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Examining the mediating roles of binge eating and emotional eating in the relationships between stress and metabolic abnormalities

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Abstract To test whether binge eating and emotional eating mediate the relationships between self-reported stress, morning cortisol and the homeostatic model of insulin resistance and waist circumference. We also explored the moderators of gender and age. Data were from 249 adults (mean BMI = 26.9 ± 5.1 kg/m^2; mean age = 28.3 ± 8.3 years; 54.2 % male; 69.5 % white) recruited from the community who were enrolled in a cross-sectional study. Participants completed a comprehensive assessment panel of psychological and physiological assessments including a morning blood draw for plasma cortisol. We found negative relationships between stress and morning cortisol (r = −0.15 to −0.21; $p < 0.05$), and cortisol and the homeostatic model of insulin resistance and waist circumference (r = −0.16, −0.25, respectively; $p < 0.05$). There was not statistical support for binge eating or emotional eating as mediators and no support for moderated mediation for either gender or age; however, gender moderated several paths in the model. These include the paths between perceived stress and emotional eating (B = 0.009, $p < 0.001$), perceived stress and binge eating (B = 0.01, $p = 0.003$), and binge eating and increased HOMA-IR (B = 0.149, $p = 0.018$), which were higher among females. Among women, perceived stress may be an important target to decrease binge and emotional eating. It remains to be determined what physiological and psychological mechanisms underlie the relationships between stress and metabolic abnormalities.

Keywords Stress · Binge eating · Emotional eating · Insulin resistance · Abdominal obesity

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Introduction

Obesity continues to be a leading health issue, impacting 34.9 % of adults in the US (Ogden et al., 2014). This rate is closely tied to the high prevalence of metabolic abnormalities. For example, the prevalence of abdominal obesity is 54.2 % (Ford et al., 2014) and impaired fasting glucose is 26.6 % among adults (Ioannou et al., 2007). These abnormalities are major predictors of coronary heart disease, stroke, and type 2 diabetes (Kannel et al., 1991; Nathan et al., 2007; Rexrode et al., 1998; Santaguida et al., 2005).

Several interventions for prevention and treatment have been developed. These interventions are multifaceted and heterogeneous though many result in only modest and short-term changes in obesity-related metabolic abnormalities (Galani & Schneider, 2007; Lemmens et al., 2008; Seo & Sa, 2008). A more nuanced understanding of the mechanisms related to metabolic abnormalities is necessary to help us amplify, optimize, and target interventions to relevant processes.

Stress and coping

One such relationship in which further clarification is needed is that between stress, coping, and metabolic abnormalities. Stress is a complex and multidimensional concept referring to a real or perceived disruption in homeostasis (Chrousos & Gold, 1992). While there is suggestion that stress increases the risk for obesity-related metabolic abnormalities including insulin resistance and abdominal obesity (Räikkönen et al., 1996, 2007), the mechanisms underlying this process are unclear. There are several potential mechanisms that may contribute to this relationship including decreased physical activity, changes in eating patterns and behaviors, differences in food reward, motivation, and food cravings, and changes in stress-related hormones (Sinha & Jastreboff, 2013). However, the interplay and relationships between these factors have not been well explored, particularly in regards to different types of stress, stress-related hormones, and eating phenotypes such as binge eating and emotional eating.

There is theoretical and empirical support for the thesis that binge eating and emotional eating may be coping mechanisms that mediate the relationship between stress and metabolic abnormalities. Yet, few studies have examined the collective relationships among these variables or the relationships with different types of stress.

Binge eating is a behavior characterized by consumption of an unambiguously large amount of food in a discrete period of time, and a feeling a loss of control over eating (American Psychiatric Association, 2013). Binge eating is common among adults: 5–11 % of adults in wider community samples reporting binge eating (Abraham et al., 2014; Mitchison et al., 2014; Striegel et al., 2012).

Emotional eating is eating that occurs as a response to a range of negative emotions (i.e., stress, anxiety, anger, depression, and loneliness) (Arnow et al., 1995). Although emotional eating is positively associated with binge eating, there is a subgroup of individuals who are emotional eaters but not binge eaters (Ricca et al., 2009). This means that these individuals eat in response to negative affect but may not overeat or feel a loss of control (Linderman & Stark, 2001).

Binge eating and emotional eating may mediate associations between stress and metabolic abnormalities. Eating as a coping mechanism in response to stress is frequently reported among individuals across the weight spectrum, though there is wide variability between studies with estimates of individuals who eat more when stressed ranging from 4 to 55 % (Macht, 2008; Weinstein et al., 1997). This wide variability is not surprising given that the concept of stress-induced eating is not well defined and multiple measures and paradigms of stress have been used when examining these relationships. Examining specific eating phenotypes (i.e., binge eating and emotional eating), will allow for a deeper understanding of these processes.

