Cue reactivity, habituation, and eating in the absence of hunger in children with loss of control eating and attention-deficit/hyperactivity disorder

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Abstract

Objective: Childhood loss of control (LOC) eating and attention-deficit/hyperactivity disorder (ADHD) are highly comorbid conditions and present with disordered eating behaviors, such as overeating. This study sought to delineate shared and specific abnormalities in physiological, cognitive-motivational, and behavioral components of food-specific impulsivity in children with LOC eating and ADHD. Specifically, children's reactivity and habituation to food and eating in the absence of hunger were examined. **Methods:** Within this community-based study, four groups of 8-13 year old children with LOC eating (*n*=24), ADHD (*n*=32), comorbid LOC eating/ADHD (*n*=9), and matched controls (*n*=34) received a standard laboratory test meal to establish satiety and were then exposed to their favorite snack food in a cue exposure/reactivity trial, while salivation and desire to eat were repeatedly assessed. Subsequently, they were offered a variety of snack foods ad libitum. **Results:** Children with LOC eating, ADHD, and LOC/ADHD did not differ from controls in salivary reactivity and habituation to food cues. Children with LOC eating and ADHD showed greater cue reactivity of the desire to eat than controls, but groups did not differ in its longer-term increments. At free access, only children with LOC/ADHD consumed significantly more energy than controls. Longer-term increments of desire to eat predicted greater energy intake beyond LOC/ADHD group status. **Discussion:** Desire to eat among children with comorbid LOC eating and ADHD was associated with overeating in the absence of hunger, which may contribute to excess weight gain. Delineation of the specific features of childhood LOC eating versus ADHD warrants further study.

Keywords: binge-eating disorder, attention deficit disorder with hyperactivity, cue, salivation, craving, meal, eating

Binge-eating disorder (BED) is defined by recurrent binge-eating episodes, involving eating an objectively large amount of food accompanied by a sense of loss of control (LOC) over eating, which occur in the absence of regular inappropriate compensatory behavior (APA, 2013). To capture childhood presentation of BED, age adaptations of diagnostic criteria were proposed, focusing on both objectively and subjectively large binge-eating episodes, termed LOC eating episodes (Hilbert & Czaja, 2009; Marcus & Kalarchian, 2003; Tanofsky-Kraff et al., 2008). Based on clinical interview, 27.3% of children from the community (Vannucci et al., 2014) and 36.4% of treatment-seeking overweight and obese children (Matheson et al., 2015) reported at least one LOC eating episode during the previous month. LOC eating is cross-sectionally characterized by pronounced eating disorder and general psychopathology, marked distress, obesity, and related metabolic symptoms (e.g., Hilbert & Czaja, 2009; Matherne et al., 2015; Radin et al., 2015; Shomaker et al., 2010). Longitudinally, childhood LOC eating predicts full-syndrome BED and, at least in vulnerable youth, excess weight gain (Hilbert et al., 2013; Hilbert & Brauhardt, 2014; Stojek et al., 2017; Tanofsky-Kraff et al., 2009, 2011).

Increased physiological (e.g., salivation) and psychological reactions (e.g., desire to eat, DTE) in response to the exposure to appetizing food are normal learned responses in humans (Pavlov, 1927). According to the classical condition model of binge eating (Jansen, 1998), validated for children with overweight (Jansen et al., 2003), cues that repeatedly accompany food (e.g., the sight of food) can be conditioned to elicit the same set of physiological or psychological responses that were elicited by the food itself, thereby increasing the likelihood of binge eating. Laboratory evidence found children and adults with overweight and adults with BED to be especially sensitive to food exposure, as indicated by increased food cue-induced salivation (Ferriday & Brunstrom, 2011; Jansen, Stegerman, Roefs, Nederkoorn, & Havermans, 2010; Meyer, Risbrough, Liang, & Boutelle, 2015) and DTE (Karhunen, Lappalainen, Tammela, Turpeinen, & Uusitupa, 1997; Naumann,

Trentowska, & Svaldi, 2013; Sobik, Hutchison, & Craighead, 2005), and a slower decline of these parameters during prolonged food presentations compared to controls (Aspen, Stein, & Wilfley, 2012; Bond, Raynor, McCaffery, & Wing, 2010; Epstein et al., 2008; Epstein, Paluch, & Coleman, 1996; Temple, Giacomelli, Roemmich, & Epstein, 2007). These findings indicate alterations in physiological and cognitive-motivational cue reactivity, referring to initial reactions to food cues that are involved in the onset of eating, and habituation processes, describing reductions in response to repeated food exposure, that are concerned with eating cessation (Epstein, Temple, Roemmich, & Bouton, 2009). Habituation to the same food may recover upon presentation of a novel food cue, termed dishabituation (Epstein, Rodefer, Wisniewski, & Caggiula, 1992). Behaviorally, increased salivary food cue reactivity and slower rates of salivary habituation were associated with greater energy intake in overweight, but not normal-weight children (Epstein et al., 2008; Jansen et al., 2003). However, in childhood LOC eating, these associations remain unexplored.

