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Prospective Relations between Overeating, Loss of Control Eating, Binge Eating, and Depressive Symptoms in a School-Based Sample of Adolescents

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Abstract A sample of 353 community adolescents (grades 9 to 12, 57.6 % female) participated in a 2-wave longitudinal study of eating behaviors (overeating, loss of control eating [LOC], and binge eating) and depression. The study addresses 4 hypotheses. (1) The prospective relations between eating behaviors and depressive symptoms will be reciprocal, with each predicting the other over time. (2) These relations will be stronger for girls than for boys. (3) These relations will be stronger for adolescents with high (not low) body mass index (BMI). (4) LOC will show incremental predictive utility in relation to depressive symptoms over and above overeating. Evidence supported reciprocal relations between binge eating and depressive symptoms and between overeating and depressive symptoms, but not between LOC and depressive symptoms. Sex and BMI did not substantially moderate these relations. Taken separately, overeating but not LOC predicted depressive symptoms. Taken together, neither predictor was significant controlling for the other. Results raise questions about the importance of LOC alone in predicting depressive symptoms in adolescence.

Keywords Binge eating · Loss of control eating · Overeating · Depressive symptoms · Adolescents

Depressive symptoms are significantly associated with three types of unhealthy eating behaviors in adolescents: overeating, loss of control eating (LOC), and binge eating (Goossens et al. 2010; Morgan et al. 2002; Stice and Agras 1998; Zaider et al. 2002). Although related, these three behaviors have key differences. Overeating is defined as eating an objectively large amount of food. LOC is the subjective experience of feeling out of control while eating. An objectively large amount of food does not have to be eaten. Binge eating is an eating episode that includes both overeating and LOC and is defined in the DSM-5 as eating significantly more food in a discrete period of time than most people would eat under similar circumstances, with episodes marked by feelings of lack of control (American Psychiatric Association 2013).

Estimates of maladaptive eating behaviors, including overeating, LOC, and binge eating, range from 7.8 % to 26.0 % (e.g., Ackard et al. 2003; Johnson et al. 2002; Marcus and Kalarchian 2003). Nationally representative studies find that about one in six adolescents in the United States report some level of depressive symptoms (e.g., Saluja et al. 2004). Although these adolescents may not meet clinical criteria for diagnoses based on their symptom levels, they are at risk for a host of associated negative outcomes, including the development of major depression, eating disorders, weight gain, obesity, substance use, and poor health and education outcomes (e.g., Field et al. 2003; Fergusson and Woodward 2002; Goldschmidt et al. 2015; Sonneville et al. 2013). Understanding the mechanisms that connect depressive symptoms and eating behavior for nonclinical populations of adolescents is a critical area of developmental psychopathology research. The overarching purpose of this study is to (1) examine the longitudinal reciprocal relations between depressive

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symptoms and three eating behaviors: overeating, LOC, and binge eating in adolescents, (2) examine potential moderators of these relations, and (3) examine the predictive utility of LOC over and above overeating.

Theoretical Framework

The leading theory to describe the onset and maintenance of binge eating behaviors is the cognitive-behavioral model of binge eating (Fairburn 2008). In this theory, poor self-esteem leads to body-focused concerns, resulting in restrictive eating. The restrictive diet is impossible to maintain and eventually the individual breaks the diet, eating in an out of control way that may culminate in a binge eating episode and low mood (Goldschmidt et al. 2016). If this pattern is maintained, continued binge eating may then become a maladaptive attempt to escape emotional distress. In line with this view, a second and connected theory is the affect regulation model (Hawkins and Clement 1984). This model suggests that although binge eating provides temporary relief from negative emotions, it ultimately increases negative emotions as the individual feels guilt or shame about losing control over their eating (e.g., Allen et al. 2008; Hawkins and Clement 1984; Marcus and Kalarchian 2003). Indeed, research suggests that the experience of shame after engaging in disordered eating behaviors can increase risk of depression (e.g., Keel et al. 2000). In addition, rumination about one's inability to control food intake is another risk factor for depression (e.g., Nolen-Hoeksema et al. 2007).

Evidence of Potential Reciprocal Relations

Only one study has examined the effects of depression on eating behaviors and vice versa. Focusing on a large sample of young adult and adolescent females from the Growing Up Today study, a four-year prospective study, Skinner et al. (2012) found significant reciprocal relations, as depression predicted later binge eating and overeating, and overeating predicted later depressive symptoms. Unfortunately, most variables were assessed via single-item measures (e.g., depression was represented by one question, "In the past year how often did you feel 'down in the dumps' or 'depressed'?"), and LOC without binge eating was not measured. Not measuring LOC alone means that this study did not examine the experience of feeling out of control while eating in the absence of overeating and that LOC's specific connection to depression was not addressed. Nevertheless, the results provide excellent preliminary evidence of reciprocal prospective relations, at least for females.