Cortisol dysfunction has been linked to both binge eating and emotional eating, though there is a paucity of literature examining these relationships and study results have been inconsistent (Lo Sauro et al., 2008). While some researchers have found that females who are obese and have a binge eating disorder have higher basal cortisol compared to females who are obese without binge eating disorder (Gluck et al., 2004), others have found no difference in cortisol levels between women who are obese with and without binge eating disorder (Coutinho et al., 2007). There is also indication that high cortisol reactivity is positively associated with stress-induced eating (Epel et al., 2001).

There is evidence that the characteristics of binge eating are important determinants of metabolic abnormalities including abdominal obesity and hyperglycemia; however, the relationship between emotional eating and metabolic abnormalities is unclear. Evidence suggests that individuals who have binge eating disorder are at increased risk for developing metabolic abnormalities (Blomquist et al., 2012; Hudson et al., 2010; Roehrig et al., 2009). While some researchers have found that emotional eating is associated with increased weight (Geliebter & Aversa, 2003), others have demonstrated no association (Masheb & Grilo, 2006; Nguyen-Rodriguez et al., 2008).

Along with the above evidence, the Transactional Model of Stress and Coping and Selye's Theory of Stress were used as a framework in this study (Fig. 1). In brief, according to Lazarus and Folkman's Transactional Model of Stress and Coping, if an individual appraises an event as stressful, coping ensues (Lazarus, 1966; Lazarus & Folkman, 1984). Individuals may cope or attempt to adapt to a stressful situation by binge eating or emotional eating. Individuals may use eating as a form of emotion-based coping to reduce the negative emotional responses associated with stress. Selye posited that stress results in physiological responses to prepare the body to cope with stress, including activation of the hypothalamic-pituitary-adrenocortical (HPA) axis (Selye, 1956). Activation of the HPA axis results in the secretion of cortisol, a steroid hormone that regulates eating behaviors and choices (Pacák & Palkovits, 2001). In certain individuals the effects of cortisol result in increased caloric intake, particularly of carbohydrates and fats (Duong et al., 2012; Vicennati et al., 2011; Zellner et al., 2006), which may be classified as binge or emotional eating. Without elucidation and clarification of the role and effects of such mechanisms, the utility of targeting stress as opposed to potential mediators such as binge eating and emotional eating is uncertain.

The purpose of this study was to examine the relationships among self-reported stress, morning cortisol, binge eating, emotional eating, and the metabolic abnormalities

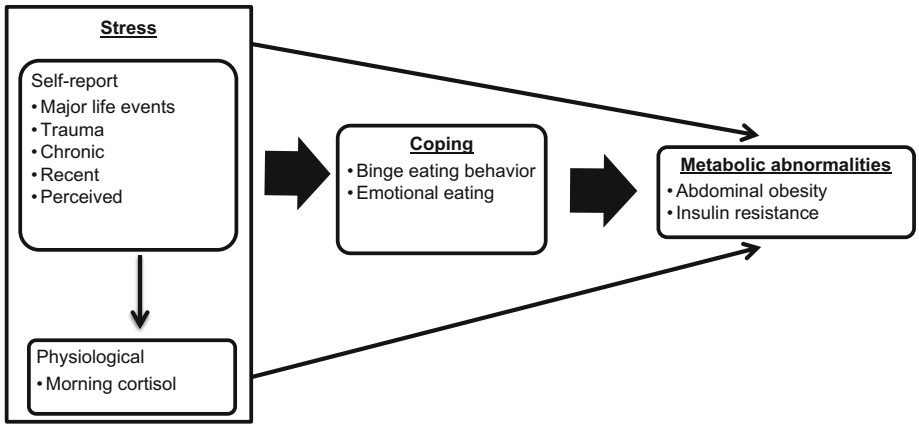

Fig. 1 Conceptual framework

of insulin resistance and abdominal obesity in adults. Insulin resistance was estimated using a well-established surrogate measure, the homeostatic model of insulin resistance (HOMA-IR; Matthews et al., 1985). Based on the above theories and empirical evidence, we hypothesized that: (1) self-reports of stress (major life events, trauma, and chronic, recent, and perceived stress) and cortisol are related to HOMA-IR and waist circumference (WC), (2) binge eating and emotional eating will mediate the relationships between self-reports of stress and HOMA-IR and WC, and (3) binge eating and emotional eating will mediate the relationships between morning cortisol and HOMA-IR and WC. We also explored moderators [gender and age] of these relationships.

Methods

This study is a secondary analysis of data collected from the Interdisciplinary Research Consortium on Stress, Self-Control, and Addiction (IRCSSA; the National Institutes of Health grants PL1-DA024859 and UL1-DE019859). In brief, the IRCSSA is a collaborative, interdisciplinary, cross-sectional set of studies examining the interactions between stress, self-control, and addiction across multiple brain, body, behavioral, and social systems.

