Childhood Loss of Control Eating Over Five-Year Follow-up

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CHILDHOOD LOSS OF CONTROL EATING OVER FIVE YEARS

ABSTRACT

Objective: Emerging prospective evidence from mixed samples, mostly covering short-term follow-up periods, suggests that childhood loss of control (LOC) eating predicts significant impairment in mental and physical health. This study sought to investigate the natural course of childhood LOC eating over the long term and in relation to binge eating disorder (BED) diagnosis, psychopathology, and body weight trajectory in the community.

Method: A total of 60 children (8-13 years) with LOC eating within the past three months and 60 demographically-matched children without LOC history were assessed with the Eating Disorder Examination adapted for Children and self-report questionnaires over a 5.5 year follow-up period. Missing data were imputed.

Results: Over follow-up, 38.3% of children showed persistent LOC eating, and 28.3% revealed an onset of LOC eating. Persistent LOC eating significantly predicted onset of partial-/full-syndrome BED at follow-up. Negative prognostic effects on eating disorder psychopathology, depressive symptoms, and body mass index were non-significant.

Discussion: The results indicate a moderate stability of LOC eating over the long term. LOC eating, especially if stable, was suggested as a variable risk factor of clinically relevant eating disturbances. In contrast, a prognostic value for psychopathology and body mass index was not confirmed.

Keywords: binge eating disorder; loss of control eating; depression; childhood; adolescence

CHILDHOOD LOSS OF CONTROL EATING OVER FIVE-YEAR FOLLOW-UP

Loss of control (LOC) eating, defined as eating an objectively or subjectively large amount of food accompanied by a sense of being unable to stop or control one's eating (1), affects 9.3% of 6-12 year old normal- and overweight children (2). Emerging prospective evidence suggests childhood LOC eating to result in significant mental and physical health impairments, although varying levels of persistence over the transition into adolescence were found. While two studies (including our previous work) reported moderate LOC persistence in children over the course of 4.7 and two years, respectively (52.2% and 45.4%) (3,4), another study found only 16.0% to engage in persistent LOC eating over one year (5). Notwithstanding, LOC eating predicted partial-/full-syndrome binge-eating disorder (BED) and increased eating disorder psychopathology, while results were inconsistent for the prediction of depressive symptoms and excess weight gain (3,4). As two of these studies oversampled overweight and obese children (3,5), further long-term evidence on the course of childhood LOC eating in the community is warranted.

This study extends our previous two-year natural course study of childhood LOC eating in the community (4) by a long-term follow-up after 5.5 years. Moderate persistence of LOC eating was expected. We hypothesized baseline LOC eating and LOC persistence (i.e., LOC eating at baseline and follow-up) to predict follow-up psychopathology, body weight, and BED.

METHOD

Participants and Procedure

Sixty 8-13 year old children with at least one episode of LOC eating over the previous three months (LOC+) and a demographically-matched control group without current or

lifetime LOC eating (LOC-) were recruited from the community (4,6-8). A long-term follow-up (t6) was conducted at an average of 5.47 ± 0.60 years after baseline (t1). At t1, participants were 10.77 ± 1.46 years old and had a body mass index (BMI, kg/m^2) of 23.02 ± 5.04 kg/m^2. At t6, participants were 16.89 ± 1.55 years old and had a BMI of 26.35 ± 9.86 kg/m^2. The sample was 56.6% (68/120) female.

At t6, data were collected from 63.3% (76/120) of the participants, with significantly lower completion in the LOC+ group than in the LOC- group (53.3% [32/60] vs. 73.3% [44/60]; $p < 0.02$). Reasons for drop-out (i.e., non-participation) were lack of contact information, decline to participate, or incapacitation. Drop-outs and completers did not differ in demographic and anthropometric characteristics, depressive symptoms, and LOC episodes (in LOC+ only) at t1. However, drop-outs reported significantly higher eating disorder psychopathology ($p < 0.001$).

Participants were assessed in person at t1, while at t6 telephone interviews were conducted (compensation €15). Ethical approval was granted by the ethics committees of the German Psychological Society and the Medical Faculty of the University of Leipzig, Germany.

Measures

At t1 and t6, LOC eating over the past 28 days was assessed by trained assessors with the semi-structured *Eating Disorder Examination adapted for Children* (*ChEDE*; diagnostic version) (9). The ChEDE was used to determine full-syndrome BED according to DSM-5 and partial-syndrome BED (i.e., at least one episode of LOC eating per week over the past three months; at least two out of five behavioral symptoms; some distress; absence of compensatory behaviors, anorexia nervosa, or bulimia nervosa) (4).

