



Negative and positive emotional eating uniquely interact with ease of activation, intensity, and duration of emotional reactivity to predict increased binge eating

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ABSTRACT

Binge eating is present in obesity and clinical eating disorder populations and positively associated with poor health outcomes. Emotional eating may be related to binge eating, but relationships with emotional reactivity remain unexplored. The present study examined the relationships between negative and positive emotional eating and emotional reactivity in predicting binge eating. A cross-sectional study was employed using an online community sample in the United States. Participants ($N = 258$) completed surveys assessing negative (Emotional Eating Scale-Revised, depression subscale) and positive emotional eating (Emotional Appetite Questionnaire), negative and positive emotional reactivity (Perth Emotional Reactivity Scale), and binge eating (Binge Eating Scale). Six moderation analyses were calculated with negative and positive emotional reactivity (ease of activation, intensity, and duration) as moderators of the relationship between negative and positive emotional eating, respectively, and binge eating. Increased negative emotional eating was associated with increased binge eating when duration of negative emotional reactivity was 1 standard deviation above average ($p < .001$), but at 1 standard deviation below average ($p < .001$), increased negative emotional eating was associated with decreased binge eating. Increased positive emotional eating was associated with increased binge eating when intensity ($p < .01$) of positive emotional reactivity was 1 standard deviation above average and when activation ($p < .05$) of positive emotional reactivity was slightly above 1 standard deviation above average. Increased positive emotional eating was associated with decreased BE when intensity of positive emotional reactivity was 1 standard deviation below ($p < .05$) average. Emotional reactivity may uniquely impact the relationship between emotional eating and binge eating. Research and clinical implications for the contribution of negative and positive emotional eating and emotional reactivity on binge eating are discussed.

1. Introduction

Binge eating (BE) is characterized as the consumption of an unusually large amount of food over a short period of time coupled with feelings of loss of control during the eating episode (American Psychiatric Association [APA], 2013). Some research holds that BE is the most common eating disorder symptom (Mitchison & Mond, 2015). BE has gained research attention because of its relevance across all clinical eating disorders (APA, 2013) and significant psychological comorbidity (Bulik, Sullivan, & Kendler, 2002; Yanovski, Nelson, Dubbert, & Spitzer, 1993). In addition, BE has emerged as a significant public health concern because it is a risk factor for weight gain, obesity, and related secondary health conditions such as high blood pressure, type 2 diabetes, and heart disease (Bankier, Januzzi, & Littman, 2004; Bulik

et al., 2002; Hasler et al., 2004; Telch, Agras, & Rossiter, 1988; Yanovski et al., 1993). Understanding factors related to the development and maintenance of BE is critical to improving prevention and treatment outcomes.

Emotional eating, or eating triggered by emotions in the absence of physiological hunger (Arnow, Kenardy, & Agras, 1995), is one factor that may be related to BE. Emotional eating and BE are distinct constructs. For example, emotional antecedents are not required to trigger BE (APA, 2013), nor is eating an unusually large amount of food over a short period of time required for emotional eating (Arnow et al., 1995). Even more, BE has marked feelings of loss of control during the eating episode (APA, 2013), a clinical feature unshared by emotional eating. That said, emotional eating and BE share negative health consequences (e.g., weight gain and obesity; Fairburn et al., 1998; Hudson, Hiripi,

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Pope Jr, & Kessler, 2007; Koenders & van Strien, 2011; Raman, Smith, & Hay, 2013; Udo & Grilo, 2018). Emotional eating is further conceptualized along negative and positive emotional dimensions (Nolan, Halperin, & Geliebter, 2010), leading some to consider how such diverse emotions relate to eating behavior including BE.

Negative and positive emotional eating may be associated with overeating and BE. In the context of negative emotional eating, several theories have been put forth to explain the relationship with BE. According to affect regulation theories of BE, BE is elicited to reduce the internal experience of negative emotions (Heartherton & Baumeister, 1991; Pine, 1985; Polivy & Herman, 1993). While research into the mechanisms of positive emotional eating is limited, some research holds that positive emotional eating may be driven by hedonic (Macht, 1999) or external influences (van Strien, Konttinen, Homberg, Engels, & Wikens, 2016). Research has demonstrated that consumption of highly palatable foods (e.g., both savory and sweet foods) was greater after positive mood inductions than negative mood inductions (Cardi, Leppanen, & Treasure, 2015) and that positive emotions, compared to negative emotions, are related to increased pleasantness and motivation to eat food (Macht, 1999; Macht, Roth, & Ellgring, 2002). Because some estimates hold that positive emotional eating occurs just as frequently as negative emotional eating (Macht, Haupt, & Salewsky, 2004), more research is needed to understand how the full range of emotions influence eating behavior, specifically BE.

Research has revealed negative correlations between negative and positive emotional eating across various emotional eating instruments (e.g., Emotional Appetite Questionnaire and Dutch Eating Behavior Questionnaire; Geliebter & Aversa, 2003; Nolan et al., 2010; van Strien, Frijters, Bergers, & Defares, 1986; van Strien, Rookus, Bergers, Frijters, & Defares, 1986). For example, Nolan et al. (2010) found significant negative correlations between negative and positive emotional eating dimensions on the Emotional Appetite Questionnaire, consistent with other research using the Emotional Eating Scale (Braden, Musher-Eizenman, Watford, & Emley, 2018). In experimental settings, individuals who are more likely to endorse negative rather than positive emotional eating consumed more snacks after a negative mood induction (van Strien et al., 2013). Interestingly, a recent meta-analysis of experimental paradigms demonstrated that the link between negative emotions and food consumption was not present while a small, significant effect for increased eating was found for positive emotions (Evers, Dingemans, Junghans, & Boevé, 2018). It should be noted, however, this review did not consider the effects of negative and positive emotions on maladaptive eating behaviors such as BE.

Negative emotional eating has been found to be positively associated with BE. For example, research has identified that individuals with binge eating disorder were more likely to engage in negative emotional eating than both individuals with eating disorder not otherwise specified and control participants (Eldredge & Agras, 1996). These early data set the stage for continued research on negative emotional eating and BE, which has been replicated in the literature (Agras & Telch, 1998; Fischer et al., 2007; Tanofsky, Wilfley, Spurrell, Welch, & Brownell, 1997; Telch & Agras, 1996a, 1996b). What is less clear is the relationship between positive emotional eating and BE.

