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Cortisol Response to an Induction of Negative Affect among Adolescents with and without Loss of Control Eating

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Abstract

Background—Adults with binge-eating disorder may have an exaggerated or blunted cortisol response to stress. Yet, limited data exist among youth who report loss of control (LOC) eating, a developmental precursor to binge-eating disorder.

Methods—We studied cortisol reactivity among 178 healthy adolescents with and without LOC eating. Following a buffet lunch meal adolescents were randomly assigned to watch a neutral or sad film clip. After, they were offered snacks from a multi-item array to assess eating in the absence of hunger. Salivary cortisol was collected at –80, 0, 30, and 50 minutes relative to film administration, and state mood ratings were reported before and after the film.

Results—Adolescents with LOC had greater increases in negative affect during the experimental paradigm in both conditions ($p > .05$). Depressive symptoms, but not LOC, related to a greater cortisol response in the sad film condition ($p > .05$). Depressive symptoms and state LOC were

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Conflict of Interest Statement

All authors declare that they have no potential biomedical conflicts of interest, financial or otherwise, relevant to this article (R.M.R., L.B.S., M.T.K., N.R.K., C.K.P., K.A.T., S.M.B., A.D., O.G., A.A., L.M.S., S.Z.Y., and J.A.Y.)

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related to different aspects of eating behavior, independent of film condition or cortisol response ($p>.05$).

Conclusions—A film clip that induced depressed state affect increased salivary cortisol only in adolescents with more elevated depressive symptoms. Adolescents with and without LOC were differentiated by greater increases in state depressed affect during laboratory test meals, but had no difference in cortisol reactivity. Future studies are required to determine if adolescents with LOC manifest alterations in stress reactivity to alternative stress-inducing situations.

Keywords

Binge-Eating; Loss of Control Eating; Cortisol; Depressive Symptoms; Adolescence

Introduction

Binge-eating disorder is uncommon among adolescents. In contrast, loss of control (LOC) eating, irrespective of the amount consumed, is reported by 5-50% of youth.¹ Compared to youth without LOC, those with LOC have more depressive symptoms, anxiety, and greater adiposity.¹ Prospective studies indicate that youth who endorse even infrequent LOC are at-risk for gaining excess weight,² worsening of metabolic syndrome components,³ and developing binge-eating disorder.⁴

Escape and affect theories propose that LOC results from deficient skills to cope adaptively with negative affect.⁴ LOC purportedly serves as a maladaptive coping mechanism to escape emotional distress. In support of these models are studies finding that stressful situations in women with binge-eating produce negative affect that, in turn, triggers binge-eating.⁵ A meta-analysis of primarily adult studies found that increases in acute negative affect preceded binge-eating.⁶ Despite fewer studies in youth, parallel affective-LOC patterns have been described. Children and adolescents with LOC exhibit more dysfunctional emotion regulation, including eating in response to adverse moods.⁷

Developmental models for how LOC leads to binge-eating disorder and/or obesity and metabolic problems necessitate an understanding of the interplay of biological, affective, and eating behavioral mechanisms.⁸ Despite the prominence of negative affect in conceptual models of LOC etiology and maintenance, we have very limited knowledge about the biological basis of LOC.⁸ The hypothalamic-pituitary-adrenal (HPA)-axis is critical to regulation of negative affect and to governing energy intake that occurs in response to non-homeostatic factors like negative affect. Dysregulation in cortisol, a primary stress hormone and output of the HPA-axis, has been associated with disordered eating and obesity in complex ways. Prior data suggest an exaggerated cortisol response to an acute stressor among adults with binge-eating,⁹ although this pattern has not been observed in all studies, with some investigations reporting a blunted response.¹⁰ Likewise, in children and adolescents, both hyper- and hypo-cortisol responses to stress relate to greater BMI cross-sectionally and longitudinally.¹¹

The possible effect of a mood-altering stressor on HPA-axis reactivity has not been evaluated in adolescents with and without LOC. Elucidating physiological underpinnings of