Strikingly, in the above-mentioned studies on food cue reactivity and habituation, food cue exposure followed several hours of fasting, which is not ideal for investigating cue reactivity in situations which may be central to LOC eating, such as eating without feeling hungry. In fact, eating in the absence of hunger (EAH), operationalized as ad libitum food intake after having established satiety (Fisher & Birch, 2002), has been proposed as diagnostic criterion of childhood presentations of BED or LOC eating disorder (Marcus & Kalarchian, 2003; Tanofsky-Kraff et al., 2008). In treatment-seeking 8-12-year-old children with overweight and obesity, EAH and LOC eating were significantly associated (Boutelle et al., 2014). Likewise, a questionnaire-based study in 6-17-year-old youths across the weight spectrum revealed that EAH was associated with the presence of objective and subjective binge-eating episodes (Shomaker et al., 2010). Notably, the limited available evidence did not clarify the underlying mechanisms of EAH, for example, whether abnormal food cue reactivity and habituation of salivation and DTE account for EAH in those with LOC eating.

Experimental evidence indicated that children from the community with higher versus lower levels of impulsivity were more prone to overeating, particularly eating high-energy dense foods (Nederkoorn, Dassen, Franken, Resch, & Houben, 2015). General impulsivity is a common feature of both LOC eating (Hartmann et al., 2010; Hartmann, Rief, & Hilbert, 2013) and attention-deficit/hyperactivity disorder (ADHD; APA, 2013). Recent findings suggested that childhood ADHD increases the susceptibility to later binge eating (Levin & Rawana, 2016; Yilmaz et al., 2017), which may be based on neurobiological mechanisms, such as dopamine deficiency (Levin & Rawana, 2016), and shared genetic risk factors (Capusan et al., 2017). Both conditions are longitudinally associated with obesity (Nigg et al., 2016; Tanofsky-Kraff et al., 2009, 2011), and their comorbidity seems to be exceedingly high (Kaisari, Dourish, & Higgs, 2017).

In the few comparative test meal studies, 8-13-year-old children with LOC eating and ADHD did not consume more food at a lunch test meal than individually matched controls after a 4-hour fast, although their subjective ratings of DTE were significantly higher (Kurz et al., 2017). Importantly, in this study, a subset of children with comorbid LOC eating/ADHD was found to consume the greatest amount of food. In a snack food meal study, Hartmann et al. (2012) found greater energy intake in 10-14-year-old youths with ADHD than those with LOC eating or controls after a 2-hour fast. In both studies, potential effects of stimulant medication were controlled for by including stimulant-naïve children only, or children had to abstain from medication for 48h. Overall, it remains unclear whether children with ADHD are more likely than children with LOC eating to consume more energy in an EAH paradigm as well. Additionally, other indicators of food-specific impulsivity, such as increased salivation and DTE, have not yet been compared between these conditions. Experimentally elucidating physiological, cognitive-motivational, and behavioral responses to food cues in childhood LOC eating and ADHD would allow for the identification of shared and distinctive

characteristics to further delineate both entities, and help clarify the diagnostic validity of childhood LOC eating (Goldschmidt, 2017).

In this experimental investigation of children with and without LOC eating and ADHD when satiated, we thus hypothesized that children with LOC eating and ADHD would show greater cue reactivity and slower habituation of salivation and DTE than controls when exposed to snack food. Based on Kurz et al. (2017), we did not expect children with LOC eating and ADHD to differ in these variables. An additional goal was to objectively determine EAH, hypothesizing that LOC and ADHD children, and especially those with both conditions (Kurz et al., 2017), would show greater EAH than controls, while LOC and ADHD children were not expected to differ in EAH. An exploratory goal was to test the hypothesis that greater cue reactivity and less habituation of salivation and DTE in LOC and ADHD children would predict greater EAH.

Methods

Recruitment and Sample

Children were recruited through school screenings in Switzerland as part of the Swiss University Study of Nutrition (SUN), approved by the cantonal board of education, the school board, and the parents (Kurz et al., 2017; Munsch et al., 2017). After ascertaining eligibility via telephone interview, the child and one parent were invited to the laboratory where written informed consent and assent were obtained, followed by clinical interviews to determine the child's diagnostic status. The study was approved by the local Ethics Committee.

Inclusion in the LOC group required ≥3 LOC eating episodes during the previous 3 months, accompanied by at least some degree of distress and/or 2 of 5 behavioral symptoms (APA, 2013; Hilbert & Czaja, 2009; Marcus & Kalarchian, 2003; Tanofsky-Kraff et al., 2008), based on the Eating Disorder Examination adapted for Children (Bryant-Waugh,

