



# Impact of the neural correlates of stress and cue reactivity on stress related binge eating in the natural environment



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## ABSTRACT

Women with symptoms of bulimia nervosa (BN) exhibit decreased response to visual food cues in several limbic and frontal regions compared to controls. Stress causes decreased blood oxygenation level dependent (BOLD) response in these regions in non-clinical samples; there is a lack of data on this topic in BN. This study examined the impact of individual differences in neural reactivity to palatable food cues following acute stress on stress-binge trajectories in everyday life. 16 women with BN symptoms viewed palatable food cues prior to and immediately following an acute stress induction in the scanner. Participants then responded to a series of prompts assessing daily ratings of stress and binge episodes for a period of two weeks. Decreased BOLD signal was observed in response to food cues pre to post stress in the anterior cingulate cortex (ACC), amygdala, and ventromedial prefrontal cortex (vmPFC). Ecological momentary assessment data collection demonstrated that stress increased prior to binge episodes in the natural environment, and decreased following. Changes in activation in the ACC, precuneus, and dorsolateral prefrontal cortex (dlPFC) significantly moderated the relationship of stress to binge eating in daily life, such that women who exhibited decreased response reported significantly increasing stress prior to binges, while women who did not exhibit decreases reported no significant change in stress prior to binges. Individual differences in neural response to food cues under stress appear to underlie distinct antecedents to binge eating.

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## 1. Introduction

Bulimia nervosa (BN) is characterized by binge eating (i.e., eating an objectively large amount of food with a sense of loss of control), compensatory behaviors, and overvaluation of shape and weight (American Psychiatric Association, 2013). Acute stress, defined as an experience in which the demands incurred by deviation from one's normal state exceed one's coping resources, is an important precipitant of binge episodes in BN (Lazarus, 1993; Smyth et al., 2007). Emotion regulation models of BN posit that acute ego-threatening stress leads to aversive cognitions about the self.

Individuals with BN distract from these cognitions via focus on a more concrete stimuli, food (Heatherton and Baumeister, 1991; Pearson et al., 2015). Consistent with this theory, acute stress is linked to later negative affect, binge eating and purging, and unplanned ingestion of high fat/high sugar foods (Smyth et al., 2007; Freeman and Gil, 2004; O Connor et al., 2008; Goldschmidt et al., 2014; Crowther et al., 2001; Roemmich et al., 2011). Acute stress may also promote disinhibition of cognitive attempts at dietary restraint in BN. For example, individuals high (versus low) in restraint exhibit greater loss of control over eating following acute stress (Freeman and Gil, 2004; Tanofsky-Kraff et al., 2000). The goals of this pilot study were to conduct a within-person examination of the neural correlates of food cue processing under acute stress conditions, and investigate how these functions impact trajectories of stress to binge eating in daily life.

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### 1.1. The impact of acute ego-threatening stress on inhibition and reward processing

Acute stress responses are initiated by cognitive evaluations of the stressor, and the release of glucocorticoids from the hypothalamic pituitary adrenal (HPA) axis (Dickerson and Kemeny, 2004; Ulrich-Lai and Herman, 2009). These processes provoke reduced response in prefrontal regions and regions involved in self-appraisal, such as areas of the pre-frontal cortex (PFC) and precuneus (Arnsten, 2015; Soares et al., 2013). Functions of the PFC that are impacted by acute stress are inhibition of pre-potent (yet potentially maladaptive) responses and attentional control (Arnsten, 2015). The release of glucocorticoids and dampening of frontal regions also promotes increased dopamine projection into limbic regions (Dedovic et al., 2009; Nieuwenhuizen and Rutters, 2008). In sum, acute stress facilitates both inhibited action in frontal regions and increased action in regions associated with reward and habit (Arnsten, 2015). This facilitates a shift from goal-oriented, planned behavior into habit-based behavior (Arnsten, 2015; Ossewaarde et al., 2011; Porcelli et al., 2012; Schwabe and Wolf, 2009; Schwabe et al., 2010; Bogdan and Pizzagalli, 2006).