The *Eating Disorder Examination-Questionnaire adapted for Children* (*ChEDE-Q*) (10) and the *Children's Depression Inventory (CDI*) (11) were used to assess global eating disorder psychopathology over the past 28 days and depressive symptoms at t1 and t6. Participants' BMI (kg/m^2) was calculated from measured height and weight at t1 and from self-reported height and weight at t6.

Data Analytic Plan

Analyses were performed using IBM® SPSS® Version 20.0. Missing data at t6 were imputed through multiple imputation. The missing data model included demographic variables at t1 (age, sex, education) and t1 and t6 anthropometric (BMI) and clinical variables (LOC eating, diagnosis of partial-/full-syndrome BED, eating disorder psychopathology, depressive symptoms). Overall, ten data sets were produced (12).

Dependent samples *t* tests and χ^2 tests were conducted to compare LOC+ vs. LOC- on demographic, anthropometric, and clinical variables. Independent samples *t* tests served to examine differences for persistent vs. non-persistent LOC eating. Stepwise hierarchical regression analyses, adjusted for demographic and anthropometric variables, were conducted examining predictive effects of LOC eating (t1) on psychopathology and BMI (t6). Predictive effects of categorical LOC eating (t1; absence vs. presence) or persistence of LOC eating (absence vs. presence) on partial-/full-syndrome BED (t6; absence vs. presence) were examined using adjusted logistic regression analyses. Overall, two-tailed $p < 0.05$ was considered significant.

RESULTS

Differences in LOC episodes between LOC+ and LOC- were no longer significant at t6 ($p < 0.34$). While at t1 the LOC+ group displayed greater eating disorder psychopathology,

and depressive symptoms, but not BMI than the LOC- group (4), these differences were no longer significant at t6 (all $p > 0.05$). The number of LOC eating episodes at t1 did not predict eating disorder psychopathology ($B = 0.00$, $SE = 0.01$, $p < 0.88$), depressive symptoms ($B = -0.08$, $SE = 0.09$, $p < 0.38$), or BMI ($B = -0.01$, $SE = 0.04$, $p < 0.73$) at t6.

Persistent LOC eating at t1 and t6 occurred in 38.3% (23/60) of participants from the LOC+ group (remission: 61.7% [37/60]). In the LOC- group, 28.3% (17/60) reported onset of LOC eating from t1 to t6 (no LOC eating: 71.7% [43/60]). Persistence and onset of LOC eating were not predicted by demographic, anthropometric, and clinical variables at t1 (all $p > 0.05$). Persistent LOC eaters reported significantly more episodes of LOC eating than non-persistent LOC eaters at t6, but there were no differences regarding demographic variables, eating disorder psychopathology, depressive symptoms, and BMI (Table 1). However, small effect sizes indicated slightly increased eating disorder psychopathology and depressive symptoms in persistent LOC eaters.

At t1, 7.5% (9/60) of the LOC+ participants were diagnosed with partial BED and 2.5% (3/60) with full-syndrome BED. At t6, 10.0% (6/60) and 6.7% (4/60) of LOC+ participants (including 4/60 [6.7%] with persistent partial-/full-syndrome BED[2]) as well as 6.4% (4/60) and 3.3% (2/60) of LOC- participants were diagnosed with partial- or full-syndrome BED (age: 16.82 ± 2.29 years; female 56.3% [9/16]). The presence of LOC eating at t1 did not predict onset of partial-/full-syndrome BED at t6 ($n = 116$[2]; $OR = 1.39$, 95% CI[0.19-10.17], $p < 0.74$), however, persistent LOC eating significantly predicted the onset of partial-/full-syndrome BED from t1 to t6 ($n = 116$[2]; $OR = 11.51$, 95% CI[1.28-103.61], $p < 0.03$).

The results were largely similar when based on non-imputed data. The only difference was that significantly more LOC eating episodes and depressive symptoms were found in the LOC+ vs. LOC- group at t6 ($p < 0.001$)

DISCUSSION

This controlled natural course study of LOC eating in 8-13 year old children from the community showed a moderate stability of LOC eating over 5.5 years. The 38.3% stability rate is plausibly somewhat lower than that of a previous study using a more extended timeframe of ChEDE assessment (any past LOC eating vs. in the past three months) and oversampling of overweight children (3). Consistent with this study, children with LOC eating, especially if persistent, were at increased risk to show onset of partial-/full-syndrome BED in adolescence. In addition, persistent LOC eating had small effects on increased eating disorder psychopathology and depressive symptoms. Overall, the results confirm previous findings, despite methodological differences in the use of measures (ChEDE-Q vs. ChEDE global score; standardized vs. non-standardized CDI scores) and the definition of LOC eating as discussed above. In contrast to prior research (3,13), LOC eating, whether persistent or not, did not have any predictive effect on BMI. This could result from individual matching for anthropometric and demographic variables that likely decreased BMI variance at baseline and, thereby, at follow-up, and by self-report measurement of BMI at follow-up.

