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Emotion regulation and loss of control eating in community-based adolescents

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Abstract

The current study investigated concurrent and prospective associations between emotion-related constructs and loss of control (LOC) eating in adolescents. Community-based females ($N=588$) completed annual self-report assessments of LOC eating, emotional awareness, emotion regulation strategies, and neuroticism from ages 16–18 years. Linear regressions and a regression-based multiple mediation model using bootstrapping were computed to examine the relationships among emotion-related constructs and LOC eating frequency. In the concurrent model, age 18 emotional awareness and emotion regulation strategies were associated with age 18 LOC eating, $F(6, 416)=12.11$, $p<.001$, accounting for 4.5% of the variance after controlling for demographics, body mass index, and neuroticism, $F\text{change}=10.81$, $p<.001$. In the prospective model, age 17 emotional awareness predicted age 18 LOC eating, $F(7, 425)=11.67$, $p<.001$, accounting for 1.7% of unique variance beyond the effects of age 16 LOC eating and age 17 demographics, body mass index, and neuroticism, $F\text{change}=4.26$, $p=.015$. In the multiple mediation model, age 18 emotion regulation strategies mediated the association between age 17 neuroticism and age 18 LOC eating, indirect effect estimate=.003, 95% confidence interval=.001–.005, after controlling for age 16 LOC eating and age 17 demographics, body mass index, and emotion regulation variables. Results suggest that deficient emotion regulation may contribute to the onset and maintenance of LOC eating in adolescence (although effects were small), and may partially explain the well-established prospective relationship between negative emotionality and later LOC eating. Prevention and early intervention programs should seek to improve adaptive coping in at-risk populations.

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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 was obtained from all individual participants included in the study.

Keywords

Loss of control; binge eating; emotion regulation; negative affect; risk factors

Loss of control (LOC) while eating, characterized by the sense that one cannot control what or how much one is eating, is a key feature of binge eating (American Psychiatric Association, 2013). LOC eating is associated with obesity, excess weight gain, and psychosocial impairment (Goldschmidt, Aspen, Sinton, Tanofsky-Kraff, & Wilfley, 2008), independent of the amount of food consumed (Goldschmidt et al., 2012; Goldschmidt, Jones, et al., 2008; Shomaker et al., 2010). Although LOC eating is reported by approximately 10–15% of community-based adolescents (Johnson, Rohan, & Kirk, 2002; Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011), there is an insufficient understanding of its contributing factors and, consequently, a relative dearth of effective interventions for this age group (Lock, 2015). Preliminary data suggest that difficulties with emotion regulation may underlie LOC eating behavior (Leehr et al., 2015), but research in youth is scant (Czaja, Rief, & Hilbert, 2009) and prospective data linking emotion regulation deficits to subsequent LOC eating are virtually nonexistent.

Negative affect is one construct that has been consistently associated with LOC eating across the age spectrum (Goldschmidt, Aspen, et al., 2008; Haedt-Matt & Keel, 2011). Negative affect often refers to a state construct describing the experience of distressing or unpleasant emotions, but it is highly correlated with the temperamental trait of neuroticism/negative emotionality, representing one's proclivity towards negative affect (Watson & Naragon-Gainey, 2014). Negative affect (at both the state and trait level) is elevated in adolescents with LOC eating in cross-sectional studies (Goldschmidt, Jones, et al., 2008; Shomaker et al., 2010), and also has been identified as a risk factor for the development of LOC eating in adolescence (Stice, 2002). LOC eating has been conceptualized as a maladaptive method of alleviating negative affect, a theory that is supported by multiple naturalistic and experimental studies indicating that increases in negative affect serve as a momentary precipitant to LOC eating episodes (Haedt-Matt & Keel, 2011). However, the nature of the relationship between negative affect and LOC eating is poorly understood and it is unclear whether other factors, such as a lowered capacity to modulate or cope with negative affect, may explain this relationship.