This sequence of events may facilitate binge eating. Binge eating is considered by several researchers to be a habit based behavior (Voon et al., 2015). The act of binge eating is thought to be influenced by associations with previous reward, instead of future behavioral goals (Voon et al., 2015). Neurobiological conceptualizations of BN posit that dysregulation in brain regions subsuming inhibitory control and reward processing contribute to the development and or maintenance of binge eating (Berner and Marsh, 2014; Kaye et al., 2013). Evidence suggests that women with BN differ from healthy controls in limbic response to visual food cue (Joos et al., 2011; Uher et al., 2004; Radeloff et al., 2012; Brooks et al., 2011). Within subjects studies find that women with BN exhibit increased BOLD response to food cues compared to neutral cues. Some studies indicate that women with BN exhibit increased response to visual food cues compared to non-eating disordered controls, while others show that non-eating disordered controls exhibit greater BOLD response to this stimuli (Joos et al., 2011; Brooks et al., 2011; Schienle et al., 2009; Kim et al., 2012). None of these studies have examined response to food cues in BN following acute stress, and many have small sample sizes. Thus, the effect of stress on the neurobiological correlates of visual food cue processing in women with BN symptoms is unclear.

Studies utilizing behavioral tasks also indicate that women with BN have impaired inhibitory control and dampened response in regions associated with regulation of eating behavior, such as the ACC (Berner and Marsh, 2014; Marsh et al., 2009). Thus, inhibitory control deficits related to activity within these regions may influence binge eating vulnerability. As acute stress may facilitate shifts into habit based behavior, and the presence of food cues may trigger lapses in dietary restraint, it is important to examine how the neural correlates of acute stress on food cues in individuals who binge eat.

### 1.2. Integration of fMRI and EMA to study binge eating

The integration of ecological momentary assessment (EMA) with neuroimaging enhances our understanding of how neurobiological dysregulation impacts the association between stress and behavior outside of the lab (Wilson et al., 2014; Forbes et al., 2009; Morgane et al., 2005). During EMA data collection, participants use smartphones or handheld devices to receive prompts for information throughout the day. This repeated assessment of mood and cognition throughout the day reduces recall bias and allows researchers to examine changes in psychological processes in 'real

time' prior to behavior. EMA allows researchers to examine temporal shifts in emotions prior and following specific behaviors in the natural environment. The integration of individual difference data gathered via fMRI allows us to examine how dampened activation in prefrontal regions may limit one's ability to inhibit binge responses to stress in daily life. Thus, the goal of the current study was to understand how individual differences in the impact of acute stress on the neural processing of food cues relates to the naturalistic association between stress and binge eating in BN.

### 1.3. Current study

We first used fMRI to examine blood oxygen level dependent (BOLD) response in women with BN symptoms to visual food cues prior to and immediately following an acute stress induction. Next, we used EMA to examine the naturalistic and momentary relationship of stress to binge eating over two weeks. Changes in activation in *a priori* regions of interest during the fMRI task were examined as moderators of the trajectory of stress around binge eating, derived from EMA data. The following hypotheses were tested: 1. BOLD response to food cues in the following regions of interest (vmPFC, ACC, amygdala, and precuneus) would decrease following stress induction. We also examined changes in the dorsolateral PFC. The dlPFC is involved in impulse control and planning (Unterrainer and Owen, 2006). Thus, we did not anticipate that this region would exhibit changes in BOLD activation during food cue presentation following stress. However, given that this region may be impacted by acute stress, and is related to pre-potent response inhibition, we wished to examine if individual responses in this region moderated the relationship of stress to binge eating. 2. Stress, measured via EMA, would increase prior to and decrease following binge eating. 3. Changes in activation in *a priori* ROIs from pre- to post-stress in the fMRI environment would moderate the trajectory of stress prior to binge eating as captured via EMA. This aim was exploratory and no directional hypotheses were made because of a lack of previous literature on this topic.

## 2. Methods

### 2.1. Inclusion and exclusion criteria

Inclusion criteria were:  $\geq$  one episode of binge eating and compensatory behavior (self-induced vomiting, laxative use, fasting for 24 h, excessive exercise) in the past month, female sex, age 18–45 years, able to access and use a 'smartphone', and BMI between 18.5 and 29.9 kg/m<sup>2</sup>. Exclusion criteria were: active substance use disorder (SUD) within past 12 months, psychotic disorder, left-handedness, and contraindications for scanning (e.g., metal implants). Active SUDs were included as an exclusion criteria because of potential alterations in regions of the brain associated with reward processing due to substance use.

### 2.2. Recruitment

Participants were recruited via flyers from a large, Mid-Atlantic university, an outpatient eating disorder clinic, and internet advertisements in the greater Washington, D.C. area. The goal of recruitment was to obtain a sample with a range of frequency of binge eating and compensatory behaviors. A total of 279 people responded to study advertisements, with 211 completing diagnostic telephone screens. Subsequently, 36 participants were invited to the laboratory to complete structured clinical interviews, 19 of which were ruled out (three with active SUD; one with a BMI below the minimum; nine with too infrequent episodes of binge eating and/or compensatory behaviors; two without significant

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