 (84.6%)	690.85 (*110.35*)

Table 4

Respiratory sinus arrhythmia, skin conductance responses and tonic skin conductance levels for Bulimia Nervosa, Binge Eating Disorder, Anorexia Nervosa and Healthy Controls at baseline, during PASAT-C, and during recovery from PASAT-C.

	True Baseline *M* (SD)	PASAT-C *M* (SD)	Recovery *M* (SD)
	Respiratory sinus arrhythmia (in ms^2)		
Bulimia Nervosa	6.25 (1.16)	6.05 (1.06)	6.32 (1.09)
Binge Eating Disorder	5.25 (1.36)	5.16 (1.39)	5.40 (1.32)
Anorexia Nervosa	5.83 (1.16)	5.69 (1.12)	6.16 (1.12)
Healthy Control	6.21 (1.36)	6.06 (1.30)	6.38 (1.27)
	Skin conductance response		
Bulimia Nervosa	1.24 (1.70)	3.54 (3.45)	2.77 (2.39)
Binge Eating Disorder	2.13 (1.90)	3.78 (3.02)	3.88 (2.75)
Anorexia Nervosa	2.12 (2.64)	4.22 (4.12)	4.16 (2.76)
Healthy Control	1.83 (1.72)	2.90 (2.54)	3.94 (2.94)
	Tonic skin conductance levels (in μs)		
Bulimia Nervosa	2.35 (3.89)	5.28 (6.51)	4.98 (6.40)
Binge Eating Disorder	2.94 (2.27)	5.14 (4.06)	5.06 (4.05)
Anorexia Nervosa	2.67 (2.75)	6.93 (6.58)	7.27 (5.86)
Healthy Control	2.40 (3.11)	5.37 (6.04)	5.77 (6.76)