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Puberty and the Manifestations of Loss of Control Eating in Children and Adolescents

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Abstract

Objective—We investigated the manifestations of pediatric loss of control (LOC) eating at different stages of pubertal development.

Methods—Participants were a non-clinical sample of 468 youth (8–17y). Physical examination determined pubertal stage. LOC eating and disordered eating attitudes were assessed with the Eating Disorder Examination. In a randomized crossover design, a subset ($n=244$) ate *ad libitum* from two test meals designed to capture normal and LOC eating.

Results—There were no differences in the prevalence rates or frequency of reported LOC eating episodes across pubertal stages ($p=.50$). There were, however, puberty by LOC eating interactions in disordered eating attitudes and palatable food consumption ($p=.05$), even after adjusting for age and body composition. LOC eating was associated with elevated global disordered eating attitudes, weight concern, and shape concern in post-pubertal youth ($p=.001$), but not pre-pubertal youth ($p=.49$). In late-puberty, youth with LOC eating consumed less energy from protein ($p<.001$) and more from carbohydrate ($p=.003$) and snack-type foods ($p=.02$) than

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those without LOC eating, whereas endorsement of LOC eating in pre- or early-to-mid-puberty was not associated with differences in eating behavior ($p = .20$).

Conclusions—Findings suggest that puberty may be a critical risk period, when LOC eating behaviors in boys and girls may become accompanied by greater weight and shape concerns and more obesogenic food consumption patterns. Interventions for LOC eating during pre-puberty should be evaluated to determine if they are particularly beneficial for the prevention of exacerbated eating disorder psychopathology and adverse weight outcomes.

Keywords

Loss of control eating; binge eating; puberty; body image; weight and shape concern; eating disorders

Loss of control (LOC) eating refers to the subjective experience of being unable to control what or how much is eaten, regardless of the amount reportedly consumed (1). LOC eating has been well phenotyped, as LOC meals typically are low in protein, high in carbohydrate, and are comprised of highly palatable, energy dense foods (2–4). Pediatric LOC eating is a precursor to partial- and full-syndrome binge eating disorder (BED) and is predictive of exacerbated general and eating disorder psychopathology (5, 6), excessive weight and fat gain (7, 8), and metabolic dysfunction (9). At least one recent episode of LOC eating is reported by 5% to 50% of youth, with higher estimates among adolescent (vs. child), female (vs. male), and overweight (vs. non-overweight) samples (1). LOC eating typically emerges during middle childhood (10) or early adolescence (11, 12). While the presence of a lifetime history of LOC eating has been associated with adverse outcomes (6, 13), only about 50% of youth with LOC eating go on to experience persistent and exacerbated LOC eating patterns during middle to late adolescence and beyond (5, 6, 14). To improve the identification of high-risk youth with LOC eating and refine intervention targets, research is needed to elucidate developmental factors that may contribute to exacerbated disordered eating.

Puberty has been identified as a critical risk period for the development of eating disorders and their symptoms, including binge and LOC eating (15). Research on puberty and binge eating lags behind the wealth of data examining the relationship between puberty and other forms of eating disorder psychopathology (15). Pubertal onset, as identified by self-reports of secondary sex characteristics, has been associated with concurrent binge eating among preadolescent girls and boys assessed in the fifth through seventh grades (16, 17). When examining this preadolescent cohort of boys and girls over time, pubertal onset predicted subsequent increases in binge eating frequency (17). Binge eating prevalence was higher at more advanced stages of pubertal development among girls, but not boys, in a large population-based sample, even after accounting for age (18). However, other cross-sectional studies failed to find a significant association between pubertal status and binge eating in girls and boys (19–21). Prior studies' reliance upon self-report questionnaires to determine both pubertal stage and binge eating severity may have limited reliability and validity relative to interview methods, physical examination, and food intake assessed via laboratory test meals. Moreover, we know of no prior human studies investigating the relationship between pubertal stage and LOC eating.