Recent research has shown that increased positive emotional eating may be related to overeating and BE (Bongers, de Graaff, & Jansen, 2016; Bongers, Jansen, Havermans, Roefs, & Nederkoorn, 2013; Bongers, Jansen, Houben, & Roefs, 2013; Evers, de Ridder, & Adriaanse, 2009; Sultson, Kukk, & Akkermann, 2017). A majority of this research has examined if positive emotional eating leads to overeating (Bongers, Jansen, Havermans et al., 2013; Bongers, Jansen, Houben et al., 2013; Bongers et al., 2016; Evers et al., 2009) not BE, leaving only one study that has directly explored the relationship between positive emotional eating and BE (Sultson et al., 2017). Sultson and colleagues identified a moderate, positive correlation between positive emotional eating and BE, with positive emotional eating continuing to predict BE after controlling for negative emotional eating in healthy adults (Sultson et al.,

2017). Although previous research cites evidence for associations between negative and positive emotional eating and BE, no research has examined factors that may strengthen the relationship between emotional eating and BE.

Emotional reactivity is one factor that may interact with emotional eating to influence BE. Emotional reactivity is characterized as an emotional response to a stimulus that varies by individual and by ease of activation, intensity, and duration (Davidson, 1998; Rothbart & Derryberry, 1981). These levels of variability in the time-course progression of emotional responses are supported by a body of research (Becerra, Preece, Campitelli, & Scott-Pillow, 2017; Davidson, 1998; Gruber, Harvey, & Purcell, 2011; Mauss, Cook, Cheng, & Gross, 2007) and significant variance in emotional reactivity underscores many models of psychopathology (Gross & Jazaieri, 2014; Linehan, 1993; Rottenberg & Johnson, 2007). This research also holds that returning emotional reactivity to baseline is a central tenant across diverse psychotherapeutic treatment programs (Barlow et al., 2010; Linehan, 1993). Thus, exaggerated emotional reactivity across both negative and positive emotions could have additive effects on emotional eating in which eating is engaged in response to these very emotions.

In the context of negative emotional reactivity, experience sampling methods show that negative emotional reactivity played an important role in the experience of negative emotions in overweight adults with BE (Lingswiler, Crowther, & Stephens, 1987). This research also identified that duration of negative emotions, as opposed to intensity of negative emotions, was related to BE severity (Lingswiler et al., 1987). More recent research has demonstrated that negative reactivity plays an important role in the development of disordered eating attitudes in adolescents, important antecedents to disordered eating behaviors including BE (Evans et al., 2019). To the understanding of the authors, no research to date has directly examined the moderating influence of negative emotional reactivity on the relationship between negative emotional eating and BE. Similarly, limited research has endeavored to understand the relationships between positive emotional reactivity and BE.

One recent laboratory-based study showed that positive, but not negative, emotional reactivity to food images was positively associated with BE among college students (Racine, Hebert, & Benning, 2018). Thus, positive emotional reactivity and reward valuation of food and eating cues may be related to BE (Racine et al., 2018; Schag, Schönleber, Teufel, Zipfel, & Giel, 2013; Simon et al., 2016). Like research on negative emotional reactivity, positive emotional reactivity maps onto the experience of these emotions in everyday life (Becerra et al., 2017; Davidson, 1998; Rothbart & Derryberry, 1981); thus, this general index of emotional reactivity could be useful in assessing emotional eating in which general emotions, not food-specific emotions, are purported antecedents to eating behavior. Indeed, despite evidence that positive emotional reactivity may be related to increased BE, this study (Racine et al., 2018) primarily focused on food-specific positive emotions as opposed to positive emotionality more broadly. Therefore, the present study builds on this work by examining negative and positive emotional reactivity (i.e., ease of activation, intensity, and duration of emotions) as potential moderators of the relationships between negative and positive emotional eating and BE.

1.1. The present study

The present study used theories of negative and positive emotional eating and emotional reactivity (Arnou et al., 1995; Becerra et al., 2017; Davidson, 1998; Macht, 1999; Macht et al., 2002; Rothbart & Derryberry, 1981) to examine these variables as potential correlates of BE. Because no research to date has examined negative and positive emotional eating and emotional reactivity together in the prediction of BE, the present study was exploratory in nature. While research on the positive association between negative emotional eating and BE is more clear (Agras & Telch, 1998; Fischer et al., 2007; Tanofsky, Wilfley,

Spurrell, Welch, & Brownell, 1997; Telch & Agras, 1996a, 1996b), the present study added to this understanding by exploring how negative emotional reactivity moderated this relationship. The present study also added to limited work identifying that increased positive emotional eating was associated with BE (Sultson et al., 2017), as well as limited work identifying a positive association between positive emotional reactivity and BE (Racine et al., 2018). To this end, we aimed to understand if negative and positive emotional reactivity (i.e., ease of activation, intensity, and duration of emotional reactivity) *strengthened* the relationship between emotional eating and BE.

2. Methods

2.1. Participants

Participants ($N = 258$) were recruited through Amazon Mechanical Turk (MTurk; a paid participant database run through [Amazon.com](https://www.amazon.com)) to participate in a cross-sectional study on eating behaviors, childhood experiences, and health. The current study is a secondary analysis of a primary study that examined correlates of emotional eating (Braden et al., under review). Inclusion criteria were that participants were adults (≥ 18 years), residents of the U.S., and fluent in English. Initially, 638 participants were recruited, and from these 380 were excluded from the final sample. Participants were excluded due to failing to provide consent (0.3% of the initial sample), reporting the presence of a medical condition impacting weight (e.g., insulin dependent diabetes, cancer requiring active chemotherapy; 14.6%), reporting current pregnancy or breast-feeding (11.1%), or reporting a current (8%) or past diagnosis of bulimia nervosa or anorexia nervosa (10%). Given that one of the goals of the primary study was to inform treatment development for adults with emotional eating who do not have anorexia nervosa or bulimia nervosa, individuals with a current or past diagnosis of anorexia nervosa or bulimia nervosa were excluded from study enrollment. Participants were also excluded if their response patterns indicated random responding (i.e., failed to correctly answer at least 2 out of 3 attention gauge items; 15.5% of the initial sample).