Emotion regulation refers to intentional or automatic strategies that influence the type, intensity, and duration of emotions one experiences (Gross & Barrett, 2011). Emotion regulation is believed to develop as a function of interacting temperamental (e.g., negative emotionality) and learned factors (e.g., familial socialization; learned beliefs that negative emotions are intolerable; John & Gross, 2004). LOC eating may be one behavioral outcome resulting from poor emotion regulation, emerging in part based on learned expectancies that eating will alleviate negative emotions (Combs, Smith, & Simmons, 2011). Experiencing eating as an *effective* (albeit *maladaptive*) emotion regulation strategy may strengthen this expectancy, such that LOC eating eventually operates through negative reinforcement (Pearson, Wonderlich, & Smith, 2015). In this way, emotion regulation and LOC eating are distinct, but overlapping, developmental processes. Given age-related changes in emotion

regulation and propensity towards LOC eating (Swanson et al., 2014; Zeman, Cassano, Perry-Parrish, & Stegall, 2006), there may be developmental windows in which the link between deficient emotion regulation and LOC eating is particularly strong. For example, this link may become more evident throughout adolescence and adulthood as associations between LOC eating and distress relief strengthen over time with repeated pairings, whereas the link may be weaker in early/middle childhood due to less consolidated learned associations between LOC eating and distress relief (Nevin & Grace, 2000). However, normative developmental changes in emotion regulation and relevant brain regions may also complicate associations between these constructs during adolescence.

Individuals with binge eating disorder (BED), who engage in recurrent overeating accompanied by LOC (American Psychiatric Association, 2013), report poorer emotion regulation than healthy, overweight, or depressed controls (Brockmeyer et al., 2014; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012). Moreover, poor emotion regulation (specifically, difficulties identifying and understanding one's emotions, and having limited access to emotion regulation strategies) predicts LOC eating severity among college students (Markey & Vander Wal, 2007; Whiteside et al., 2007) and adults (Carano et al., 2006; Gianini, White, & Masheb, 2013). Experimental data suggest that adults with BED are more likely to regulate negative emotions via maladaptive strategies such as suppression (i.e., inhibition of emotion-expression behavior), and less likely to utilize adaptive strategies such as reappraisal (i.e., generating alternative interpretations of an emotional stimulus), than overweight controls (Svaldi, Tuschen-Caffier, Trentowska, Caffier, & Naumann, 2014). Furthermore, some (but not all; Dingemans, Martijn, Jansen, & van Furth, 2009) data suggest that suppression, but not reappraisal, increases the desire to engage in LOC eating (Svaldi, Caffier, & Tuschen-Caffier, 2010) and is associated with greater energy intake (Svaldi et al., 2014).

In contrast to this growing body of literature in adults, there is a relative dearth of research linking emotion regulation constructs to LOC eating in adolescents. Several studies have shown that the dispositional tendency to behave rashly when distressed (i.e., negative urgency) predicts LOC eating onset in youth (Combs, Pearson, & Smith, 2011; Fischer, Peterson, & McCarthy, 2013; Kelly, Cotter, & Mazzeo, 2014; Pearson, Combs, Zapolski, & Smith, 2012), but to our knowledge, only one study has examined dimensions of emotion regulation more broadly among youth with LOC eating (Czaja et al., 2009). In this small cross-sectional study of pre-adolescent children, those with LOC eating reported greater use of maladaptive emotional regulation strategies such as perseverating and giving up than controls. Prospective data collected in adolescence, a key developmental timeframe for the emergence of LOC eating (Kessler et al., 2013), are needed to further explore the relationship between emotion regulation and LOC eating in order to inform prevention and early intervention strategies.

The purpose of the current study was to examine the extent to which theoretically relevant dimensions of emotion regulation (i.e., emotional awareness, access to effective emotion regulation strategies) are concurrently and prospectively associated with LOC eating in adolescents. A secondary aim was to investigate whether these constructs mediate the relationship between negative emotionality and LOC eating. We hypothesized that lower

emotional awareness and limited access to emotion regulation strategies would predict more frequent LOC eating both concurrently and prospectively, as well as mediate the prospective relationship between negative emotionality and LOC eating.

Methods

Participants

Data were drawn from the Pittsburgh Girls Study, a community-based prospective study of psychosocial functioning from childhood to adulthood (Loeber et al., 2002). A stratified, random household sampling method, with oversampling of households in low-income neighborhoods, was used to identify girls for the first wave of data collection, when girls were ages five to eight years. All homes in neighborhoods in which at least 25% of the residents were living at or below the poverty level were contacted in person to determine if an age-eligible girl was living in the home. A randomly selected 50% of the households in non-risk neighborhoods were contacted during 1998 and 1999. 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 were ultimately included in the study; the other 668 households were either excluded (due to intellectual disability in the eligible girl or imminent plans to relocate) or were unwilling to participate. 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.