Existing developmental risk models suggest that pubertal development may increase risk for LOC eating through direct and indirect pathways that involve biological and psychosocial factors. Traditional theories posit that the physical changes associated with puberty lead to body dissatisfaction and negative mood, which in turn promote disordered eating behaviors such as classic binge episodes or LOC eating (22, 23). It is hypothesized that girls may be at greater risk for these psychosocial disturbances, as puberty-induced increases in adiposity move girls away from the socially reinforced “thin” body size ideals whereas gains in muscle mass propel boys closer to masculine body ideals (22, 23). Recent theories, by contrast, focus on biological factors and suggest that increases in gonadal hormone levels during puberty play a central role in activating and/or organizing genetic and phenotypic effects that exacerbate disordered eating risk (15). It is also conceivable that pubertal shifts in metabolic hormone levels, which dually regulate eating behaviors and the hypothalamic-pituitary-gonadal axis, may directly promote LOC eating and/or the phenotypic manifestation of LOC eating (24, 25). To date, no known studies have examined the interplay among puberty, LOC eating, and theoretically-driven LOC correlates such as disordered eating attitudes and eating behavior.

The current study sought to investigate the association between pubertal development and manifestations of LOC eating in a community sample of children and adolescents. We hypothesized that youth in early-to-mid- and late-pubertal stages would exhibit higher rates of LOC eating than youth in pre-puberty. Based on the mechanisms put forth by psychosocial developmental risk models (22, 23), we hypothesized that associations between LOC eating and concerns about weight and shape would be more robust among post-pubertal youth relative to pre-pubertal youth. Given hypotheses made by biologically-driven developmental risk theories (15), we also expected post-pubertal youth with LOC eating to have greater palatable food consumption in the laboratory, even after adjusting for age and adiposity, relative to pre-pubertal youth with LOC eating and all youth without LOC eating.

Methods

Participants

Participants were children and adolescents who were enrolled in non-intervention protocols investigating eating behaviors in youth spanning a broad weight range (ClinicalTrials.gov Identifiers [ID]: NCT00320177, NCT00631644). Protocols were approved by the Institutional Review Board at the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development.

Participants were recruited through flyers posted on public bulletin boards at the National Institutes of Health (NIH), local libraries, supermarkets, and school parent email listservs in the Washington, DC greater metropolitan area. Youth enrolled in previous non-treatment studies were also invited to participate in the current study. Study flyers were mailed to parents of children within the studies' age range in counties within the Washington, DC metropolitan area. Notices requested participation of children for studies investigating eating behaviors in children and adolescents. Healthy boys and girls between 8 and 17 years with any BMI percentile were eligible for participation. Individuals were excluded if they (1) had

a significant medical condition; (2) had abnormal hepatic, renal, or thyroid function; (3) were taking medications known to impact body weight; (4) experienced greater than five pounds (2.3 kg) of weight loss in the past three months; (5) were undergoing weight loss treatment; or (6) had current suicidality or a psychiatric disorder (e.g., psychosis, bipolar disorder, major depression) that warranted a referral or hospitalization.

Procedures

Participants and a parent/guardian were seen during outpatient visits at the NIH Hatfield Clinical Research Center. Informed parental consent and child assent were obtained for all studies. Families understood that treatment would not be received, but that youth would be financially compensated for their participation. All participants underwent a medical history and a physical examination performed by an endocrinologist or nurse practitioner. Fasting anthropometric measures were taken. Children and parents completed questionnaires. Youth also participated in a semi-structured interview regarding eating disorder psychopathology. A subset of participants ($n = 244$) were scheduled for laboratory test meal visits at the NIH, as described previously (4). All study visits took place following an overnight fast.

Measures

Eating Disorder Psychopathology—The Eating Disorder Examination (EDE), versions 12.0D (26) and C.2 (27), was used to determine the presence or absence of objective binge episodes (OBE; consumption of an unambiguously large amount of food with a sense of loss of control) and subjective binge episodes (SBE; the experience of loss of control while consuming an ambiguously large amount of food) in the month prior to assessment. Consistent with prior research (28–30), the presence of LOC eating in the past month was coded if youth reported any OBEs, any SBEs, or both. The EDE has demonstrated very good inter-rater reliability and discriminant validity for all episode types (30–32).