Sample and Setting

The sample for the IRCSSA is a convenience sample of men and women who were recruited from online and print advertisements in local newspapers, community centers and churches in New Haven, Connecticut. The sample includes individuals with the addictive behaviors of cigarette smoking, alcohol drinking, and overeating, as well as those without these disorders, from a variety of racial and socioeconomic backgrounds. Inclusion criteria were that participants were between the ages of 18–50 years and were able to read English at least at the sixth grade level. Exclusion criteria were dependence on any drug other than alcohol or nicotine, use of prescribed medications for any psychiatric disorders, pregnancy, and medical conditions or medications that may influence cortisol levels. Additionally, individuals identified on the Eating Disorder Examination-Questionnaire as engaging in compensatory mechanisms (i.e., laxative use, self-induced vomiting) were excluded as biobehavioral processes may differ in this subgroup of individuals. Study procedures were conducted at the Yale Stress Center, and participants received compensation for participation in assessment sessions.

Procedures

The Yale University Institutional Review Board approved this study. Potential participants completed an initial screening over the telephone or in person to determine eligibility based on inclusion and exclusion criteria. Following screening, eligible participants met with a research assistant for a 2-h intake session to obtain informed consent and begin assessments. After the intake session, participants had three to four additional sessions. During these sessions, the research staff performed a comprehensive assessment battery including physical examinations, diagnostics, cognitive and psychological assessments, and conducted blood work. This paper is a secondary data analysis; however, the sample meets the suggested "critical" sample size of 200 for structural equation modeling (Hoelter, 1983).

Measures

Demographics/BMI Data were collected with self-report forms designed for this study that provided data on age, gender, and race/ethnicity. A research nurse or trained research team member measured heights and weights during the physical examination. These were used to calculate BMI.

Stress

Self-report Trained interviewers administered the Cumulative Adversity Interview (Turner & Wheaton, 1995; Turner et al., 1995), a well-established, 140-item interview that assesses major life events, traumas, recent life stress, and chronic stress. Major life events are social adversities that are non-violent but severe and have potential for long-term consequences (e.g., loss of a child, significant other with substance use). Recent life stress measures discrete stressful events occurring in the past 12 months to the participant or participant's immediate family member (e.g., physical assault, abortion or miscarriage, serious accident or injury). Chronic stress measures the subjective experience of continuous stressors or ongoing life problems and hassles. For traumas and recent life stress, items were scored as being present or absent and are summed to form the subscales. For chronic stressors, items were scored using a 3-point Likert scale ranging from not true to very true. Three-month test–retest reliability was measured in subsamples of individuals participating in the IRCSSA (Stults-Kolehmainen et al., 2014). Reliability coefficients for major life events, trauma, recent life events, and chronic stress were 0.91, 0.83, 0.94, and 0.92 respectively (Stults-Kolehmainen et al., 2014). Use of interview techniques is recommended to decrease participant recall bias (Dohrenwend, 2006).

Perceived stress The Perceived Stress Scale (PSS-14) is a 14-item self-report scale that assesses the degree to which individuals appraise situations in their lives as stressful during the previous month (Cohen et al., 1983). Participants are asked how often they felt or thought a certain way on a 5-point Likert Scale of 0 (never) to 4 (very often). Higher scores indicate greater perceived stress. The PSS has good internal reliability ($\alpha = 0.84$–0.86) and concurrent validity with the Daily Stress Inventory ($r = 0.62$) (Cohen et al., 1983; Machulda et al., 1998).

Cortisol Plasma cortisol levels were collected on participants who fasted for 13 h prior to blood draw and were instructed to come to the study site immediately after waking (Kudielka & Wüst, 2009; Nicolson, 2008). Upon arrival, participants had a peripheral intravenous line inserted, and cortisol levels were collected at 15-min intervals for an hour. Morning cortisol was operationalized as the mean plasma cortisol value of the repeated measurements taken over the hour. Morning cortisol has high intra-individual stability ($r = 0.63$) (Hucklebridge et al., 2005; Wüst et al., 2000) with a "normal" morning range of 6–23 mcg/dL (Varon & Fromm, 2014).

Coping

Binge eating Binge eating was assessed with the Eating Disorder Examination-Questionnaire with instructions (EDE-Q-I) (Fairburn & Beglin, 1994; Goldfein et al., 2005). The EDE-Q is a self-report version of the interviewer-based EDE that assesses dimensional aspects of eating disorders (i.e. binge eating, vomiting). The 36-item questionnaire uses 7-point, forced-choice Likert scales to assess eating attitudes and also asks about the frequency of overeating episodes that are unusually large (overeating) and accompanied by a sense of loss of control (binge eating) over the past 28 days. The EDE-Q has recently been revised with instructions about the concepts of binge eating (EDE-Q-I) to improve reliability, and has psychometric support for assessing binge eating in both community and clinical populations (Celio et al., 2004; Goldfein et al., 2005; Mond et al., 2004; Reas et al., 2006).

Emotional eating Emotional eating was measured using the raw score from the emotional eating subscale of the Dutch Eating Behavior Questionnaire (Van Strien et al., 1986). This subscale measures the tendency to overeat in response to negative emotions. The subscale contains 13 questions measured on a 5-point Likert scale ranging from never to very often and has good internal reliability ($\alpha = 0.93$) in adults (Van Strien et al., 1986).

