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Binge eating disorder and obesity: Preliminary evidence for distinct cardiovascular and psychological phenotypes

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HIGHLIGHTS

- Obese women with binge eating disorder (BED) were compared to obese and normal weight non-BED women.
- Obese BED group reported greater depression, perceived stress, and eating psychopathology.
- Obese BED showed greater blood pressure, negative affect, and anxiety irrespective of stress task.
- Stress-induced change in hunger was associated with cardiovascular measures in obese BED only.
- Results indicate that BED and obesity are distinct in terms of psychological and physiological stress-related factors

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ABSTRACT

This study investigated cardiovascular functioning, mood, and eating-related psychological factors at rest and in response to mental stress in three groups of women: 1) Obese women with binge eating disorder (BED; n = 9); 2) obese non-BED women (n = 15); and 3) normal weight (NW) non-BED women (n = 15). Compared to both obese and NW non-BED women, obese women with BED showed heightened overall blood pressure and reported greater depression symptoms, perceived stress, and eating-related psychopathology. Additionally, obese women with BED reported greater overall negative affect and state anxiety compared to obese non-BED women. The heart rate response to stress was blunted in the obese BED group compared to the other groups, but this effect was no longer significant after controlling for baseline differences in depression. Correlational analyses revealed a positive association between stress-induced changes in hunger and cardiovascular measures only in obese women with BED. Longitudinal studies are needed to determine if stress dysregulation and stress-induced increases in hunger contribute to the onset and/or maintenance of BED. In particular, studies utilizing an additional NW BED control group are warranted in order to further examine the impact of BED above and beyond the impact of obesity on psychophysiological functioning and to inform the growing literature regarding stress-related factors that distinguish the BED and obesity phenotypes.

1. Introduction

Over one-third of adults in the United States are obese [34]. Obesity and obesity-related health conditions such as heart disease, stroke, type 2 diabetes, and certain cancers [33] constitute a significant public health problem costing the United States an estimated $147 billion annually [10]. Recent findings project that cumulative lifetime exposure to excess weight and related comorbid conditions will increase due to earlier onset of obesity [52]. Thus, there is a clear need for a greater understanding of the etiology of obesity and its correlates in order to inform prevention and treatment efforts.

One such correlate of obesity is binge eating. Binge eating involves the consumption of an amount of food that is definitely larger than most people would consume under like circumstances over a relatively brief (e.g., within any 2-hour) discrete period of time. When binge eating occurs with sufficient regularity and is accompanied by various hallmark behavioral and psychological symptoms, an individual may be diagnosed with binge-eating disorder (BED). BED is characterized by recurrent (at least 1 day per week for 3 months) binge-eating episodes that occur in the absence of regular compensatory behaviors such as purging. These binge episodes often involve eating more rapidly than normal and until uncomfortably full and they are associated with feeling depressed, disgusted, or guilty after overeating [1]. In the U.S., approximately 3.5% of women and 2.0% of men suffer from BED in their lifetimes [24]. BED and obesity are highly comorbid, as individuals with BED are more likely to be obese [37] and the prevalence of BED is nearly 2-fold

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greater in obese compared to non-obese adults. Moreover, BED becomes more prevalent with greater obesity severity [7,47] and the prevalence of BED is higher among individuals who are seeking treatment for obesity (20–30%) compared to those in the general population [24,49,50].

BED is associated with considerable psychological and physiological dysfunction that distinguishes it from obesity [4,20,35,40,50,55,56]; it aggregates in families independently of obesity and is caused in part by familial factors distinct from those for obesity [23]. Yet, additional studies are needed to further differentiate the BED phenotype from the obesity phenotype in order to inform treatment development [50,55]. The present study addressed this need by examining cardiovascular and psychological stress reactivity in individuals with and without BED across the weight spectrum.

Stress has been implicated in the etiology of BED [31,39,45,46] as well as in the onset and maintenance of obesity (for review, see [48]). The release of glucocorticoids following stress increases appetite as well as the preference for comfort foods, a combination of factors that nurture the development of binge eating and obesity (for reviews, see [29,48]). Binge eating is associated with elevated levels of perceived stress [39,54], and perceived stress is a precursor to binge eating in non-clinical populations [5,13,21,38]. Furthermore, following a laboratory stressor, obese women with BED show increased hunger, desire to binge eat, and rates of consumption of highly palatable foods compared to obese non-BED women [17,42].

Studies comparing the physiological stress response in obese BED and non-BED individuals have been scarce and results have been mixed, including greater reductions in parasympathetic activity [14], blunted autonomic responses to stress and impaired stress recovery [30], as well as heightened [17], blunted [41], or no differences [43] in hypothalamic–pituitary–adrenal (HPA) axis responses in obese BED groups. One potential reason for the discrepancy in the literature may be the lack of consistency in the comparison group(s). The vast majority of prior studies have compared obese BED to obese non-BED control groups, reporting significant differences in cardiovascular, HPA-axis, and psychological factors and thus evidence that it is the disorder, and not obesity, contributing to the dysregulation seen in obese BED individuals [4,14,17,30,40,43,56]. In the absence of a normal weight (NW) control group, however, the relative importance of these differences cannot be determined. That is to say, the impact of BED above and beyond the impact of obesity on physiological and psychological dysfunction remains unclear.

