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Depressive Symptoms and Observed Eating in Youth

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Abstract

Depressive symptoms in youth may be a risk factor for obesity, with altered eating behaviors as one possible mechanism. We tested whether depressive symptoms were associated with observed eating patterns expected to promote excessive weight gain in two separate samples. In Study 1, 228 non-treatment-seeking youth, ages 12–17y (15.3 ± 1.4y; 54.7% female), self-reported depressive symptoms using the Beck Depression Inventory. Energy intake was measured as consumption from a 10,934-kcal buffet meal served at 11:00am after an overnight fast. In Study 2, 204 non-treatment-seeking youth, ages 8–17y (13.0 ± 2.8; 49.5% female), self-reported depressive symptoms using the Children's Depression Inventory. Energy intake was measured as consumption from a 9,835-kcal buffet meal served at 2:30pm after a standard breakfast. In Study 1, controlling for body composition and other relevant covariates, depressive symptoms were positively related to total energy intake in girls and boys. In Study 2, adjusting for the same covariates, depressive symptoms among girls only were positively associated with total energy intake. Youth high in depressive symptoms and dietary restraint consumed the most energy from sweets. In both studies, the effects of depressive symptoms on intake were small. Nevertheless, depressive symptoms were associated with significantly greater consumption of total energy and energy from sweet snack foods, which, over time, could be anticipated to promote excess weight gain.

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Keywords

Depression; Obesity; Eating; Child; Adolescent

A growing body of literature supports a connection between depressive symptoms and overweight (body mass index [BMI] ≥85th percentile for age and sex) or obesity (BMI ≥95th percentile) in youth. Overweight and obese children and adolescents generally report more depressive symptoms than their peers who are not overweight (Bell et al., 2011; BeLue, Francis, & Colaco, 2009; Erermis et al., 2004; Goldfield et al., 2010; Sjoberg, Nilsson, & Leppert, 2005). Several studies have shown that BMI and percentage body fat are positively associated with symptoms of depression in pediatric samples (Erickson, Robinson, Haydel, & Killen, 2000; Hillman, Dorn, & Bin, 2010; Katon et al., 2010). Furthermore, many, though not all (Bardone et al., 1998; Larsen, Otten, Fisher, & Engels, 2013; Pine, Cohen, Brook, & Coplan, 1997) longitudinal studies suggest that elevated depressive symptoms or major depressive disorder predict excessive increases in children's and adolescents' BMI over time (Pine, Goldstein, Wolk, & Weissman, 2001; Roberts & Duong, 2013; Rofey et al., 2009). In a meta-analysis of 16 longitudinal studies that included samples of adolescents and adults, depressive symptoms were significantly associated with an increased risk of developing obesity, even after accounting for the potential confounding factors of age, socioeconomic status, baseline BMI, health behaviors, and parental depression (Blaine, 2008). The association between depressive symptoms and obesity onset was particularly pronounced in adolescent girls, such that those with elevated depressive symptoms were 2.5 times more likely to become obese compared to girls with low symptoms (Blaine, 2008).

Findings are more mixed with regard to the reverse relationship – the role of obesity in promoting depressive symptoms – with some longitudinal studies reporting that obesity was associated with increases in depressive symptoms (Anderson, Cohen, Naumova, Jacques, & Must, 2007; Boutelle, Hannan, Fulkerson, Crow, & Stice, 2010) and other studies finding that initial BMI was not related to future depression (Roberts & Duong, 2013; Stice & Bearman, 2001; Stice, Hayward, Cameron, Killen, & Taylor, 2000).

Despite the consistent evidence supporting a link between depressive symptoms and obesity, the mechanisms that explain this relationship are not well understood. One possibility is that depressive symptoms alter eating behaviors that promote excessive weight gain. According to affective theories of disordered eating, eating may serve as a coping mechanism, temporarily reducing negative emotions or distracting an individual from distressing emotional states (Bruch, 1969; Heatherton & Baumeister, 1991; Kaplan & Kaplan, 1957). Among adults, individuals who report dietary restraint—cognitive intent and/or behavioral attempts to cut back on food intake for the purpose of maintaining or losing weight (regardless of effectiveness)—are especially likely to eat more in response to emotional distress. However, the opposite or no effect on eating has been observed in adults who do not endorse restraint (Heatherton, Herman, & Polivy, 1991; Polivy & Herman, 1976). According to theoretical models of restraint and hunger, cognitive and/or behavioral restriction of eating may increase vulnerability to overeating when dietary rules have been violated or through heightened responsiveness to environmental food cues (Lowe & Levine, 2005; Polivy & Herman, 1985).

In youth, emotional eating, referring to overeating in reaction to negative affect (Thayer, 2001), more frequently occurs in overweight or obese children and adolescents than normal weight youth, as measured by parent report (Braet & Van Strien, 1997; Eloranta et al., 2012; Santos et al., 2011; Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009). Similarly, adolescent boys and girls who are distressed by frequent overeating episodes accompanied

by feelings of loss of control report higher depressive mood than those without overeating or frequent overeating (Ackard, Neumark-Sztainer, Story, & Perry, 2003). Females may be especially likely to turn to food in an attempt to cope with negative affect (Dube, LeBel, & Lu, 2005; Mikolajczyk, El Ansari, & Maxwell, 2009). In adolescent girls and young adult women, depressive symptoms predict the onset of perceived overeating or overeating accompanied by feelings of loss of control (Skinner, Haines, Austin, & Field, 2012; Stice, Presnell, & Spangler, 2002).

