Examining two prevailing models of loss of control eating among community-based girls

Andrea B. Goldschmidt, Ph.D.[1], **Jason M. Lavender, Ph.D.**[2], **Alison E. Hipwell, Ph.D.**[3,4], **Stephanie D. Stepp, Ph.D.**[3,4], and **Kate Keenan, Ph.D.**[5]

[1]Department of Psychiatry and Human Behavior, Warren Alpert Medical School of Brown University/The Miriam Hospital, Providence, RI, USA

[2]Department of Psychiatry, University of California, San Diego, La Jolla, CA, USA

[3]Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA

[4]Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

[5]Department of Psychiatry and Behavioral Neuroscience, The University of Chicago, Chicago, IL, USA

Abstract

Objective—Cognitive-behavioral and interpersonal models of loss of control (LOC) eating have been under-explored in adolescents.

Methods—Using data from community-based adolescent girls assessed annually over 4 years, we examined the cognitive-behavioral ($n = 416$) and interpersonal ($n = 418$) models using a regression-based bootstrapping approach.

Results—Body dissatisfaction at 14 years prospectively predicted LOC eating at 18 years, both directly (direct effect = −0.039; *S.E.* = 0.017; $p = .02$) and indirectly via dieting (indirect effect = −0.010; 95% CI = −0.022 to −0.003). Interpersonal functioning at 14 years was negatively associated with negative emotionality at 17 years, which in turn was prospectively associated with LOC eating at 18 years (indirect effect = 0.001; 95% CI = −0.001 to −0.0003); however, the direct association between age 14 interpersonal functioning and age 18 LOC eating was not significant (direct effect = −0.001; *S.E.* = 0.001; $p = .47$).

Conclusions—These findings support the cognitive-behavioral model, and partially support the interpersonal model, with the latter findings implying that over time, negative emotionality may promote LOC eating, independent of the effects of prior social functioning. Prevention and early intervention efforts for LOC eating may benefit from integrating these theoretical frameworks.

Corresponding author: Andrea B. Goldschmidt, Department of Psychiatry and Human Behavior, Alpert Medical School of Brown University, The Miriam Hospital/Weight Control and Diabetes Research Center, 196 Richmond St., Providence, Rhode Island, 02903; TEL: (401) 793-8251; FAX: (401) 793-8944; andrea.goldschmidt@lifespan.org.

Disclosure of potential conflicts of interest: The authors declare that they have no conflict of interest.

Introduction

Loss of control (LOC) while eating, characterized by feeling that one cannot control what or how much one is eating, is a key feature of binge eating and is a transdiagnostic eating disorder construct (1). LOC eating is associated with significant distress and impairment independent of the amount of food consumed, and predicts excess weight gain as well as the development of full-syndrome eating disorders (2). LOC eating typically develops during mid- to late-adolescence (3), and 10–15% of adolescent boys and girls in population-based samples report engaging in this behavior (2). Although boys and girls of all racial/ethnic backgrounds are affected (4), gender and race/ethnicity may be associated with unique patterns of onset and maintenance related to variability in risk and maintaining factors (5, 6). Cognitive-behavioral and interpersonal models are two prevailing theories in the literature that explain the development and maintenance of LOC eating via different core constructs (7, 8), and inform current treatment recommendations (9). However, there are limited prospective data to support these models in relation to risk and maintenance of LOC eating in adolescent females, particularly those belonging to racial minority groups, impeding understanding of their relevance to prevention and early intervention efforts.