The EDE also assessed disordered eating attitudes. The EDE generates four subscales from items different than those used for the OBE and SBE assessment, including restraint (cognitive and behavioral dietary restraint), and eating, shape, and weight concern, which average to create a global score of eating disorder psychopathology. The EDE has demonstrated good internal consistency, inter-rater reliability, and convergent and discriminant validity for the subscales and global score in samples of children and adolescents (13, 30).

Pubertal Stage—An endocrinologist or trained nurse practitioner performed a physical examination to determine pubertal stage (33). In boys, testicular volume (mL) was estimated by using a set of orchidometer beads as standards according to Prader (34). Breast development was assigned according to the five stages of Tanner (35, 36) using visual inspection augmented by palpation to separate lipomastia from gynecomastia as previously described (33). Testicular volume for males and Tanner breast staging for females were used to categorize youth as those in pre-puberty (males: testes < 3 mL; females: Tanner stage 1), early- to mid-puberty (males: testes 4–12 mL; females: Tanner stages 2–3), or late puberty

(males: testes >12 mL; females: Tanner stages 4–5). For cases in which the stage was discordant between the right and left testis/breast, the higher stage was assigned.

Body Composition—Participants' height and weight were measured in a fasting state using calibrated electronic instruments. BMI was calculated as weight in kilograms divided by the square of height in meters. BMI standard deviation (BMI-z) scores adjusted for age and sex were calculated according to the Centers for Disease Control and Prevention 2000 growth charts (37). Total fat mass (kg) and fat-free mass (kg) were measured by air displacement plethysmography (Bod Pod; Life Measurement Inc., Concord, CA) with participants wearing only underclothes or a form-fitting bathing suit, as recommended (38).

Laboratory Test Meals

Participants from one of the study protocols were asked to consume their lunch *ad libitum* from a buffet test meal on two separate days. The proportion of youth from this laboratory study represents over half of the current sample (52.1%; $n = 244$). In random order, youth participated in a "Normal" meal (at which they were instructed to "eat as much as you would at a normal meal") and an "LOC" meal (at which they were instructed to "let yourself go and eat as much as you want"). Apart from the instructions, test meal conditions were identical. On the day of each test meal, youth were provided with a standard 288-kcal breakfast (7% protein, 19% fat, 74% carbohydrate). Participants were observed at the NIH Clinical Center for the next six hours to ensure no calorie-containing foods or beverages were consumed and no moderate-to-vigorous activity was performed. Each participant was then presented with a multiple-item, 9,835-kcal food array varied in macronutrient composition (51% carbohydrate, 12% protein, 37% fat, across all foods) and containing a wide assortment of foods (39). These paradigms are standard, well-validated approaches in pediatric and adult samples (40, 41). All presented food items were weighed to the nearest 0.1 g before and after the test meal sessions. Energy content and macronutrient composition were calculated as previously described (4).

Analytic Plan

Data were screened for normality. Logarithmic transformations were made for total energy intake and arcsine transformations were made for the percentage of macronutrient content intake. Extreme, but plausible, outliers were recoded to fall 1.5 times the interquartile range below or above the 25th or 75th percentile so they were no longer outliers (42). This procedure places the outliers in the lower or upper 1–2% of the distribution. This *a priori* strategy was used because it minimizes outliers' influence on the characteristics of the distribution, minimally changes the distribution overall, and avoids potential bias associated with eliminating outliers altogether (42). No more than 3% of the data comprising any variable was considered an outlier. After adjusting outliers, skew and kurtosis were confirmed to be satisfactory for all variables.