Existing data suggest that individuals with elevated depressive symptoms also may be prone to overeating specific types of foods that promote obesity. For example, in adults, greater depression, perceived stress, and negative emotions have been associated with higher self-reported intake of carbohydrates, sweets and fast food, especially in females (Dube et al., 2005; Jeffery et al., 2009; Konttinen, Mannisto, Sarlio-Lahteenkorva, Silventoinen, & Haukkala, 2010; Mikolajczyk et al., 2009). In adolescents, depressive symptoms or stress have been related to higher self-reported consumption of sugary foods, percent calories from fat, or soft drinks (Fulkerson, Sherwood, Perry, Neumark-Sztainer, & Story, 2004; Kim, Yang, Kim, & Lim, 2013).

The current investigations had three aims. The first aim was to determine the relationship between depressive symptoms and observed eating behaviors in youth. In contrast to self-report measures of food intake, which may be limited by inaccurate recall and biased by social desirability (Fisher, Johnson, Lindquist, Birch, & Goran, 2000; Wolkoff et al., 2011), laboratory test meal studies have the advantage of providing observational data of participants' eating behaviors in a standardized environment (Hadigan, Kissileff, & Walsh, 1989; Tanofsky-Kraff, Haynos, Kotler, Yanovski, & Yanovski, 2007). We hypothesized that depressive symptoms would be related to greater observed total energy intake and to greater intake of energy from sweet snack foods, even when accounting for body composition.

The second aim was to test whether sex and dietary restraint moderated the relationship between youth's depressive symptoms and eating behavior. Prior literature supports an especially strong link between depressive symptoms and obesity in girls (Blaine, 2008; Larsen et al., 2013; Rofey et al., 2009). Also, females may be more likely than males to consume sweet snack foods in response to negative emotions (Dube et al., 2005; Mikolajczyk et al., 2009). Therefore, we hypothesized that the link between depressive symptoms and observed energy intake might be more pronounced in girls than boys. In addition, based upon adult data illustrating that individuals with dietary restraint are likely to overeat in response to negative mood and depressive symptoms (Heatherton et al., 1991; Polivy & Herman, 1976), we predicted that youth with both high depressive symptoms and high dietary restraint would eat more total energy and more energy intake from sweet snack foods as compared to youth with either high depressive symptoms or high restraint alone.

The third aim was to investigate eating behavior as a proposed mediator of the association between youths' depressive symptoms and adiposity. Based upon the notion that depressive symptoms, in theory, may contribute to excess weight gain via their effect on eating behavior, we hypothesized (i) that there would be a positive association between depressive symptoms and adiposity and (ii) that measured food intake patterns would mediate the depressive symptoms-adiposity relationship. To examine these aims and hypotheses, we conducted secondary analyses of two separate large, laboratory feeding studies.

Study 1

Methods

Participants and Procedure—Participants were a convenience sample of adolescent boys and girls who took part in an observational study of eating behavior in youth recruited through flyers and school parent e-mail listservs in the Washington, DC and greater metropolitan area (ClinicalTrials.Gov ID: NCT00631644). The study was advertised as an investigation of eating behaviors in adolescents, and materials indicated that no treatment would be provided. Eligibility criteria were ages 13–17 years and good general health as indicated by a physical examination and medical history conducted by a nurse practitioner or endocrinologist. Adolescents who had chronic illnesses, were on medications likely to affect energy intake, were pregnant, in ongoing weight-loss treatment, had a psychiatric condition that would interfere with adherence to study procedures, or reported liking fewer than 50% of foods offered at the test meal were excluded. Participants provided written assent and their parents or legal guardians gave written consent for participation. The study was approved by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD) Institutional Review Board. Participants were given monetary compensation for their participation. Participants attended an initial outpatient screening visit at the National Institutes of Health (NIH) Hatfield Clinical Research Center. Eligible adolescents returned for an outpatient test meal appointment on a separate day. On both days, adolescents were instructed to fast after 10:00pm the night prior to the visit.

Measures

Pubertal assessment: Testicular volume (mL) was measured by using a set of orchidometer beads as standards according to Prader (Tanner, 1981) and breast development was assigned according to the five stages of Tanner (Marshall & Tanner, 1969, 1970). Testicular volume and breast development staging were utilized to categorize youth as in prepuberty or early/midpuberty (boys; testes<15mL; girls: breast Tanner stages 1–3) or in late puberty (boys: testes ≥ 15mL; girls: breast Tanner stages 4–5).

Body composition: Three heights were collected to the nearest millimeter using a stadiometer (Holtain, Crymmych, United Kingdom) calibrated before each participant's measurement. Fasting weight was measured to the nearest 0.1 kg with a calibrated digital scale (Scale-Tronix, Wheaton, IL). Average height and weight were used to compute BMI, calculated as weight (kg) divided by the square of height (m). BMI standard deviation (BMI *z*) scores for sex and age were calculated according to the Centers for Disease Control and Prevention 2000 standards (Kuczmarski et al., 2000). Youth were classified as non-overweight if BMI < 85^{th} percentile and overweight or obese if BMI ≥ 85^{th} percentile. Fat-free mass (kg) and percentage body fat were assessed with air displacement plethysmography (Life Measurement Inc, Concord, CA). Body composition measurements were obtained while participants were fasting and wearing underclothes only (Nicholson et al., 2001).