Other studies have examined relations in just one direction. Studies of the prospective relation of depressive symptoms to binge eating have found that depressive symptoms predict binge eating at 10 month follow-up (Stice et al. 2002; Zaider et al. 2002) and at 2 and 4 year follow up (Chen et al. 2009). A 5 year longitudinal study examining both overeating and binge eating found that higher depressive symptoms predicted greater overeating and binge eating for boys and greater binge eating for girls (Goldschmidt et al. 2016). Finally, another study found a prospective effect of depressive symptoms on LOC 6 years later in a sample of overweight male and female adolescents who participated in a weight loss program (Goossens et al. 2011).

In the opposite direction, studies that examine the eating to depressive symptom relation also find significant relations. LOC predicted depressive symptoms approximately 4.7 years later in children under 13 (Tanofsky-Kraff et al. 2011). In a prospective cohort study design, Sonneville et al. (2013) measured binge eating, overeating, and depressive symptoms every 12 to 24 months between 1996 and 2005 for adolescents who were 9 to 15 at the start of the study. They found that binge eating, but not overeating, predicted increases in depressive symptoms. In sum, although there is one contradictory result for overeating, evidence exists to suggest that each of the three eating behaviors is prospectively related to depressive symptoms and vice versa.

Potential Moderators

In the current study, we also examined the potential moderator effects of two variables: sex and body mass index (BMI). Historically, studies on depressive symptoms and disordered eating in children and adolescents have focused on females (e.g., Carlat et al. 1997). Adolescent boys are not immune to either depressive symptoms or eating concerns, although they have lower rates of both disorders than do adolescent girls (Olivardia et al. 2004). In addition, eating behaviors endorsed by males and females differ. For example, Striegel et al. (2009) found that men were more likely to self-report overeating whereas women were more likely to self-report LOC. One possible reason for this finding is that social attitudes may support overeating by males but not females, resulting in men being less likely to consider their eating as being out of control and women being less likely to engage in overeating (e.g., Cohn and Lemberg 2013). Previous research findings regarding sex differences in eating pathology are mixed, with some studies finding no sex differences and some studies finding differences. For example, in a correlational study, Goldschmidt et al. (2015) reported that for both males and females binge eating was more strongly associated with depressive symptoms than was overeating without loss of control. Conversely in a 5 year longitudinal study, Goldschmidt et al. (2016) found that higher depressive symptoms predicted greater binge eating for both girls and boys but predicted greater problems with overeating only for boys. The authors

interpreted their findings to suggest that there were no unique risk factors for eating pathology in boys; however, for girls, depressive symptoms uniquely predicted the most extreme form of eating pathology studied (i.e., binge eating). They suggested that hormonal changes of puberty put girls at unique risk for developing the more pathological form of maladaptive eating over time. Although empirical results are mixed, theoretical evidence supports the possibility that depressive symptoms may be more strongly related to eating pathology for females than males. Sociocultural models of eating disorders describe the pressure placed on females to achieve the Western thin body ideal and how this pressure puts girls at increased risk for eating pathology as well as increased psychological distress (Striegel-Moore and Bulik 2007). In sum, although evidence is mixed, some support accrues for the idea that the prospective relation between depressive symptoms and problematic eating behaviors may be stronger for girls.

Other evidence suggests that BMI may also moderate these relations. BMI is a number calculated from a person's weight and height and a fairly reliable indicator of body fatness for most people (Mei et al. 2002). Evidence suggests that children with higher BMIs engage in more overeating, LOC, and binge eating than children with lower BMIs (e.g., Decaluwé et al. 2003; Stice et al. 2002); however, whether BMI affects the longitudinal relation between these eating behaviors and depressive symptoms in adolescence is unclear. One correlational study showed that overweight adolescents who reported binge eating also reported greater depressive symptoms than did overweight adolescents who did not endorse binge eating, (Glasofer et al. 2007); however, a low BMI group was not included in the study, preventing the examination of BMI as a moderator. Other research has shown that adolescents with higher BMIs reported the use of eating to cope with negative emotions more so than did adolescents with lower BMIs (e.g., Martyn-Nemeth et al. 2009). In sum, research literature suggests that the reciprocal relations between depressive symptoms and the eating behaviors are stronger for adolescents with higher BMIs than for adolescents with lower BMIs.