LOC in adolescents could inform our understanding of biological mechanisms through which LOC impacts weight and metabolic health. Similarly, cortisol patterns in and of themselves, and/or in conjunction with other vulnerability factors, are increasingly gaining attention as potential risk factors for developmental psychopathology and metabolic disease.¹² The objective of the current study was to determine the relationship of LOC to cortisol and psychological responses to an experimental paradigm, designed to assess the effect of negative affect on eating in the absence of hunger. We hypothesized that adolescents with LOC would exhibit a heightened cortisol response and greater negative affect than youth without LOC. We anticipated that these differences would persist after accounting for trait negative affect (depressive symptoms) and adiposity. We also sought to characterize the relationship of LOC and cortisol response to eating in the absence of hunger after an induction of negative affect, independent of depressive symptoms and adiposity.

Methods

Sample and participant selection

This study is a secondary analysis of healthy 13-17-year-olds, recruited for a study of eating in adolescents (ClinicalTrials.gov ID: NCT00631644). Findings from the primary aim are reported elsewhere.¹³ This manuscript is the first presentation of the study's cortisol data, and the only paper to evaluate the relationship of adolescent LOC to cortisol. All participants were in good health, determined by medical history and physical exam. Participants were excluded if they had a major medical or psychiatric condition, medication affecting appetite/weight, pregnancy, ongoing weight-loss treatment, or dislike of >50% test meal foods. Adolescents and parents/guardians provided written assent and consent, respectively. The NICHD IRB approved all procedures. Participants were financially compensated for their time.

Procedure

Adolescents attended a screening at the NIH Clinical Center and underwent a physical exam. An average 5.8 ± 4.3 weeks later, they returned for a test meal visit on a separate day for which they were instructed to adhere to a 10-hour overnight fast. The experimental paradigm is depicted in **Figure 1**. Fasting salivary cortisol was obtained at 10:30am (–80 minutes before the film). At 11:00am, participants were served a multi-item buffet meal of lunch-type foods consisting of 11,000 kcal that varied in macronutrients.¹³ Participants were instructed to eat until no longer hungry.¹³ Forty-five minutes after initiation of the buffet (11:45am), adolescents were randomized to either a sad mood induction or a control condition. The sad induction involved viewing a 4-minute 20-sec film clip of “The Champ” (1979), demonstrated to induce temporary sadness.¹³ Adolescents in the control condition viewed a 4-minute 20-sec film clip of a bird documentary, “Winged Migration” (2003). Immediately following the film (11:50am), a salivary sample was obtained (0 minutes post-film clip). At 12:00pm, each participant was exposed to a multi-item snack array consisting of 4,500 kcal to measure eating in the absence of hunger.¹³ Saliva samples were repeated at 30 (12:20pm) and 50 minutes after the film (12:40pm).

Measures

LOC eating—At the screening, the *Eating Disorder Examination version 12OD/C.2*¹⁴ was administered to determine LOC in the month prior. Participants were categorized as those who endorsed 1 episode of: i) unambiguous overeating with LOC (objective binge); ii) LOC, with episode size being ambiguous (subjective binge); iii) overeating without LOC (objective overeating); or iv) no episodes. Consistent with research in non-clinical youth, LOC was defined by 1 objective and/or subjective binge. The Eating Disorder Examination has good inter-rater reliability for all episode types in adolescents.¹⁵

At the test meal, participants reported perceived, state experience of LOC while eating. They responded to, “How much was your eating at today's visit like a meal when you feel loss of control?” using a Likert-scale from *1=Extremely* to *5=Not at all*, such that lower scores reflected greater state LOC. Responses showed convergent validity with interview-assessed LOC; those with LOC in the past month reported greater state LOC at the test meal than those without LOC in the past month (3.3 ± 1.3 versus 4.3 ± 0.9 , $p < .001$).