Depressive symptoms: Depressive symptoms were assessed by adolescent report on the reliable and well-validated Beck Depression Inventory (BDI-II), a 21-item questionnaire used to measure the severity of depressive symptoms over the past two weeks (Beck, Steer, & Brown, 1996). The BDI-II has been used in adolescents as young as age 13 years (Steer, Ranieri, Kumar, & Beck, 2003). Each item was rated on a scale from 0 (absence of symptom) to 3 (high severity of symptom), and a total score (range = 0–63) was derived from the sum of all items. Scores that exceed 13 may be indicative of elevated depressive symptoms (Beck et al., 1996).

Dietary restraint: Dietary restraint was measured with the restraint subscale score of the Eating Disorder Examination (EDE), a semi-structured interview instrument administered by trained interviewers (Fairburn & Cooper, 1993). This subscale contains 5 items rated on a 7-point Likert-scale from 0–7. The items are averaged to measure an individual's reported degree of behavioral and/or cognitive restraint over food intake in the past month. The EDE has demonstrated satisfactory discriminant validity, internal consistency, concurrent validity, and test-retest reliability (Glasofer et al., 2007; Tanofsky-Kraff et al., 2004).

Eating behavior: Adolescents were instructed to adhere to an 11-hour overnight fast to minimize any acute influences of food consumed outside of the laboratory on eating behavior measured in the laboratory. Adherence to overnight fasting was encouraged by reminder calls from research staff the night before each appointment. Compliance was assessed upon adolescents' arrival to the NIH Clinical Research Center. Any adolescent who did not adhere to fasting was re-scheduled for a different day. Participants were served a buffet meal of lunch-type foods at 11:00am in a private room and given the tape-recorded instruction: "Please eat until you are no longer hungry. Take as much time as you need and open the door when you're done." The meal consisted of a 10,934-kcal multi-item buffet with individual items that differed in macronutrient composition (overall: 54% carbohydrate, 12% protein, 33% fat). For a detailed list of food items and their amounts, refer to Shomaker et al. (2010). Total energy consumption was the difference in weight (g) to one tenth of a gram of each food and beverage item measured on a digital scale before and after the meal. Consumption of energy from sweet snacks was determined by difference in the weight of the sweet snack food items on the buffet, which included sandwich cookies, vanilla wafer cookies, jellybeans, and chocolate candy. Energy (kcal) intakes for total energy and items classified as sweet snacks were calculated with data from the US Department of Agriculture (USDA) National Nutrient Database for Standard Reference (USDA, Agricultural Research Service, Beltsville, MD) and food manufacturer nutrient information obtained from food labels.

Statistical Plan—Descriptive information was generated about study participants by sex. Fat-free mass was log transformed to correct for problems with skew and kurtosis; all other variables approximated a normal distribution. Influence statistics were indicative of no significant multivariate outliers in regression models (e.g., Cook's distance ≤ .15). Independent samples t-tests were used to describe differences between boys and girls on age (years), height (cm), percentage fat mass, fat-free mass (log kg), BMI *z* score, and depressive symptoms. Chi-square analyses were used to examine sex differences in race/ethnicity (non-Hispanic White vs. Other), overweight status (non-overweight vs. overweight or obese), and depressive symptoms considered categorically (low vs. elevated). Pearson correlations were conducted to describe the bivariate associations among continuous measures of depressive symptoms, dietary restraint, and body measurements (percentage fat mass, fat-free mass, and BMI *z*). To test the relationship between depressive symptoms and eating behavior and to test whether sex and dietary restraint moderated the relationship of depression and eating behavior, hierarchical multiple linear regressions were conducted with the dependent variables of total energy intake (kcal) and sweet snacks intake (kcal). The independent variables were depressive symptoms, dietary restraint, and sex, and the interactions of depressive symptoms by sex, depressive symptoms by dietary restraint, and dietary restraint by sex. Following recommendations for testing moderation with continuous variables, depressive symptoms and dietary restraint were mean-centered in the regression equations and prior to their entry in computing interaction terms (Cohen, 2003). Covariates were age (years), race (non-Hispanic White vs. Other), height (cm), percentage fat mass, and fat-free mass (log kg). Since a portion of participants studied for the current project had participated in additional test meal assessments (n = 53, 23%), we also accounted for meal

exposure (no prior test meal vs. prior test meal). In the regression predicting sweet snacks intake, we accounted for total energy intake (kcal). Pubertal stage also was considered as a covariate but was not included in the final models because it was non-significant in all analyses (*p*s = .22–.71).

Mediation was examined with a distribution-of-products approach (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). A confidence interval for the indirect effect of depressive symptoms (independent variable) on percentage body fat (dependent variable) via eating behavior (mediator: total energy intake or sweet snacks intake) was derived based upon the product of α, the effect of the independent variable on the mediator, and β, the effect of the mediator on the dependent variable. Estimates for α were obtained by regressing eating behavior (total energy or sweet snacks intake) on depressive symptoms, dietary restraint, age, race, height, fat-free mass, and meal exposure. Estimates for β were obtained by regressing percentage body fat on depressive symptoms, dietary restraint, age, race, height, fat-free mass, meal exposure, total energy intake, and sweet snacks intake. If the interaction effects were significant for depression by sex or depression by restraint, we estimated mediation effects for each level of the moderator separately.