The cognitive-behavioral model suggests that overvaluation of shape, weight, and control over eating (meaning that shape, weight, and control over eating constitute one of the primary means upon which one bases one's self-worth) contributes to stringent and inflexible dieting behaviors and related attitudes/cognitions associated with body shape/weight (e.g., attempts to follow strict rules about eating). LOC eating is posited to occur when such attempts are interrupted (e.g., by breaking a dietary rule), a causal link that is compounded by the experience of stressful events and negative affect (7) either through an interactive or mediational effect (e.g., stressful events and negative affect co-occur with interruptions in dietary restraint to promote LOC eating, or interruptions in dietary restraint lead to the experience of stress and/or negative affect which promotes subsequent LOC eating, respectively). Indeed, laboratory-based and naturalistic data suggest that shape-, weight-, and dietary-related factors predict the *proximal occurrence* of LOC eating in adult females (e.g., 10, 11), but these studies fail to inform an understanding of how they influence *risk* for LOC eating development/maintenance over time. One study testing a cognitive-behavioral model of LOC eating in boys and girls (ages 10–16) using a cross-sectional design (12) revealed significant associations between appearance-related concerns and dietary restraint, the latter of which in turn was associated with LOC eating. Furthermore, related conceptualizations that incorporate constructs that overlap with the cognitive-behavioral model, including body dissatisfaction, dieting, and negative affect (e.g., the dual-pathway model; 13), have received support in the literature. However, these models have been under-explored prospectively in adolescent girls (14, 15), especially among Black girls who may be less likely to endorse appearance-related concerns and dieting behaviors (6).

Alternatively, the interpersonal model suggests that interpersonal stressors and/or dysfunction precipitate negative emotionality (a dispositional proclivity to experience distressing emotions), which results in LOC eating as a maladaptive means of coping with momentary distress (16). Both interpersonal problems (17) and negative affect (18) appear to be prospective risk factors for LOC eating in adolescent and young adult females, and have been identified as momentary precipitants of LOC eating (19, 20), suggesting that they are involved in the maintenance of LOC eating as well. Indeed, our group has previously demonstrated that negative emotionality precipitates LOC eating in adolescent girls (an association that is mediated by emotion regulation deficits; 21). Findings also suggest that negative emotionality mediates the association between interpersonal problems and LOC eating in women (22, 23) and boys and girls ages 8–17 (24), although existing evidence is based on cross-sectional data. The interpersonal model may be particularly relevant to Black girls, given its focus on social constructs (e.g., stigmatization) and related distress (25).

Despite support from cross-sectional and prospective studies of individual risk/maintenance factors, research evaluating the full cognitive-behavioral or interpersonal models of LOC eating has been limited, particularly in adolescent girls. Since full-syndrome eating disorders involving LOC are relatively rare in children and adolescents, a logical first step in providing such evidence is to examine the validity of these two models of eating disorder development and maintenance in relation to LOC eating, a prodromal symptom of eating disorders (26). As such, the overall aim of this investigation was to examine each model using longitudinal data collected in a community-based study of girls followed throughout adolescence. Specifically, this study sought to test the direct and indirect effects of 1) body image (age 14) on LOC eating (age 18) via dieting (age 16), consistent with the cognitive-behavioral model; and 2) interpersonal functioning (age 14) on LOC eating (age 18) via negative emotionality (age 17), consistent with the interpersonal model. We hypothesized that both models would be supported in relation to risk for LOC eating onset and/or maintenance in late adolescence.

Methods

Participants

Participants were enrolled in the Pittsburgh Girls Study, a community-based prospective study of psychosocial functioning from childhood to adulthood (27). Girls were identified for the first wave of data collection when they were between 5 and 8 years of age via a stratified, random household sampling method, which included oversampling of households in low-income neighborhoods (28). During 1998–1999, all homes in neighborhoods in which at least 25% of the residents were living at or below the poverty level and a randomly selected 50% of the households in non-risk neighborhoods were contacted in person to determine if an age-eligible girl was living in the home. A total of 3,118 households in which an age-eligible girl resided were identified (which represented 83.7% of all age-eligible girls in contacted neighborhoods, according to Census data), of which 2,450 agreed to participate and were enrolled in the first assessment wave. In homes in which more than one eligible girl resided, one girl was randomly selected for participation. Girls and their caregivers were then re-assessed annually in the home by trained interviewers.