Depressive symptoms—Adolescents self-reported depressive symptoms on the Beck Depression Inventory, Second Edition, a 21-item questionnaire used to assess severity of depressive symptoms over the past two weeks.¹⁶ Items are rated on a Likert scale of *0=absence* to *3=high severity of symptom*, and the total score is the sum.¹⁶

Salivary cortisol—Samples were obtained with an oral swab (Sarstedt, Newton, NC) placed under the tongue for 120 sec, to minimize variations in volume and analytes. Adolescents provided four samples at -80, 0, 30, and 50 minutes relative to film administration. Cortisol was measured using an enzyme immunoassay (Siemens Immulite 1000; sensitivity 60 ng/dL, intra- and inter-assay CVs 5.8-11.2%). Salivary cortisol reflects circulating free plasma cortisol (physiologically active cortisol). Cortisol area under the curve with respect to ground (AUC_g) and to increase (AUC_i) were calculated using formulas by Pruessner et al.¹⁷ Because cortisol was collected during a laboratory stressor and meal paradigm, cortisol output was assumed to reflect a combination of cortisol response to food intake and the film.

Depressed state affect—Adolescents completed the depression subscale of the Brunel Mood Scale,¹⁸ containing a series of mood descriptors rated on 5-point Likert scale. Respondents indicate to what extent they experience each feeling “right now.” Participants completed the Brunel Mood Scale following the buffet and just prior to film administration (11:45am), directly after (11:50am), and again 30 (12:20pm) and 50 minutes (12:40pm) following film administration. Normed T-scores for age were calculated. We determined immediate pre- to post-film change in depressed affect as post-film minus pre-film rating. We also determined AUC_g and AUC_i for depressed affectivity during the entire paradigm.

Energy intakes—Buffet and snack intakes were measured as differences in weight (g) of each item before and after eating. Energy (kcal) intakes were calculated with data from the USDA National Nutrient Database for Standard Reference (USDA, Agricultural Research Service, Beltsville, MD) and food manufacturer information.

Body composition—Height was the average of three measurements by calibrated stadiometer. Weight was obtained in a fasted state with a calibrated digital scale. BMI (kg/m^2) and BMIz were determined according to CDC 2000 standards. Body fat (kg) and percentage fat-free mass were estimated with air displacement plethysmography (Life Measurement Inc., Concord, CA).

Puberty—Breast development was assigned according to Tanner. Testicular volume (mL) was measured by orchidometry. Breast and testicular staging were used to classify adolescents as those in pre/early/midpuberty (girls: breast Tanner 1-3; boys: testes <15 mL) or late puberty (girls: breast Tanner 4-5; boys: testes $\geq$ 15 mL).

Analytic approach—This study represents a secondary analysis, performed with IBM SPSS 22.0. Cortisol was log-transformed. Independent t-tests and chi-square described adolescents with versus without ≥ 1 LOC episode in the past month. Parallel analyses compared those randomized to the sad versus neutral film. Relationship of LOC to buffet intake was described with ANCOVA, with LOC in the past month as the independent factor, total intake as the dependent variable, and depressive symptoms, state LOC, age, race, sex, puberty, fat mass, fat-free mass, and height included in the model. To evaluate effects of LOC by film on state depressed affect, two-way ANCOVAs were used to predict immediate pre- to post-film change, depressed affect AUC_g , and AUC_i from LOC, film, and their interaction, including in the model pre-film depressed affect, depressive symptoms, state LOC, cortisol AUC_g , age, race, puberty, sex, fat mass, fat-free mass, and height. ANCOVA compared differences in cortisol AUC_g and AUC_i by LOC in the past month, film, and their interaction, accounting for depressive symptoms, state LOC, baseline state depressed affect, age, race, sex, puberty, fat and fat-free mass, height, and buffet intake. Relationship of LOC to snack intake was evaluated with a two-way ANCOVA with LOC, film, and their interaction as independent factors, snack intake as the dependent variable, adjusting for depressive symptoms, state LOC, state depressed affect prior to the snack period, cortisol AUC_g , age, race, sex, puberty, fat mass, fat-free mass, height, and buffet intake. Interactions of film with depressive symptoms, state LOC, baseline state depressed affect, and cortisol AUC_g were also evaluated and reported when significant. We found no significant interactions for depressive symptoms and LOC or for the three-way interaction with film; thus, these findings are not reported.