The associations among puberty, reported LOC eating, and disordered eating attitudes were examined among the full sample ($N = 468$). Binary logistic regression models were used to investigate puberty (independent variable, coded as pre-puberty, early-to-mid-puberty, or late-puberty) as a predictor of the presence of LOC eating, OBEs, and SBEs (dependent

variables, coded as present or absent). Among the subset of youth with LOC eating ($n = 122$), general linear models were used to investigate the associations between puberty (independent variable) and the continuous frequency of LOC episodes, OBEs, and SBEs (dependent variables). A general linear model was conducted to examine the main and interactional effects of puberty and LOC eating status (independent variables) on the EDE subscales and global score (dependent variables). Covariates for all of these models included age (years), race (coded as non-Hispanic White or Other), sex (coded as male or female), height (cm), and fat mass (kg). Depression and anxiety symptoms were considered as covariates but their inclusion did not significantly contribute to any models or change the pattern of results.

For the subset of youth ($n = 244$) who participated in the laboratory test meals, linear mixed models with repeated measures were used to examine the main and interactional effects of puberty and LOC eating (independent variables) on key dimensions of observed eating behavior. Dependent variables included total energy intake (kcal; logarithm transformed), percent macronutrient content consumed (carbohydrate, fat, and protein; arcsine transformed), or total snack intake (kcal). The repeated measure in all models was meal instruction (Normal versus LOC). Covariates included age (years), race (coded as non-Hispanic White and Other), sex (coded as male or female), height (cm), fat-free mass (kg), and percent fat mass. Total energy intake was included as a covariate in the models examining percent macronutrient content intake. Meal type randomization (Normal First versus LOC First) was considered as a covariate but was removed because it did not significantly contribute to any model and did not improve overall model fit. In all models, subject was treated as a random effect to allow for within-subject correlation and the remaining independent variables were treated as fixed effects. Interactional effects among meal instruction, puberty, and LOC eating status were considered in all models, in light of prior data demonstrating differences in energy consumption between the Normal and LOC test meals (4).

Given consistent data showing that sex differences in disordered eating symptoms emerge during puberty (as reviewed by Klump, 15), we explored whether the relationships among puberty and LOC eating differed between girls and boys. Secondary analyses, therefore, included sex as a two-way (sex by puberty; sex by LOC eating) and three-way (sex by puberty by LOC eating) interaction term in all models. For significant three-way interactions, separate analyses for males and females were conducted.

All analyses were performed with SPSS version 20.0 (SPSS Inc., Chicago, IL, USA). The Benjamini and Hochberg (43) correction was applied to control for multiple comparisons. Main and interactional effects of puberty and LOC eating, were considered significant when the corrected p values were at or below .05. Follow-up analyses dividing the sample by pubertal status were conducted when interaction terms were significant. The Bonferroni-Hochberg correction was applied to each set of post hoc tests to correct for multiple comparisons in follow-up analyses. Similarly, post-hoc tests were considered significant when the corrected p values were at or below .05. All tests were two-tailed.

Results

468 children and adolescents (8 to 17 years; $M = 14.01$ years, $SD = 2.53$; 53.4% female) were studied. The racial/ethnic breakdown of the sample was 58.1% non-Hispanic White, 33.3% non-Hispanic Black or African American, 5.9% Hispanic/Latino, 2.3% Asian Origin, and 1.4% Multiple Races. The presence of LOC eating in the past month was reported by 26.1% ($n = 122$) of the sample. Youth with LOC eating reported a median of two LOC episodes in the past month. Participant characteristics across pubertal stages are presented in Table 1. There were no significant differences in demographic characteristics across pubertal stages ($ps = .06$), with the exception of age ($p < .001$), height ($p < .001$), and percent fat mass ($p = .01$).

Puberty and Reported LOC Eating – Full Sample (N = 468)

The associations between pubertal stage and the presence of LOC eating, OBEs, or SBEs in the past month, adjusting for age, sex, race, height, and fat mass, were not significant ($ps = .50-.76$; Table 1). Among youth with LOC eating ($n = 122$), no significant differences in the frequency of LOC episodes, OBEs, or SBEs across pubertal stages were revealed, adjusting for the same covariates ($ps > .20$; Table 1). Secondary analyses did not reveal any sex differences in the association between puberty and reported LOC eating, OBEs, or SBEs ($ps = .08$).