A total of 588 girls completed measures relevant to the study aims at ages 14–18 years (spanning assessment waves 10–14 of the study). Girls were assessed in person, and questionnaires were read by interviewers from a laptop screen. The girls were, on average, 14.5 years of age at the wave 10 assessment, most were Caucasian ($n = 253$; 43.0%) or African-American ($n = 302$; 51.4%; see Table 1 for descriptive characteristics of the full sample of 588 girls), and 41.3% ($n = 209$; status unknown for 82 participants) were from families receiving public assistance (i.e., Women, Infants, and Children, food stamps, welfare). Retention ranged from 85–86% in assessment waves 10–14, with attrition due in part to difficulty tracking participants. There was very little attrition due to refusal to participate. For the current analyses, only participants with complete data for all covariates and measures across the time points were included in the analyses (ns = 416–418).

All study procedures were approved by the University of Pittsburgh Human Research Protection Office and were conducted in accordance with the Declaration of Helsinki. All primary caretakers provided written informed consent, and verbal assent was obtained from the girls starting at age 10. At age 18 years, girls provided their own written, informed consent. Girls and their caregivers were reimbursed for their time completing the interviews.

Measures

Interviewer-measured height and weight were used to determine body mass index (BMI; kg/m^2) at ages 14, 16, 17, and 18 years. Girls' age, race, and socioeconomic status (SES) were derived from parent reports.

The Eating Attitudes Test-26 (EAT-26; 29), a 26-item self-report measure of eating disturbance, was administered at ages 14, 16, and 18 years to assess LOC eating and dieting. LOC eating was ascertained at ages 14 and 18 via one item asking the respondent how often, "I have gone on eating binges where I feel that I might not be able to stop." Dieting was assessed at age 16 years via a 13-item subscale (current $\alpha = .88$) inquiring about relevant behavioral (e.g., "I avoid foods with sugar in them") and cognitive/attitudinal symptoms (e.g., "I am terrified about being overweight"). Response options for the LOC and dieting items included "never," "rarely," "sometimes," "often," "very often," and "always."

Body image was assessed via figure drawings administered at age 14 years (30). Participants were shown images of an adolescent in a bathing suit as she would appear at seven different sizes (ranging from extremely thin to obese), and asked to select the image that most resembled herself, and, separately, her ideal size. Body dissatisfaction was approximated by calculating the difference between self and ideal body shape, such that more negative scores reflect greater dissatisfaction, consistent with the previous literature (31).

The 38-item Social Skills Rating System-Secondary Student Form (SSRS; 32) was completed by adolescents at age 14 years to assess interpersonal functioning. The SSRS measures cooperation (e.g., helping with household tasks), self-control (e.g., controlling one's temper during conflict), assertion (e.g., introducing oneself to peers), and empathy (e.g., feeling sorry for others). Items are scored on a 3-point rating scale ranging from 0 ("never") to 2 ("often"), with lower scores reflecting poorer interpersonal functioning. As interpersonal functioning is conceptualized broadly in the interpersonal model of LOC

eating (16), the SSRS total scale score was utilized in analyses, consistent with previous validation studies (current α = .90; 33, 34).

The neuroticism scale of the Neuroticism-Extraversion-Openness Five Factor Inventory-3 (NEO-FFI-3; 35), a self-report personality assessment, was administered at age 17 years. The neuroticism subscale contains 12 items inquiring about the respondent's tendencies towards <u>negative emotionality</u> (e.g., "I am not a worrier," "sometimes I feel completely worthless"), each rated on a 5-point scale ranging from "strongly disagree" to "strongly agree" (current α = .71).

Statistical Analysis

All data were analyzed in SPSS 22.0. Chi-square analyses and t-tests were used to evaluate demographic and psychosocial factors associated with age 18 missing LOC eating data (n = 90; 15.3%). Natural log transformations were applied to age 14 and age 18 LOC eating frequency to correct for positive skew. A regression-based bootstrapping approach with k = 10,000 re-samples and 95% bias-corrected confidence intervals (CIs) using the PROCESS macro (36) in SPSS was used to evaluate the indirect effects of 1) age 14 body dissatisfaction on age 18 LOC eating through age 16 dieting (n = 416); and 2) age 14 interpersonal functioning on age 18 LOC eating through age 17 negative emotionality (n = 418). This approach has been recommended as a powerful method for estimating indirect effects, even in large samples (37). Indirect effect estimates were considered significant if the associated 95% CIs did not contain zero.