Cooper, Taylor, & Lask, 1996), a well-established eating disorder interview (Hilbert et al., 2013). Children were included in the ADHD group if they met DSM-IV-TR or DSM-5 criteria for ADHD determined via the Schedule of Affective Disorders and Schizophrenia for School-age Children – Present and Lifetime Version (Puig-Antich & Chambers, 1978). Children with comorbid LOC eating and ADHD (LOC/ADHD) were required to meet the inclusion criteria for both the LOC and ADHD groups. Treatment for ADHD with central nervous system stimulants was not an exclusion criterion for children with ADHD if on a stable prescription during the previous 3 months. The control group (CG) was stratified to children with LOC eating for age, sex, and percentile of body mass index (BMI; kg/m^2), and required absence of lifetime or current LOC eating, compensatory behaviors, an eating disorder, and ADHD symptoms. General inclusion criteria for all groups were age between 8-13 years, regular school attendance, and sufficient German or French language skills. General exclusion criteria were compensatory behaviors (>1 episode/past 3 months), anorexia nervosa or bulimia nervosa, psychotic disorders, current treatment for overweight, intake of drugs with an effect on eating, or serious medical problems.

A total of 599 children were screened by telephone, of whom 132 children were invited to the diagnostic visit. Subsequently, 32 children were excluded according to the above-described criteria. One child with ADHD was excluded because of missing experimental data, leaving a total sample of 99 children (LOC: *n*=24; ADHD: *n*=32; LOC/ADHD: *n*=9; CG: *n*=34) with a mean age of 11.54±1.29 years (56.6% girls, *n*=56). The groups did not differ significantly in sex and age, except for a higher age in the LOC group and CG than in the ADHD group and a higher BMI in the LOC than in the ADHD group (Table 1), based on objective measurement.

Four (12.5%) children with ADHD and 1 (11.1%) child with LOC/ADHD were on a stable stimulant medication for the treatment of ADHD during the previous 3 months. These children were required to abstain from medication for at least 48h before study participation.

Procedure

After an overnight fast, the child received a standardized breakfast at 8:30 a.m., consisting of Corn Flakes with milk and orange juice (300 kcal), for homogenizing pre-experimental food intake, as reported by Kurz et al. (2017). Both the child and the parent had to ensure that the child would not consume any additional food or liquid (other than water) before returning to the laboratory for a test meal 4h later consisting of 700g of a typical lunch food (Pizza prosciutto, Pizza formaggi, Cannelloni, Lasagne verdure, Oven-fries), chosen by the child (Kurz et al., 2017). At this test meal the child was asked to eat "until feeling full." There were no significant group differences in the test meal food offered to the children (kcal, g macronutrients) or in food intake (g, kcal, g macronutrients), with the exception of a significantly greater carbohydrate intake (g) in the LOC group compared to the CG (Table S1), so that carbohydrate intake at lunch test meal was used as a covariate in data analyses.

The experimental protocol is depicted in Figure 1. For the *cue reactivity/habituation trial*, the child was seated in a light-, noise-, and temperature-controlled laboratory and received standard instructions for visual and olfactory exposure to food cues through loud speakers. At the outset, the child was asked to select the first- and second-ranking favorite food out of five typical snack foods (Prinzen Rolle cookies, kinder Chocolate, SMARTIES, Zweifel Paprika Chips, HARIBO Goldbears; Hilbert, Tuschen-Caffier, & Czaja, 2010). After a training in the correct placement of three dental cotton rolls into the mouth (Peck, 1959), the first-ranking food was placed on a plate adjustable in height to the child's mouth. The child was instructed to look at the food, smell it, and think about eating it, without actually eating it.

The cue reactivity/habituation trial provided 11 exposure trials with 1-min inter-trial intervals. A baseline trial using water as a neutral stimulus (trial 0) was followed by 8 trials of food exposure using the child's favorite food (200g). Trial 1 lasted 4 min and included salivation measurement at min 4 to determine cue reactivity (personal communication with A.

Jansen, 09.02.2010). Trials 2-8 included 1-min exposure trials to the same food in order to measure habituation (Epstein et al., 2003). To control for time effects, children were randomly and equally assigned to exposure to the second-ranking favorite food (200g), the "dishabituator," either in trials 9 and 10, or trial 10 only (Temple et al., 2006). After each trial, the food was taken out of the laboratory and the child removed the dental cotton rolls, returning them in plastic bags. The child completed psychological measures (see below) at baseline, at inter-trial intervals, and afterwards. Finally, the child was given 15-min *free access* to 200g portions of all 5 snack foods available at the cue reactivity/habituation trial. Food was served on a plate with instruction to eat as much or as little as desired and a 15-min video-sequence of The Wizards of Waverly Place was presented. To control experimental procedures, all experimental trials were videotaped.

Dependent variables. Salivation was measured by the difference in pre- and post-exposure weights of cotton rolls to 0.001g on a precision scale (CPA223; Sartorius, Switzerland) from baseline trial 0 to trial 10. DTE was rated on Visual Analog Scales (VAS, 0=*not at all* to 6=*extremely)* at baseline, at inter-trial intervals, and afterwards, as previously used in experimental reactivity research (e.g., Hilbert, Vögele, Tuschen-Caffier, & Hartmann, 2011). Cue reactivity was determined by the change from trial 0 to trial 1, and habituation was determined by the change from trial 1 through trial 8. Food intake at the free access trial was determined by the weight difference of presented minus remaining food, using a calibrated table top standard scale (Cubis® 5201S; Sartorius, Switzerland). Food intake in g was converted into kcal using nutritional information of the food producer.