The current study utilized data from 588 girls who were assessed at ages 16–18 years (spanning assessment waves 12–14 of the Pittsburgh Girls Study), when measures relevant to the study aims were administered. The girls were, on average, 16.5 years of age at the first assessment point in the current set of analyses, and most were Caucasian (52.1%) or African-American (41.7%; see Table 1 for descriptive information). Approximately one-third ($n=210$) of the families were receiving public assistance (i.e., Women, Infants, and Children, food stamps, welfare). Retention has ranged from 85–86% in data collection waves 12–14, with attrition due in part to difficulty tracking participants. There was very little attrition due to refusal to participate.

All study procedures were approved by the University of Pittsburgh Institutional Review Board. All primary caretakers provided written informed consent, and 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 16, 17, and 18 years. Girls' age, race, and socioeconomic status (SES) were based on parent-report.

The Eating Attitudes Test-26 (EAT-26; Garner, Olmsted, Bohr, & Garfinkel, 1982), a 26-item self-report measure of eating disturbance, was administered at ages 16 and 18 years. LOC eating is ascertained via one item asking the respondent how often, in the past year, "I

have gone on eating binges where I feel that I might not be able to stop” (response options include *never*, *rarely*, *sometimes*, *often*, *very often*, and *always*).

Lack of emotional awareness and limited access to adaptive emotion regulation skills were measured at ages 17 and 18 years using two subscales of the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004): *awareness* and *strategies*, respectively. The awareness subscale contains six items, and the strategies subscale contains eight items, all of which are rated on a five-point scale, with higher scores reflecting greater difficulties in the particular emotion regulation domain. In the current sample, the alpha coefficient for the awareness subscale was .88 at age 17 and .87 at age 18, while the alpha coefficient for the strategies subscale was .80 at age 17 and .82 at age 18.

The *neuroticism* scale of the Neuroticism-Extraversion-Openness Five Factor Inventory-3 (NEO-FFI-3; McCrae & Costa, 2010), a self-report personality assessment, was administered at ages 17 and 18 years. The neuroticism subscale contains 12 items inquiring about the respondent’s tendencies towards negative emotionality (e.g., “I am not a worrier,” “sometimes I feel completely worthless”), each rated on a five-point scale, ranging from *strongly disagree* to *strongly agree*. In the current study, the alpha coefficient for the neuroticism scale was .71 at age 17 and .77 at age 18.

Statistical Analysis

All data were analyzed in SPSS 22.0. Missing data were excluded from analyses in a listwise fashion. Less than 20% of the sample was missing data on any given measure at each time point ($n=102$ missing age 16 LOC eating, 17.3%; $n=97$ missing age 18 LOC eating, 16.5%; $n=104$ missing age 17 awareness and strategies, 17.7%, respectively; $n=97$ missing age 18 awareness and strategies, 16.5%, respectively; $n=102$ missing age 17 neuroticism, 17.3%; $n=95$ missing age 18 neuroticism, 16.2%). Chi-square analyses and *t*-tests were used to evaluate demographic and psychosocial factors associated with missing LOC eating data. Logarithmic transformations were applied to age 16 and age 18 LOC eating frequency to correct for positive skew. Two separate linear regression models were computed to examine concurrent and prospective associations between emotion regulation constructs and LOC eating frequency. For the concurrent model ($n=417$), emotional awareness and emotion regulation strategies at age 18 were examined as correlates of age 18 LOC eating frequency, controlling for race, SES, BMI, and neuroticism at age 18. For the prospective model ($n=426$), emotional awareness and emotion regulation strategies at age 17 were examined as predictors of age 18 LOC eating frequency, controlling for age 16 LOC eating frequency, and race, SES, BMI, and neuroticism at age 17. A regression-based bootstrapping approach ($n=431$) with $k=5,000$ re-samples and 95% bias-corrected confidence intervals using the PROCESS macro (Hayes, 2013) in SPSS was used to simultaneously evaluate the indirect effects of age 17 neuroticism on age 18 LOC eating frequency through age 18 emotion regulation constructs, adjusting for the above-mentioned covariates, as well as for age 17 emotion regulation constructs. This approach has been recommended as the most powerful method for estimating indirect effects, even in large samples, in part because it does not rely on assumptions of normality (Preacher & Hayes, 2008). A regression-based approach was selected to maintain consistency with the preceding

sets of analyses, and its use was informed by a priori hypotheses about the directionality of effects. Given that the three main variables in the indirect effect analysis were not available at all three time points (i.e., full temporal precedence could not be established), the decision was made to establish temporal precedence between the independent variable (neuroticism) and the mediating variables (emotion regulation constructs) due to the more substantial overlap between these constructs (i.e., both emotion-related constructs; see Table 2). Significant mediation was considered to have occurred if the 95% confidence intervals for the indirect effect did not contain zero. All data were weighted to account for oversampling in low-income neighborhoods, with the exception that mediation analyses utilized unweighted data since sample weights cannot be implemented in PROCESS.