Metabolic abnormalities

Abdominal obesity WC is a commonly used index of abdominal obesity because it is predictive of cardiovascular disease, low-cost, and low-burden (Janssen et al., 2004; Zhu et al., 2004). WC was measured following the National Heart, Lung, and Blood Institute and National Health and Nutrition Education Survey protocol, at minimal inspiration midway between the last rib and the iliac crest (Janssen et al., 2002). For men, high risk is considered at a WC > 40 inches. For women, high risk is considered a WC > 35 inches (NHLBI Obesity Education Initiative Expert Panel, 1998).

Insulin resistance screen Fasting glucose and insulin were measured to calculate a well-accepted screening measure of insulin resistance, HOMA-IR (Wallace et al., 2004). The following formula was used for this calculation: (plasma glucose in mg/dL $\times$ plasma insulin in μU/mL)/405 (Ikeda et al., 2001). The HOMA-IR has strong correlations with well-validated methods that assess insulin resistance including euglycemic (Garcia-Estevez et al., 2003) and hyperglycemic clamps (Matthews et al., 1985; Wallace et al., 2004).

Data analysis

We used SPSS version 21.0 and SPSS AMOS version 22.0 to conduct analyses. We began data analysis by calculating univariate statistics for each variable to evaluate potential outliers and the distribution of data. We found a number of variables with a positive skew (major life events, trauma, recent life events, morning cortisol, emotional eating, binge eating, and HOMA-IR). These variables underwent a log transformation which helped to normalize values and are reported as such throughout this paper. Next, we conducted bivariate analyses and used Cohen's criteria to determine effect sizes (Cohen, 1992). Then we conducted more complex multivariate analyses using structural equation modeling with maximum likelihood estimation following the procedures recommended by Kline (2011). Indirect effects were estimated using 2000 bootstrapped samples and 95 % bias-corrected confidence intervals (Preacher & Hayes, 2008) using both en bloc (i.e., consideration of both mediators) and decomposed approaches for multiple mediators (Daniel et al., 2015). To test model fit we used the fit indices of root mean square error of approximation (RMSEA) and the Comparative Fit Index (CFI). A RMSEA value of $\leq$0.05 indicates a close approximate fit and a RMSEA $\geq$ 1 suggests a poor fit (Browne et al., 1993). A CFI value $\geq$0.90 and close to 1.00 indicate good fit (Hu & Bentler, 1999).

Moderation by gender was tested using a multi-group path analysis in AMOS. We compared an unconstrained model which assumes that path coefficients differ across groups to a constrained model where coefficients are set to be equal across group using a Chi square difference test. Gender moderation was tested using a parameter level analysis by comparing path coefficients between the two groups using a z-score over 1.96 (Arbuckle, 2013). To test moderation by age, we used an interaction moderation analysis (i.e., moderated mediational effects) (Bollen, 1998; Hayduk, 1988; Kline, 2011). To reduce multicollinearity, first-order terms were standardized before creating cross-product interaction terms. We entered the interaction terms as both first (i.e., each type of stress $\times$ age) and second-stage (i.e., emotional eating $\times$ age; binge eating $\times$ age) moderation (Kline, 2011).

Due to multicollinearity, BMI was not included in any of the models. However as an exploratory and post hoc analysis, we also conducted a mediation analysis testing whether binge eating and emotional eating mediated the relationships between stress and BMI.

Results

Preliminary analysis

The sample included 249 adults. The mean BMI was 26.93 (SD 5.14) kg/m^2 with 29.3 % overweight and 27.3 % obese. The mean age was 28.30 (SD 8.31) years. A little over half of the sample was male at 54.2 % and the sample was predominantly white at 69.5 %. The mean binge eating episodes was 0.87 (SD 2.26) with 22.9 % of the sample endorsing at least 1 day with a binge eating episode over the month. The mean emotional eating score was 26.64 (SD 11.89). The mean WC was 34.90 (SD 5.62) inches with 22.9 % meeting gender-specific criteria for abdominal obesity. The mean HOMA-IR was 3.09 (SD 2.00).

Bivariate correlations

The bivariate correlations are show in Table 1. There were small to moderate positive associations among the self-reported stress scales (r = 0.14–0.43). Morning cortisol

Table 1 Correlations between study variables (N = 249)

	Major life events	Trauma	Recent life	Chronic stress	Perceived stress	Morning cortisol	Emotional eating	Binge eating	HOMA-IR
Major life events	–								
Trauma	0.425**	–							
Recent life	0.363**	0.404**	–						
Chronic stress	0.393**	0.318**	0.285**	–					
Perceived stress	0.342**	0.144*	0.217**	0.401**	–				
Morning cortisol	−0.208**	−0.152*	0.059	−0.162*	−0.009	–			
Emotional eating	0.128*	0.000	0.029	0.160*	0.305**	0.057	–		
Binge eating	0.080	0.071	0.020	0.100	0.161*	0.042	0.365**	–	
HOMA-IR	0.140*	0.162*	0.047	0.109	0.040	−0.160*	−0.005	0.019	–
Waist circumference	0.197**	0.257**	0.080	0.139*	−0.012	−0.246**	−0.006	0.195**	0.489**

