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The impact of neural responses to food cues following stress on trajectories of negative and positive affect and binge eating in daily life



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ABSTRACT

Stress and affect have been implicated in the maintenance of binge eating for women with symptoms of bulimia nervosa (BN). Neuroimaging and ecological momentary assessment (EMA) have separately examined how these variables may contribute to eating disorder behavior. Though both methodologies have their own strengths, it's unclear how either methodology might inform the other. This study examined the impact of individual differences in neural reactivity to food cues following acute stress on the trajectories of positive affect (PA) and negative affect (NA) surrounding binge eating. Women (n = 16) with BN symptoms viewed palatable food cues before and after a stress induction in the scanner. For two weeks, participants responded to prompts assessing affect and binge episodes several times a day. EMA data revealed NA increased and PA decreased before binge episodes in the natural environment. Additionally, NA decreased while PA increased following binge episodes. Changes in activation in the ACC, amygdala, and the vmPFC significantly moderated the relationship of affect to binge eating. However, lateral differences of each brain region uniquely moderator the trajectory of PA, NA, or both to binge eating. Specifically, those with less change in BOLD response reported significantly increasing NA and decreasing PA prior to binges, while women with greater decreases reported no change in affect. Following binge eating, individuals with decreased change in BOLD response reported decreasing NA and increasing PA. This may suggest individual differences in neural response to food cues under stress appear to underlie affect driven theory on the antecedents to binge eating.

1. Introduction

Symptoms of bulimia nervosa (BN) include binge eating, compensatory behaviors, with shape and weight concerns (APA, 2013). Binge eating is defined as eating an objectively large amount of food in a short period of time, with associated loss of control over eating. The affect regulation model of BN symptoms suggests the cycle of binge eating is maintained via negative affect (NA) reduction (Haedt-Matt and Keel, 2011a, 2011b; Pearson et al., 2015). The development and maintenance of BN has both neurobiological and psychosocial bases (Berner and Marsh, 2014; Collins et al., 2017; Kaye et al., 2013). The neural correlates of affect regulation in BN have not been well characterized. Thus, the current study integrates neuroimaging data with daily momentary assessment of behavior in the natural environment to better understand how individual differences in brain function may be integrated into affect regulation theories of BN symptoms.

Binge eating is hypothesized to distract one from an aversive stimulus (e.g., NA) by focusing on a concrete stimulus (food; Heatherton and Baumeister, 1991). Data supports the hypothesis that increasing NA, with simultaneous decreases in positive affect (PA), precede binge eating in women with BN (Engel et al., 2016). Data collected via ecological momentary assessment (EMA) are particularly compelling, as this data occurs in the participant's natural environment in "real time." Researchers using EMA have shown that NA increases and PA decreases before binges, while NA decreases and PA increases following binge (Berg et al., 2013; Smyth et al., 2007).

1.2. Neural modulation of affect

The amygdala and the ventromedial prefrontal cortex (vmPFC) play important roles in modulating emotion (Ochsner et al., 2002; Schaefer

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^{1.1.} Affect and binge eating

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et al., 2002). The amygdala is implicated in a range of functions related to affective reactions, including the processing of emotionally salient memories (LeDoux, 2000; Phelps, 2004). Increased amygdalar activity is associated with heightened response to either emotionally aversive or emotionally appetitive stimuli, and high levels of anxiety and NA (Milad et al., 2006; Rauch et al., 2006).

The vmPFC is associated with modulating amygdalar activity, and thus, is important in understanding affect (Phan et al., 2002; Phillips et al., 2003). The vmPFC appears to process information relevant to goal-related outcomes, which can help to modulate impulsive, emotional behavior (Barbey et al., 2009). Some suggest the vmPFC regulates affect via top-down inhibition of brain regions involved in processing emotion. For example, fMRI studies have shown an inverse relationship between vmPFC activity and activation in the amygdala during negative emotion (Motzkin et al., 2015; Quirk and Beer, 2006).

Urry et al. (2006) examined PFC and the amygdala function in relation to stress and NA. Participants viewed unpleasant images and were then asked to modulate their affective response. This was followed by a week of repeated salivary cortisol collection. The results indicated that lower amygdala response after experiencing NA was associated with higher vmPFC signal. Individuals with a greater negative association between the response of the vmPFC and amygdala while modifying NA exhibited a steeper, more normative, decline in cortisol throughout the day. These findings support the idea that vmPFC regulates emotion regulation via top-down inhibitory processes of the amygdala, which mediates HPA disturbances associated with stress.