Predictive Utility of LOC

The focus on LOC is a recent development in the study of eating disorders that allows researchers to measure unhealthy eating behaviors that do not meet full criteria for a binge eating episode but are still related to psychopathology. This development is especially important for researchers who study adolescents, as caloric intake increases during puberty, and what constitutes overeating in this population may be unclear (Tanofksy-Kraff et al., 2008). Although overeating and LOC both have psychological correlates (including depressive symptoms), evidence suggests that LOC may be more strongly linked to psychopathology than is overeating. For example, a correlational study found that children who experience LOC have greater disordered eating attitudes, more serious eating behavior problems, and higher levels of internalizing symptoms than children who report overeating without LOC (Shomaker et al. 2010). Goldschmidt et al. (2008) found that adolescents who reported LOC had higher weight and shape concerns than did adolescents who reported either only overeating or had no eating pathology. In addition, adolescents who endorsed LOC had higher depression scores than those who reported no eating pathology. This evidence suggests that although both overeating and LOC are maladaptive, LOC may put adolescents at particularly high risk.

Hypotheses

Given the literature and previous research, we tested four hypotheses in this study. First, the prospective relations between the three eating behaviors and depressive symptoms will be reciprocal, with each predicting the other over time. Second, these relations will be stronger for girls than for boys. Third, these relations will be stronger for adolescents with high (not low) BMI. Fourth, LOC will show incremental predictive utility in relation to depressive symptoms over and above overeating. The current study builds upon previous research in four ways: examining prospective relations between depressive symptoms and eating behaviors in both directions in the same study, obtaining multiple (and full) measures of all three eating behavior variables (overeating, LOC, and binge eating), and including males as well as females in the study design.

Methods

Participants

Participants were freshmen through seniors taking physical education and health classes in three public high schools in middle Tennessee. The number of students who participated at T1 was 353. Four months later, we recruited 320 participants for T2. This included both the loss of 88 participants due to attrition and the addition of 55 participants who had obtained parental consent but were absent on the first day of data collection. Almost all differences between groups of participants with different patterns of missingness were nonsignificant (*ps* > 0.20), except that sophomores were slightly more likely to drop out and upperclassmen were more likely to join ($\chi^2_6 = 27.28$, $p < 0.001$). Participants were 57.6 % female, 94.1 % Caucasian, 2.5 % African-American, 5.1 % Hispanic, 0.7 % Asian, 2.2 % Native American, and 3.1 % other. (Note that ethnicities were not mutually exclusive.) These demographics are representative of the county where the schools

were located (U.S. Census Bureau 2015). Participants were 49.0 % freshmen, 37.5 % sophomores, 8.7 % juniors, and 8.9 % seniors, and 1.7 % unreported grade level. These percentages were all essentially unchanged at T2. Mean age was 15.75 years (*SD* = 1.10) at T2 and 16.06 years (*SD* = 1.08) at T2.

Procedure

Prior to data collection, principals at the participating schools were contacted in order to set up a time to speak with teachers about the study. Informed consent documents were given to teachers at this time and collected later. At the pre-arranged time, informed consent documents were distributed to all students in each participating classroom. We offered a $100 donation to each classroom if 90 % of students returned consent forms signed by a parent or guardian, either granting or denying permission for their child's participation. Informed consent documents distributed totaled 1086; of these, 520 (47.9 %) were returned. After the informed consent process, 450 parents (86.5 %) gave permission for their child to participate in the study. During regular school hours, the principal investigator and trained research assistants gathered the consented students into small groups and went over informed assent documents. We then administered the questionnaires, reading the instructions aloud and then allowing participants to complete the questionnaires at their own pace. The principal investigator and research assistants were available to answer questions before, during, and after questionnaire administration. At the end of the data collection, students were given healthy snacks and a decorated pencil for their participation. The procedure was repeated again four months later at each school.

Measures

Depressive Symptoms Participants completed the Children's Depression Inventory (CDI; Kovacs 1985), the Reynolds Adolescent Depression Scale 2 (RADS2; Reynolds, 2002), and the Patient Health Questionnaire Mood Subscale (PHQ-9; Spitzer et al. 1999). The CDI is a 27-item self-report measure assessing affective, cognitive, and behavioral symptoms. The item pertaining to suicide was removed at the request of school administrators. Each item consists of three statements scored in order of increasing severity from 0 to 2. Respondents select one sentence from each group that best describes themselves for the previous two weeks. The CDI has high levels of internal consistency and test-retest reliability, especially in nonclinical samples (Carey et al. 1987; Saylor et al. 1984; Smucker et al. 1986). Cronbach's alpha was 0.91 at T1 and 0.91 at T2.