2.2. Measures

2.2.1. Demographics

Participants self-reported demographic characteristics including age, gender, education level, race, relationship status, employment status, and income. Participants also reported similar demographic characteristics about their parents, such as marital status and education, but parent demographics were not analyzed in the present study.

2.2.2. Anthropometry

Body mass index (BMI; kg/m^2) was calculated using self-reported height and weight. Participants reported their height in feet and inches and weight in pounds.

2.2.3. Emotional Appetite Questionnaire (EAQ)

Positive emotional eating was measured using the Emotional Appetite Questionnaire (Nolan et al., 2010). The EAQ contains 22 items measuring the tendency to eat in response to positive and negative emotions and situations. The first 14 items address eating in response to emotions (e.g., “confident”, “playful”), and the following 8 items address eating in response to situations (e.g., “after receiving good news”). Participants are instructed to rate how emotions and situations affect their food intake on a scale ranging from 1 (“Much less”) to 9 (“Much more”). They can also answer items with “not applicable” and “don't know”, but those responses were not included in scoring. EAQ items are separated into positive EAQ (EAQ-P) and negative EAQ (EAQ-N) scales. Each scale can be further split into positive emotion (EAQ-PE), positive situation (EAQ-PS), negative emotion (EAQ-NE), and negative situation subscales (EAQ-NS). Items from each subscale (save for the “not

applicable” and “don't know” responses) are averaged to determine subscale scores for the EAQ-PE, EAQ-PS, EAQ-NE, and EAQ-NS subscales. EAQ-PE and EAQ-PS scores are then averaged to determine the EAQ-P scale score. Similarly, EAQ-NE and EAQ-NS scores are averaged to determine the EAQ-N scale score. Higher scores indicate increased emotional eating. All EAQ items were administered, but only positive emotional eating items (not positive situation items) were analyzed in the present study. For negative emotional eating, we administered the Emotional Eating Scale-Revised (see below). Nolan et al. (2010) found adequate internal consistency for the positive emotion subscale ($\alpha = 0.75$) and weaker internal consistency for the positive situation subscale ($\alpha = 0.57$) of the EAQ. The present study, in contrast, obtained stronger internal consistency for all positive EAQ items ($\alpha = 0.93$), the positive emotional subscale ($\alpha = 0.92$), and the positive situations subscale ($\alpha = 0.83$).

2.2.4. Emotional Eating Scale-Revised (EES-R)

Negative emotional eating was assessed with the depression subscale of the Emotional Eating Scale-Revised (EES-R; Koball, Meers, Storfer-Isser, Domoff, & Musher-Eizenman, 2012). Consisting of 25 items, the EES-R assessed eating in response to negative emotions. Participants were asked to rate their urge to eat on a 5-point Likert scale; available responses for each question ranged from “No desire to eat” to “An overwhelming urge to eat.” For the present study, we used the depression subscale which consisted of 9 items assessing eating in response to depressed feelings (e.g., discouragement, sadness, feelings of being upset). Scores on the EES-D depression subscale were calculated by computing the mean of the items. In previous research, the EES-R showed good psychometric properties (Koball et al., 2012). In the present study, Cronbach's alpha was .94, indicating high internal consistency.

2.2.5. Binge Eating Scale (BES)

The Binge Eating Scale consists of 16 self-report items used to assess behavioral, emotional, and cognitive aspects of BE (Gormally, Black, Dastin, & Rardin, 1982). Each item is comprised of three to four statements about a particular characteristic of binge eating (e.g. eating while bored). Statements range in severity (e.g. “I don't have any problem stopping eating when I feel full” compared to “Because I have a problem not being able to stop eating when I want, I sometimes have to induce vomiting to relieve my stuffed feeling”). Participants are asked to select the statement that best describes their own behavior. Higher BES scores indicate greater severity of BE. Previous research supports the psychometric properties of the BES in a variety of clinical and non-clinical samples (Duarte, Pinto-Gouveia, & Ferreira, 2015; Hood, Grupski, Hall, Ivan, & Corsica, 2013; Mason & Lewis, 2015; Napolitano & Himes, 2011; Timmerman, 1999; Gordon, Holm-Denoma, Troop-Gordon, & Sand, 2012). In the present study, Cronbach's alpha was .93, indicating high internal consistency.

2.2.6. Perth Emotional Reactivity Scale (PERS)

Negative and positive emotional reactivity were measured using the Perth Emotional Reactivity Scale (Becerra et al., 2017). The PERS is a 30-item self-report scale used to assess emotional reactivity for both positive and negative emotions. This scale measures three dimensions of emotional reactivity: activation (e.g., “I tend to get happy very easily”), intensity (e.g., “When I am joyful, I tend to feel it very deeply”), and duration (e.g., “I can remain enthusiastic for quite a while”). In total, there are six subscales containing five items each. All six subscales were administered. Participants were asked to rate the extent to which the items apply to them on a 5-point Likert scale ranging from “Very unlike me” to “Very like me.” Scale and subscale scores are calculated by summing participant responses, with higher scores indicating greater emotional reactivity. Becerra et al. (2017) demonstrated strong psychometric properties of the PERS, including high internal consistency for the general positive reactivity scale ($\alpha = 0.93$), as well as

the positive-activation ($\alpha = 0.81$), positive-intensity ($\alpha = 0.89$), and positive duration ($\alpha = 0.81$) subscales. The authors also demonstrated high internal consistency for the general negative reactivity scale ($\alpha = 0.94$) and the negative-activation ($\alpha = 0.86$), negative-intensity ($\alpha = 0.87$), and negative-duration ($\alpha = 0.85$) subscales. In the present study, Cronbach's alpha also indicated high internal consistency for the general positive reactivity scale ($\alpha = 0.94$), all three positive subscales ($\alpha = 0.87$, $\alpha = 0.86$, $\alpha = 0.86$, respectively), the general negative reactivity scale ($\alpha = 0.96$), and all three negative subscales ($\alpha = 0.91$, $\alpha = 0.89$, $\alpha = 0.89$, respectively).