Results

Preliminary analyses

Participants were 178 (66% female) 13-17-year-olds ($M \pm SD$ 15.3 $\pm$ 1.4). Characteristics based on LOC status are presented in **Table 1**. Participants reporting LOC were more likely to be overweight/obese ($p=.04$) and to report higher depressive symptoms ($p<.001$) than no LOC. Ninety-five (53%) were randomized to the sad film (20 with LOC) and 83 to the neutral film (18 with LOC). Adolescents assigned to the neutral versus sad film did not differ on any variable ($ps>.26$).

Adolescents with versus without LOC in the past month did not differ in calories consumed at the buffet, nor was state LOC associated with buffet intake ($p=.40$). Significant predictors

of buffet intake were depressive symptoms ($B \pm SE$ 22.7 $\pm$ 7.2, $p=.002$), cortisol AUC_g (311.1 $\pm$ 135.0, $p=.02$), and being non-Hispanic White (-186.9 ± 71.2 , $p=.01$) and male (395.3 $\pm$ 93.9, $p<.001$).

Effects of LOC and film on depressed state affect

Main effects were observed for LOC and film in the prediction of depressed affect (**Figure 2a, 2b**). Adolescents with LOC in the past month had greater pre- to post-film increases in state depression than no LOC ($M \pm SE$ 2.8 $\pm$ 0.9 versus 0.9 $\pm$ 0.5, $p<.05$), with a trend of LOC on AUC_i ($p=.09$) and no effect on AUC_g . Among all adolescents who viewed the sad film, greater pre- to post-film increases in state depression were observed compared to those in the neutral film (3.2 $\pm$ 0.7 versus 0.5 $\pm$ 0.7, $p<.001$), with parallel effects of film on depressed affect AUC_i ($p=.02$) and AUC_g ($p=.01$). The only other predictor of state depressed affect AUC_g was depressive symptoms ($B \pm SE$ 6.5 $\pm$ 2.0, $p=.001$). State LOC did not relate to any changes in state depressed affect ($ps>.54$). The main effects of LOC and depressive symptoms on state depression did not differ as a function of film ($ps>.10$).

Effects of LOC and film on cortisol

Figure 2c and 2d display trajectories of cortisol by film and LOC. AUC_g was positive, indicating participants, on average, showed a positive gain in cortisol over the experiment. Consistent with prior data showing food increases cortisol with a peak 20-40 minutes post-ingestion,¹⁹ buffet intake related to greater cortisol AUC_g ($p=.02$). There were no main effects on cortisol AUC_g for film, LOC in the past month, state LOC, depressive symptoms, or state depressed affect ($ps>.48$). Instead, there was an interaction of depressive symptoms by film ($p=.05$). In analyses stratified by film to interpret this interaction, depressive symptoms were positively related to cortisol AUC_g in the sad film ($B \pm SE$ 0.01 $\pm$ 0.004, $p=.029$) but not the neutral film ($p=.44$). Film did not interact with LOC in the past month, state LOC, or state depressed affect in predicting cortisol AUC_g ($ps>.51$).

The only predictor of cortisol AUC_i was sex; males had greater AUC_i than females ($d=0.2$, $p<.05$). There were no effects of film, LOC in the past month, state LOC, depressive symptoms, state depressed affect ($ps>.30$) or interactions by film on cortisol AUC_i ($ps>.36$).

Effects of LOC and film on eating in the absence of hunger

There was no effect of LOC in the past month or film on eating in the absence of hunger ($p=.89$). Instead, state LOC predicted more eating in the absence of hunger; every one-unit increase in state LOC while eating related to consuming 32 $\pm$ 12.3 kcal more ($p=.01$). This effect was not modified by film ($p=.84$). The only other predictor of eating in the absence of hunger was prior buffet intake ($B \pm SE$.07 $\pm$.03, $p=.02$).