Lo Sauro and colleagues [27] conducted an extensive review of the literature on stress in eating disorders; based on their qualitative analysis, HPA-axis dysregulation observed in BED and obese individuals was attributed mainly to excess weight. This conclusion was based on data from studies assessing either BED participants only, obese participants only, or comparing BED groups to obese non-BED groups. More recently, however, Rosenberg et al. [41] found that obese BED individuals showed blunted cortisol responses to stress as well as a positive association between the cortisol stress response and stress-induced increases in the desire to binge eat and sweet cravings compared to both obese and NW non-BED groups, suggesting that stress dysregulation in BED may be a function of the disorder itself and not strictly obesity. Given that these findings pertain only to HPA-axis functioning, the current study investigated the second major component of the stress axis, the sympathetic nervous system. The goal of the present study, therefore, was to expand upon these recent findings by investigating cardiovascular and psychological stress responses in three groups of women: obese BED, obese non-BED, and NW non-BED. The primary hypotheses were that obese women with BED, as a group, would show greater cardiovascular and psychological stress dysregulation and eating-related psychopathology as well as greater overall perceived stress and depressive symptoms compared to both groups of obese and NW non-BED women. Furthermore, given the association between stress, hunger, and food consumption in BED [17,42], our secondary hypothesis was that obese women with BED would report greater increases in hunger following stress than both obese and NW non-BED women. For both our primary and secondary hypotheses, we expected that obese and NW non-BED women would not differ significantly from each other.

2. Materials & methods

2.1. Participants

The 39 women (19–50 years of age) who comprise this report were recruited via newspaper and posted advertisements online and in local businesses targeting women who were overweight and not taking blood pressure, stimulant, or psychoactive medications. These medications were exclusionary due to their influence on cardiovascular functioning and other variables of interest to the current study (e.g. hunger, negative affect, and anxiety). Importantly, no advertisements specifically targeted women with BED or any eating disorder. Our recruitment strategies ensured that participants remained blind to the study's main variable of interest, BED diagnostic status, in order to diminish demand characteristics. Participants were told that the purpose of the study was to investigate the influence of the menstrual cycle on responses to mental stress in overweight and normal weight women. A small percentage of advertisements eliminated the term "overweight" in order to obtain our normal weight (NW) sample.

The final sample of participants was composed of three groups: 1) Obese women with BED (n = 9); 2) obese non-BED women (n = 15); and 3) NW non-BED women (n = 15). All women in the BED group met full DSM-V criteria for BED and women in the non-BED groups had no history of any eating disorders. Participants in the two obese groups had a body mass index (BMI) between 29 and 47 and the NW group had a BMI between 19 and 24. The protocol was approved by the Institutional Review Board of Rhodes College and all participants provided informed, written consent. Participants received $45 compensation.

2.2. Preliminary screening protocol

Women responding to our advertisements received a link via email directing them to a set of online screening questions aimed at excluding prospective participants who were pregnant or breastfeeding, postmenopausal, taking prescription medication (excluding oral contraceptives, see Table 1), had a cardiovascular disorder, or were regular smokers. Women were also excluded if they reported seeking treatment for weight or eating issues. This exclusionary criterion was intended to

Table 1
Mean (±SEM) demographic and baseline measures as a function of BED and obesity status.

	NW non-BED n = 15	Obese non-BED n = 15	Obese BED n = 9
Body mass index [a]	21.5 (±0.5)	35.3 (±1.3)	37.9 (±1.6)
Current major depressive or anxiety disorder (%) [b]	2 (13%)	1 (7%)	8 (89%)
Age	28.8 (±1.7)	26.8 (±1.2)	33.3 (±3.1)
Minority race (%)	8 (53%)	9 (60%)	7 (78%)
Oral contraceptive use (%)[c]	5 (33%)	1 (2%)	0 (0%)
Beck depression inventory [b]	11.3 (±2.6)	5.3 (±1.2)	29.8 (±3.9)
Perceived stress scale [d]	17.8 (±2.5)	12.7 (±1.1)	24.9 (±2.4)
Restraint [b]	0.6 (±0.2)	0.3 (±0.1)	2.1 (±0.3)
Eating concern [b]	0.1 (±0.03)	0.1 (±0.04)	3.6 (±0.5)
Shape concern [b]	1.0 (±0.2)	1.5 (±0.2)	4.8 (±0.5)
Weight concern [b,e]	0.4 (±0.1)	1.2 (±0.2)	4.4 (±0.4)
Binge eating scale [b]	3.9 (±0.6)	4.6 (±1.3)	29.5 (±3.9)

[a] NW non-BED < obese BED and obese non-BED, $p < .001$.
[b] Obese BED > obese non-BED and NW non-BED, $p < .001$.
[c] NW non-BED > obese BED and obese non-BED, $p < .05$.
[d] Obese BED > obese non-BED, $p \leq .01$.
[e] Obese non-BED > NW non-BED, $p < .05$.