2.3. Procedure

Participants were recruited through MTurk's TurkPrime extension. Following electronic informed consent, participants completed a sequence of self-report questionnaires. Completion of the entire sequence lasted approximately 30 minutes in total. Participants were debriefed at the end of the present study and offered contact information for study related questions or concerns. In exchange for study completion, participants were compensated \$1.25.

Although previous research demonstrated the potential for MTurk to yield quality data from diverse samples (Casler, Bickel, & Hackett, 2013), data quality may still be compromised as a result of low participant motivation or random responses. Researchers may choose from a variety of methods to reduce the risk of collecting invalid data (Buchanan & Scofield, 2018). Throughout the present study, three manipulation checks were implemented to identify threats to data quality (e.g., "When you are done reading this question, choose 'about half the time' as your answer.").

2.4. Analytic plan

Data were screened for accuracy to ensure that the minimum and maximum values for each variable were within the expected range. Missing data were identified. Next, potential outliers were identified. Regression assumptions were then examined, followed with descriptive statistics to calculate demographic characteristics of the sample. Distributions for primary study variables were examined by calculating standard deviations and means, and correlations between primary study variables (negative and positive emotional eating, negative and positive emotional reactivity (activation, intensity, and duration subscales), and BE) were also calculated.

Moderation analyses (Model 1) were calculated using the PROCESS macro in SPSS version 25 following the methods of Hayes (2018). Bootstrapped, 95% confidence intervals with 5000 replications were calculated to estimate indirect effects. Indirect effects were considered significant if the confidence intervals did not contain zero. Six moderation analyses were conducted to examine negative and positive emotional reactivity (activation, intensity, and duration) as moderators of the relationship between negative and positive emotional eating, respectively, and BE. BE was used as the dependent variable in all analyses. Heteroscedasticity consistent standard error estimates (HC3 estimator) (Hayes & Cai, 2007) were used after inspection of data given scatterplots revealed data were slightly heteroscedastic. Use of HC3 estimator aids in correction of heteroscedasticity. Additionally, all six moderation models were calculated with BMI and gender as covariates.

3. Results

3.1. Preliminary results

Of the sample of 258 participants, 2 participants (0.78%) reported implausible age values and 20 participants (7.75%) reported height and weight values that yielded unlikely BMI values or BMI values which may indicate moderate to severe thinness (i.e., BMI < 17; World Health Organization, 1995). Unlikely BMI values were determined by

Table 1
Participant characteristics.

Variable	M(SD) or %
Age	36.5(11.0)
Gender	
Women	50.4%
BMI	26.9(7.1)
Ethnicity	
Caucasian	64.7%
Hispanic	5.4%
African American	21.7%
Asian	5.0%
American Indian or Alaska Native	1.2%
Hawaiian or other Pacific Islander	0.4%
Other/Would rather not say	1.6%
Marital Status	
Single	34.5%
Married	45.3%
Not married but living together	10.9%
Divorced/Separated	7.8%
Widowed	0.8%
Would rather not say	0.8%
Employment	
Full-time	75.6%
Part-time	10.1%
Student	2.3%
Unemployed	3.9%
Retired	2.3%
Homemaker	4.3%
Would rather not say	1.6%
Income	
< \$20,000	8.1%
\$20,000-\$50,000	38.8%
\$50,000-\$75,000	32.2%
> \$75,000	20.9%

Note. M = mean. SD = standard deviation. % = percent. BMI = body mass index.

identifying subjects who reported impossible height or weight data (e.g., weight of 3 pounds). BMI values which may indicate moderate to severe thinness were subjects whose calculated BMI was less than 17 (World Health Organization, 1995). These data were not used in moderation analyses because BMI was included as a covariate. An additional 25 participants (9.69%) were not calculated in moderation analyses due to missing data on more than one item of the EAQ. Regression assumptions of linearity, normality, additivity, and homoscedasticity were also examined. Again, scatterplots revealed that data were slightly heteroscedastic. To correct for this, heteroscedasticity consistent standard error estimates (HC3 estimator) were used in moderation analyses.

The sample of participants ($N = 258$) was nearly an even split between men ($n = 127$) and women ($n = 130$), with only one participant who listed their gender as "other." Participant ages ranged from 19 to 73 years ($M = 36.5$, $SD = 11.0$), and they were primarily White (64.7%), married (45.3%), and overweight, as indicated by their BMI ($M = 26.9$, $SD = 7.1$). See Table 1 for additional participant characteristics.

Variability across study variables was sufficient, and normality of distribution was confirmed with use of Q-Q plots and histograms. Means, standard deviations, and bivariate correlations for study variables are presented in Table 2. Results revealed significant correlations between study variables (see Table 2). To dismiss concerns of multicollinearity between emotional eating and emotional reactivity, we conducted collinearity diagnostics. Results revealed that all independent variables had a tolerance greater than 0.17 and variance inflation factors that did not exceed a value of 10, with no independent variable exceeding a variance inflation factor greater than 6 (Belsley, Kuh, & Welsch, 1980).

Table 2

Means, standard deviations, and correlations of study variables.

	Mean	SD	1.	2.	3.	4.	5.	6.	7.	8.
1. NEE	2.49	1.11	–							
2. NER-A	15.61	5.71	.57**	–						
3. NER-I	16.50	5.23	.50**	.85**	–					
4. NER-D	15.69	5.46	.53**	.87**	.86**	–				
5. PEE	5.51	1.55	.18**	.16*	.14*	.21**	–			
6. PER-A	16.25	4.47	.19**	.03	.06	.00	.28**	–		
7. PER-I	16.04	4.48	.24**	.15*	.22**	.15*	.32**	.80**	–	
8. PER-D	17.21	4.34	.05	-.16*	-.09	-.16*	.21*	.76**	.65**	–
9. BE	10.53	9.39	.60**	.49**	.43**	.46**	.13*	.16*	.20**	.02

Notes. * $p < .05$. ** $p < .01$. NEE = Negative Emotional Eating. PEE = Positive Emotional Eating. NER-A = Negative Emotional Reactivity-Activation. NER-I = Negative Emotional Reactivity-Intensity. NER-D = Negative Emotional Reactivity-Duration. PER-A = Positive Emotional Reactivity-Activation. PER-I = Positive Emotional Reactivity-Intensity. PER-D = Positive Emotional Reactivity-Duration. BE = Binge Eating. $N = 218$ (Model with NEE, NER, and BE). $N = 211$ (Model with PEE, PER, and BE).