Discussion

Adolescents with and without LOC eating did not differ in cortisol response to eating behavior before and after a sad versus neutral mood induction. Instead, trait depressive symptoms were associated with a greater cortisol response in the sad condition. Adolescents

with LOC were distinguished by greater increases in depressed affect during test meals than those without LOC. Further, state LOC related to eating more in the absence of hunger.

The current findings echo inconsistencies in the literature among adults with binge-eating. Some, but not all, studies have demonstrated that adults with binge-eating have higher basal cortisol and a hyper-reactive HPA-axis.⁹ These differences have not been observed consistently, with some investigations reporting a blunted response.¹⁰ Other individual-difference factors may explain these discrepancies. In the current study, adolescents with LOC had significantly more depressive symptoms than those without LOC, and it was depressive symptoms that showed an association with cortisol response. Trait depressive symptoms were related to greater cortisol response, only with an induction of sad mood. Thus, one explanation is that the negative affectivity elevated in adolescents with LOC is a more salient driver of biological, cortisol response to an induction of acute negative affect than LOC per se. Indeed, depressive symptoms are predictive of binge-eating onset in adolescents,²⁰ metabolic risk,^{21, 22} and have been repeatedly related to dysregulation in cortisol reactivity, with both hyperactivation and hyporesponsiveness observed depending upon measurement and sample characteristics.^{23, 24} It is also possible that cortisol abnormalities observed in adults with binge-eating are a consequence, as opposed to a cause, of repetitive overeating, concurrent depressive symptoms, and/or obesity.

Both trait-like and state assessments of LOC were important for state negative affect and for disinhibited, non-homeostatic eating. Even controlling for depressive symptoms, adiposity, and other confounds, adolescents with trait-like LOC had greater increases in depressed affect, despite no differences in basal depressed state. Further, perceived experience of state LOC while eating was associated with eating more palatable snack foods in the absence of hunger, after both a sad and neutral mood induction. These results are congruent with momentary affective models of binge-eating.⁵ Youth with uncontrolled eating clearly experience acute negative affect surrounding eating episodes, and momentary experiences of LOC relate specifically to more disinhibition, which is anticipated to promote excess weight gain.

It remains to be determined to what extent biologically-mediated stress systems play a role in developmental models of LOC and obesity/metabolic outcomes. Another possible explanation for null physiological differences in LOC may center on the type of stressor. While films are brief and easily administered, stressors that incorporate motivated performance with social-evaluative threat and uncontrollability produce a greater cortisol response,²⁵ with age and other factors complicating this picture further, particularly in youth.^{26, 27} Neuroscience data highlight that peer rejection and anxiety may be particularly salient physiological stressors for adolescents with LOC.²⁸ Future studies of biological responses to stress and eating warrant testing of alternative stressors.²⁹ Likewise, cortisol is an index of HPA-axis reactivity; other biological processes implicated in regulation of negative affect and eating may be relevant (e.g., sympathetic-adrenomedullary axis).⁸

Because a primary aim of the original study was to evaluate the effect of negative affect on eating, we evaluated cortisol response to a stressor embedded in a series of test meals. Although we accounted for energy intake, it is plausible that overall normative increases in

cortisol that accompany food intake¹⁹ masked LOC differences. We chose to examine four cortisol time points to minimize burden; more frequent sampling in a larger number of individuals with LOC may have permitted detection of LOC differences, for example, in timing of cortisol response to negative affect or intake. It would also be valuable to more carefully examine post-stress recovery.

Strengths of this study include use of standardized laboratory assessments of cortisol, trait and state-measures of LOC and depression, and direct estimation of body composition, as opposed to BMI. Limitations include reliance on a secondary-analysis data set with a small sample of adolescents with LOC, which may have limited power. Post-hoc power analyses suggest ~500 adolescents would be required for the observed effect of LOC on cortisol to reach statistical significance. We also had few boys, preventing examination of sex as a moderator. We did not assess sleep or wake time, which may be important for cortisol.³⁰

In conclusion, despite more subjective distress around eating and greater eating in the absence of hunger in adolescents with LOC, corresponding cortisol changes were not observed. In response to an experimentally-induced sad mood state, only adolescents with higher trait depressive symptoms had a more enhanced cortisol response. Future studies must determine if adolescents with LOC manifest alterations in stress reactivity to alternative stress-inducing situations.