The RADS2 is a self-report measure designed to assess depressive symptoms in adolescents. The RADS2 is comprised of 30 items and measures 4 subscales (dysphoric mood, anhedonia/negative affect, negative self-evaluation, and somatic complaints), which can be summed to form an overall depression score. The scale uses a 4-point Likert-type response format that asks respondents to indicate whether the symptom-related item has occurred, "Almost Never," "Hardly Ever," "Sometimes," or "Most of the Time." The RADS2 has high levels of internal consistency and test-retest reliability, especially in nonclinical samples (Osman et al. 2010). Cronbach's alpha was 0.94 at T1 and 0.94 at T2.

The PHQ-9 is a self-report measure of depressive symptoms taken from the larger Patient Health Questionnaire. The Patient Health Questionnaire measures a wide range of mental health problems including anxiety symptoms, mood symptoms, and somatic complaints. The Mood Subscale consists of nine items that correspond directly to the DSM defined symptoms of depression. The item pertaining to suicide was removed at the request of school administrators. The scale uses a 4-point Likert response format. The PHQ-9 has high levels of validity and internal consistency for adolescents (Martin et al. 2006). Cronbach's alpha was 0.80 at T1 and 0.86 at T2.

The three measures of depressive symptoms were highly correlated, so we converted them to z-scores and averaged them to create the depression composite. Reliability of this composite (Stanley 1971) was 0.94 at both times.

Eating Behavior Overeating, LOC, and binge eating were measured using The Youth Eating Disorder Examination-Questionnaire (YEDE-Q; Goldschmidt et al. 2007) and the How I Eat questionnaire. The instructions that precede the YEDE-Q use words and pictures to explain the definitions of overeating, LOC, and binge eating, which were adapted from instructions for adults created by Goldfein et al. (2005). The YEDE-Q generates four subscales: Restraint, Eating Concern, Weight Concern, and Shape Concern as well as a global score measuring the overall severity of eating disorder symptoms. Questions use a combination of 7-point Likert-type and yes/no formats. Subsets of questions are used to determine whether participants overeat, experience LOC in the absence of overeating, or binge eat. Participants also indicate the frequency of these behaviors. The YEDE-Q has high levels of internal consistency and test-retest reliability (Goldschmidt et al. 2007).

How I Eat is a self-report questionnaire designed for the current study to assess overeating, LOC, and binge eating. Although the YEDE-Q is typically used to place participants into four eating behavior groups (i.e., binge eaters, LOC, overeaters, and no eating pathology), How I Eat assesses eating behaviors on continua. Questions pertaining to overeating or LOC were taken directly or adapted from the Binge Eating Scale (BES; Gormally et al. 1982), Binge Scale Questionnaire (BSQ; Hawkins and Clement 1980), the Bulimic Investigatory Test (BITE; Henderson and Freeman 1987),

the Bulimia Test- Revised (BULIT-R; Thelen et al. 1991), the Eating Attitudes Test-Revised (EAT-26; Garner et al. 1982), the Multifactorial Assessment of Eating Disorder Symptoms (MAEDS; Anderson et al. 1999), and the Three Factor Eating Questionnaire (TFEQ; Stunkard and Messick 1985).[1] The scale uses a 5-point Likert-type response format that asks participants to indicate whether particular eating behaviors have occurred *Never, Hardly Ever, Sometimes, Often,* or *Always*. The original scale consisted of 20 items and had 2 subscales: overeating and LOC. The scale also measures binge eating, as binge eating is the combination of these two factors.

Principal axis factoring with oblique rotation of the items on How I Eat revealed two factors: Overeating and LOC. The factor structure replicated from T1 to T2. Questions that loaded highly onto Overeating at both time points but did not cross-load onto LOC became the new Overeating subscale. Questions that loaded highly onto LOC at both time points but did not cross-load onto overeating became the new LOC subscale (Table 1). After dropping items with small loadings and crossloadings, the final How I Eat scale contained thirteen items, eight measuring overeating and five measuring LOC. The factor structure was the same for boys and girls at T1 and T2. Every item loaded onto the same factor (with no crossloadings) in both sexes and at both time points.[2]

We formed composites measures of overeating, LOC, and binge eating from the YEDE-Q and How I Eat. YEDE-Q and How I Eat items were standardized to z-scores (to put them on the same metric). All items related to overeating formed the overeating composite variable and all items related to LOC formed the LOC composite. Finally, all items from How I Eat as well as the binge-specific items from the YEDE-Q formed the binge eating composite. Reliabilities of these composites were good: for LOC, $\alpha = .79$ at T1 and .80 at T2; for overeating, $\alpha = .87$ at T1 and .85 at T2; for binge eating, $\alpha = .90$ at T1 and .89 at T2.