Manipulation Check: Control Variables

The experimental procedure was piloted in 15 non-clinical children (data not shown). Control variables and univariate analyses of variance (ANOVAs) by group are displayed in Table 2. An olfactory function test (Sniffin' Sticks, Burghart, Germany), conducted prior to

the experimental trials, did not show significant group differences. All children recognized more than 10 out of 12 odors correctly, except for 1 LOC child (4.2%) and 1 LOC/ADHD child (11.1%) who recognized 7 out of 12 odors correctly. Baseline scores of hunger, satiety, and mood were assessed using VAS (0=*not at all* to 6=*extremely)*. While baseline hunger was significantly greater in the LOC/ADHD group than in all other groups, baseline satiety and mood did not differ by group. Groups did not differ in the first and second selection of their favorite food, or in the energy content presented during cue exposure. Hunger, satiety, and mood ratings did not differ by groups immediately before the free access trial. Post-experimentally, children reported low stress caused by cue exposure and free access, high post-experimental mood, and good compliance with the experimental procedures across groups, without any significant group difference.

Data Analytic Plan

Generalized linear mixed models (GLMM) of Group×Time were used for analyzing salivation and DTE regarding cue reactivity and habituation. Participants were nested within time, and a first-order autoregressive covariance structure was assumed. Univariate ANOVAs were conducted for kcal and g food intake, including group status (dummy coded with CG as reference), cue reactivity, and habituation as predictors. For the latter predictor variables, significant cue reactivity and habituation effects from the GLMM were entered into the analyses. All models were re-computed with covariates, considering age, sex, BMI, carbohydrate intake at lunch test meal, baseline hunger, and olfactory function; the results were reported only in case of changing significance of effects. For analyzing dishabituation in salivation and DTE as an experimental manipulation check, GLMM examined Group×Time(trials 8, 9, 10)×Dishabituation condition(dishabituator in trials 9 and 10, or trial 10 only) effects. To control for stimulant ADHD medication, all models were re-computed excluding the 5 children with stimulant medication; the results were reported only in case of

changing significance of effects. Sample size was based on an a priori power analysis for the entire SUN project. A post-hoc power analysis for this study revealed 1-β≥.98 for medium or large effects (f=0.25 or 0.40) of cue reactivity or habituation in a repeated measures ANOVA of Group×Time as a proxy for GLMM. For food intake, a power analysis revealed 1-β=.76 or .99 for medium or large effects (f^2=0.15 or 0.35) for five predictors in a linear multiple regression. Statistical significance was determined with a two-tailed α of .025, adjusted for the use of two indicators of food-specific impulsivity (salivation, DTE), and snack food intake (g, kcal). Follow-up analyses, conducted for explanation of significant effects, were Bonferroni-corrected. All analyses were performed with SPSS 23 (SPSS Inc., Chicago, IL).

Results

The course of salivation and DTE by group is depicted in Figure 2.

Salivation

The analyses on salivary cue reactivity did not show any significant effect of group, F(3,190)=0.78, p=.507, time, F(1,190)=1.20, p=.275, or Group×Time, F(3,190)=1.83, p=.144. Likewise, salivary habituation did not differ by group, F(3,759)=0.41, p=.743, time, F(7,759)=1.54, p=.150, or Group×Time, F(21,759)=0.51, p=.966.

Desire to Eat

The cue reactivity analysis of DTE showed a significant Group×Time interaction, F(3,188)=5.06, p<.002, modifying significant effects of group, F(3,188)=4.93, p<.003, and time, F(1,188)=9.91, p<.002. Follow-up analyses documented a significant increase of DTE in the ADHD group when exposed to food than when exposed to water (p<.001), and a trend of an increase in the LOC group (p=.072), whereas there was no change in the LOC/ADHD

group or CG (both $p>.263$). While groups' DTE did not differ at baseline (all $p>.186$), trial 1 evidenced a greater DTE in the LOC and ADHD groups than in the CG (both $p<.020$).

The habituation analysis of DTE showed significant effects of group, $F(3,759)=3.48$, $p<.016$, and time, $F(7,759)=3.24$, $p<.025$, but no significant interaction effect, $F(21,759)=0.29$, $p=.999$. Follow-up analyses did not reveal any differences between specific groups (all $p>.153$). Across groups, DTE increased from trial 2 to trial 5 (all $p<.025$). Thus, sensitization in terms of an increase of DTE, rather than habituation, was observed. Upon inclusion of covariates, the group effect became non-significant ($p=.311$), with baseline hunger being the only significant covariate ($p<.001$).