Consistent with prior studies (1, 15), significant main effects of puberty and LOC eating emerged for disordered eating attitudes. Youth in early-to-mid-puberty and in late-puberty reported greater global disordered eating attitudes than youth in pre-puberty, and also did not significantly differ from each other ($p = .02$). This pattern of results was the same for eating concern ($p = .003$), weight concern ($p = .007$), and shape concern ($p = .01$), but there was no significant main effect of puberty for restraint ($p = .48$). Youth with LOC eating reported greater global disordered eating attitudes than youth without LOC eating ($p < .001$). This pattern of results was the same for restraint, eating concern, weight concern, and shape concern ($ps < .001$).

There were significant interactional effects of puberty by LOC eating for global disordered eating attitudes ($p = .05$), weight concern ($p = .03$), and shape concern ($p = .01$) (Figure 1). Among youth in early-to-mid-puberty and in late-puberty, those with LOC eating reported higher global disordered eating attitudes ($ps = .003$), weight concern ($ps = .001$), and shape concern ($ps < .001$) than those without LOC eating. Among pre-pubertal youth, however, youth with LOC eating did not differ on these variables from those without LOC eating ($ps = .49-.99$). No significant interactional effects of puberty by LOC eating were revealed for restraint ($p = .39$) or eating concern ($p = .13$). In secondary analyses, sex did not serve as a moderator of the puberty by LOC eating interactions.

Puberty, LOC Eating, and Laboratory Eating Behavior – Sample Subset (n = 244)

Independent main effects of puberty (44) and LOC eating (4) for laboratory eating behavior across both test meals in this sample have been reported elsewhere. There were significant interactional effects of puberty by LOC eating for the percentage of energy consumed from

protein ($p = .02$) and carbohydrate ($p = .03$), adjusting for age, sex, race, height, percent fat mass, fat-free mass, and total energy intake (Figure 2). Among late-pubertal youth, those with LOC eating consumed a lesser percentage of energy from protein ($p < .001$) and a greater percentage of energy from carbohydrate ($p = .003$) than those without LOC eating. Among youth in pre-puberty and in early-to-mid puberty, those with LOC eating did not differ from those without LOC eating with regard to percent protein or carbohydrate consumption (p s = .13–.88). The puberty by LOC eating interaction was non-significant for percent fat intake ($p = .66$) and overall energy intake ($p = .20$). Neither meal instruction nor sex served as significant moderators of the puberty by LOC eating interactions in secondary analyses.

There was a significant puberty by LOC eating interaction for overall snack intake ($p = .004$). Late-pubertal youth with LOC eating consumed more energy from snack-type foods than those without LOC eating (LOC: $M = 509.0$, $SD = 357.8$ vs. No-LOC: $M = 401.9$, $SD = 312.1$ kcal; $p = .02$). However, there were no differences in snack consumption between those with and without LOC eating among pre-pubertal youth (LOC: $M = 354.1$, $SD = 230.1$ vs. No-LOC: $M = 373.2$, $SD = 217.7$ kcal; $p = .42$) and early-to-mid-pubertal youth (LOC: $M = 361.4$, $SD = 252.3$ vs. No-LOC: $M = 339.6$, $SD = 235.9$ kcal; $p = .46$). Meal instruction significantly moderated these findings at a trend level ($p = .057$), such that the puberty by LOC eating interaction persisted at the LOC meal ($p = .02$), but not the Normal meal (p s = .19). In secondary analyses, there was a significant sex by puberty by LOC eating interaction for total snack intake ($p = .01$). Specifically, the pattern of results observed in the puberty by LOC eating interaction remained significant among girls ($p = .006$), but not boys ($p = .35$) (Figure 3).