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Abbreviations

ANCOVA	analysis of covariance
BMI	body mass index
HPA	hypothalamic-pituitary-adrenal
LOC	loss of control

References

1. Tanofsky-Kraff, M.; Yanovski, SZ.; Yanovski, JA. Loss of control over eating in children and adolescents.. In: Striegel-Moore SAW, R.; Walsh, BT.; Mitchell, JE., editors. Developing an evidence-based classification of eating disorders: scientific findings for DSM-5. American Psychiatric Association Press; 2011. p. 221-36.
2. Tanofsky-Kraff M, Yanovski SZ, Schvey NA, Olsen CH, Gustafson J, Yanovski JA. A prospective study of loss of control eating for body weight gain in children at high risk for adult obesity. *Int J Eat Disord*. 2009; 42:26–30. [PubMed: 18720473]
3. Tanofsky-Kraff M, Shomaker LB, Stern EA, Miller R, Sebring N, Dellavalle D, et al. Children's binge eating and development of metabolic syndrome. *Int J Obes*. 2012; 36:956–62.
4. Tanofsky-Kraff M, Shomaker LB, Olsen C, Roza CA, Wolkoff LE, Columbo KM, et al. A prospective study of pediatric loss of control eating and psychological outcomes. *J Abnorm Psychol*. 2011; 120:108–18. [PubMed: 21114355]
5. Arnow B, Kenardy J, Agras WS. Binge eating among the obese: a descriptive study. *J Behav Med*. 1992; 15:155–70. [PubMed: 1583679]
6. Haedt-Matt AA, Keel PK. Revisiting the affect regulation model of binge eating: a meta-analysis of studies using ecological momentary assessment. *Psychol Bull*. 2011; 137:660–81. [PubMed: 21574678]
7. Ranzenhofer LM, Hannallah L, Field SE, Shomaker LB, Stephens M, Sbrocco T, et al. Pre-meal affective state and laboratory test meal intake in adolescent girls with loss of control eating. *Appetite*. 2013; 68C:30–37. [PubMed: 23603224]
8. Vannucci A, Nelson EE, Bongiorno DM, Pine DS, Yanovski JA, Tanofsky-Kraff M. Behavioral and neurodevelopmental precursors to binge-type eating disorders: support for the role of negative valence systems. *Psychol Med*. 2015:1–16.
9. Gluck ME, Geliebter A, Hung J, Yahav E. Cortisol, hunger, and desire to binge eat following a cold stress test in obese women with binge eating disorder. *Psychosom Med*. 2004; 66:876–81. [PubMed: 15564352]
10. Rosenberg N, Bloch M, Ben Avi I, Rouach V, Schreiber S, Stern N, et al. Cortisol response and desire to binge following psychological stress: comparison between obese subjects with and without binge eating disorder. *Psychiatry Res*. 2013; 208:156–61. [PubMed: 23083917]
11. Ruttelle PL, Javaras KN, Klein MH, Armstrong JM, Burk LR, Essex MJ. Concurrent and longitudinal associations between diurnal cortisol and body mass index across adolescence. *J Adolesc Health*. 2013; 52:731–7. [PubMed: 23402983]
12. Doom JR, Gunnar MR. Stress physiology and developmental psychopathology: past, present, and future. *Dev Psychopathol*. 2013; 25:1359–73. [PubMed: 24342845]
13. Kelly NR, Shomaker LB, Pickworth CK, Grygorenko MV, Radin RM, Vannucci A, et al. Depressed affect and dietary restraint in adolescent boys' and girls' eating in the absence of hunger. *Appetite*. 2015; 91:343–50. [PubMed: 25936291]
14. Fairburn, CG.; Cooper, Z. The Eating Disorders Examination.. In: Fairburn, CG.; G.T., W., editors. Binge eating: nature, assessment, & treatment. Guilford; New York, NY: 1993. p. 317-60.
15. Glasofer DR, Tanofsky-Kraff M, Eddy KT, Yanovski SZ, Theim KR, Mirch MC, et al. Binge eating in overweight treatment-seeking adolescents. *J Pediatr Psychol*. 2007; 32:95–105. [PubMed: 16801323]
16. Beck, AT.; Steer, RA.; Brown, GK. Beck Depression Inventory. 2nd edn.. Psychological Corporation; San Antonio: 1996.
17. Pruessner JC, Kirschbaum C, Meinlschmid G, Hellhammer DH. Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology*. 2003; 28:916–31. [PubMed: 12892658]
18. Terry PC, Lane AM, Lane HJ, Keohane L. Development and validation of a mood measure for adolescents. *J Sports Sci*. 1999; 17:861–72. [PubMed: 10585166]
19. Al-Damluji S, Iveson T, Thomas JM, Pendlebury DJ, Rees LH, Besser GM. Food-induced cortisol secretion is mediated by central alpha-1 adrenoceptor modulation of pituitary ACTH secretion. *Clin Endocrinol*. 1987; 26:629–36.