Table 3

Positive emotional reactivity as a moderator of the relationship between positive emotional eating and binge eating.

	b	t	p	LLCI	ULCI
Model 1					
PEE	.33	.72	.47	-.57	1.24
PER-A	.26	1.98	.05	.00	.53
PEE * PER-A	.18	2.20	.03*	.02	.33
<i>Conditional Effects</i>					
1 SD below average	-.45	-.79	.43	-1.58	.68
Average PER-A	.33	.72	.47	-.57	1.24
1 SD above average	1.12	1.89	.06	-.05	2.28
Model 2					
PEE	.59	1.22	.22	-.36	1.55
PER-D	-.08	-.54	.59	-.37	.21
PEE * PER-D	.14	1.66	.10	-.03	.30
Model 3					
PEE	.00	.01	.44	-6.55	2.89
PER-I	.38	2.64	.01**	.10	.67
PEE * PER-I	.33	3.36	.00***	.14	.52
<i>Conditional Effects</i>					
1 SD below average	-1.47	-2.14	.03*	-2.82	-.12
Average PER-I	.00	.01	.99	-.90	.91
1 SD above average	1.50	2.57	.01*	.34	2.61

Notes. * $p < .05$. ** $p < .01$. *** $p < .001$. PER-A, $R^2 = 0.14$. PER-D, $R^2 = 0.13$. PER-I, $R^2 = 0.19$. BE = Binge Eating. PEE = Positive Emotional Eating. PER-A = Positive Emotional Reactivity-Activation. PER-D = Positive Emotional Reactivity-Duration. PER-I = Positive Emotional Reactivity-Intensity. Covariates included in moderation analyses: BMI and gender.

3.2. Positive emotional reactivity as a moderator

3.2.1. Model 1: Ease of activation of positive emotional reactivity

When examining BE, the overall model assessing activation of positive emotional reactivity was significant ($R^2 = 0.14$, $p < .001$; Table 3). The interaction between positive emotional eating and activation of positive emotional reactivity were entered into the model, which accounted for a significant proportion of the variance in BE ($b = 0.18$, $p = .03$). Conditional moderation was observed such that increased positive emotional eating was approaching a significant association with BE when activation of positive emotional reactivity was 1 SD above average ($b = 1.12$, $p = .06$; activation of positive emotional reactivity score = 4.50; Fig. 1). To investigate this effect further, we examined the Johnson-Neyman significance region. Increased positive emotional eating was significantly associated with BE when activation of positive emotional reactivity was slightly above 1 SD (activation of positive emotional reactivity score = 5.50; $b = 1.29$, $p = .04$), and this effect on BE continued to strengthen as positive emotional eating and activation of positive emotional reactivity increased. Activation of positive emotional reactivity did not moderate the association between positive emotional eating and BE when activation of positive emotional

reactivity was average ($b = 0.33$, $p = .47$) or below average ($b = -0.45$, $p = .43$). Conditional moderation remained significant with the inclusion of BMI ($b = 0.37$, $p < .001$) and gender ($b = 1.31$, $p = .30$) as covariates in the model.

3.2.2. Model 2: Duration of positive emotional reactivity

When examining BE, the overall model assessing duration of positive emotional reactivity was significant ($R^2 = 0.13$, $p < .001$; Table 3). However, duration of positive emotional reactivity did not moderate the association between positive emotional eating and BE ($b = 0.14$, $p = .10$).

3.2.3. Model 3: Intensity of positive emotional reactivity

When examining BE, the overall model assessing intensity of positive emotional reactivity was significant ($R^2 = 0.19$, $p < .001$; Table 3). The interaction between positive emotional eating and intensity of positive emotional reactivity were entered into the model, which accounted for a significant proportion of the variance in BE ($b = 0.33$, $p < .001$). Conditional moderation was observed such that increased positive emotional eating was associated with increased BE when intensity of positive emotional reactivity was 1 SD above average ($b = 1.50$, $p = .01$; Fig. 2). Conditional moderation was also observed such that increased positive emotional eating was associated with decreased BE when intensity of positive emotional reactivity was 1 SD below average ($b = -1.47$, $p = .03$; Fig. 2). Intensity of positive emotional reactivity, coupled with increased positive emotional eating, strengthened the effect on BE as intensity reached 1 SD above average while at 1 SD below average, the relationship between these variables weakened the effect on BE. Intensity of positive emotional reactivity did not moderate the association between positive emotional eating and BE when intensity of positive emotional reactivity was average ($b = .00$, $p = .99$). Conditional moderation remained significant with the inclusion of BMI ($b = 0.36$, $p < .001$) and gender ($b = 1.32$, $p = .27$) as covariates in the model.

3.3. Negative emotional reactivity as a moderator

3.3.1. Model 1: Ease of activation of negative emotional reactivity

When examining BE, the overall model assessing duration of positive emotional reactivity was significant ($R^2 = 0.46$, $p < .001$; Table 4). However, activation of negative emotional reactivity did not moderate the association between negative emotional eating and BE ($b = 0.13$, $p = .13$).

3.3.2. Model 2: Duration of negative emotional reactivity

When examining BE, the overall model assessing duration of negative emotional reactivity was significant ($R^2 = 0.47$, $p < .001$; Table 4). The interaction between negative emotional eating and duration of negative emotional reactivity were entered into the model, which