Discussion

The current study evaluated the manifestation of LOC eating at different pubertal stages in children and adolescents. We found no differences in the prevalence or frequency of reported LOC eating patterns across puberty. However, there were marked differences in disordered eating attitudes and palatable food consumption that emerged among post-pubertal youth with LOC eating. Specifically, in early-to-mid- and late-puberty, youth with LOC eating reported higher weight and shape concerns than those without LOC eating, whereas there were no reported differences in pre-pubertal youth with and without LOC eating. In late-puberty, and independent of age and body composition, endorsement of LOC eating was associated with consumption of less energy from protein and more from carbohydrate and snack-type foods. Overall, findings suggest that puberty is associated with body image disturbances and obesity-promoting eating behaviors in boys and girls with LOC eating.

The prevalence of reported LOC eating (~26%), objective binge episodes (~13%), and subjective binge episodes (~20%) did not differ across pubertal stages of development. These findings are consistent with retrospective data indicating that the presence of LOC eating often emerges during middle childhood (10), which is distinct from other forms of disordered eating behaviors (e.g., overly restrictive eating practices, compensatory behaviors) that are rarely seen prior to adolescence (45, 46). A number of psychosocial risk

factors for binge and LOC eating onset during childhood have been identified in prospective (5, 47) and case control (48) studies that may account for this distinction, including emotional eating, weight-related teasing, parental underinvolvement, and the critical life event of changing schools. Among all the youth with LOC eating in our study, there were no differences in self-reported LOC episode frequency across pubertal stages. While these data are contrary to findings that binge eating severity is greater among youth in advanced pubertal stages (16–18), they support other studies that reported no direct association between puberty and binge eating (19–21). It is also possible that the self-reported frequency of LOC episodes is not a reliable marker of severity during childhood and adolescence. Indeed, several studies report that the mere presence of LOC eating is associated with (4, 28–30), and predictive of (5–9), adverse outcomes. Studies are needed to evaluate biologically-based developmental risk factors for the emergence of LOC eating during childhood, as well as alternative measures of LOC severity.

LOC eating was associated with elevated weight and shape concerns in post-pubertal youth but not pre-pubertal youth, which is consistent with hypotheses put forth by developmental risk theories of eating disorders (15, 23). Body dissatisfaction during middle childhood does not predict LOC eating outcomes in early adolescence (6), whereas body dissatisfaction during early-to-middle adolescence consistently predicts LOC eating onset and persistence in late adolescence and young adulthood (49). In light of these prospective data and the current findings, we put forth the intriguing possibility that the causal relationship between body image disturbances and LOC eating may emerge during puberty. According to psychosocial developmental risk theories (22, 23, 50), the co-occurrence of salient stressors during adolescence, including puberty-related body changes, poor body esteem, and dating onset, interact to cause negative mood states and eating disturbances. Notably, associations among puberty, weight and shape concerns, and LOC eating persisted even after controlling for age, body composition, and general psychopathology, which may support emerging evidence that puberty-induced genetic and hormonal effects exert significant influence on eating disorder risk (15). Multi-modal studies are required to evaluate biopsychosocial factors that may account for the observed associations between body image disturbances and LOC eating during puberty.

Puberty was associated with observed differences in palatable food consumption among youth with and without LOC eating, even after adjusting for body composition, age, and sex. Among late-pubertal youth, endorsement of LOC eating was associated with consumption of less energy from protein and more energy from carbohydrate across both normal and LOC test meals. However, there were no such differences in intake among pre- and early-to-mid-pubertal youth with and without LOC eating. Findings from the current study are comparable to a prospective study examining puberty-induced differences in binge eating proneness among female rats (51). Although such animal models have limitations for predicting results found in human studies, the similarities between animal and human studies support the notion that genetic and neuroendocrine risk factors for disordered eating may emerge during puberty (15), since rats are immune to psychosocial influences that promote LOC eating during human adolescence (25). Findings underscore the utility of laboratory paradigms to characterize the phenotype of LOC eating across stages of development, particularly in the absence of validated self-report measures of LOC severity for use in

children and adolescents. However, validation and extension of our cross-sectional observations through longitudinal studies are needed.