20. Stice E, Presnell K, Spangler D. Risk factors for binge eating onset in adolescent girls: a 2-year prospective investigation. *Health Psychol.* 2002; 21:131–8. [PubMed: 11950103]
21. Hannon TS, Li Z, Tu W, Huber JN, Carroll AE, Lagges AM, et al. Depressive symptoms are associated with fasting insulin resistance in obese youth. *Pediatr Obes.* 2014; 9:e103–7. [PubMed: 24861610]
22. Shomaker LB, Tanofsky-Kraff M, Stern EA, Miller R, Zocca JM, Field SE, et al. Longitudinal study of depressive symptoms and progression of insulin resistance in youth at risk for adult obesity. *Diabetes Care.* 2011; 34:2458–63. [PubMed: 21911779]
23. Dockray S, Susman EJ, Dorn LD. Depression, cortisol reactivity, and obesity in childhood and adolescence. *J Adolesc Health.* 2009; 45:344–50. [PubMed: 19766938]
24. Keenan K, Hipwell A, Babinski D, Bortner J, Henneberger A, Hinze A, et al. Examining the developmental interface of cortisol and depression symptoms in young adolescent girls. *Psychoneuroendocrinology.* 2013; 38:2291–9. [PubMed: 23726646]
25. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol Bull.* 2004; 130:355–91. [PubMed: 15122924]
26. Gunnar MR, Talge NM, Herrera A. Stressor paradigms in developmental studies: what does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology.* 2009; 34:953–67. [PubMed: 19321267]
27. Stroud LR, Foster E, Papandonatos GD, Handwerker K, Granger DA, Kivlighan KT, et al. Stress response and the adolescent transition: performance versus peer rejection stressors. *Dev Psychopathol.* 2009; 21:47–68. [PubMed: 19144222]
28. Jarcho JM, Tanofsky-Kraff M, Nelson EE, Engel SG, Vannucci A, Field SE, et al. Neural activation during anticipated peer evaluation and laboratory meal intake in overweight girls with and without loss of control eating. *Neuroimage.* 2015; 108:343–53. [PubMed: 25550068]
29. Shankardass K, McConnell R, Jerrett M, Lam C, Wolch J, Milam J, et al. Parental stress increases body mass index trajectory in pre-adolescents. *Pediatr Obes.* 2014; 9:435–42. [PubMed: 24311567]
30. Ly J, McGrath JJ, Gouin JP. Poor sleep as a pathophysiological pathway underlying the association between stressful experiences and the diurnal cortisol profile among children and adolescents. *Psychoneuroendocrinology.* 2015; 57:51–60. [PubMed: 25889840]
31. Yanovski SZ, Yanovski JA, Gwirtsman HE, Bernat A, Gold PW, Chrousos GP. Normal dexamethasone suppression in obese binge and nonbinge eaters with rapid weight loss. *J Clin Endocrinol Metab.* 1993; 76:675–9. [PubMed: 8445025]
32. Lo Sauro C, Ravaldi C, Cabras PL, Faravelli C, Ricca V. Stress, hypothalamic-pituitary-adrenal axis and eating disorders. *Neuropsychobiology.* 2008; 57:95–115. [PubMed: 18552511]