Missing item-level data were imputed using scale means when <33% of items on a given questionnaire were completed; when 33% of item-level data were missing, the respective scale was considered incomplete. Since the SPSS PROCESS macro cannot accommodate missing data, participants who had incomplete data on any of the measures included in each model were excluded from that respective model. Overall, missing scale-level data ranged from 14 to 18% for each measure across the time points: at age 14, missing data for each of the covariates and the independent variables for each model ranged from approximately 14 to 18% (missing public assistance data=14.0%; missing BMI data=18.4%; missing LOC data=17.9%; missing SSRS data=14.6%; and missing body dissatisfaction data=14.6%); at age 16 (cognitive-behavioral model) 16.3% of dieting data were missing; at age 17 (interpersonal model) 16.3% of negative emotionality data were missing; and at age 18 15.3% of LOC data were missing. As demographic and anthropometric factors may influence patterns of onset/maintenance factors for LOC eating in girls (38, 39), both models adjusted for age 14 race, SES, and BMI (as a continuous variable), as well as age 14 LOC eating.

Results

Descriptive Characteristics

Table 1 provides descriptive information on the sample, including frequency of LOC eating. Individuals who were missing data on LOC eating were more likely to be Caucasian than those with complete LOC data [χ^2 (N = 588) = 7.10; p = .03]. Missing data were not

significantly associated with age 14 SES, BMI, body dissatisfaction, or interpersonal functioning, age 16 dieting, or age 17 negative emotionality.

Cognitive-Behavioral Model

Results of both indirect effect models are presented in Table 2. Findings from the first analysis revealed a significant indirect effect of age 14 body dissatisfaction on age 18 LOC eating through age 16 dieting (estimate = −0.010; 95% CI = −0.022 to −0.003; effect ratio = .20), controlling for age 14 race, SES, BMI, and LOC eating, such that body dissatisfaction at age 14 was positively associated with dieting at age 16, which in turn was positively associated with LOC eating at age 18. The total effect of age 14 body dissatisfaction on age 18 LOC eating was significant (estimate = −0.049; *S.E.* = 0.017; *p* = .003), as was the direct effect (estimate = −0.039; *S.E.* = 0.017; *p* = .02).

Interpersonal Model

A significant indirect effect of age 14 interpersonal functioning on age 18 LOC eating through age 17 negative emotionality was also found (estimate = 0.001; 95% CI = −0.001 to −0.0003; effect ratio = .52), controlling for age 14 race, SES, BMI, and LOC eating frequency, such that interpersonal functioning at age 14 was negatively associated with negative emotionality at age 17, which in turn was positively associated with LOC eating at age 18. However, the total effect of age 14 interpersonal functioning on age 18 LOC eating was not significant (estimate = −0.002; *S.E.* = 0.001; *p* = .14), nor was the direct effect of age 14 interpersonal functioning on age 18 LOC eating (estimate = −0.001; *S.E.* = 0.001; *p* = .47).

Discussion

The current study was designed to provide a preliminary evaluation of two widely recognized but under-examined theories of LOC eating in adolescents. In support of the cognitive-behavioral model (7), dieting scores at age 16 accounted for the significant association between body dissatisfaction at age 14 and LOC eating at age 18. These results suggest that greater body dissatisfaction prospectively predicts greater LOC eating both directly and via increased dieting behavior. For the interpersonal model (8), the prospective association between age 14 interpersonal functioning and age 18 LOC eating was not significant; however, there was a significant indirect effect via negative emotionality at age 17. Interpersonal functioning at age 14 was negatively associated with negative emotionality at age 17, and in turn negative emotionality was positively associated with LOC eating at age 18. Thus, poorer social functioning may be associated with future LOC eating through negative emotionality, and negative emotionality may sustain LOC eating behavior, independent of the effects of prior social functioning.