The effects of a common single nucleotide polymorphism (rs9939609) of the *FTO* (fat mass and obesity-associated) gene may serve as one compelling genetic mechanism that accounts for the puberty-related differences in the phenotypic manifestation of LOC eating. The presence of rs9939609 *FTO* risk alleles is more common among youth with LOC eating (52), associated with fat intake and snack consumption (52, 53), and modulates neural responses in homeostatic and food reward regions upon exposure to palatable food images (54). Alterations in leptin and ghrelin signaling, which are metabolic hormones involved in both energy homeostasis and reproductive functioning (24), have been linked to the rs9939609 *FTO* risk alleles (54, 55) and implicated in LOC eating and BED (25). Furthermore, the influence of the rs9939609 *FTO* risk alleles on body weight shifts throughout childhood and may peak in late adolescence (55). Taken together, it is conceivable that youth who are genetically prone to LOC eating may begin to exhibit obesogenic eating behaviors more consistently across all meals during late-puberty. Research is required to evaluate whether pubertal variation in the expression of the *FTO* gene influences risk for LOC eating and BED via the proposed hormonal and neural mediators.

Sex differences in the prevalence of LOC eating and BED in adolescence are much more modest (3:2 ratio of females to males) relative to other forms of disordered eating (1, 56). It therefore was unsurprising that few puberty-related sex differences in the manifestations of LOC eating emerged in the current study. Among girls only in the current study, late-pubertal youth with LOC eating consumed more energy from snack-type foods than those without LOC eating. These findings were only observed at the test meal designed to capture LOC-like eating, suggesting that girls with LOC eating behave differently at an LOC meal during late-puberty. These data are consistent with findings from human and animal studies suggesting that ovarian hormones mediate binge eating behaviors (15). Indeed, estrogen regulates gene transcription for serotonergic signaling pathways and the production of brain-derived neurotrophic factor, which have been implicated in the etiology of binge-type eating disorders (15). Estrogen also exerts organizational effects on brain reward neural circuitry, including opioid and dopaminergic neurotransmission, which may be linked to increases preferences for palatable snack foods that distinguish the LOC eating phenotype (15). Future data are required to explore alternative biological mechanisms that could exacerbate LOC eating throughout pubertal development in both boys and girls, such as changes in leptin concentration (57) or in brain regions that enhance stress reactivity (58), to inform developmental risk models of binge-type eating disorders.

The current study has several notable strengths, including a fairly large and diverse community sample of boys and girls spanning a broad age and weight range, use of semi-structured clinical interviews to assess LOC eating, use of air displacement plethysmography to measure body composition, and the determination of pubertal status by physical examination. All analyses controlled for age and adiposity to increase confidence that findings were related to pubertal stage rather than other extraneous confounds. However, the cross-sectional design limits our capacity to make causal inferences about the

effects of pubertal development on LOC eating. It is possible that participants would exhibit changes in their own LOC eating patterns as they advance through puberty. Longitudinal studies investigating developmental risk factors for LOC eating are warranted.

The use of a randomized crossover design to assess eating behavior at two laboratory meals represents another important strength. Laboratory studies of eating behavior have several benefits, including the ability to control the conditions, precisely measure energy intake, and avoid recall biases (59). Participants' intake during test meal studies have been shown to reflect their eating behavior outside the laboratory (60, 61). However, the large multi-item buffet may have promoted increased energy intake. Conversely, social desirability effects may have been associated with decreased energy intake. Although two distinct test meal instructions were designed to capture normal and LOC-like eating, inferences regarding the actual experience of LOC eating are limited because there were no measures assessing youth's experience of LOC during or after each test meal. Therefore, future studies of puberty and LOC eating would benefit from incorporating naturalistic test meal designs, measures of the LOC experience during test meals, and ecological momentary assessment to capture LOC eating behaviors.

Overall, findings from the current study suggest that no pubertal developmental stage independently promotes emergence of LOC eating. However, puberty appears to be associated with more body image disturbances and obesity-promoting eating behaviors in youth with LOC eating. In light of these data, interventions for LOC eating during pre-puberty should be evaluated to determine if they are particularly beneficial for the targeted prevention of exacerbated disordered eating pathology and excessive weight gain.