* Correlation is significant at the 0.05 level (2-tailed)

** Correlation is significant at the 0.01 level (2-tailed)

Table 2 Results of the structural equation model testing the relationships between self-report stress, morning cortisol, binge eating, emotional eating, insulin resistance, and waist circumference (N = 249)

Variables of interest	B	SE	β	*p* value	95 % Bias-corrected confidence interval
Major life events → emotional eating	0.049	0.053	0.069	0.350	−0.103, 0.129
Major life events → binge eating	0.027	0.084	0.024	0.751	−0.121, 0.217
Trauma → emotional eating	−0.033	0.046	−0.052	0.466	−0.095, 0.096
Trauma → binge eating	0.061	0.073	0.062	0.400	−0.052, 0.230
Recent life events → emotional eating	−0.039	0.044	−0.063	0.369	−0.127, 0.060
Recent life events → binge eating	−0.059	0.070	−0.061	0.396	−0.284, 0.088
Chronic stress → emotional eating	0.002	0.002	0.064	0.373	−0.003, 0.008
Chronic stress → binge eating	0.002	0.004	0.043	0.565	−0.004, 0.012
Perceived stress → emotional eating	**0.006**	**0.002**	**0.280**	**<0.001**	**0.003, 0.010**
Perceived stress → binge eating	**0.005**	**0.002**	**0.142**	**0.044**	**0.001, 0.011**
Morning cortisol → emotional eating	0.083	0.063	0.084	0.190	−0.031, 0.216
Morning cortisol → binge eating	0.109	0.101	0.071	0.281	−0.051, 0.311
Emotional eating → HOMA−IR	−0.010	0.060	−0.012	0.862	−0.132, 0.127
Emotional eating → waist circumference	−1.600	2.011	−0.051	0.427	−4.719, 3.345
Binge eating → HOMA-IR	0.008	0.037	0.013	0.835	−0.078, 0.117
Binge eating → waist circumference	**3.976**	**1.249**	**0.196**	**0.002**	**1.195, 6.915**
Major life events → HOMA-IR	0.041	0.049	0.063	0.406	−0.108, 0.103
Major life events → waist circumference	2.409	1.459	0.109	0.145	−2.023, 5.771
Trauma → HOMA-IR	0.067	0.043	0.115	0.118	0.017, 0.199
Trauma → waist circumference	**2.928**	**1.434**	**0.147**	**0.041**	**0.791, 6.397**
Recent life events → HOMA-IR	−0.014	0.041	−0.025	0.733	−0.104, 0.075
Recent life events → waist circumference	−0.516	1.370	−0.027	0.707	−3.712, 1.988
Chronic stress → HOMA-IR	0.001	0.002	0.037	0.617	−0.004, 0.006
Chronic stress → waist circumference	0.051	0.074	0.050	0.487	−0.079, 0.188
Perceived stress → HOMA-IR	−0.002	0.001	−0.008	0.909	−0.003, 0.002
Perceived stress → waist circumference	−0.068	0.047	−0.103	0.149	−0.175, 0.013
Morning cortisol → HOMA-IR	*−0.108*	*0.059*	*−0.120*	*0.070*	*−0.206, 0.059*
Morning cortisol → waist circumference	**−6.466**	**1.992**	**−0.209**	**0.001**	**−10.624, −1.561**

[a] The following variables are log transformed: major life events, trauma, recent life events, morning cortisol, emotional eating, binge eating, and HOMA-IR

[b] Tests significant at the 0.05 level are shown in bold; tests with trend-significance at the 0.10 level are shown in italics

[c] Values are from 95 % bias-corrected confidence intervals from 2000 bootstrapped samples

was significantly correlated with major life events, chronic stress, and trauma with a small effect size (r = −0.15 to −0.21). There were significant relationships between major life events, trauma, and increased HOMA-IR and WC (r = 0.14–0.26). Chronic stress was significantly associated with increased WC (r = 0.14). Morning cortisol was inversely related to HOMA-IR (r = −0.16) and WC (r = −0.25).

Mediation analysis

We used confirmatory modeling to test our hypotheses about the relationships between self-reported stress, morning cortisol, binge eating, emotional eating, HOMA-IR, and WC as shown in Fig. 1. The results of the model are provided in Table 2. Perceived stress was positively associated with both emotional eating and binge eating (B = 0.006, SE = 0.002, $p < 0.001$ and B = 0.005, SE = 0.002, $p = 0.04$, respectively). Binge eating was also associated with increased WC (B = 3.976, SE = 1.249, $p = 0.002$). Trauma was associated with increased WC (B = 2.928, SE = 1.434, $p = 0.041$) and lower morning cortisol was associated with increased WC (B = −6.466, SE = 1.992, $p = 0.001$). However, despite attempts to improve the model fit, there is some evidence of lack of fit with the data with RMSEA = 0.335 and CFI = 0.921 (Kline, 2011). There was no evidence of mediation either en bloc or decomposed with all indirect