The cognitive-behavioral and interpersonal models inform current treatment recommendations for individuals who already exhibit LOC eating behaviors (9), but our findings suggest that prevention efforts could be improved by integrating these theoretical frameworks as well. Although it is unclear from the current data whether different subgroups of girls demonstrate distinct patterns of onset (i.e., a moderated-mediation model), or

whether both models operate simultaneously or sequentially within the same individual, more broadly integrating appearance-related and interpersonal themes (e.g., self-control, assertion, insofar as they may improve later negative emotionality) into preventive interventions could address both possibilities. Existing prevention programs targeting eating disorders (e.g., dissonance-based and cognitive-behavioral interventions; 40) typically incorporate interventions addressing body dissatisfaction, dieting, and negative emotionality, but few have focused on interpersonal functioning, and most have been delivered to college-aged populations, thus potentially bypassing a peak period for the emergence of risk factors. However, given that the current study assessed interpersonal functioning broadly, future research should explore specific interpersonal domains that could be incorporated into such interventions. Since LOC eating is associated with the onset of full-syndrome bulimia nervosa and binge eating disorder (26), designing prevention and early intervention programs targeting this behavior may be an expedient way to impede the development of multiple binge eating-related disorders.

This study had several important strengths, particularly the large, diverse, community-based sample, and the use of longitudinal data. However, several limitations warrant mention. First, given the limited existing longitudinal data on cognitive-behavioral and interpersonal models in young people, the aim of this investigation was to provide preliminary support through separately evaluating these models, rather than examining an integrated framework or conducting a head-to-head comparison. However, based on the findings reported herein, which support prospective associations (either direct or indirect) of body dissatisfaction, dieting, interpersonal functioning, and negative emotionality with onset/maintenance of LOC eating, these alternative approaches would be useful directions for future research by elucidating how much variance in the onset/maintenance of LOC eating is accounted for by these constructs. Second, because this was a large study, self-report assessments within the context of in-person interviews were used to assess psychological constructs. These included including dieting, which is often conceptualized as a multidimensional construct involving both attempted and actual restriction; LOC eating, which was operationalized using a single questionnaire item assessing current patterns that did not specify onset or duration (which could have resulted in overlapping responses across time points, i.e., the same LOC episodes could have been reported in multiple years of the study); and interpersonal functioning, which was broadly defined as one general construct and thus should be assessed in a more nuanced manner in future studies to determine the relevance of particular domains (e.g., interpersonal mistrust, social anxiety) in relation to risk for LOC eating. The use of self-report to ascertain the presence of these psychological constructs could have introduced measurement bias, and thus results should be replicated using more rigorous investigator-based assessments. Third, given the small number of participants endorsing LOC eating at age 18, we were unable to examine the validity of the cognitive-behavioral and interpersonal models in relation to new onset LOC eating (although LOC eating at age 14 was covaried in all analyses, which somewhat addresses this issue). Indeed, the assessment intervals spanned middle to late adolescence, with analyses including predictors of LOC eating at age 18, bypassing the peak age of onset for LOC eating (3). Thus, results are best characterized as describing models of risk and/or maintenance of LOC eating. Fourth, although assessments were administered longitudinally, due to the absence of a measure of negative emotionality

at age 14, we were unable to examine whether *changes* in negative emotionality from age 14 to 17 mediated the prospective association between age 14 interpersonal functioning and age 18 LOC eating (and, to be consistent across models, to assess whether changes in dieting from ages 14 to 16 mediated the prospective association between age 14 body dissatisfaction and age 18 LOC eating). Therefore, mechanisms of change related to the mediator could not be determined. Similarly, in the absence of an earlier measure of negative emotionality, the reverse direction of causality, whereby negative emotionality operates through interpersonal functioning to influence later LOC eating, cannot be excluded. Finally, the sample was exclusively female which precludes generalizing the findings to males, who may show different patterns of onset/maintenance (5).

Conclusion

In summary, results provide support for cognitive-behavioral and interpersonal-affective constructs as predictors of LOC eating during middle to late adolescence. This investigation is the first to test longitudinal models based on cognitive-behavioral and interpersonal frameworks for LOC eating. Future next steps include testing these models in relation to the development of full-syndrome eating disorders in heterogeneous samples, extending the timeframe to identify the optimal timing of preventive interventions, and assessing whether individual difference variables may influence the relevance of certain domains within these models in relation to risk for LOC eating.