Strengths of this study include a well-controlled population-based sample of children with recent LOC eating and demographically-matched controls as well as the use of the validated ChEDE to determine LOC eating and BED and well-established self-report questionnaires on psychopathology. A limitation was the self-report follow-up assessment of height and weight, which commonly leads to overestimation of height and underestimation of weight (14). Further, study drop-out was substantial and selective; more children with baseline LOC eating and higher eating disorder psychopathology failed to participate. However, this drop-out was comparable to previous research (3) and we used multiple imputation in order to estimate missing data based on observed data distributions.

In conclusion, childhood LOC eating, if persistent, was suggested to be a variable risk factor (15) for partial-/full-syndrome BED, the most prevalent eating disorder with onset in youth (16,17). Additional evidence with larger samples and longer time periods is warranted, using multiple assessments to clearly establish temporal precedence of LOC eating before first onset of BED. Clinically, evidence on the treatment and prevention of LOC eating and implications for excess weight gain (18-20) should be extended. Such evidence on the efficacy of interventions addressing LOC eating for both eating disorder and weight outcome would provide a critical test of LOC eating as a causal risk factor (15) of both BED and obesity.

Footnotes

[1] For diagnosis of BED, the DSM-5 requires recurrent episodes of binge eating (eating a definitely large amount of food accompanied by a sense of lack of control over eating) at least once per week over three months, three out of five behavioral symptoms (eating more rapidly, until uncomfortably full, when not physically hungry, alone because of embarrassment, and feeling disgusted, depressed or guilty), marked distress, and absence of recurrent compensatory behaviors, anorexia nervosa, or bulimia nervosa.

[2] Persistent partial-/full-syndrome BED was defined as presence of a diagnosis of either partial- or full-syndrome BED at both t1 and t6. Four participants showed partial-/full-syndrome BED at t1 and t6. Full-syndrome BED was diagnosed in two participants at t1 and t6, and partial-syndrome BED at t1 shifted to full-syndrome BED at t6 in another two participants. These four participants were excluded from the logistic regression analysis predicting the onset of partial-/full-syndrome BED from presence of LOC eating at t1 and persistent LOC eating.

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Table 1. Comparison of demographic, anthropometric, and clinical variables at follow-up (t6) for participants who reported at least one episode of LOC eating at t1 (LOC+) vs. participants without LOC eating history (LOC-) and for participants with vs. without persistent LOC eating.

	LOC+ (t6; *n* = 60)	LOC- (t6; *n* = 60)				Persistence (t6; *n* = 23)	Non-persistence (t6; *n* = 97)			
	n (%)	n (%)	$\chi^2(1, n = 120)$	*p*	φ	n (%)	n (%)	$\chi^2(1, n = 120)$	*p*	φ
Sex (female)	35 (58.3)	33 (55.0)	0.14	0.85	0.03	13 (56.5)	55 (56.7)	0.00	0.99	0.00
	M (SD)	*M (SD)*	*t(59)*	*p*	*d*	*M (SD)*	*M (SD)*	*t(118)*	*p*	*d*
Age (years)	10.62 (1.46)	10.92 (1.47)	1.12	0.26	0.20	16.67 (1.68)	16.62 (1.48)	0.12	0.91	0.03
BMI (kg/m^2)	26.89 (9.84)	25.81 (7.13)	-1.06	0.30	0.14	27.09 (10.98)	26.15 (9.16)	0.40	0.69	0.09
LOC eating[a]	4.77 (15.49)	2.55 (8.21)	-0.98	0.34	0.13	12.13 (16.07)	1.59 (6.70)	3.45	0.005	1.16
ChEDE-Q	1.38 (3.41)	0.99 (1.78)	-1.22	0.24	0.16	1.90 (3.84)	0.99 (1.87)	1.35	0.21	0.29
CDI	48.68 (24.17)	48.40 (14.56)	-0.10	0.92	0.01	53.85 (24.65)	47.22 (15.96)	1.60	0.14	0.26

Note. LOC eating = loss of control eating; persistent LOC eating = LOC eating at t1 and t6; ChEDE-Q = Eating Disorder Examination-Questionnaire adapted for Children global score (range 0-6); CDI = Children's Depression Inventory (standardized *T* scores); BMI = body mass index, kg/m^2; $\chi^2 = \chi^2$ statistic; *t* = *t* statistic; *p* = probability; φ = effect size Phi-coefficient; *d* = effect size Cohen's d. [a] Number of LOC eating episodes over the past three months.