Results

Descriptive Characteristics and Intercorrelations

At age 16, 23.9% of the sample ($n=141$) reported ever engaging in LOC eating (*rarely to always*), and 23.8% ($n=140$) reported ever engaging in LOC eating at age 18 (see Table 1 for descriptive characteristics). Individuals who were missing data on LOC eating did not significantly differ from the rest of the sample on race, SES, BMI, emotional awareness, emotion regulation strategies, or neuroticism.

Correlations among the independent, dependent, and mediating variables were mostly in the medium range (see Table 2).

Concurrent Prediction of Loss of Control Eating Frequency

Poor emotional awareness and deficient emotion regulation strategies at age 18 each were associated with LOC eating frequency at the same age, accounting for the effects of race, SES, BMI, and neuroticism at age 18. The full model, including both emotion regulation constructs, accounted for 15.0% of the variance in age 18 LOC eating frequency, $F(6, 416)=12.11$, $p<.001$, with emotional awareness and emotion regulation strategies accounting for an additional 4.5% of the variance beyond the effects of age 18 control variables, F change=10.81, $p<.001$ (see Table 3).

Prospective Prediction of Loss of Control Eating Frequency

Poor emotional awareness at age 17 independently predicted age 18 LOC eating frequency, controlling for the effects of age 16 LOC eating frequency and race, SES, BMI, and neuroticism at age 17. The full model, including both age 17 emotion regulation constructs, accounted for 16.3% of the variance in age 18 LOC eating frequency, $F(7, 425)=11.67$, $p<.001$, with emotional awareness and emotion regulation strategies accounting for an additional 1.7% of the variance beyond the effects of age 16 and 17 control variables, F change=4.26, $p=.015$. However, deficient emotion regulation strategies at age 17 was not an independent predictor, $p=.340$ (see Table 4).

Mediation Analyses

Multiple mediation analysis indicated that age 18 emotional awareness and emotion regulation strategies mediated the relation between age 17 neuroticism and age 18 LOC

eating frequency, after controlling for age 16 LOC eating frequency and race, SES, BMI, and emotion regulation constructs at age 17. Deficient emotion regulation strategies was a significant independent mediator, indirect effect estimate=.003, 95% confidence interval=.001–.005, effect ratio=.600, while poor emotional awareness was not, indirect effect estimate=.001, 95% confidence interval=–.000–.005, effect ratio=.111 (see Table 5). The direct effect of age 17 neuroticism on age 18 LOC eating frequency was nonsignificant.

Discussion

The purpose of the current study was to explore the concurrent and prospective relationships between emotion regulation, negative emotionality, and LOC eating in a community-based sample of adolescent girls. We found that poor emotional awareness and limited access to adaptive emotion regulation strategies were uniquely associated with greater LOC eating concurrently at age 18, and that poor emotional awareness at age 17 uniquely predicted greater LOC eating frequency at age 18. Furthermore, limited access to adaptive emotion regulation strategies at age 18 significantly mediated the relationship between neuroticism at age 17 and LOC eating frequency at age 18 (accounting for 60% of the effects of neuroticism on LOC eating frequency), suggesting that poorer emotion regulation at least partially explains the well-established association between negative affect and later LOC eating (Stice, 2002). Overall, results provide support for the role of emotion dysregulation in the development and severity of adolescent LOC eating, although it should be noted that effect sizes were mostly small, suggesting that other variables that were not assessed in the current study (e.g., interpersonal dysfunction, negative urgency; Elliott et al., 2010; Pearson et al., 2012) could account for unexplained variance. Our findings add to the growing literature suggesting that emotion regulation is a relevant construct in the etiology and maintenance of eating disorders (Leehr et al., 2015; Svaldi et al., 2010; Svaldi et al., 2012), supporting the use of interventions aimed at improving one's understanding, tolerance, and management of negative emotions.