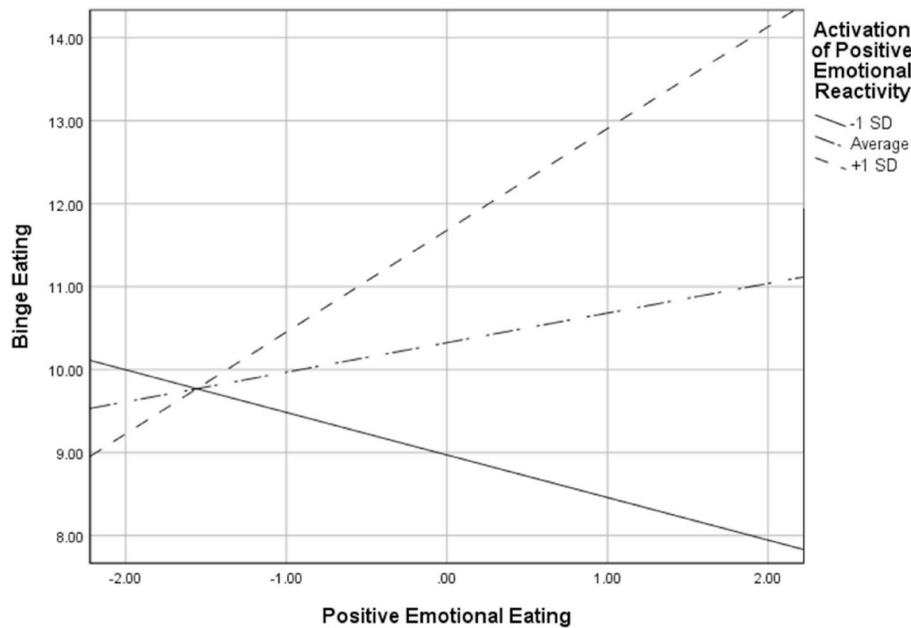


Fig. 1. Moderation of positive emotional eating and binge eating by activation of positive emotional reactivity.

accounted for a significant proportion of the variance in BE ($b = 0.17, p < .05$). Conditional moderation was observed such that increased negative emotional eating was associated with increased BE when duration of negative emotional reactivity was 1 SD above average ($b = 4.94, p < .001$; Fig. 3). Conditional moderation was also observed such that increased negative emotional eating was associated with decreased BE when duration of negative emotional reactivity was 1 SD below average ($b = 3.10, p < .001$; Fig. 3). Finally, conditional moderation was also observed such that increased negative emotional eating was associated with BE when duration of negative emotional eating was average ($b = 4.02, p < .001$; Fig. 3). As can be seen in Fig. 3, this interaction between negative emotional eating and negative emotional reactivity at the mean positioned the variance in BE between the ± 1 SDs. Conditional moderation remained significant with the inclusion of BMI ($b = 0.31, p < .001$) and gender ($b = 0.91, p = .34$)

as covariates in the model.

3.3.3. Model 3: Intensity of negative emotional reactivity

When examining BE, the overall model assessing intensity of negative emotional reactivity was significant ($R^2 = 0.44, p < .001$; Table 4). However, intensity of negative emotional reactivity did not moderate the association between negative emotional eating and BE ($b = 0.05, p = .61$).

4. Discussion

Binge eating cuts across all clinical eating disorders (APA, 2013) and is positively associated with a range of negative health outcomes including weight gain and obesity (Fairburn et al., 1998; Hudson, Hiripi, Pope, & Kessler, 2007; Raman et al., 2013; Udo & Grilo, 2018).

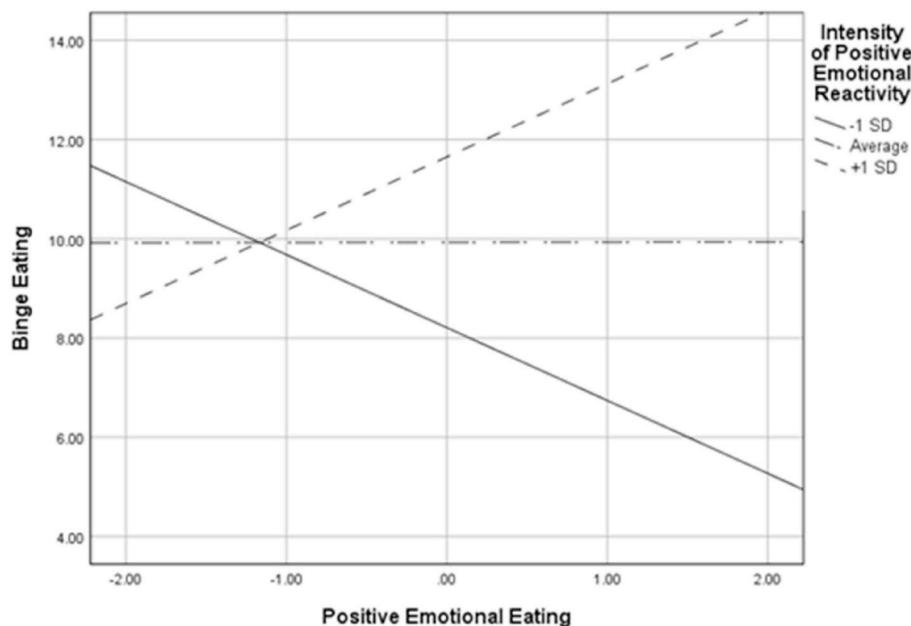


Fig. 2. Moderation of positive emotional eating and binge eating by intensity of positive emotional reactivity.

Table 4

Negative emotional reactivity as a moderator of the relationship between negative emotional eating and binge eating.

	b	t	p	LLCI	ULCI
Model 1					
NEE	3.92	6.38	.00***	2.71	5.13
NER-A	.44	4.24	.00***	.23	.64
NEE * NER-A	.13	1.54	.13	-.04	.30
Model 2					
NEE	4.03	7.20	.00***	2.92	5.13
NER-D	.44	4.53	.00***	.25	.63
NEE * NER-D	.17	2.05	.04*	.01	.33
<i>Conditional Effects</i>					
1 SD below average	3.10	3.93	.00***	1.55	4.67
Average NER-D	4.02	7.20	.00***	2.92	5.13
1 SD above average	4.94	7.80	.00***	3.70	6.20
Model 3					
NEE	4.48	7.18	.00***	3.25	5.71
NER-I	.36	3.25	.00**	.14	.57
NEE * NER-I	.05	.51	.61	-.14	.24

Notes. * $p < .05$. ** $p < .01$. *** $p < .001$. NER-A, $R^2 = 0.46$. NER-D, $R^2 = 0.47$. NER-I, $R^2 = 0.44$. BE = Binge Eating. NEE = Negative Emotional Eating. NER-A = Negative Emotional Reactivity-Activation. NER-D = Negative Emotional Reactivity-Duration. NER-I = Negative Emotional Reactivity-Intensity. Covariates included in moderation analyses: BMI and gender.