Acknowledgments

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5. Washington, DC: American Psychiatric Association; 2013.

2. Goldschmidt AB. Are loss of control while eating and overeating valid constructs? A critical review of the literature. Obes Rev. 2017; 18:412–449. [PubMed: 28165655]

3. Swanson SA, Crow SJ, Le Grange D, Swendsen J, Merikangas KR. Prevalence and correlates of eating disorders in adolescents. Results from the national comorbidity survey replication adolescent supplement. Arch Gen Psychiatry. 2011; 68:714–723. [PubMed: 21383252]

4. Lee-Winn AE, Reinblatt SP, Mojtabai R, Mendelson T. Gender and Racial/Ethnic Differences in Binge Eating Symptoms in a Nationally Representative Sample of Adolescents in the United States. Eat Behav. 2016; 22:27–33. [PubMed: 27085166]

5. Calzo JP, Horton NJ, Sonneville KR, et al. Male Eating Disorder Symptom Patterns and Health Correlates From 13 to 26 Years of Age. J Am Acad Child Adolesc Psychiatry. 2016; 55:693–700. e692. [PubMed: 27453083]

6. Croll J, Neumark-Sztainer D, Story M, Ireland M. Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: relationship to gender and ethnicity. J Adolesc Health. 2002; 31:166–175. [PubMed: 12127387]

7. Fairburn, CG. Eating disorders: The transdiagnostic view and the cognitive behavioral theory. In: Fairburn, CG., editor. Cognitive behavior therapy and eating disorders. New York: Guilford Press; 2008. p. 7-22.

8. Wilfley, DE., Stein, RI., Welch, RR. Interpersonal psychotherapy. In: Treasure, J.Schmidt, U., van Furth, E., editors. Handbook of Eating Disorders. London: John Wilfley & Sons; 2003.

9. Kass AE, Kolko RP, Wilfley DE. Psychological treatments for eating disorders. Curr Opin Psychiatry. 2013; 26:549–555. [PubMed: 24060917]

10. Zunker C, Peterson CB, Crosby RD, et al. Ecological momentary assessment of bulimia nervosa: Does dietary restriction predict binge eating? Behav Res Ther. 2011; 49:714–717. [PubMed: 21764036]

11. Agras WS, Telch CF. The effects of caloric deprivation and negative affect on binge eating in obese binge-eating disordered women. Behav Ther. 1998; 29:491–503.

12. Decaluwe V, Braet C. The cognitive behavioural model for eating disorders: A direct evaluation in children and adolescents with obesity. Eat Behav. 2005; 6:211–220. [PubMed: 15854867]

13. Stice E. A prospective test of the dual-pathway model of bulimic pathology: mediating effects of dieting and negative affect. J Abnorm Psychol. 2001; 110:124–135. [PubMed: 11261386]

14. Dakanalis A, Timko CA, Carra G, et al. Testing the original and the extended dual-pathway model of lack of control over eating in adolescent girls. A two-year longitudinal study. Appetite. 2014; 82:180–193. [PubMed: 25058649]

15. Sonneville KR, Grilo CM, Richmond TK, et al. Prospective association between overvaluation of weight and binge eating among overweight adolescent girls. J Adolesc Health. 2015; 56:25–29. [PubMed: 25438968]

16. Arcelus J, Haslam M, Farrow C, Meyer C. The role of interpersonal functioning in the maintenance of eating psychopathology: A systematic review and testable model. Clin Psychol Rev. 2013; 33:156–167. [PubMed: 23195616]

17. Hamann DM, Wonderlich-Tierney AL, Vander Wal JS. Interpersonal sensitivity predicts bulimic symptomatology cross-sectionally and longitudinally. Eat Behav. 2009; 10:125–127. [PubMed: 19447355]

18. Stice E, Presnell K, Spangler D. Risk factors for binge eating onset in adolescent girls: A 2-year prospective investigation. Health Psychol. 2002; 21:131–138. [PubMed: 11950103]