Snack Food Intake

At the free access trial, the LOC/ADHD group showed a significantly greater snack food intake in g and kcal than the CG (both $p<.021$; Table 3, Figure 3), while the LOC and ADHD groups did not significantly differ from the CG (all $p>.066$). For predictor analysis, only a greater sensitization of DTE was a significant additional predictor of snack food intake in g and kcal (both $p<.019$), beyond group status. Upon inclusion of covariates, the group difference between the LOC/ADHD group and GC on snack food intake in g, but not in kcal, became non-significant ($p=.036$); however, without significant covariate effects (all $p>.032$).

Manipulation Check: Dishabituation

The dishabituation analysis of salivation did not show any significant effects of dishabituation, $F(1,273)=3.55$, $p=.061$, and interactions with group, $F(6,273)=0.38$, $p=.892$, time, $F(4,273)=0.86$, $p=.487$, or both, $F(12,273)=0.64$, $p=.806$.

The dishabituation analysis of DTE revealed a significant dishabituation effect, $F(1,272)=6.00$, $p<.015$, and a significant interaction with time, $F(4,272)=4.25$, $p<.002$, whereas Group×Dishabituation, $F(6,272)=2.33$, $p=.033$, and Group×Time×Dishabituation

effects, $F(12,272)=0.97$, $p=.482$, were non-significant. Follow-up analyses indicated that if the dishabituator was presented in trial 9, DTE increased from trial 8 to trial 9 ($p<.005$), but not from trial 9 to trial 10 ($p=.503$), lending support to the dishabituation of DTE. Upon inclusion of covariates, the main effect of dishabituation and its interaction with time became non-significant (both $p>.135$); however, without significant covariate effects (all $p>.063$).

Discussion

This study provides novel evidence on physiological, cognitive-motivational, and behavioral aspects of food-specific impulsivity in children with LOC eating, ADHD, their comorbidity, and matched controls from the community. Specifically, we assessed children's food cue reactivity and habituation of salivation and DTE in satiety and children's snack food intake using an EAH paradigm. Children with LOC eating, ADHD, and comorbid LOC/ADHD did not show physiological food-specific impulsivity as indicated by normal salivary responding to their favorite snack food. However, children with LOC eating and ADHD demonstrated shared abnormalities in cognitive-motivational food-specific impulsivity as characterized by greater cue reactivity of DTE compared to controls. The comorbidity of LOC eating and ADHD accounted for marked EAH, which was further predicted by the sensitization of DTE.

Against expectations, children with LOC eating, ADHD, comorbid LOC/ADHD, and controls did not differ in physiological aspects of food-specific impulsivity. Specifically, the absence of salivary cue reactivity in each of the groups contrasted previous findings in food-deprived samples (e.g., Ferriday et al., 2011), but may mirror the pre-established satiety in this study. Likely, the lack of salivary cue reactivity had an impact on absent salivary habituation because both cue reactivity and habituation are assumed to act upon one stimulus-response pathway (Groves & Thompson, 1970). It is possible that salivary effects would have been more pronounced if overweight had been an inclusion criterion; previous evidence on

altered salivary short- and long-term responding to food was derived from samples with overweight or obesity (e.g., Aspen et al., 2012). Consistent with non-significant effects on salivary cue reactivity and habituation, no significant dishabituation was observed.

Children with ADHD showed significant increases of DTE when exposed to their favorite snack food, as expected, while the increase in DTE was only marginally significant in children with LOC eating, which might be related to descriptively higher levels of the DTE in children with LOC eating at the water exposure trial. Although all groups did not significantly differ in initial levels of DTE in satiety, the LOC and ADHD groups reported a greater DTE than the CG at the first food exposure trial, suggesting increased cognitive-motivational cue reactivity in satiety in these conditions. Of note, all groups showed gradually increasing levels of DTE throughout, suggesting that sensitization rather than habituation had occurred, which typically emerges when the stimulus has strong reinforcement value (Groves & Thompson, 1970). The fact that all children were exposed to their favorite snack foods and informed that they would have access to them afterwards may have increased the food's attractiveness. As previous studies found that food cue exposure increased the accessibility of hedonic thoughts about food and eating (e.g., Roefs, Herman, MacLeod, Smulders, & Jansen, 2005), sensitization of DTE might result from the reinforcing value of implicit expectations that the food is going to be eaten. Regarding dishabituation of DTE, the results were consistent with expectations that introducing a novel food would increase DTE (Epstein et al., 2003).

For EAH, we found that only children with comorbid LOC/ADHD ate significantly more snack food than controls. The result is consistent with that from Kurz et al. (2017), who examined energy intake during a laboratory test meal in the present sample. They showed that children with comorbid LOC/ADHD consumed the greatest amount of lunch food, whereas food intake in LOC eating or ADHD did not differ from that of controls (Kurz et al., 2017). Based on findings from a 10-15-year-old sample with LOC eating and ADHD, those with comorbid LOC/ADHD reported significantly greater levels of general impulsivity and showed

a trend to exhibit more LOC eating episodes than those with LOC eating only (Hartmann et al., 2012), presumably placing them at risk for increased food intake (Reinblatt, Mahone et al., 2015). The fact that children with LOC eating consumed a similar amount of snack food (in g) in satiety compared to controls is unexpected, but consistent with findings from Hilbert et al. (2010). LOC children in that study, particularly those with highly recurrent LOC eating, consumed significantly more energy (in kcal) than controls, while the effect in the present study was only marginally significant. As LOC children's weight status was found to be positively associated with snack food intake (Hilbert et al., 2010), the lack of effects might be related to the lower number of children with overweight and obesity in the present study (42%) versus Hilbert et al. (2010) study (69%). The sensitization of DTE significantly predicted EAH, which supported available evidence in children with overweight and normal weight demonstrating that sensitization of instrumental responding for food was associated with increased energy intake (Epstein et al., 2008).