Results

Descriptive information: Participants were 228 adolescents, 123 girls with an average age of 15.4 years (SD = 1.4) and 105 boys age 15.1 years (SD = 1.3; Table 1). The racial/ethnic composition of the sample approximated that of the DC metro area (U.S. Department of Commerce, 2011). Boys were taller than girls (170.7 ± 8.9 vs. 162.7 ± 6.8 cm, $p < .001$) and had greater fat-free mass (53.0 ± 12.7 vs. 45.2 ± 11.0 kg, $p < .001$). Conversely, girls had greater percentage fat mass than boys (29.8 ± 10.5 vs. 18.3 ± 9.3 %, $p < .001$). Depressive symptoms were significantly higher in girls as compared to boys (6.1 ± 5.8 vs. 4.0 ± 3.5, $p = .001$). A greater percentage of girls compared to boys reported elevated depressive symptoms (BDI-II > 13, 13.0% vs. 1.9%, $p = .002$).

Depressive symptoms and dietary restraint were positively correlated ($r = .17$, $p = .01$). Depressive symptoms also were positively correlated with percent body fat ($r = .23$, $p < .001$) and inversely correlated with fat-free body mass ($r = -.16$, $p = .01$). Depressive symptoms were not related to BMI *z* ($p = .53$). Dietary restraint was positively correlated with percent body fat ($r = .39$, $p < .001$) and BMI *z* ($r = .38$, $p < .001$), but was unrelated to fat-free mass ($p = .34$). These relationships were similar when girls and boys were examined separately.

Depressive symptoms, dietary restraint, sex, and eating behavior: Demographic and anthropometric factors accounted for a combined 14% of the variance in total energy intake in Step I ($p < .001$; Table 2). Adding depressive symptoms, dietary restraint, and sex to the prediction of total energy intake in Step II explained an additional 11% of the variance ($p < .001$). If depressive symptoms alone were added to the model in Step II, 2% additional variance in total energy consumed was explained ($p = .07$). Accounting for all other variables in the model, depressive symptoms were positively related to total energy intake, such that adolescents who reported higher depressive symptoms consumed more energy in the laboratory than those with lower symptoms ($p < .05$). Accounting for all other variables in the equation, each 1-unit increase in BDI-II depressive symptoms total score was associated with consumption of an additional 13 total kcal. Said differently, with all covariates being equal, adolescents with a BDI-II total score of 10 (1 SD above the sample mean) ate 1344 kcal, whereas adolescents with a BDI-II total score of 0 (1 SD below the sample mean) ate 1214 kcal ($d = 130$ kcal).

Also, boys consumed 443.2 ± 82.9 kcal more than girls ($p < .001$). Dietary restraint was not a significant predictor of total intake. The addition of the interactions of depressive symptoms by sex, depressive symptoms by dietary restraint, and dietary restraint by sex to the model in Step III was not significant ($\Delta R^2 = .00$, $p = .75$), indicating that the association between depressive symptoms and total energy intake did not significantly differ for boys compared to girls or by adolescents' level of dietary restraint.

When predicting energy from sweet snacks, demographic and anthropometric variables combined explained 39% of the variance in adolescents' sweet snacks intake in Step I ($p < .001$; Table 2). In Step II, depressive symptoms, dietary restraint, and sex explained an additional 4% of the variance ($p = .002$). The additional variance explained was driven by sex differences. Girls consumed 76.0 ± 21.5 kcal more from sweet snacks than boys, accounting for all other variables in the model ($p = .001$). The main effects of depressive symptoms and dietary restraint on sweet snacks intake were not significant ($p = .19$ and .84, respectively). Likewise, in Step III, the interactions between depressive symptoms and sex, depressive symptoms and restraint, and restraint by sex in Step III were not significant ($\Delta R^2 = .01$, $p = .33$).

Depressive symptoms-eating behavior-adiposity mediational model: The confidence intervals for the indirect effects of depressive symptoms on percentage body fat via total energy intake (CI = −.36, .32) or sweet snacks intake (CI = −.29, .25) were not significant. In the regression model predicting adolescent adiposity, depressive symptoms remained significantly associated with adiposity ($\beta = .15$, $p = .02$), even when total energy intake and sweet snacks intake were included in the model, along with all additional covariates.