WHAT IS ALREADY KNOWN ABOUT THIS SUBJECT

- Among adults with binge-type behaviors, both an exaggerated and blunted cortisol response to an acute laboratory stressor have been observed
- No studies have characterized cortisol reactivity to negative affect or food intake among adolescents with loss of control eating

WHAT THIS STUDY ADDS

- Adolescents who reported loss of control had more increases in depressed state affect during laboratory test meals, regardless of mood induction
- In a sad (versus neutral) mood induction, depressive symptoms, but not loss of control eating, related to a greater cortisol response
- State loss of control was associated with eating more in the absence of hunger, after a sad and neutral mood induction

Time	10-10:30 am	10:30 am	11-11:40 am	11:45-11:50 am	11:50 am	12-12:15 pm	12:20 pm	12:40 pm
Event	Rest period		Buffet lunch	Sad or neutral film exposure		Snacks array		
Cortisol or Food Intake Measure		Salivary cortisol (-80 ^l)	Meal total kcal/ macronutrient intakes		Salivary cortisol (0 ^l)	Snacks total kcal intake	Salivary cortisol (30 ^l)	Salivary cortisol (50 ^l)
Subjective Rating			Mood rating (pre-film)		Mood rating (post-film)		Mood rating	Mood rating

^lNumber in parentheses refers to minutes relative to film administration

Figure 1.
Flow of experimental paradigm.

Figure 2.

Geometric means and standard errors of state depressed affect and salivary cortisol throughout the experiment, depicted for youth without loss of control (LOC) eating (square) and with LOC eating (filled circle), adjusted for age, race, sex, puberty, fat mass, fat-free mass, height, buffet meal intake, depressive symptoms, state LOC, and basal state depressed affect (for cortisol only) among adolescents exposed to the sad film (Panel A/C) or neutral film (Panel B/D). Film had a main effect on state depressed affect measured as pre- to post-film change ($p < .001$), AUC_g ($p = .01$), and AUC_i ($p = .02$). LOC had a main effect on state depressed affect measured as pre- to post-film change ($p < .001$). The film by LOC interaction for depressed state affect was not significant, and there were no effects of film, LOC, or their interaction on cortisol.

Table 1

Characteristics of adolescents with and without loss of control (LOC) eating

Variable	LOC Eating ^a	No LOC Eating	t or χ^2	p-Value
<i>n</i>	38	140		
Age (y) ^b	15.4±1.3	15.3±1.4	0.37	.71
Sex (% female)	76.3	63.6	2.17	.14
Race (%)			6.15	.19
Non-Hispanic white	40	61		
Non-Hispanic black	37	23		
Hispanic	11	6		
Multiple or other	8	5		
Asian	5	6		
BMI (kg/m ²) ^{b,c}	26.4±8.4	23.9±6.5	1.91	.06
BMIz ^{b,d}	0.99±1.0	0.65±1.0	1.84	.07
Overweight or obese (%)	53	34	4.26	.04
Fat mass (kg) ^b	22.6±18.1	18.1±12.6	1.45	.15
Fat-free mass (%)	71.0±12.4	73.8±11.4	1.80	.07
Late puberty (%)	78	78	.00	.96
Depressive symptoms ^b	9.0±5.8	4.9±4.7	4.59	<.001
Buffet meal total intake (kcal) ^b	1290±564	1207±493	0.89	.37
Snack array total intake (kcal) ^b	341.1±157.2	311.5±151.5	1.06	.29

^a LOC=Loss of control eating assessed on the Eating Disorder Examination as presence of 1 episode in month prior to assessment.^b M±SD.^c BMI=body mass index.^d BMIz=body mass index standard deviation score for age and sex, calculated according to the Centers for Disease Control and Prevention 2000 growth charts.^{31, 32}