Regarding strengths and limitations, this well-controlled experimental study focused on clinically relevant presentations of childhood LOC eating, identified using age-adapted DSM-5 diagnostic criteria of BED and full-syndrome ADHD, and determined through clinical interviews conducted by trained raters. The experimental procedure used methodology elaborated in previous research on EAH (Fisher & Birch, 1999). Although the child's age did not have an effect on the variables under investigation, other physical determinants of children's food intake were not assessed, such as children's pubertal status (e.g., Vannucci et al., 2014) or an objective measure of fat mass. Consistent with other population-based studies in LOC eating and ADHD (Reinblatt, Leoutsakos et al., 2015), we did not exclude children with ADHD using stimulants with stable dosage in the last 3 months, but ensured that stimulants were not taken 48h prior to the experiment. In addition, a re-analysis without the 5 children receiving stimulant medication was conducted, making it unlikely that the results were influenced by medication. However, future studies could systematically examine the role

of stimulants on LOC eating onset in children with ADHD, as there is inconclusive evidence thus far as to whether appetite-suppressant effects of stimulants in ADHD may induce LOC eating as their effects wear off (Reinblatt, Leoutsakos et al., 2015). As we only included children from the population with the majority of them being normal-weight, the results may not generalize to treatment-seeking children with overweight and LOC eating. Finally, although the statistical power to detect medium-to-large effects in the primary analysis of cue reactivity/habituation and EAH was adequate, the probability to identify small effects was limited, highlighting the necessity to examine larger sample sizes in further research.

This study did not provide evidence for dysfunctional salivary responding to food in 8-13-year-old children from the community with LOC eating, ADHD, and comorbid LOC/ADHD when satiated, but revealed increased cue reactivity of DTE in children with LOC eating and ADHD. Behaviorally, children with comorbid LOC/ADHD showed greater snack food intake in an EAH paradigm than healthy controls. The results thus support recently adjusted research criteria for childhood BED (Matherne et al., 2015), proposing that EAH may be included as a behavioral feature, but not as the key diagnostic criterion of childhood BED as initially suggested (Marcus & Kalarchian, 2003). The results further suggest that in identifying the discriminant validity of childhood LOC eating, which has thus far been addressed by only a few studies (Goldschmidt, 2017), ADHD needs to be considered. Longitudinal studies could help to identify shared and distinctive risk factors for abnormal eating behaviors and excess weight trajectory in childhood LOC eating and ADHD.

Clinically, children with LOC eating and ADHD, particularly those with comorbid LOC/ADHD, may require specific interventions directly targeting reductions in the strength of learned associations among food cues, DTE, and food intake. For example, cue exposure treatment has demonstrated effectiveness in reducing EAH and LOC eating in children with overweight and obesity (Boutelle et al., 2011) and for improving eating disorder psychopathology and weight loss in adults with obesity and binge eating (Boutelle, Knatz,

Carlson, Bergman, & Peterson, 2017). However, the efficacy of these interventions in childhood LOC eating and ADHD warrants further study.

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Table 1

Sociodemographic and Clinical Characteristics

	LOC (*N*=24)		ADHD (*N*=32)		LOC/ADHD (*N*=9)		CG (*N*=34)			
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*	$\chi^2(df=3)$	*p*
Sex, girls	16	66.7	13	40.6	4	44.4	23	67.7	6.54	.088
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*(3,95)	*p*
Age, years	11.97[a]	1.02	10.88[b]	1.44	11.43[ab]	1.36	11.87[a]	1.08	4.99	.003
BMI, kg/m^2	21.51[a]	4.57	18.67[b]	3.60	21.46[ab]	2.96	19.76[ab]	3.04	3.33	.023
BMI-SDS	0.78	1.26	0.21	1.17	1.09	0.62	0.44	0.92	7.69	.092
Weight status	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	$\chi^2(df=9)$	*p*
Underweight (BMI<10th percentile)	2	8.3	3	9.4	0	0.0	1	2.9	8.75	.460
Normal-weight (BMI≥10th–90th perc.)	12	50.0	22	68.8	6	66.7	28	82.4		
Overweight (BMI≥90th–97th perc.)	6	25.0	5	15.6	2	22.2	3	8.8		
Obesity (BMI≥97th percentile)	4	16.7	2	6.3	1	11.1	2	5.9		

Notes. LOC, group with loss of control eating; ADHD, group with attention-deficit/hyperactivity disorder; LOC/ADHD, group with comorbid LOC eating and ADHD; CG, control group; BMI, body mass index; BMI-SDS, BMI standard deviation score and weight status, defined

according to the Workgroup for Adiposity in Childhood and Adolescence (Wabitsch & Kunze, 2015). In the ADHD group, 17 children (53.1%) were diagnosed with the inattentive type, 4 (12.5%) had the impulsive type, and 11 (34.3%) had the mixed type. For the LOC/ADHD group, the respective ADHD subgroups were diagnosed in 2 (22.2%), 1 (11.1%), and 6 (66.7%) children. [a,b] Different superscripts indicate significant post-hoc tests, $p<.05$.