Discussion

Consistent with our first hypothesis, the results of Study 1 suggest that depressive symptoms are related to greater overall energy intake in adolescents. Girls and boys who displayed higher levels of depressive symptoms consumed more energy overall at a lunch-type buffet meal in the laboratory, even after accounting for important covariates such as fat-free mass and adiposity. Nonetheless, depressive symptoms explained only a small percentage (2%) of the variability in total energy intake. Contrary to our second set of hypotheses, we found no evidence to support a stronger relationship between depressive symptoms and eating behavior in girls compared to boys or among adolescents with higher relative to lower levels of dietary restraint. These patterns are inconsistent with some adult studies. Women who experience stress or depressive symptoms report that they gravitate toward sweets more so than men (Jeffery et al., 2009; Konttinen et al., 2010; Mikolajczyk et al., 2009). Likewise, among adults, eating more in response to depressive symptoms or negative mood has been shown to occur among individuals who report dietary restraint, whereas no effect or the reverse has been shown in unrestrained adult eaters (Heatherton et al., 1991; Polivy & Herman, 1976). With respect to the third hypothesis, adolescents' depressive symptoms and adiposity were positively related in bivariate as well as multivariate models. However, we found no evidence that adolescents' eating behaviors mediated the association between depressive symptoms and adiposity. Depressive symptoms and adiposity remained significantly related to each other, independent of food intake.

Since the eating paradigm in Study 1 involved adolescents being served a buffet meal at 11:00am following an overnight fast, it is possible that those who would normally consume breakfast or who would typically eat lunch later in the day ate uncharacteristically. We, therefore, sought to replicate the findings from Study 1 using secondary analyses of a separate laboratory eating paradigm, at which youth's consumption from a similar lunch-type buffet meal was assessed following a standard breakfast.

Study 2

Methods

Participants and Procedure—Participants were children and adolescents, ages 8–17 years with a BMI > 5th percentile, recruited for a study of eating behavior (ClinicalTrials.Gov ID: NCT00320177). Recruitment and inclusion/exclusion criteria (other than the wider age range) were identical to Study 1. Written assent and consent was obtained from children and parents, respectively. The study was approved by the NICHD Institutional Review Board. Participants were financially compensated for participation. After an initial outpatient screening visit at the NIH Hatfield Clinical Research Center, eligible children and adolescents returned for an outpatient test meal appointment on a separate day. On both days, youth were instructed to adhere to a fast after 10:00pm the night before the visit.

Measures

Pubertal assessment: Testicular volume (for males) and breast development (for females) were assessed as described in Study 1.

Body composition: As in Study 1, height and fasting weight were used to compute BMI and BMI *z* score. Fat-free mass and percentage body fat were assessed with air displacement plethysmography.

Depressive symptoms: Depressive symptoms were assessed with the widely-used, reliable, and well-validated Children's Depression Inventory (CDI), a 27-item questionnaire measuring the severity of depressive symptoms over the past two weeks in children age 7–17 years (Kovac, 1992; Kovacs, 1985). Items were rated on a scale from 0 (absence of symptom) to 3 (high severity of symptom). A total score (range = 0–54) was derived from the sum of all items. Scores that exceed 12 may be indicative of elevated depressive symptoms (Kazdin, Colbus, & Rodgers, 1986).

Dietary restraint: As in Study 1, dietary restraint was measured with the restraint subscale score of the EDE or (for youth < 13 years) the EDE adapted for children (Bryant-Waugh, Cooper, Taylor, & Lask, 1996).

Eating behavior: Following a 10-hour overnight fast, participants were provided with a standard breakfast of 288 kcal, consisting of 240 mL apple juice, 1 English muffin, and 6 g butter, at 8:40am. After breakfast, youth took part in sedentary activities and were observed to ensure no further food intake until the buffet lunch meal was served at 2:30pm. Children were asked to eat from a 9,835-kcal buffet with a variety of individual lunch food items that differed in macronutrient composition (51% carbohydrate, 12% protein, and 37% fat). The buffet meal food items were very similar to those in Study 1. Tanofsky-Kraff et al. (2009) presents a detailed list of all food items served in Study 2 and their amounts. Participants were instructed: "Eat as you would at a normal meal." They were then left alone to eat ad libitum while viewing pre-taped episodes of a G-rated television show devoid of food, eating, or weight-related content. Total energy consumed was calculated by weighing each food and beverage item before and after the meal. Energy intake from sweet snacks was classified as consumption of the same food items as in Study 1. Energy content and nutrient composition for each food were determined using the USA Department of Agriculture Nutrient Database for Standard Reference as well as nutrient information supplied by food manufacturers.

Results

The statistical plan paralleled the analyses outlined in Study 1.

Descriptive information—There were 204 participants, 101 girls with an average age of 13.1 ± 2.6 years and 103 boys age 12.9 ± 2.9 years (Table 3). Girls had greater percentage fat mass than boys (31.1 ± 12.0 vs. 23.7 ± 12.9, $p < .001$). Similar percentages of girls and boys had elevated depressive symptoms (CDI > 12, 10.9% vs. 7.8%, $p = .44$). No other sex differences were observed in key variables.

Depressive symptoms and dietary restraint were positively correlated ($r = .37$, $p < .001$). In addition, depressive symptoms were positively related to percent body fat ($r = .21$, $p = .002$) and BMI *z* ($r = .18$, $p = .009$). Depressive symptoms were not related to fat-free mass ($p = .52$). Youth's dietary restraint was positively related to all body measurements, including body fat ($r = .53$, $p < .001$), fat-free mass ($r = .22$, $p = .002$), and BMI *z* ($r = .48$, $p < .001$). These correlations showed a very similar pattern among girls and boys.