Sex and BMI Participants filled out a demographic questionnaire that asked them to self-report their sex, height, and weight. From these data, BMI was calculated using the formula BMI = (weight in pounds)/(height in inches^2) × 703. Research with adolescents has indicated that correlations between BMIs calculated from self-reported height and weight and BMIs calculated from measured height and weight range from .80 to .89 (e.g., Brener et al. 2003; Elgar et al. 2005; Fonseca et al. 2010). BMI-for-age percentiles were then calculated using the Centers for Disease Control and Prevention's (Centers for Disease Control and Prevention 2010) BMI-for-age growth charts for children and adolescents. Percentiles indicate the relative position of the BMIs among children of the same sex and age.

Analyses

To test our hypotheses about reciprocal prospective relations and possible moderator effects, we used hierarchical regression (with full information maximum likelihood estimation, to handle missing data). In one set of analyses, depressive symptoms predicted one of three eating behaviors (overeating, LOC, or binge eating); in the other set of regressions, an eating disorder variable predicted depressive symptoms. In each analysis, we first tested the three-way Predictor x Sex x BMI interaction. If it was nonsignificant (or very small) it was eliminated, and we then tested the three component two-way interactions. If those were also nonsignificant, we eliminated them and then tested the three main effects. In every analysis, the T1 measure of the dependent variable served as a covariate. Due to our directional hypotheses, we conducted one-way tests. Because each set of analyses involved three regressions, each with one of the three eating variables, we protected for family-wise error rate by dividing alpha (0.05) by 3. For our hypothesis about the incremental predictive utility of LOC versus overeating, we regressed our T2 depression composite onto our LOC and overeating composites, while controlling for our T1 depression composite. The relative size and significance of the partial beta weights for the LOC and overeating composites provide evidence of the incremental value of each over and above the other in the prediction of depressive symptoms over time.

Results

Table 2 contains correlations among all study variables, as well as descriptive statistics. Depressive symptoms at both time points were significantly correlated with all three eating variables at both time points. Eating variables were also significantly correlated with each other. Sex at T1 and T2 was significantly correlated with LOC at T1 and T2, such that girls reported more LOC than did boys. BMI was correlated with T1 depressive symptoms, T2 binge eating, and T1 and T2 LOC.

Depressive Symptoms as a Predictor of the three Eating Behaviors In the regressions of T2 binge eating and LOC onto the T1 depressive symptoms on composite, the Sex x BMI x Depression interactions were statistically significant. In the binge eating regression, the beta weight for the three-way interaction was −0.01 ($SE = 0.002$, $p = 0.040$). In the LOC regression, the standardized beta weight for the three-way interaction was 0.004 ($SE = 0.002$, $p = 0.049$). Both were

[1] A list of appropriate and freely available measures was created after researching eating disorder assessment in the *Handbook of Eating Disorders Assessment and Treatment (*Walsh et al. 2015). Additional information about the measure is available from the first author upon request.

[2] Additional information about the factor analysis is available from the first author upon request.

Table 1 How I eat factor analysis waves 1 and 2

How I Eat Items	T1 LOC Eating	T1 Overeating	T2 LOC Eating	T2 Overeating
1. I eat until completely stuffed.	−0.056	**0.605**	−0.145	**0.650**
2. I eat even when not hungry.	0.263	**0.419**	0.032	**0.527**
3. How hungry I feel determines how much I eat.	0.332	−0.466	0.422	−0.370
4. I eat until I can't eat anymore.	0.090	**0.638**	−0.142	**0.751**
5. I eat and eat until I am physically uncomfortable.	0.239	**0.416**	0.218	**0.612**
6. Sometimes, when I start eating, I just can't seem to stop.	0.485	0.467	0.264	0.633
7. I eat amounts of food that others would consider unusually large.	0.145	**0.570**	−0.082	**0.612**
8. I eat alone because I am embarrassed of how much I'm eating.	0.369	0.075	0.246	0.261
9. I rarely eat so much food that I feel uncomfortably stuffed afterwards.	0.274	−0.035	0.302	0.029
10. There are times when I rapidly eat a very large amount of food.	0.192	**0.556**	0.038	**0.724**
11. When I see my favorite food, I find it very difficult to keep from eating it, even if I have just finished a meal.	0.117	**0.626**	0.048	**0.605**
12. Some things just taste so good that I keep on eating even when I am no longer hungry.	0.140	**0.685**	0.049	**0.706**
13. I lose control when eating.	0.459	0.315	0.362	0.593
14. I have control over the amount of food I consume.	**0.582**	0.120	**0.566**	−0.007
15. Compared to most people, my ability to control my eating behavior seems to be good.	**0.584**	0.162	**0.484**	0.170
16. It is not difficult for me to leave something on my plate.	0.199	−0.053	0.422	−0.052
17. I feel that food controls my life.	0.476	0.294	0.187	0.487
18. I display self-control around food.	**0.466**	0.076	**0.526**	0.145
19. I can stop eating when I want to.	**0.466**	0.013	**0.425**	0.065
20. I have no control over how much I eat.	**0.520**	0.122	**0.307**	0.263