Research has endeavored to understand risk and maintenance factors associated with BE, including the complex role of emotional eating. Adding to this research, the present study investigated the interaction between negative and positive emotional eating and emotional reactivity (ease of activation, intensity, and duration) to predict BE.

First, we examined if the relationship between negative and positive emotional eating and BE would be strengthened by increased activation of negative and positive emotions. We failed to find evidence for this effect when considering the relationship between negative emotional eating and activation of negative emotions on BE. However, we found evidence for the relationship between positive emotional eating and activation of positive emotions to impact BE when activation of positive emotional reactivity was slightly above 1 SD. Increased activation of positive emotional reactivity equates to increased ease with which positive emotions are activated (Davidson, 1998; Rothbart & Derryberry, 1981), thus facilitating the experience of positive emotions. Increased

ease in activating positive emotions may put people who eat in response to positive emotions at additional risk for BE. Further, these findings may help develop positive emotional eating theory in relation to BE. With positive emotional eating, it appears that the positive state imposed by these emotions may not be enough to drive BE. Indeed, the relationship between positive emotional eating and BE was strengthened by activation of positive emotional reactivity, thereby highlighting a potential pathway by which BE may emerge in the presence of positive emotions. Given the correlational nature of the present study, experimental manipulation with mood induction of emotional eaters who experience increased ease of activation of emotions may allow for confirmatory conclusions regarding the role of emotions (e.g., negative and positive) and temporal nature of these variables on BE.

Next, we examined if the relationship between negative and positive emotional eating and BE would be strengthened by increased intensity of negative and positive emotions. Again, we failed to find evidence for this effect when considering the relationship between negative emotional eating and intensity of negative emotions on BE, consistent with previous research (Lingswiler et al., 1987). However, with positive emotional eating we found support for this hypothesis such that increased positive emotional eating was associated with increased BE when intensity of positive emotional reactivity was 1 SD above average. This finding adds to the data on activation of positive emotional reactivity and may aid our understanding of positive emotional eating theory in that the experience of positive emotions (increased activation and intensity) may be important factors related to positive emotional eating and BE. In addition to this finding, it was identified that increased positive emotional eating was associated with decreased BE when intensity of positive emotional reactivity was 1 SD below average. These findings are important given they add an important layer of evidence to the claim that positive emotional reactivity strengthens the relationship between positive emotional eating and BE: when intensity of positive emotions decreased, positive emotional eating had a weakened effect on BE. Thus, exaggerated, not blunted, experiences of positive emotions, notably activation and intensity of positive emotions, coupled with a tendency to eat in response to positive emotions may be important factors underlying the presentation of BE.

Finally, we examined if relationships between negative and positive emotional eating and BE would be strengthened by increased duration of negative and positive emotions. We found evidence for the

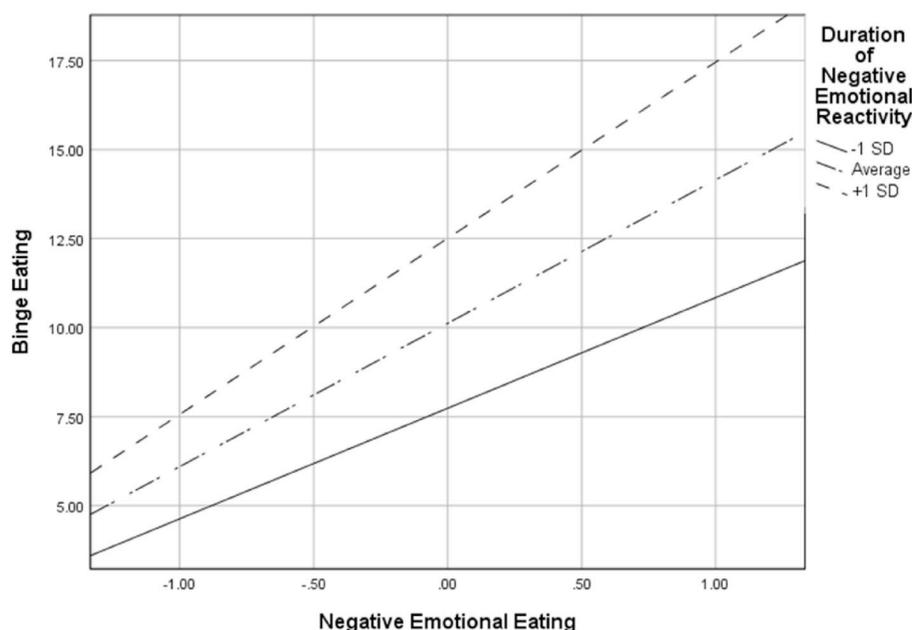


Fig. 3. Moderation of negative emotional eating and binge eating by duration of negative emotional reactivity.

relationship between negative emotional eating and BE to be strengthened by increased duration of negative emotions. In addition, we also observed that increased negative emotional eating was associated with decreased binge eating when duration of negative emotions was 1 standard deviation below average. Thus, like the relationship between positive emotional eating and positive emotional reactivity, it appears that exaggerated, not blunted, duration of negative emotions coupled with a tendency to eat in response to negative emotions are important factors underlying the presentation of BE. Interestingly, the absence of effects of activation and intensity of negative emotions to relate to negative emotional eating and BE are in line with research that holds that duration of negative emotions play a specific role in BE severity (Lingswiler et al., 1987). These data on duration of negative emotions also overlap with a meta-analytic review of negative emotional antecedents and BE such that negative emotions appear to occur before and after the eating episode (Haedt-Matt & Keel, 2011; Lynch, Everingham, Dubitzky, Hartman, & Kasser, 2000). Moving to positive emotional eating, we failed to find evidence for duration of positive emotions to interact with positive emotional eating to impact BE.

An important distinction between duration and ease of activation/intensity of emotional reactivity can be made in terms of how these emotional reactive states temporally progress. In other words, activation and intensity are quick, discrete experiences of emotion while duration is a slower, less discrete experience of emotion (Davidson, 1998; Rothbart & Derryberry, 1981). Indeed, such experiences of emotion are dichotomized in the literature along lines of emotion and mood. Emotions tends to be conceptualized as fast-paced, discrete experiences (e.g., experience of sadness, happiness, anger) while mood tends to be conceptualized as slow-paced, more diffuse and less distinct experiences (e.g., depression, euphoria) (Gross, 1998). Along these lines, activation and intensity of positive emotional reactivity may relate more to emotions while duration of positive emotional reactivity may relate more to mood.