Depressive symptoms, dietary restraint, sex, and eating behavior—Demographic and anthropometric variables combined in Step I accounted for 35% of the variance in total energy intake ($p < .001$; Table 4). Adding depressive symptoms, dietary restraint, and sex to the model in Step II explained an additional 8% of the variance in total energy intake ($p < .001$). The additional variability explained was due to sex differences; after accounting for all other variables in the model, boys consumed 369.3 ± 71.4 total kcal more than girls ($p < .001$). The main effects of depressive symptoms ($p = .74$) and restraint ($p = .83$) on total energy were not significant. The interactions terms added in Step III together added only a marginal amount of explained variability ($\Delta R^2 = .01$, $p = .12$). However, there was a significant interaction between depressive symptoms and sex. When this interaction was probed, depressive symptoms were related to greater total energy intake among girls ($\beta = .18$, $p = .05$), but not among boys ($\beta = -.14$, $p = .10$). If the non-significant interactions terms (depression by restraint and restraint by sex) were removed from the model, depressive symptoms by sex itself explained statistically significant additional variability ($\Delta R^2 = .01$, $p = .03$). Each 1-unit increase in girls' CDI depressive symptoms total score was associated with consumption of an additional 17 total kcal. Said differently, with all else being equal, girls with a CDI total score of 12 (1 SD above the sample mean) consumed 1309 total kcal, whereas girls with a CDI score of 0 (1 SD below the sample mean) consumed 1125 total kcal ($d = 184$ kcal).

Demographic and anthropometric factors combined in Step I explained 33% of the variance in energy consumed from sweet snacks ($p < .001$; Table 4). Depressive symptoms, dietary restraint, and sex together in Step II added a marginal 3% of explained variance in sweet snacks intake ($p = .10$). Depressive symptoms were positively associated with sweet snacks intake ($p = .02$). If this variable alone was added in Step II, a statistically significant 2% of variance in sweet snacks intake was explained ($p = .02$). There was no main effect of restraint or sex (ps > .33). In Step III, the inclusion of interaction terms added explained variance in the model ($\Delta R^2 = .03$, $p = .01$). Specifically, there was a significant depressive symptoms by restraint effect. When this effect was probed, we found that among youth who endorsed relatively higher levels of restraint, depressive symptoms were associated with eating more sweet snacks ($\beta = .30$, $p < .01$), whereas among youth with no restraint, depression and snack intake were not related ($\beta = -.06$, $p = .43$). The interactions of depression by sex and depression by restraint did not reach significance (ps > .21). Also, we verified that a three-way interaction of depression by restraint by sex was not significant ($p = .79$), indicating that the observed effect of depressive symptoms on sweet snacks intake among youth with dietary restraint did not differ significantly by sex.

Depressive symptoms-eating behavior-adiposity mediational model—The confidence intervals for the indirect effects of depressive symptoms on percentage body fat via total energy intake were estimated separately by sex because of the significant

depression by sex interaction in predicting total intake. These confidence intervals did not reach significance for either boys (CI = −.40, .55) or girls (CI = −.38, .56). The confidence intervals for the indirect effects of depressive symptoms on percentage body fat via sweet snacks intake were estimated separately for youth low compared to high in dietary restraint because of the significant depression by restraint interaction in predicting sweet snacks intake. These confidence intervals were not significant for youth low in restraint (CI = −.16, .16) or high in restraint (CI = −.49, .51).

Discussion

Consistent with our hypotheses for aims 1 and 2, only girls, but not boys, in Study 2 who reported greater depressive symptoms consumed more total energy overall than girls with lower symptoms, even after accounting for differences in body composition and other covariates. In addition, both girls and boys with greater depressive symptoms consumed more energy from sweet snack foods than those with lower depressive symptoms. However, this effect was modified by dietary restraint. Youth with higher levels of depressive symptoms only ate more energy from sweets if they also were restrained eaters. With respect to the third aim, youths' depressive symptoms and adiposity were positively related. However, as in Study 1, we found no support for eating behavior as a mediator of the depression-adiposity association.

General Discussion

Taken together, the results of Study 1 and Study 2 provide replicate findings that children and adolescents with higher levels of depressive symptoms consume more energy as measured objectively during laboratory test meals. In both studies, this effect was small, but statistically significant. In Study 1, this effect did not differ by sex, whereas in Study 2, only girls with elevated depressive symptoms ate more total energy. In Study 1, participants were adolescents ages 13–17 years, whereas in Study 2, we studied a broader age spectrum of children and adolescents ages 8–17 years. It is possible that depressive symptoms, which tend to rise during adolescence (Rutter, Graham, Chadwick, & Yule, 1976), begin to show a connection with eating behavior in boys as well as girls, but only among teens. In contrast, lower depressive symptoms during pre-adolescence might have made it harder to detect an effect for boys in Study 2. Although the adolescent rise in depressive symptoms is more pronounced in girls (Hankin et al., 1998), there are data to suggest that negative affect increases during adolescence in boys as well (Derdikman-Eiron et al., 2011; Lindsey, Joe, & Nebbitt, 2010).