LOC = Loss of Control. Factor loadings are in boldface if they loaded > 0.3 onto the same factor at both time points and did not crossload onto the other factor at either time point. Only these items were retained

ordinal interactions in which the relation between depression and the eating variables was significant in males as well as females and in high-BMI as well as low-BMI adolescents. Which subgroup evinced the largest effect varied from analysis to analysis. Furthermore, although both interactions were statistically significant, their effects were small ($\Delta R^2 < 0.01$ for both).[3] With small, inconsistent, and marginally significant effects, we conservatively elected to regard these effects as Type 1 errors, remove the three-way interaction, and test the lower-order effects. None of the two-way interactions were significant. We then tested the main effects. As shown in Table 3, the T1 depressive symptoms composite was a significant predictor of binge eating, LOC, and overeating, even after controlling for T1 levels of the eating behaviors.

Binge Eating as a Predictor of Depressive Symptoms In the regression of T2 depressive symptoms onto binge eating, none of the three-way or two-way interactions were significant. In the main effects model (see Table 4), significant effects emerged for binge eating and sex. Binge eating was a significant predictor of the T2 depressive symptoms while controlling for T1 depressive symptoms, sex, and BMI. Sex was a significant predictor of T2 depressive symptoms while controlling for T1 depressive symptoms, binge eating, and BMI. Females reported larger increases in depressive symptoms than did males.

LOC as a Predictor of Depressive Symptoms In the regression of T2 depressive symptoms onto LOC, none of the three-way or two-way interactions were significant. In the main effects model (see Table 4), a significant main effect for sex emerged, while controlling for T1 depressive symptoms, LOC, and BMI. Females reported larger increases in depressive symptoms than did males. LOC was not a significant predictor of depressive symptoms at T2.

Overeating as a Predictor of Depressive Symptoms In the regression of T2 depressive symptoms onto overeating, the three-way interaction and two-way interactions were nonsignificant. For the main effects model, significant effects for overeating and sex emerged (see Table 4). Overeating was a significant predictor of T2 depressive symptoms while controlling for T1 depressive symptoms, sex, and BMI. Sex was

[3] With the current sample size and one-tailed alpha set at .05, we had 0.80 power to detect even a small three-way interaction (Cohen's $f^2 = .02$) and 0.81 power to detect a small two-way interaction (Cohen 1988). We also tested interaction effects involving age; age was not a statistically significant moderator in any analysis. Full regression models are available from the first author upon request.

Table 2 Correlations, means, and standard deviations

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Depressive symptoms (T1)	1									
2. Depressive symptoms (T2)	0.802**	1								
3. Binge eating (T1)	0.365**	0.372**	1							
4. Binge eating (T2)	0.428**	0.467**	0.753**	1						
5. LOC (T1)	0.386**	0.348**	0.763**	0.576**	1					
6. LOC (T2)	0.400**	0.419**	0.549**	0.711**	0.642**	1				
7. Overeating (T1)	0.316**	0.336**	0.917**	0.704**	0.486**	0.391**	1			
8. Overeating (T2)	0.375**	0.412**	0.701**	0.927**	0.430**	0.431**	0.722**	1		
9. Sex (0 = female, 1 = male)	−0.282**	−0.321**	−0.027	−0.088	−0.155**	−0.183**	0.028	−0.046	1	
10. BMI Percentile	0.107*	0.107	0.098	0.126*	0.245**	0.206**	0.014	0.038	0.002	1
Means	1.50	1.43	−0.005	−0.128	0.002	−0.082	0.001	−0.121	–	65.62
SD	2.29	2.44	0.581	0.535	0.591	0.586	.673	.687	–	27.21

LOC = Loss of Control eating, T1 = Time 1, T2 = Time 2, BMI Percentile = Body Mass Index percentile

*$p < 0.05$ ** $p < 0.01$

also a significant predictor of T2 depressive symptoms. Again, females reported larger increases in depressive symptoms than did males.

Incremental Predictive Utility of LOC and Overeating In the preceding analyses, where LOC and overeating were examined separately, overeating significantly predicted change in depressive symptoms, but LOC did not. To test the incremental predictive utility of these two variables over and above one another, we conducted a final regression of T2 depressive symptoms onto LOC and overeating, controlling for T1 depressive symptoms. Although the combined effects of LOC and overeating were significant ($p < 0.05$), neither partial beta was significant at $\alpha = 0.05$ (for LOC, $\beta = 0.03$; for overeating, $\beta = 0.06$). Controlling for one another, neither T1 overeating nor T1 LOC were significant predictors of change in depressive symptoms over time.