Table 2

Manipulation Check

	LOC (*N*=24)		ADHD (*N*=32)		LOC/ADHD (*N*=9)		CG (*N*=34)			
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*	*p*
Baseline cue exposure										
Olfactory function, *n* errors	0.95	1.16	1.65	0.98	2.00	1.58	1.29	1.03	2.76	.081
Hunger, 0-6	0.50[a]	1.06	0.45[a]	0.93	1.67[b]	1.94	0.21[a]	0.48	5.23	.002
Satiety, 0-6	5.58	0.78	5.35	1.33	5.67	1.00	5.67	0.60	0.59	.592
Mood, 0-6	5.08	1.56	5.06	1.31	5.44	1.13	4.97	1.31	0.29	.835
Selection of food for cue exposure										
First favorite food, kcal	852.46	192.62	942.61	190.25	929.37	187.85	888.56	180.91	1.06	.371
Second favorite food, kcal	1009.02	111.46	975.54	172.05	1060.94	73.16	924.92	180.32	2.06	.111
Baseline free access										
Hunger, 0-6	0.64	1.26	1.17	1.47	1.00	1.66	0.68	0.98	1.04	.381
Satiety, 0-6	5.45	1.22	4.76	1.77	5.67	1.00	5.23	1.28	1.48	.225

Table 2 (cont.)

	LOC (*N*=24)		ADHD (*N*=32)		LOC/ADHD (*N*=9)		CG (*N*=34)			
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*	*p*
Mood, 0-6	5.41	1.22	5.52	1.06	5.78	0.67	5.16	1.13	0.96	.416
Post-experimental										
Stress through cue exposure, 0-6	0.67	1.37	1.10	1.60	2.00	2.29	0.74	1.11	2.18	.096
Stress through free access, 0-6	0.08	0.41	0.06	0.36	0.22	0.44	0.03	0.17	0.83	.483
Mood, 0-6	5.38	0.82	5.32	0.83	5.63	0.52	5.29	1.03	0.32	.813
Compliance with procedures, 0-6	4.79	1.18	5.00	1.03	4.67	2.00	4.74	1.40	0.29	.834

Notes. LOC, group with loss of control eating; ADHD, group with attention-deficit/hyperactivity disorder; LOC/ADHD, group with comorbid LOC eating and ADHD; CG, control group. Visual Analog Scales, 0=*not at all* to 6=*extremely*. Univariate Analyses of Variance with df_1=3 and df_2=87-94. [a,b] Different superscripts indicate significant post-hoc tests, p<.05.

Table 3

Snack Food Intake at Free Access Trial and Prediction by Group, Cue reactivity, and Sensitization of Desire to Eat

	B	*SE*	*t*	*p*	95% CI	R^2
Snack food intake, g						
LOC versus CG	17.36	10.44	1.66	.100	-3.39 – 38.10	13.0
LOC/ADHD versus CG	34.66	14.73	2.35	.021	5.39 – 63.92	
ADHD versus CG	12.86	10.03	1.28	.203	-7.07 – 32.79	
Cue reactivity desire to eat	3.68	4.20	0.88	.383	-4.66 – 12.01	
Sensitization desire to eat	-6.34	2.24	-2.83	.006	-10.78 – -1.89	
Constant	54.78	6.99				
Snack food intake, kcal						
LOC versus CG	98.32	52.86	1.86	.066	-6.70 – 203.33	11.2
LOC/ADHD versus CG	182.81	74.58	2.45	.016	34.64 – 330.98	
ADHD versus CG	71.36	50.79	1.41	.163	-29.53 – 172.26	
Cue reactivity desire to eat	19.02	21.24	0.90	.373	-23.18 – 61.23	
Sensitization desire to eat	-27.14	11.33	-2.40	.019	-49.64 – -4.64	

Table 3 (cont.)

	B	*SE*	*t*	*p*	95% CI	R^2
Constant	252.24	35.38				

Notes. B, regression coefficient; *SE*, standard error; *t* test; CI, confidence interval; R^2, adjusted multiple R^2. LOC, group with loss of control over eating; ADHD, group with attention-deficit/hyperactivity disorder; LOC/ADHD, group with comorbid LOC eating and ADHD; CG, control group. The overall models for snack food intake in g, $F(5,90)=3.83$, $p<.003$, and kcal, $F(5,90)=3.48$, $p<.006$, were significant, $p<.025$.