19. Goldschmidt AB, Wonderlich SA, Crosby RD, et al. Ecological momentary assessment of stressful events and negative affect in bulimia nervosa. J Consult Clin Psychol. 2014; 82:30–39. [PubMed: 24219182]

20. Ranzenhofer LM, Engel SG, Crosby RD, et al. Using ecological momentary assessment to examine interpersonal and affective predictors of loss of control eating in adolescent girls. Int J Eat Disord. 2014

21. Goldschmidt AB, Lavender JM, Hipwell AE, Stepp SD, Keenan K. Emotion regulation and loss of control eating in community-based adolescent girls. J Abnorm Child Psychol. 2017; 45:183–191. [PubMed: 27040409]

22. Ivanova IV, Tasca GA, Hammond N, et al. Negative affect mediates the relationship between interpersonal problems and binge-eating disorder symptoms and psychopathology in a clinical sample: a test of the interpersonal model. Eur Eat Disord Rev. 2015; 23:133–138. [PubMed: 25582510]

23. Ansell EB, Grilo CM, White MA. Examining the interpersonal model of binge eating and loss of control over eating in women. Int J Eat Disord. 2012; 45:43–50. [PubMed: 21321985]

24. Elliott CA, Tanofsky-Kraff M, Shomaker LB, et al. An examination of the interpersonal model of loss of control eating in children and adolescents. Behav Res Ther. 2010; 48:424–428. [PubMed: 20074702]

25. Wilfley D, Pike K, Striegel-Moore R. Towards an integrated model of risk for binge eating disorder. Journal of Gender, Culture, and Health. 1997; 2:1–3.

26. Tanofsky-Kraff M, Shomaker LB, Olsen C, et al. A prospective study of pediatric loss of control eating and psychological outcomes. J Abnorm Psychol. 2011; 120:108–118. [PubMed: 21114355]

27. Loeber R, Stouthamer-Loeber M, Farrington DP, et al. Editorial introduction: three longitudinal studies of children's development in Pittsburgh: The Developmental Trends Study, the Pittsburgh

Youth Study, and the Pittsburgh Girls Study. Crim Behav Ment Health. 2002; 12:1–23. [PubMed: 12357254]

28. Hipwell AE, Loeber R, Stouthamer-Loeber M, et al. Characteristics of girls with early onset disruptive and antisocial behaviour. Crim Behav Ment Health. 2002; 12:99–118. [PubMed: 12357260]

29. Garner DM, Olmsted MP, Bohr Y, Garfinkel PE. The eating attitudes test: Psychometric features and clinical correlates. Psychol Med. 1982; 12:871–878. [PubMed: 6961471]

30. Collins ME. Body figure perceptions and preferences among preadolescent children. Int J Eat Disord. 1991; 10:199–208.

31. Thompson JK, Altabe MN. Psychometric qualities of the figure rating scale. Int J Eat Disord. 1991; 10:615–619.

32. Gresham, F., Elliott, S. Social skills rating system: Manual. Circle Pines, MN: American Guidance Service; 1990.

33. Mota CP, Matos PM, Lemos MS. Psychometric properties of the Social Skills Questionnaire: Portuguese adaptation of the student form (grades 7 to 12). Span J Psychol. 2011; 14:486–499. [PubMed: 21568204]

34. Van der Oord S, Van der Meulen EM, Prins PJ, et al. A psychometric evaluation of the social skills rating system in children with attention deficit hyperactivity disorder. Behav Res Ther. 2005; 43:733–746. [PubMed: 15890166]

35. McCrae, RR., Costa, P. NEO Inventories professional manual. Lutz, FL: Psychological Assessment Resources; 2010.

36. Hayes, AF. Introduction to mediation, moderation, and conditional process analysis: A regression-based approach. New York: Guilford Press; 2013.

37. Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav Res Methods. 2008; 40:879–891. [PubMed: 18697684]

38. Sonneville KR, Calzo JP, Horton NJ, et al. Body satisfaction, weight gain and binge eating among overweight adolescent girls. Int J Obes (Lond). 2012; 36:944–949. [PubMed: 22565419]

39. Bodell LP, Wildes JE, Cheng Y, et al. Associations between Race and Eating Disorder Symptom Trajectories in Black and White Girls. J Abnorm Child Psychol. 2017

40. Le LK, Barendregt JJ, Hay P, Mihalopoulos C. Prevention of eating disorders: A systematic review and meta-analysis. Clin Psychol Rev. 2017; 53:46–58. [PubMed: 28214633]

What is already known about this subject?

- Cognitive-behavioral and interpersonal-affective constructs have been associated with the onset and maintenance of loss of control eating in youth

- The full cognitive-behavioral and interpersonal models have undergone limited prospective evaluation in adolescents

What does this study add?

- Supporting the cognitive-behavioral model, body dissatisfaction at 14 years prospectively predicted loss of control eating at 18 years, both directly and indirectly via dieting

- Partially supporting the interpersonal model, interpersonal functioning at 14 years was negatively associated with negative emotionality at 17 years, which in turn was prospectively associated with loss of control eating

- The direct association between interpersonal functioning at age 14 and loss of control eating at age 18 was not significant

Table 1

Descriptive statistics (presented as $M\pm SD$, unless otherwise indicated)

Variable		Age 14	Age 16	Age 17	Age 18
Demographic factors					
Race, % (*n*)	African-American	51.4 (302)	---	---	---
	Caucasian	43.0 (253)	---	---	---
	Asian	1.4 (8)	---	---	---
	Multi-racial/other	4.3 (25)	---	---	---
Age, y		14.5±0.4	16.5±0.4	17.3±0.4	18.3±0.3
Public assistance, % (*n*) receiving		41.3 (209)	38.2 (188)	36.7 (178)	7.3 (36)
Body mass index (kg/m²)		24.45±6.63	25.52±6.99	25.86±7.14	26.57±7.20
Psychological factor					
Loss of control eating frequency, % (*n*)	Never	76.6 (370)	72.1 (354)	---	72.5 (361)
	Rarely	14.7 (71)	18.3 (90)	---	13.7 (68)
	Sometimes	4.1 (20)	4.4 (26)	---	7.0 (35)
	Often	1.9 (9)	2.2 (11)	---	3.8 (19)
	Very often	0.8 (4)	1.2 (6)	---	1.0 (5)
	Always	1.9 (9)	0.8 (4)	---	2.0 (10)
Body dissatisfaction (range=−4 to 4)		−0.3±0.9	−0.3±0.9	---	−0.4±0.9
Dieting (range=13 to 78)		21.9±8.9	22.7±9.5	---	23.0±9.7
Interpersonal functioning (range=0 to 80)		57.1±10.7	---	---	---
Negative emotionality (range=0 to 60)		---	---	20.6±6.0	20.3±6.5

Note: Descriptive characteristics reflect the full sample of 588 girls. Differences in public assistance before and after age 18 may reflect differences in eligibility at minor and major age status

Table 2

Summary of indirect effect models

Independent variable (IV)	Mediating variable (MV)	Dependent variable (DV)	Effect of IV on MV (a)	Effect of MV on DV (b)	Direct effect (c′)	Indirect effect (a* b)	Total effects (c)
Age 14 body dissatisfaction	Age 16 dieting	Age 18 loss of control eating	-2.872^{***} ($S.E.$=0.719)	0.003^{**} ($S.E.$=0.001)	-0.039^{*} ($S.E.$=0.017)	$-0.010^{\dagger}$ ($S.E.$=0.005)	-0.049^{**} ($S.E.$=0.017)
Age 14 interpersonal functioning	Age 17 negative emotionality	Age 18 loss of control eating	-0.094^{***} ($S.E.$=0.028)	0.008^{***} ($S.E.$=0.002)	-0.001 ($S.E.$=0.001)	$-0.001^{\dagger}$ ($S.E.$=0.000)	-0.002 ($S.E.$=0.001)

Note:

$\dagger$ Significant (associated 95% confidence interval does not contain zero). $n = 416$ for the cognitive-behavioral model and $n = 418$ for the interpersonal model.

$^{*}p<.05$;

$^{**}p<.001$