Table 3 Results of the structural equation model testing moderation by gender (N = 249)

Variables of interest	Male		Female		*z* score
	Estimate	*p* value	Estimate	*p* value	
Major life events → emotional eating	0.087	0.216	−0.060	0.409	−1.455
Major life events → binge eating	0.062	0.591	0.008	0.946	−0.326
Trauma → emotional eating	0.001	0.987	−0.028	0.690	−0.321
Trauma → binge eating	0.069	0.461	0.009	0.940	−0.403
Recent life events → emotional eating	−0.041	0.493	−0.009	0.876	0.380
Recent life events → binge eating	**0.064**	**0.513**	**−0.214**	**0.026**	**−2.031****
Chronic stress → emotional eating	0.006	0.110	−0.002	0.579	−1.570
Chronic stress → binge eating	0.002	0.736	0.005	0.340	0.375
Perceived stress → emotional eating	**0.004**	**0.060**	**0.009**	**0.000**	**1.918***
Perceived stress → binge eating	**0.001**	**0.728**	**0.010**	**0.003**	**1.868***
Morning cortisol → emotional eating	−0.026	0.805	0.094	0.187	0.947
Morning cortisol → binge eating	0.144	0.405	0.106	0.369	−0.180
Emotional eating → HOMA-IR	**0.125**	**0.099**	**−0.212**	**0.034**	**2.683*****
Emotional eating → waist circumference	3.568	0.154	−2.622	0.416	−1.517
Binge eating → HOMA-IR	**−0.103**	**0.023**	**0.149**	**0.013**	**3.353*****
Binge eating → waist circumference	2.010	0.179	5.162	0.007	1.292
Major life events → HOMA-IR	0.050	0.416	0.015	0.847	−0.359
Major life events → waist circumference	1.483	0.463	4.073	0.097	0.814
Trauma → HOMA-IR	**0.124**	**0.011**	**−0.048**	**0.515**	**−1.943***
Trauma → waist circumference	4.057	0.012	−0.664	0.781	−1.638
Recent life events → HOMA-IR	−0.016	0.759	0.032	0.608	0.591
Recent life events → Waist circumference	−1.864	0.269	0.885	0.661	1.046
Chronic stress → HOMA-IR	−0.002	0.610	0.004	0.252	1.182
Chronic stress → Waist circumference	0.007	0.945	0.171	0.092	1.160
Perceived stress → HOMA-IR	−0.001	0.650	0.001	0.703	0.575
Perceived stress → waist circumference	−0.042	0.455	−0.116	0.124	−0.797
Morning cortisol → HOMA-IR	−0.212	0.019	−0.026	0.737	1.577
Cortisol → Waist circumference	**−10.126**	**0.000**	**−2.923**	**0.234**	**1.862***

[a] The following variables are log transformed: major life events, trauma, recent life events, morning cortisol, emotional eating, binge eating, and HOMA-IR

[b] Tests showing a significant difference between genders at the 0.10 level are shown in bold

[c] *** *p* value <0.01; ** *p* value <0.05; * *p* value <0.10

effects at $p > 0.05$, two-tailed ($p = 0.180$–0.881). Similar to the results above, our post hoc analysis using BMI as the outcome variable in lieu of HOMA-IR and WC demonstrated no statistical evidence of mediation either en bloc or decomposed with all indirect effects at $p > 0.05$ ($p = 0.140$–0.550; data not shown), two-tailed.

Moderation analyses

Gender Next we examined the extent to which gender moderated these relationships. The Chi square difference test between the completely unconstrained and constrained models was significant, $\Delta\chi^2(10) = 32.15$, $p < 0.001$, suggesting the model is different across gender. Differences were then tested between the parameter estimates from the full model, demonstrating some significant differences in some paths by gender (Table 3). For females only, there was a significant relationship between perceived stress and emotional eating (B = 0.009, $p < 0.001$), and perceived stress and binge eating (B = 0.01, $p = 0.003$). There was an inverse relationship between recent life events and binge eating (B = −0.214, $p = 0.026$). There was a significant relationship between binge eating and increased HOMA-IR among females (B = 0.149, $p = 0.013$). For males only, there was a significant relationship between trauma and increased HOMA-IR (B = 0.124, $p = 0.011$). Also, lower morning cortisol was associated with higher WC among males (B = −10.126, $p < 0.001$). When mediation analy-

ses were conducted separated by gender, we did not find statistical support for mediation.

Age We did not find support for evidence of moderated mediation (i.e., conditional indirect effects) at either the first or second stage in the model. There were main effects of age on WC (B = 0.11 SE = 0.04, $p = 0.008$). There was an interaction effect between age and perceived stress on the relationship with HOMA-IR (B = −0.022, SE = 0.009, $p = 0.018$). All other interaction terms were not statistically significant ($p > 0.05$, two-tailed; p values ranged from 0.103 to 0.987).