Further research is needed to clarify the means by which deficient emotion regulation contributes to LOC eating in adolescents at both the trait- and state-level. For example, LOC eating may develop in the context of a lack of adaptive emotion regulation strategies, in part based on learned expectancies that negative emotions are intolerable (Corstorphine, Mountford, Tomlinson, Waller, & Meyer, 2007) and that eating will alleviate negative emotions (Combs, Smith, et al., 2011). Experiencing eating as an efficient means of alleviating negative affect, and perhaps failing to develop alternative (more adaptive) strategies as a consequence, may strengthen these expectancies, such that emotion regulation ultimately promotes the momentary occurrence of LOC eating through negative reinforcement contingencies (Pearson et al., 2015). This, and other possible models, should be explored in future studies.

This study was marked by several important strengths, including the large, racially and socioeconomically diverse sample of community-based girls, and the prospective nature of the design. However, there are also several limitations. First, because of the epidemiological nature of the study, self-report questionnaires were used to assess LOC eating (which was operationalized using a single questionnaire item, which, although consistent with previous

research on LOC eating in children, could have introduced measurement bias; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008) and emotion-related constructs. Because adolescents may tend to over-report LOC eating behaviors on self-report questionnaires (Decaluwe & Braet, 2004; Goldschmidt, Doyle, & Wilfley, 2007) due to various forms of bias (De Los Reyes & Kazdin, 2005), it is unclear whether similar findings would apply to LOC eating as identified by practitioners in clinical settings, or using more objective assessment tools. Second, due to the absence of a measure of neuroticism at age 16, we were unable to establish full temporal precedence in our mediation model (i.e., age 17 emotion regulation mediating the association between age 16 neuroticism and age 18 LOC eating frequency). Thus, the directionality of the relationship between emotion regulation and LOC eating is unclear, such that having few adaptive strategies may promote LOC eating, and LOC eating may simultaneously impair the use of adaptive strategies, perhaps via increased reliance on LOC for coping resulting in reduced opportunities to implement more adaptive skills (Pearson et al., 2015). Additional research is needed to investigate the directionality of effects. Third, due to the small number of participants endorsing LOC eating at each time point (i.e., ~24% reporting any LOC eating in the past year at ages 16 and 18), we were unable to examine whether emotion regulation is a risk factor for new onset LOC eating, which is important in terms of identifying foci for preventive interventions and should be investigated in future studies. Fourth, the PROCESS macro did not allow for the application of sample weights, thus the findings from our indirect effects analyses cannot be generalized beyond our sample, which was oversampled for low income. However, of note, when we re-ran our mediation analyses using an alternate approach (Baron & Kenny, 1986) which allowed us to implement sample weights, results were consistent with the findings from our regression-based bootstrapping analysis. Finally, the exclusively female sample precludes generalization of our findings to males.

In general, deficits in emotion regulation may serve as a precipitating and/or maintaining factor for LOC eating in adolescents, especially when preceded by higher overall negative emotionality, and thus should be further explored in future studies, including extending findings to eating disorder samples. Importantly, the findings expand on the extant literature by suggesting that negative emotionality may not directly impact on loss of control eating, but rather operates indirectly through poor emotion regulation. Prospective data collected over a longer timeframe are needed to understand the optimal timing of interventions designed to prevent the onset of LOC eating, and momentary data could elucidate whether emotion dysregulation serves as an acute trigger for LOC eating episodes, which could further inform the development of effective interventions for this problem. Moreover, given that dialectical behavior therapy, which focuses on reducing emotion dysregulation and related maladaptive behaviors, has produced encouraging results in adult BED (Berg & Wonderlich, 2013) and has been shown to reduce LOC eating pathology in adolescents in a small pilot study as well (Fischer & Peterson, 2015), the current findings should be further explored in the context of prevention and early intervention research.

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Table 1Descriptive statistics ($M \pm SD$, unless otherwise indicated)