1.3. Integration of fMRI and EMA

Integrating EMA and fMRI provides an avenue to understand how neurobiological dysregulation is associated with behavior in daily life (Forbes et al., 2009; Wilson et al., 2014). Neuroimaging studies in eating disorders typically compare brain structure or function across clinical groups, or examined functional responses within groups. These studies yielded important findings regarding brain function and psychopathology. However, like all laboratory studies, they do not provide ecologically valid information regarding how differences in brain function affect real-world behavior. On the other hand, EMA provides information on how within person changes in emotions influence behavior in the natural environment (Wonderlich et al., 2014). Thus, integration of these methodologies provides an ideal mechanism to examine how differences in brain function may be incorporated into psychosocially based affect regulation models of the disorder.

1.4. Current study

One recent study examined how functional responses to food cues (collected using fMRI) following acute stress influenced trajectories of stress to binge eating (collected using EMA; Fischer et al., 2017). During an fMRI scan, women with viewed neutral and food cues, completed a stress induction, and again viewed food cues. Individual differences in BOLD response in the precuneus, ACC, and dlPFC moderated the relation between stress to binge eating in EMA (Fischer et al., 2017). Specifically, decreased BOLD response to food cues from pre to post stress induction was associated with greater increases in stress before binge eating in the natural environment.

Although stress is linked to NA, it has distinct physiological and neurobiological correlates. Acute stress involves coordination of both the limbic system and HPA axis (Born et al., 2010). Affect appears to be modulated by vmPFC and amygdalar activity, as described by Urry et al. (2006). Individuals may experience a variety of NA states as distinct from acute stress processing. Additionally, there may be temporal differences in the processing of stress and affect relevant to binge eating. For example, Goldschmidt et al. (2014) demonstrated that NA mediates the relation between acute stress and binge eating in women with BN.

Thus, one limitation of Fischer et al. (2017) study was that it did not examine how NA and PA impact subsequent binge eating. Given our understanding that stressful events followed by subsequent change in affect increase the likelihood of a binge eating episode (Goldschmidt et al., 2014) this study expands upon the previous work to address this gap. Using the same dataset as Fischer et al., 2017, we examined how individual differences in response to visual food cues in the vmPFC and amygdala pre to post stress induction may impact the relation between the trajectory of NA and PA before and after a binge, while controlling for momentary changes in stress. We hypothesized the following: (1) NA, measured via EMA, would increase prior to and decrease following binge eating. (2) PA, measured via EMA, would decrease prior to and increase following binge eating. (3) Changes in BOLD response to food cues in a priori regions of interest (ROIs) from pre to post acute stress would moderate the trajectories of affect preceding and following binge eating (vmPFC; amygdala; ACC). Specifically, based on previous findings, we hypothesize that greater decreases in bold activity when looking at food cues following a stress induction would be associated with a greater, and more rapid, increase of negative affect and a greater and more rapid decrease in positive affect prior to binge eating. Following a binge eating episode, greater decrease in BOLD activity from pre to post stress induction when viewing food cues, would be associated with a greater, and more rapid, decrease in negative affect and a greater and more rapid increase in positive affect. We also explored the potential moderating effect of the ACC on these trajectories.

2. Methods

2.1. Inclusion and exclusion criteria

Participant recruitment and participation has previously been described (Fischer et al., 2017; Wonderlich et al., 2017; see Fig. 1.) Participants were recruited from the community. The goal of participant recruitment for the parent study (Fischer et al., 2017) was to recruit women with a range of BN symptoms, in order to examine the relationship of individual differences in the neural correlates of stress and food cue exposure to a range of symptom severity. Inclusion criteria were: \geq one episode of binge eating and compensatory behavior (self-induced vomiting, laxative use, fasting, excessive exercise) in the past month, female sex, age 18-45 years, able to use a 'smartphone', and BMI between 18.5 and $29.9\,\mathrm{kg/m^2}$. Exclusion criteria were: substance use disorder (SUD) within past 12 months, psychotic disorder, left-handedness, and contraindications for scanning (e.g., metal implants).

2.2. Participants

Participants were 16 right-handed women with symptoms of BN and normal or corrected-to-normal vision. Participants ranged in age from 18 to 40 years (mean = 22.85, sd = 5.42). BMI ranged from 20.0 to 29.4 with a mean of 24.47 (sd = 3.25). As per self-report, 71.4% described themselves as Caucasian, 14.3% as Hispanic-American, and 14.3% as Asian-American. Only 2 participants reported being in treatment at the time of the study, and 50% report any previous treatment for disordered eating. Two participants reported taking psychiatric medications (SSRIs). During the baseline assessment, participants reported a mean number of 7.1 objective binge episodes in the past month (range = 1-15; sd = 3.8), a mean number of 4.4 purging episodes (vomiting, laxative use, or diuretic use) (range = 0-16, sd = 5.0), a mean number of 6.6 excessive exercise episodes (range = 0-25, sd = 6.1), and a mean of 1.0 days of fasting (range = 0-4, sd = 1.5). Of the 16 participants, 12 met DSM-5 criteria for BN, while the remainder were diagnosed with Otherwise Specified Feeding and Eating Disorder (OSFED).

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