Discussion

This study is the first multimethod attempt to examine reciprocal prospective relations, incremental predictive utility, and potential moderators in the relations between depressive

Table 3 Depressive symptoms as a predictor of eating behaviors

Predictor	Unst. B	SE(B)	β	t	$p <$
Dependent variable = Binge eating (T2)					
Binge eating (T1)	0.668	0.037	0.706	17.829	0.001
Depressive symptoms (T1)	0.042	0.010	0.175	4.117*	0.001
Sex (0 = female, 1 = male)	0.011	0.043	0.010	0.263	0.396
BMI (T1)	0.001	0.001	0.046	1.889	0.117
Dependent variable = Loss of Control Eating (T2)					
Loss of Control Eating (T1)	0.564	0.049	.566	11.529	0.001
Depressive symptoms (T1)	0.047	0.013	0.182	3.611*	0.001
Sex (0 = female, 1 = male)	−0.039	0.054	−0.032	−0.724	0.235
BMI (T1)	0.001	0.001	0.059	1.241	0.108
Dependent variable = Overeating (T2)					
Overeating (T1)	0.704	0.043	.686	16.371	0.001
Depressive symptoms (T1)	0.047	0.014	0.153	3.380*	0.001
Sex (0 = female, 1 = male)	0.033	0.058	0.024	0.576	0.283
BMI (T1)	0.001	0.001	0.025	0.606	0.272

T1= Time 1, T2 = Time 2, BMI = Body Mass Index percentile

*alpha = 0.05/3

Table 4 Eating behaviors as predictors of depressive symptoms

Predictor	Unst. B	SE(B)	β	t	$p <$
Dependent variable = Depressive symptoms (T2)					
Depressive symptoms (T1)	0.804	0.042	0.743	19.205	0.001
Binge Eating (T1)	0.409	0.164	0.096	2.494*	0.007
Sex (0 = female, 1 = male)	−0.449	0.179	−0.090	−2.507	0.006
BMI (T1)	0.004	0.003	0.049	1.353	0.088
Dependent variable = Depressive symptoms (T2)					
Depressive Symptoms (T1)	0.829	0.042	0.767	19.858	<0.001
Loss of Control Eating (T1)	0.142	0.167	0.034	0.848	0.198
Sex (0 = female, 1 = male)	−.413	0.180	−0.083	−2.294	0.011
BMI (T1)	0.004	0.003	0.047	1.257	0.105
Dependent variable = Depressive symptoms (T2)					
Depressive Symptoms (T1)	0.810	0.041	0.750	19.582	< 0.001
Overeating (T1)	0.303	0.139	0.083	2.184*	0.0145
Sex (0 = female, 1 = male)	−0.457	0.180	−0.092	−2.532	0.006
BMI (T1)	0.005	0.003	0.057	1.566	0.059

T1 = Time 1, T2 = Time 2, BMI = Body Mass Index percentile

*alpha = 0.05/3

symptoms and three eating behaviors (overeating, LOC, and binge eating) in adolescent girls as well as boys. First, we found that depressive symptoms predicted binge eating, overeating, and LOC over time, even after controlling for prior levels of the eating behaviors. Conversely, binge eating and overeating (but not LOC) predicted T2 depressive symptoms, even after controlling for T1 depressive symptoms. Taken together, these effects provide moderate support for our hypothesis about reciprocal predictive relations. Second, sex and BMI did not appear to moderate these relations in any substantial or consistent way. Finally, although overeating did predict change in depressive symptoms, neither LOC nor overeating predicted change in depressive symptoms over and above one another. Each of these results is elaborated below.

In support of our reciprocal relations hypothesis, we found that (a) depressive symptoms significantly predicted both overeating and binge eating and (b) overeating and binge eating both predicted depressive symptoms. These results extend previous research on the longitudinal relations between eating behaviors and depressive symptoms in adolescents, which (despite the numerous strengths of the studies) sometimes focused on relations in only one direction or the other, sometimes studied only females, or tended to use single measures (and sometimes only single items) to represent key constructs (e.g., Goldschmidt et al. 2016; Skinner et al. 2012; Sonneville et al. 2013). Only with regard to LOC were reciprocal relations not evident. Although depressive symptoms predicted LOC, the reciprocal effect of LOC on depressive symptoms was not significant. Taken together, these findings suggest that depression erodes individuals' abilities to control their food intake and results in both maladaptive overeating as well as the awareness that one's eating is out of control. Conversely, in the prediction of depression, overeating per se (and not the evaluation of oneself as losing control) appears to be the preeminent predictor.