To our knowledge, no prior studies examining the relationship of depression and eating behavior in youth have utilized laboratory measurements of energy intake. The current findings illustrate that depressive symptoms relate to greater overall intake, particularly in girls. Previous studies in adults relating depressed mood to measured eating behavior in the laboratory have yielded inconsistent findings, with some studies finding no relationship (Friedman & Brownell, 1995; Hallstrom & Noppa, 1981), others a positive relationship (Heo, Pietrobelli, Fontaine, Sirey, & Faith, 2006; Roberts, Kaplan, Shema, & Strawbridge, 2000), and others an inverse relationship such that adults with depression were observed to eat less (Baucom & Aiken, 1981; Beck, 1967). Individual differences in dietary restraint may be a part of this equation (Heatherton et al., 1991; Polivy & Herman, 1976). In Study 2, youth with more depressive symptoms consumed more energy from sweet snacks, but only if they also reported restraint over their eating. Theoretical frameworks for restraint and hunger purport that individuals who attempt to restrain their intake are inadvertently more vulnerable to overeating when dietary rules have been violated or through heightened responsiveness to environmental food cues (Lowe & Levine, 2005; Polivy & Herman, 1985). Yet, this pattern was not found in Study 1, and further, restraint had no main effect on

eating in either study. One limitation is that restraint was measured as a combination of cognitive or behavioral intent to control eating, likely blending individuals with unsuccessful and successful diet control attempts. Contemporary models call for a distinction between restraint and dieting (Stice, Cooper, Schoeller, Tappe, & Lowe, 2007; Stice, Fisher, & Lowe, 2004). Therefore, further work clarifying the role of restraint and dieting behavior in depressed mood and eating behavior will be necessary.

In both studies, children's and adolescents' depressive symptoms were significantly associated with having greater adiposity. Although the current data are cross-sectional, limiting causal inferences, this depression-adiposity connection is consistent with meta-analytic analyses of longitudinal studies finding that depressive symptoms are associated with an increased risk of becoming obese, particularly among girls (Blaine, 2008). The mechanisms explaining this relationship are not fully understood. In theory, individuals with negative feelings may be more likely to turn to comforting foods as a way to relieve sadness (Wurtman & Wurtman, 1995). Over time, this pattern may develop into a routine coping mechanism for dealing with depressed mood. Such increased energy consumption overall and of sweet snack foods high in sugar and fat would be expected to promote excessive weight and fat gain (Hooper et al., 2012). Yet, in neither study did we find support for the proposed mediational model that depressive symptoms would be related to adiposity via measured food intake patterns of either total energy or sweet snacks consumption. It is possible that behavioral factors (e.g., physical inactivity or sleep) and/or physiological mechanisms (e.g., hyperactivated stress systems) other than eating behavior explain depression's connection to adiposity. However, it is important to point out that the current studies measured "normative" eating behavior in the laboratory rather than acute, negative-affect induced eating behavior, and the latter could be expected to relate even more strongly to depressive symptoms. Moreover, our evaluation of a mediational process, which inherently would unfold over time, with cross-sectional rather than longitudinal data is limiting and requires examination with prospective data.

It is important to underscore that the cross-sectional nature of the data in both studies reported here precludes drawing causal inferences. Indeed, depressive symptoms could lead to greater intake, but alternatively, some evidence suggests that eating behaviors can affect mood states (Hendy, 2012; Sanchez-Villegas et al., 2012). Similarly, the correlational nature of the data means that the observed association between depressive symptoms and eating behavior could be accounted for by other variables. For instance, some (Anderson et al., 2007; Boutelle et al., 2010; Mustillo et al., 2003), but not all (Goodman & Whitaker, 2002; Stice & Bearman, 2001; Stice et al., 2000) longitudinal studies have found that obesity is associated with increases in future depressive symptoms and major depression among adolescents. In addition, the effects of depression on eating behavior were small relative to the effects of anthropometric variables. Another limitation of both studies includes the measurement of depressive symptoms using a self-report questionnaire, which does not permit a diagnostic assessment of depression as would a clinical interview. Since this sample was comprised of healthy adolescent volunteers with, at most, subthreshold depressive symptoms, the findings cannot be generalized to adolescents with clinical depression. Study of the connection between depression and eating behavior in adolescent samples with clinically elevated symptomatology may shed greater light on the magnitude of the relationship between depression and eating behavior. Likewise, although the samples approximated community norms for levels of elevated depressive symptoms and the racial/ethnic distribution of a large metropolitan area, the samples were convenience samples, which might limit generalizability.

Strengths of the current studies include their large sample sizes consisting of boys and girls varied in age and body weight. Also, body composition and eating behaviors were obtained

with objective measurements. Laboratory assessments of food intake often are more accurate than self-report measures of eating (Fisher et al., 2000; Wolkoff et al., 2011). Future studies should examine the depression-eating behavior relationship longitudinally and study experimentally how manipulation of negative affect acutely affects energy intake in children and adolescents. Furthermore, we suspect that individual difference factors such as genetic predispositions to depression or obesity likely play a moderating role in determining who is susceptible to overeating versus undereating when depressed or stressed. Identification of such factors will help to facilitate the development of targeted and coordinated preventive and intervention approaches for obesity and for depression.

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Highlights

- Depressive symptoms were linked to youths' observed eating behavior in 2 studies.
- In Study 1, girls and boys with higher symptoms ate more calories during a meal.
- In Study 2, girls, but not boys, with higher symptoms ate more total calories.
- In Study 2, youth with higher depressive symptoms and restraint ate more sweets.