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Figure 1. Association between Loss of Control (LOC) Eating and Global Disordered Eating Attitudes Across Pubertal Stages

Disordered eating attitudes were assessed with the Eating Disorder Examination (26; 27).

Among youth in early-to-mid-puberty and in late-puberty, those with LOC eating reported higher global disordered eating attitudes than those without LOC eating, adjusting for age, sex, race, height (cm), and fat mass (kg) ($p < .001$). Pre-pubertal youth reported similar global disordered eating attitudes, regardless of LOC status ($p = .53$). This pattern of results was the same for weight concern ($p = .03$) and shape concern ($p = .01$).

Figure 2. Macronutrient Consumption across Normal and Loss of Control (LOC) Test Meals in Youth with and without LOC Eating at Different Pubertal Stages

Among late-pubertal youth, those with LOC eating consumed a lower percentage of energy from protein ($p < .001$) and a greater percentage of energy from carbohydrate ($p = .003$) than those without LOC eating, adjusting for age, sex, race, height (cm), fat mass (%), fat-free mass (kg), and total energy intake (kcal, logarithm transformed). Among youth in pre-puberty and early-to-mid-puberty, there were no significant differences between those with and without LOC eating with regard to percent protein or carbohydrate consumption ($ps > .05$).

13). Endorsement of LOC eating was not associated with percent fat intake at any pubertal stage ($ps > .14$).

Figure 3. Consumption of Snack-Type Foods at the Loss of Control (LOC) Test Meal

A significant Puberty by LOC eating interaction among girls ($p = .006$), but not boys ($p = .35$), adjusting for age, race, height (cm), fat mass (%), and fat-free mass (kg). Among girls in late-puberty, those with LOC eating consumed more energy from snack-type foods than those without LOC eating ($p = .001$), whereas there were no differences in snack intake among pre- and early-to-mid-pubertal girls with and without LOC eating ($ps = .13$). Among boys, there were no significant differences between youth with and without LOC eating at any pubertal stage ($ps = .14$).

Table 1

Participant Characteristics Across Pubertal Stages.

	Pre-Puberty (<i>n</i> = 55)	Early- to Mid-Puberty (<i>n</i> = 122)	Late-Puberty (<i>n</i> = 291)	
Variable	M (SD)	M (SD)	M (SD)	<i>p</i> value
Age (years)	9.67 ^a (1.36)	12.50 ^b (2.07)	15.37 ^c (1.45)	<.001
BMI-z	0.43 (1.08)	0.70 (1.18)	0.82 (1.05)	.06
Height (cm)	138.03 ^a (9.94)	154.97 ^b (11.38)	167.30 ^c (8.67)	<.001
Fat Mass (%) ¹	20.08 ^a (10.94)	26.66 ^b (12.02)	26.41 ^b (12.20)	.01
LOC Episode Frequency ²	1.92 (1.24)	3.07 (4.87)	3.17 (3.22)	.55
OBE Frequency ²	0.58 (0.90)	1.77 (5.11)	1.08 (2.04)	.27
SBE Frequency ²	1.33 (1.30)	1.29 (1.01)	2.09 (2.07)	.48
	% (n)	% (n)	% (n)	
Race (% White)	49.1 (27)	59.0 (72)	59.4 (173)	.39
Sex (% Female)	43.6 (24)	52.5 (64)	55.7 (162)	.24
LOC eating (% Present)	27.3 (15)	28.7 (35)	24.7 (72)	.72
OBEs (% Present)	12.7 (6)	13.9 (17)	11.3 (33)	.76
SBEs (% Present)	18.2 (10)	22.1 (27)	17.2 (50)	.50

Notes: Superscripted letters (a, b, c) that differ represent significant differences between the pubertal stages using a Bonferroni-Hochberg post hoc test. Abbreviations include: BMI = Body mass index (kg/m²); LOC = Loss of control; OBE = Objective binge episode; SBE = Subjective binge episode.

¹ Adjusted for height (cm);

² Analyses conducted only among youth with LOC eating (*n* = 122).