Although we found some statistical evidence of three-way Sex x BMI x Depression interactions, the effects were small, inconsistent, and did not substantively qualify the relation of depressive symptoms to eating behaviors. The relation of depression to binge eating and LOC was significant for high- and low-BMI males and females, suggesting that the connection between depressive symptoms and maladaptive eating is present in both sexes and across the weight spectrum. Two implications are important. First, this generalizability supports the importance of including both males and females in studies of problematic eating behaviors in adolescents (e.g., Dominé et al. 2009; Muise et al. 2003). Second, even though binge eating is often associated with being overweight or obese in adolescents (e.g., Stice et al. 2002), the current results suggest that depression is related to problematic eating among both normal weight and overweight individuals. A possible developmental implication could be that depression-related overeating, binge eating, and LOC when one is younger (and not yet obese) could lead adolescents to become overweight or obese over time. All three eating behaviors are associated with other risk factors for obesity, such as overconsumption of high-carbohydrate and high-fat foods and inadequate intake of fruits and vegetables (Neumark-Sztainer et al. 1996; Tanofsky-Kraff et al. 2009). Future research should examine these behaviors as mediators of the longitudinal relation between depression and weight gain.

Finally, this study provided no evidence of the incremental utility of LOC over and above overeating (or vice versa) in the prediction of depressive symptoms over time. Although

overeating significantly predicted depressive symptoms, neither LOC nor overeating predicted depressive symptoms over and above one another. The fact that LOC and overeating are highly correlated may leave relatively little unique variance in either to predict change in depression. Tanofsky-Kraff et al. (2008) focused on children under twelve in their initial research on LOC. Our study is one of the first to assess the LOC concept in adolescents. The effects of pubertal growth on both appetite and caloric consumption could change the meaning of these variables, as well as their relation to depression, during adolescence. Other factors, such as weight gain and body shape, may be more powerful predictors than LOC during these years. Previous longitudinal studies with differing results assessed LOC by asking whether participants felt they could control themselves during episodes of overeating, thus conflating LOC with overeating (Sonneville et al. 2013 & Goldschmidt et al. 2016). We elected to define LOC as an explicit intent to control food intake and an awareness of one's inability to do so. Overeating was defined as a completely separate construct: eating a large amount of food with no mention of intent to control intake. Other researchers may elect to define these constructs differently. Changes in the operational definitions of these constructs could affect their incremental utility.

The results of the current study have possible clinical implications. The potentially reciprocal nature of the relations between eating behavior and depressive symptoms may create an unfortunate cycle in which one set of symptoms exacerbates the other (e.g., Skinner et al. 2012). Clinicians treating adolescents should assess their clients for both depressive symptoms and eating disorder symptoms, even if their primary focus is on just one of these problem areas. Positively, the potential reciprocal nature of the symptoms also suggests that reductions in one area could result in reductions in the other. For example, helping adolescent clients improve eating habits may be a supplemental technique for reducing depressive symptoms, a method proven useful with adults (e.g., Thomson et al. 2010).

Limitations of the current study suggest avenues for future research. First, assessment of depressive and eating disorder symptoms relied on self-report questionnaires. Use of clinical interviews and physical measures could have enabled a truly multi-method assessment of eating behaviors, potentially controlling for the effects of response bias. Second, the duration of the current study was four months. This timeframe is similar to those used in research on eating disorder and depressive symptoms, which vary from ecological momentary assessment studies that assess symptoms "in real time" to studies that follow participants for years (e.g., Goldschmidt et al. 2016; Haedt-Matt and Keel 2011; Stice and Agras 1998). Future research could measure symptoms on multiple timeframes in order to assess how long it takes for depressive symptoms to affect eating behaviors and vice versa. LOC may require more time to impact depressive symptoms than what was used in the current study. Third, this study did not measure weight change. Distress regarding weight gain due to overeating and binge eating may be another mechanism through which the eating behaviors are connected to depression. Perhaps LOC predicts depressive symptoms for adolescents who experience weight gain but does not impact depressive symptoms for those who do not, a distinction not addressed in the current study. Fourth, this study focused on high school adolescents. The relations between eating behaviors and depressive symptoms may differ with age. Future research should include wider age ranges or follow cohorts for longer periods of time (e.g., Sonneville et al. 2013) to determine if relations between eating behaviors and depressive symptoms change as adolescents move into adulthood. Finally, this study focused on just two moderators, sex and BMI. Pubertal status is another potentially important moderator (e.g., Culbert et al. 2009). Future research could examine how this and other moderators may impact the potential reciprocal pathways between depressive symptoms and eating-related problem behaviors.

Compliance with Ethical Standards

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Conflict of Interest Keneisha Sinclair-McBride, Ph.D., declares that she has no conflict of interest. David A. Cole, Ph.D., declares that he has no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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