Table 1

Demographic and anthropometric characteristics of Study 1 participants

	Girls	Boys
N	123	105
Age (years)	15.4 ± 1.4	15.1 ± 1.3
Race/ethnicity (% non-Hispanic White)	54.5	55.2
Height (cm)***	162.7 ± 6.8	170.7 ± 8.9
Fat mass (%)***	29.8 ± 10.5	18.3 ± 9.3
Fat-free mass (log kg)***	45.2 ± 11.0	53.0 ± 12.7
BMI *z*	.7 ± 1.1	.5 ± .9
Overweight (%)	38.3	29.0

*** $p < .001$.

Table 2

Hierarchical multiple linear regressions examining main and interactional effects of depression, dietary restraint, and sex on Study 1 adolescents' total and sweet snacks intake (kcal)

	Total Energy Intake			Sweet Snacks Intake		
	B	**SE**		**B**	**SE**	
I. Age (y)	−51.31	26.06	−.14*	−6.60	6.19	−.06
Race/ethnicity	74.49	66.94	.07	−10.65	15.80	−.04
Height (cm)	.84	5.69	.01	.37	1.34	.02
Fat mass (%)	−4.78	3.02	−.11	−.82	.72	−.07
Fat-free mass (log kg)	1842.92	510.59	.35***	−218.53	123.77	−.15
Meal exposure	44.91	77.18	.04	34.07	18.18	.10
Total intake (kcal)	--	--	--	.17	.02	.62***
		R^2 = .14*** ΔR^2 = .14***			R^2 = .39*** ΔR^2 = .39***	
II. Depression	13.05	6.51	.13*	2.13	1.60	.07
Dietary restraint	−40.33	47.92	−.06	2.35	11.71	.01
Sex	−443.22	82.90	−.43***	75.95	21.54	.27***
		R^2 = .25*** ΔR^2 = .11***			R^2 = .43*** ΔR^2 = .04**	
III. Depression x sex	8.72	16.04	.07	−7.08	3.89	−.21
Depression x restraint	−9.68	10.00	−.06	.49	2.43	.01
Restraint x sex	−29.20	92.00	−.03	−.61	22.33	.00
		R^2 = .25*** ΔR^2 = .00xxx			R^2 = .44*** ΔR^2 = .01xx	

B = Unstandardized regression coefficient. SE = Standard error. β = Standardized regression coefficient. Race was dummy coded: 0 = non-Hispanic White vs. 1 = Other. Sex was dummy coded: 0 = Male vs. 1 = Female. Meal exposure was dummy coded: 0 = prior meal vs. 1 = no prior meal.

* $p < .05$

** $p < .01$

*** $p < .001$.

Table 3

Demographic and anthropometric characteristics of Study 2 participants

	Girls	Boys
N	101	103
Age (years)	13.1 ± 2.6	12.9 ± 2.9
Race (% non-Hispanic White)	56.4	64.1
Height (cm)	155.3 ± 12.6	157.8 ± 17.3
Fat mass (%)***	31.1 ± 12.0	23.7 ± 12.9
Fat-free mass (log kg)	41.7 ± 13.5	44.4 ± 16.7
BMI z	1.0 ± 1.1	.9 ± 1.1
Overweight (%)	49.5	45.6

*** $p < .001$.

Table 4

Hierarchical multiple linear regressions examining main and interactional effects of depression, dietary restraint, and sex on Study 2 participants' total and sweet snacks intake (kcal)

	Total Intake			Sweet Snacks Intake		
	B	**SE**		**B**	**SE**	
I. Age (y)	−54.85	23.97	−.25*	−2.74	8.77	−.04
Race	318.43	76.33	.26***	−30.24	28.78	−.07
Height (cm)	13.91	5.64	.35**	−.22	2.07	−.02
Fat mass (%)	5.52	2.84	.12*	1.18	1.04	.07
Fat-free mass (log kg)	1256.21	490.38	.32**	−153.30	179.94	−.11
Meal exposure	51.09	72.00	.04	64.39	26.01	.15**
Total intake (kcal)	--	--	--	.21	.03	.60***
			$R^2 = .35^{***}$ $\Delta R^2 = .35^{***}$			$R^2 = .33^{***}$ $\Delta R^2 = .33^{***}$
II. Depression	2.28	6.98	.02	6.10	2.64	.15*
Restraint	12.51	58.00	.02	−1.74	21.95	−.01
Sex	−367.82	72.84	−.31***	28.21	29.42	.07
			$R^2 = .43^{***}$ $\Delta R^2 = .08^{***}$			$R^2 = .36^{***}$ $\Delta R^2 = .03$
III. Depression x sex	37.88	15.55	.25*	6.28	5.89	.12
Depression x restraint	−7.91	7.34	−.08	6.33	2.75	.18*
Restraint x sex	−35.10	111.94	−.04	9.63	41.77	.03
			$R^2 = .44^{***}$ $\Delta R^2 = .01$			$R^2 = .39^{***}$ $\Delta R^2 = .03^{**}$

B = Unstandardized regression coefficient. SE = Standard error. β = Standardized regression coefficient. Race was dummy coded: 0 = non-Hispanic White, 1= Other. Sex was dummy coded: 0 = Male, 1=Female. Meal exposure was dummy coded: 0 =no prior meal, 1= prior meal.

* $p < .05$

** $p < .01$

*** $p < .001$.