Discussion

Our study assesses the relationships among self-reported stress, morning cortisol, binge eating, emotional eating, HOMA-IR, and WC in a sample of adults recruited from the community. Several key contributions emerge from our results. Our first hypothesis, that self-reports of stress and morning cortisol are related to metabolic abnormalities, was partially supported. Similar to previous studies, we found significant associations between increased major life events, trauma and increased HOMA-IR and WC (Heppner et al., 2009; Lee et al., 2014). Chronic stress was associated with increased WC. We also found that lower morning cortisol was correlated with higher HOMA-IR and WC. These results corroborate the association between lower morning cortisol and metabolic disturbances, which may be indicative of impaired cortisol regulation (Bruehl et al., 2009; Lasikiewicz et al., 2008). Contrary to our hypothesis, we did not find evidence that binge eating or emotional eating statistically mediated the relationship between any of the types of stress and HOMA-IR or WC.

Our results support the importance of perceived stress as a correlate of binge eating and emotional eating (Greeno & Wing, 1994; Groesz et al., 2012; Polivy & Herman, 1993), and our results extend this literature by demonstrating a gender moderation effect. Similar to our results, researchers have found associations between perceived stress and binge and emotional eating in samples with only women (Tomiyama et al., 2011). Few studies have examined correlates of eating disorder pathology in samples that include men. However, researchers have found no significant difference in perceived stress between males who did and did not binge eat (Rosenberger & Dorflinger, 2013). Additionally, our results are congruent with a prior cross-sectional study demonstrating that there is a gender moderation effect in the relationship between perceived stress and emotional eating among middle school students (Nguyen-Rodriguez et al., 2009). Our results also support results from a large, cross-sectional study of adults who were participating in a health risk self-assessment screening. One item from the Perceived Stress Scale was used to assess perceived stress and this study demonstrated a similar moderation effect as our results, with women having a higher association between stress and binge eating (Striegel et al., 2012). The results presented add to the growing body of literature on stress and eating phenotypes through use of the complete Perceived Stress Scale and also demonstrate a gender moderation effect for emotional eating.

Numerous measures have been used to measure and quantify stress throughout the literature, with no clear gold standard. In this study we incorporate environmental, psychological, and biological perspectives related to stress measurement (Cohen et al., 1997). While we found a significant relationship between binge and emotional eating and perceived stress as assessed by the Perceived Stress Scale (Cohen et al., 1983), we did not find consistent relationships with major life events, trauma, recent life events, and chronic stress as assessed by the Cumulative Adversity Interview (Turner & Wheaton, 1995). The Perceived Stress Scale is a global measure that assesses the degree to which one appraises situations in one's life as stressful, unpredictable, overloading, and uncontrollable (Cohen et al., 1983). This originates from a more psychological perspective (i.e., focusing on an individuals' subjective abilities to cope with stress) (Cohen, 2000; Cohen et al., 1997). On the other hand, the Cumulative Adversity Interview asks more specific and event-specific questions about stress exposure in terms of interpersonal, social and financial issues, work and home environment and relationships with family and significant others. This originates from the environmental perspective, which focuses on environmental events and experiences, which objectively are related with substantial adaptive burdens (Cohen, 2000). Further exploration of the convergent and discriminant validity of these two measures, particularly related to the concept of chronic stress, is warranted.

Lower morning cortisol was associated with higher HOMA-IR and WC, especially in men. This is congruent with prior studies suggesting that men cope with stress differently than females (Ljung et al., 2000). These results also may signal a perturbed function of the hypothalamic–pituitary–adrenal axis due to "burn-out" (Björntorp, 1999) or "allostatic overload" (McEwen, 2005). Though we did not include other measures of allostasis, there is suggestion that with allostatic overload there are disturbances in other systems (e.g., gonadal, sympathetic or autonomic nervous systems). Thus, the associations with HOMA-IR and abdominal obesity may be a result of perturbations in those systems given that morning cortisol was low (Björntorp & Rosmond, 2000).

There have been few studies on the relationships between biological markers of stress in binge and emo-

tional eaters, and results have been mixed (Lo Sauro et al., 2008). In this study, we found no statistically significant relationship between morning cortisol levels and emotional and binge eating. Another type of cortisol response that has been frequently examined is cortisol reactivity; however, there is no consensus regarding the relationships. In studies examining cortisol reactivity, some have demonstrated increased cortisol reactivity (Gluck et al., 2004; Rosenberger & Dorflinger, 2013), others have demonstrated a blunted cortisol response (Rosenberg et al., 2013), while others have demonstrated no difference (Schulz et al., 2011) based on binge eating. There is a paucity of studies examining the relationships between biological markers of stress and emotional eating. The studies that have been conducted have been mainly with undergraduate, female-only populations. Of the studies that have examined cortisol and emotional eating, most have looked at cortisol reactivity and found no difference in cortisol reactivity between emotional and non-emotional eaters (Raspopow et al., 2014; van Strien et al., 2013). Some of these discrepancies in the literature are likely due to variable measures and cortisol reactivity paradigms such as the dexamethasone suppressor test, Trier Social Stress Test (Kirschbaum et al., 1993) and further replication with larger samples is necessary. Additionally, the physiological mechanisms related to stress are complex and imaging techniques have demonstrated that a complex integration of brain networks influence feeding behavior (Dallman, 2010). It is possible that other physiological stress pathways play a role related to obesity-related eating phenotypes, and exploration of the relationships between other stress biomarkers is needed. For example, a study examining aspects of sympathetic nervous system functioning demonstrated that among obese women with binge eating disorder, stress-induced changes in hunger were associated with greater stress-induced changes in systolic and diastolic blood pressure ($r = 0.76$, 0.78, respectively). These relationships were not seen in obese or normal weight women without binge eating disorder (Klatzkin et al., 2015). Additionally, the influence of stress on other areas in the brain such as the amygdala and hippocampus, which are involved in learning and memory, may play an important role in these relationships (Dallman, 2010).