Variable		Age 16	Age 17	Age 18
<i>Demographic factors</i>				
Race, % (<i>n</i>)	Caucasian	52.1 (253)		
	African-American	41.7 (302)		
	Asian	1.7 (8)		
	Multi-racial/other	4.5 (25)		
Age, y		16.5 $\pm$ 0.4	17.3 $\pm$ 0.4	18.3 $\pm$ 0.3
Public assistance, % (<i>n</i>) receiving		32.0 (210)	30.3 (204)	6.1 (42)
BMI		25.14 $\pm$ 6.78	25.53 $\pm$ 6.97	26.27 $\pm$ 7.05
<i>Psychological factors</i>				
Loss of control eating frequency in the past year, % (<i>n</i>)	Never	71.1 (347)		71.5 (353)
	Rarely	19.2 (94)		14.6 (72)
	Sometimes	5.4 (26)		7.6 (37)
	Often	2.3 (11)		3.6 (18)
	Very often	1.2 (6)		1.0 (5)
	Always	0.8 (4)		1.7 (8)
Neuroticism (range=0–60)			20.6 $\pm$ 6.0	20.2 $\pm$ 6.7
Emotional awareness (range=6–36)			15.1 $\pm$ 5.8	14.3 $\pm$ 5.5
Emotion regulation strategies (range=8–40)			14.9 $\pm$ 5.3	14.3 $\pm$ 5.2

Note: BMI=body mass index (kg/m^2). All *ns* are raw, and all percentages/means are weighted to account for oversampling in low-income neighborhoods.

Table 2

Pearson bivariate correlations among the dependent, independent, and mediator variables

Variable	1	2	3	4	5	6	7	8
1.Age 17 neuroticism	---	---	---	---	---	---	---	---
2.Age 18 neuroticism	.63***	---	---	---	---	---	---	---
3.Age 17 emotional awareness	.14**	.23***	---	---	---	---	---	---
4.Age 17 emotion regulation strategies	.50***	.47***	---	---	---	---	---	---
5.Age 18 emotional awareness	.21***	.24***	.55***	.12*	---	---	---	---
6.Age 18 emotion regulation strategies	.41***	.59***	.19***	.55***	.14**	---	---	---
7.Age 16 LOC eating frequency	.19***	.20***	.03	.28***	.03	.25***	---	---
8.Age 18 LOC eating frequency	.24***	.31***	.15***	.21***	.17***	.32***	.33***	---

Note: LOC=loss of control

* $p < .05$;

**

$p < .01$;

$p < .001$

Summary of concurrent hierarchical regression analysis for age 18 variables predicting age 18 loss of control eating frequency

Table 3

Predictor	B	SE	β	p-value	R
Step 1					.106***
Race	0.00	0.01	-0.00	.951	
Public assistance	-0.04	0.04	-0.05	.307	
BMI	0.00	0.01	0.03	.483	
Neuroticism	0.01	0.01	0.32	<.001	
Step 2					.045***
Race	0.00	0.01	0.00	.987	
Public assistance	-0.04	0.04	-0.06	.234	
BMI	0.00	0.01	0.03	.588	
Neuroticism	0.01	0.00	0.16	.007	
Emotional awareness	0.01	0.00	0.14	0.003	
Emotion regulation strategies	0.01	0.00	0.21	<.001	

Note: BMI=body mass index (kg/m^2)

 $p < .001$

Table 4

Summary of prospective hierarchical regression analysis for age 17 variables predicting age 18 loss of control eating frequency

Predictor	B	SE	β	p-value	R ²
Step 1					.146***
Race	−0.00	0.01	0.00	.998	
Public assistance	0.01	0.02	0.02	.718	
BMI	−0.00	0.00	−0.05	.321	
Loss of control eating frequency	0.33	0.05	0.30	<.001	
Neuroticism	0.01	0.00	0.19	<.001	
Step 2					.017*
Race	−0.00	0.01	−0.00	.925	
Public assistance	−0.00	0.02	−0.00	.979	
BMI	−0.00	0.00	−0.06	.224	
Loss of control eating frequency	0.33	0.05	0.29	<.001	
Neuroticism	0.01	0.00	0.15	.005	
Emotional awareness	0.01	0.00	0.13	.006	
Emotion regulation strategies	0.00	0.00	0.05	.34	

Note: BMI=body mass index (kg/m^2). All predictors were measured at age 17 with the exception that loss of control eating frequency was measured at age 16.

* $p < .05$;

*** $p < .001$

Table 5

Summary of multiple mediation model results

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 17 neuroticism	Age 18 emotional awareness	Age 18 loss of control eating frequency	.185 *** (S.E.=.045)	.003 (S.E.=.002)	.003 (S.E.=.002)	.001; (95% CI= -.000–.005)	.005 * (S.E.=.002)
	Age 18 emotion regulation strategies		.346 *** (S.E.=.039)	.008 *** (S.E.=.002)		.003 ^a (95% CI= .001–.005)	

Note. CI=confidence interval^aSignificance levels are determined by 95% confidence intervals not containing zero* $p < .05$;*** $p < .001$