These data on emotional reactivity introduce the idea that the relationship between emotional eating and BE may be valence-specific: more discrete, fast-paced experiences of emotion (e.g., ease of activation and intensity) interact with positive emotional eating to predict variance in BE while less discrete, slower-paced experiences of emotion (e.g., duration) interact with negative emotional eating to predict variance in BE. These data may also add to scientific commentary on emotional eating instruments in which recent research has called attention to the validity of laboratory-based self-reported emotional eating instruments (Bongers & Jansen, 2016; Braden, Emley, Watford, Anderson, & Musher-Eizenman, 2019; Domoff, Meers, Koball, & Musher-Eizenman, 2014; Evers et al., 2018). Future research is needed to better distinguish if these emotional eating instruments are tapping into emotions or mood, their overlap, and subsequent implications for the assessment of eating behavior including emotional eating and BE. There are several important practical implications to consider when reviewing these findings.

First, identifying an increased tendency to experience negative emotions for long periods of time might be important to the clinician interested in targeting emotional eating among populations who engage in BE. These data paired with a meta-analysis revisiting the affect regulation model of BE (Haedt-Matt & Keel, 2011) demonstrate that duration of negative emotions may be an important factor to consider throughout the BE episode. Second, eating in response to positive emotions, when paired with an increased tendency to quickly and intensely experience these same emotions, might be important and otherwise innocuous targets for intervention in similar populations. Finally, the experience of positive emotions might be important in the context of treatment and relapse prevention of BE given such emotions may re-emerge in the absence of eating pathology. Clinicians should be adept in recognizing the impact of the full range of emotions on maladaptive eating behaviors including emotional eating and BE.

4.1. Limitations and future directions

There were several limitations and future directions to consider when interpreting results. First, the cross-sectional nature of the present study prevents attributions of causality between emotional eating, emotional reactivity, and BE. Second, the present study did not include clinical presentations of BE as found in samples of people with clinical eating disorders to control for factors related to these health conditions— but unrelated to our study questions (e.g., body image, compensatory mechanisms such as purging and excessive exercise, fear of gaining weight, etc.)— that may have impacted outcomes. Future research should explore the role of emotional eating and emotional reactivity to predict BE in these populations given this eating disorder symptom is of marked concern in relation to the development and maintenance of obesity and clinical eating disorders (Fairburn et al., 1998; Hudson et al., 2007; Raman et al., 2013). More specifically, future research should address these variables in anorexia nervosa and bulimia nervosa given BE is a transdiagnostic eating disorder symptom present across the full spectrum of eating pathology. Some research with bulimia nervosa holds that purging after BE results in meaningful reductions in negative emotions (Haedt-Matt & Keel, 2011), which may illustrate an important constraint to these findings that sampled sub-clinical levels of BE. This research could add rich information on the transdiagnostic trends of BE across the full spectrum of eating pathology. Third, the construct dietary restriction, the intentional restriction of food intake, has been shown to increase food intake among people who engage in negative emotional eating (Cardi et al., 2015; Greeno & Wing, 1994); however, among people who engage in positive emotional eating, the role of dietary restraint is less clear (Cools, Schotte, & McNally, 1992; Yeomans & Coughlan, 2009). Future research should examine the role of dietary restraint on positive emotional eating, positive emotional reactivity, and BE to demonstrate if this effect is present across the full range of emotional experiences. Fourth, the present study considered negative and positive emotional eating broadly and did not tease out specific positive emotions (e.g., discouragement, sadness, feelings of being upset; enthusiasm, excitement, happiness, etc.) that may provide more information about the interaction between negative and positive emotional reactivity and BE. Fifth, non-WEIRD sampling which includes participants who are not White, Educated, Industrialized, Rich, and Democratic (Henrich, Heine, & Norenzayan, 2010) may be of interest in future research to test the parameters of these exploratory findings. In the same vein, sex differences may be of interest in future research. For example, sex differences have emerged in mood induction studies across negative and positive emotional eating in that men show higher motivation to eat in response to positive emotions (Nolan et al., 2010) and reported higher motivation to eat in positive mood inductions compared to women (Macht et al., 2002). While our demographics did account for race/ethnicity and education of participants, other factors such as sampling from diverse age groups, non-industrialized regions, religious and spiritual background, and sexuality were not captured and may constrain the generality of findings of the present study (Simonds, Shoda, & Lindsay, 2017). It is important, however, to reflect on sample size limitations when considering several predictors and covariates in models tested and how inclusion of such variables could be related to overfitting of models tested and lack of generalizability. Thus, it is imperative that future research parse out these effects with use of large, diverse sample sizes. Finally, future research should consider the use of additional methods and novel measures of emotional eating and BE – including use of ecological momentary assessment methods– to replicate these findings given recent concerns regarding the validity of laboratory-based self-reported emotional eating instruments and methodological concerns related to assessment of BE in the real-world (Bongers & Jansen, 2016; Braden et al., 2019; Domoff et al., 2014; Evers et al., 2018; Kockler, Santangelo, & Ebner-Priemer, 2018).

5. Conclusions

Taken together, the results of the present study provide initial evidence for the interaction between negative and positive emotional eating and aspects of emotional reactivity to predict BE. Here, we show that people who engage in increased positive emotional eating, and who also feel increased ease of activation and intensity of positive emotions, were predicted to have increased BE. We also show that people who engage in increased negative emotional eating, coupled with increased duration of negative emotions, were predicted to have increased BE. These data provide initial evidence for valence-specific effects across negative and positive emotional eating in relation to emotional reactivity, yielding important effects on BE. Because targeting BE results in promising eating disorder (Carter et al., 2003; Wonderlich, de Zwann, Mitchell, Peterson, & Crow, 2003) and obesity (Lillis, Hayes, & Levin, 2011) treatment outcomes, these findings may be of use to clinicians and researchers alike. Screening for negative and positive emotional eating and emotional reactivity may be of use to eating- and weight-related treatments of BE.

Declaration of competing interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.appet.2020.104688>.

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