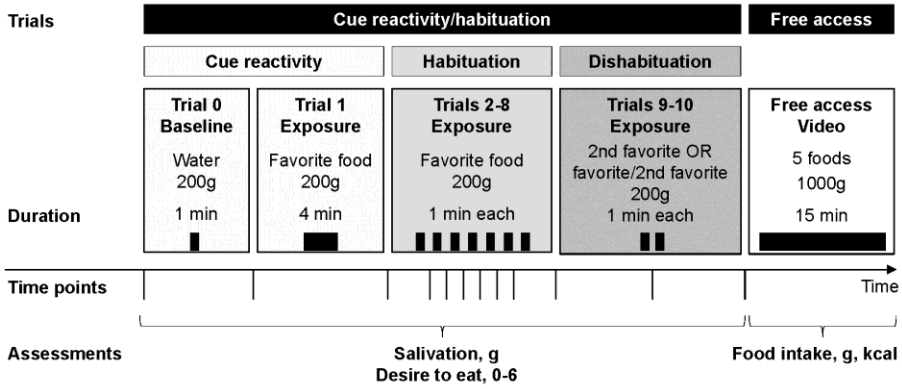

Figure 1. Experimental protocol.

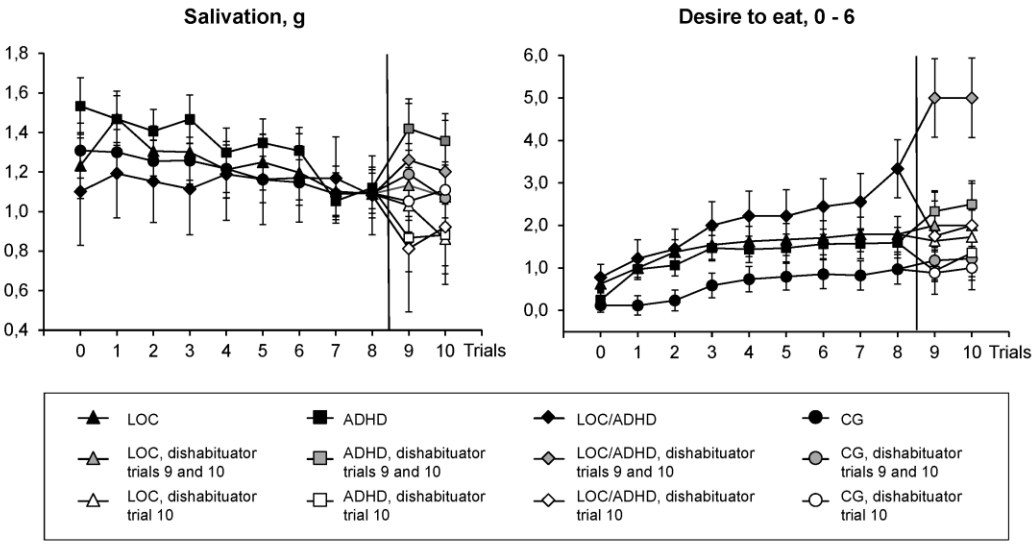

Figure 2. Salivation and desire to eat over ten experimental trials of exposure to snack food in children with loss of control (LOC) eating, with attention-deficit/hyperactivity disorder (ADHD), with comorbid LOC eating and ADHD (LOC/ADHD), and in control children (CG). Children were randomly presented a snack food dishabituator in trials 9 and 10 or in trial 10. Desire to eat, 0=*not at all* to 6=*extremely*. Displayed are means ± standard errors. Cue reactivity and habituation of salivation did not differ by group, time, or Group×Time (all $p>.025$). Cue reactivity of desire to eat differed significantly by group, time, and Group×Time (all $p<.003$), and habituation differed by group and time (both $p<.025$), but not by Group×Time ($p>.025$).

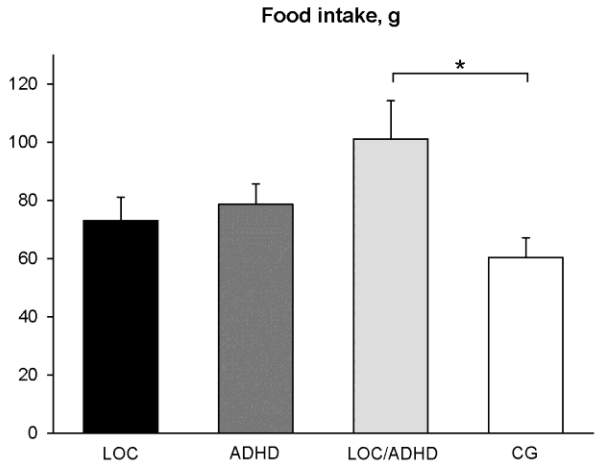

Figure 3. Ad libitum snack food intake in the absence of hunger in children with loss of control (LOC) eating, with attention-deficit/hyperactivity disorder (ADHD), with comorbid LOC eating and ADHD (LOC/ADHD), and in control children (CG). Displayed are means and standard errors with adjustment by body mass index. *p<.025.