Similar to previous literature demonstrating a relationship between binge eating and increased risks of metabolic abnormalities (Roehrig et al., 2009), our findings support that individuals who binge eat have an increased WC. Yet, the relationship between binge eating and HOMA-IR was significant in females only. This may be related to the types of food consumed during binges as well as the rapid speed that is common during a binge. Females tend to favor high carbohydrate/high fat foods whereas males tend to prefer high protein/fat foods (Drewnowski et al., 1992). Thus, the types of foods consumed and resultant effects on physiology may account for this moderation effect.

Among phenomena that may explain the relationship between stress and metabolic abnormalities, hedonic eating (i.e., eating for pleasure and not due to energy deprivation) is a prime candidate. Nevertheless, our hypothesis that two types of hedonic eating, binge eating and emotional eating, would mediate the relationship between stress and HOMA-IR and WC was not supported. It is possible that there is a relationship, though it may be weak and thus require a larger sample size to detect. Additionally, these effects may be restricted to specific subgroups. For example, while we did not find any moderation effect of age, the mean age of our population was fairly young at 28.3 years. Since the risk of diabetes increases with age, a longitudinal approach or a sample of older participants may be necessary to see these relationships. Also, we included individuals across the weight spectrum which may have attenuated significant results. Future studies may benefit from directly studying these relationships among individuals who are overweight or obese. Furthermore, a combination of multiple mediators—such as physical activity and sedentary behavior—acting jointly may be involved.

Lastly, researchers have been exploring different types of psychologically-motivated and hedonic eating types including binge eating, emotional eating, and stress-induced eating (Lowe & Butryn, 2007). Future research is needed to examine the potential physiological and behavioral mechanisms and conceptual overlaps between different types of eating phenotypes (e.g., binge eating, emotional eating, stress-induced eating). It is possible that only a subset of individuals who are binge or emotional eaters would also be classified as "stress-eaters". Further research is needed to examine whether subtyping binge eating based on stress or physiological stress responses would aid with phenomenology, etiology, outcomes, course and treatment. For example, researchers have used the opioidergic antagonist, naltrexone, to examine changes in hedonic eating following a mindfulness-based weight loss intervention. Participants who reported naltrexone-induced nausea and who were randomized to a mindfulness group had greater improvements in hedonic eating following the mindfulness intervention compared to individuals randomized to the control group (Mason et al., 2015). This finding was also observed in another sample of obese women (Daubenmier et al., 2014).

Limitations

This study is not without its limitations. First, the data are cross-sectional and non-experimental, thus this study does not lead to casual inferences. Second, there are limitations resulting from the sampling methods used in the IRCSSA

study. The sample is a convenience sample and individuals with medical conditions or taking medications that may disrupt cortisol levels were excluded. This was done to control the homogeneity of the sample; however, decreases external generalizability. Third, morning cortisol was used in this study and is not representative of the daily fluctuations of cortisol, the cortisol awakening response or of cortisol reactivity; however, these may be important potential areas for further exploration. Fourth, due to multicollinearity, BMI was not included in this model. Thus the independent effects on WC and HOMA-IR beyond the effect on BMI are not known. Despite these limitations, the results from this study provide the foreground for additional longitudinal and experimental studies exploring these variables.

In conclusion, we did not find evidence that binge eating and emotional eating statistically mediate the relationship between stress and the metabolic abnormalities of insulin resistance and abdominal obesity. We found significant negative relationships between stress and morning cortisol levels, and cortisol and HOMA-IR and WC. Our results support previous research demonstrating gender moderation effects among the relationships of perceived stress, and binge and emotional eating and the relationship with binge eating and increased HOMA-IR. These findings suggest that interventions that target perceived stress targeted may improve binge and emotional eating among females.

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Compliance with ethical standards

Conflict of interest Ariana Chao, Margaret Grey, Robin Whittemore, Jonathan Reuning-Scherer, Carlos M. Grilo and Rajita Sinha declare that they have no conflict of interest.

Human and animal rights and Informed consent All procedures followed were in accordance with ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all patients for being included in the study.

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