Cue exposure therapy reduces overeating of exposed and non-exposed foods in obese adolescents

J. Behav. Ther. & Exp. Psychiat. 58 (2018) 68–77

Contents lists available at ScienceDirect

Journal of Behavior Therapy and Experimental Psychiatry

journal homepage: www.elsevier.com/locate/jbtep

Cue exposure therapy reduces overeating of exposed and non-exposed foods in obese adolescents

Ghislaine Schyns [a, *], Anne Roefs [a], Fren T.Y. Smulders [b], Anita Jansen [a]

[a] Department of Clinical Psychological Science, Faculty of Psychology and Neuroscience, Maastricht University, P.O. Box 616, 6200 MD Maastricht, The Netherlands

[b] Department of Cognitive Neuroscience, Faculty of Psychology and Neuroscience, Maastricht University, P.O. Box 616, 6200 MD Maastricht, The Netherlands

ARTICLE INFO

Article history:
Received 13 February 2017
Received in revised form
17 August 2017
Accepted 4 September 2017
Available online 5 September 2017

Keywords:
Exposure therapy
Habituation
Cue reactivity
Obesity
Expectancy violation
Eating in the absence of hunger

ABSTRACT

Background and objectives: This study tested whether two sessions of food cue exposure therapy reduced eating in the absence of hunger (EAH), specified for exposed and non-exposed food, in overweight and obese adolescents, and whether habituation of food cue reactivity and reduced CS-US expectancies predicted a decrease in EAH.

Methods: 41 overweight adolescents (aged 12–18 years) were randomly assigned to a cue exposure intervention or a lifestyle intervention (control condition). Habituation of food cue reactivity (self-reported desire to eat and salivation) and CS-US expectancy were measured during both sessions, and EAH was measured at the end of session two.

Results: Compared to the control condition, the cue exposure condition showed less EAH for the exposed food item as well as for the non-exposed food items. Larger within-session (WSH) and between-session habituation (BSH) of cue reactivity were not related to less EAH, change in CS-US expectancy was unrelated to EAH.

Limitations: The study was underpowered, and compliance to homework instructions between sessions was poor, intervention effects might have been larger when participants adhered to daily homework exercises.

Conclusions: Food cue exposure was effective to reduce EAH of exposed and non-exposed food items, indicating generalisability of the exposure effect. In line with exposure effects in anxiety disorders, habituation was not found to benefit outcome, though the present data do also not provide evidence that CS-US expectancy violation predicts EAH.

1. Introduction

Exposure-based interventions have widely received empirical support for the treatment of anxiety disorders (Deacon & Abramowitz, 2004). In eating disorders and obesity, far less evidence is available on the effectiveness of exposure therapy, in spite of an alarming need for effective treatments. Worldwide, overweight and obesity prevalences increase disturbingly, with estimated increases of 27.5% for adults and as much as 47.1% for children falling in the overweight range between 1980 and 2013 (Ng et al., 2014). Calorie intake beyond physiological needs, also referred to as eating in the absence of hunger (EAH), is a major determinant of overweight. Several studies have reported on the significant association between EAH and adiposity in children (e.g. Butte et al., 2007; Kral et al., 2013). Triggers for EAH seem to be a core problem of overeating: increased food cue reactivity, which refers to anticipatory psychological (e.g., eating desires) and physiological responses (e.g., salivary production) that prepare for food intake, supposedly forms a major obstacle to resist palatable food and successful weight loss (A. Jansen, 1998). Indeed, a meta-analysis showed that cue-induced food cravings were prospectively related to eating and weight gain, with similar (medium) effect sizes for children and adults (Boswell & Kober, 2016). Moreover, a pilot study showed that cue reactivity was significantly reduced in successful dieters (formerly obese) compared to unsuccessful (still obese) dieters (A. Jansen, Stegerman, Roefs, Nederkoorn, & Havermans, 2010). These findings suggest that successfully refraining oneself from consuming high-caloric foods

* Corresponding author.
E-mail address: ghislaine.schyns@maastrichtuniversity.nl (G. Schyns).

http://dx.doi.org/10.1016/j.jbtep.2017.09.002

is related to decreased food cue reactivity, which in turn might make it easier to resist tempting foods and hence improve successful weight loss and relapse prevention.

Models of overeating state that food cue reactivity can be acquired through classical conditioning (A. Jansen, 1998): by associating food intake (unconditioned stimulus; US) with predictive cues (conditioned stimulus; CS), such as the smell and sight of food, presentations of merely the CS are capable of inducing cue reactivity (conditioned response). Results of human laboratory studies confirm that associations between food intake (US) and neutral stimuli (CS) are easily learned (Bongers, van den Akker, Havermans, & Jansen, 2015; van den Akker, Havermans, Bouton, & Jansen, 2014). If CS-US associations are acquired in daily life through classical conditioning, extinction of food cue reactivity, by repeated exposure to CSs without the US (eating), could be helpful to reduce overeating. During food cue exposure, participants are exposed to intake-predicting cues (CSs), like the sight of favourite food, while food intake (US) is not permitted, allowing conditioned motivation to eat to decrease over time. Pilot studies in bulimia nervosa patients show that cue exposure therapy is indeed effective in decreasing food cravings and binge eating (A. Jansen, Broekmate, & Heymans, 1992; A. Jansen, Van den Hout, De Loof, Zandbergen, & Griez, 1989; Martinez-mallen et al., 2007; McIntosh, Carter, Bulik, Frampton, & Joyce, 2011; Toro et al., 2003). With regard to overweight samples, cue exposure has shown to prevent weight regain after successful weight loss in adults (Mount, Neziroglu, & Taylor, 1990). Interestingly, cue exposure is also effective for obese children to reduce EAH in a behavioural task (Boutelle et al., 2011, 2014). Investigating exposure therapy in children is of great importance, as obesity at a young age is not only associated with serious medical issues, it also increases the chance of being obese as an adult – along with the health consequences later in life (Serdula et al., 1993). In addition, children are less burdened with an ingrained learning history, and suggested to be more malleable than adults (e.g. faster adaptation of behavioural patterns), which make them an important target group for interventions (Wilson, 1994). Although the studies by Boutelle et al. (2011, 2014) found interesting result on EAH in general, it is of interest to further investigate the specificity of cue exposure effects on EAH: it has been shown in overweight adult women that a single session of cue exposure, as compared to an active control intervention, led to less EAH for the exposed foods, but no generalisation to non-exposed food items occurred (Schyns, Roefs, Mulkens, & Jansen, 2016).

Given the limited research available, the food cue exposure domain could greatly benefit from the advances in the anxiety research field. A fascinating line of research on working mechanisms of exposure in anxiety disorders has shown that, in contrast to the original assumption that anxiety levels should habituate during therapy sessions, habituation of anxiety during (within session habituation; WSH) and between treatment sessions (between session habituation; BSH) is not a good predictor of treatment outcome (Craske et al., 2008). It is now well established that extinction learning is not erasing old CS-US memories, but instead creating new CS-noUS associations; inhibitory learning (Bouton & King, 1983; Bouton, 1993). Exposure should aim to make new CS-noUS associations as strong as possible, which can for example be achieved by violating US expectancies during exposure (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014). Exposure for panic disorder patients aimed at violating US expectancies has shown to be more effective than aiming at habituation of fear (Salkovskis, Hackmann, Wells, Gelder, & Clark, 2006). Strengthening inhibitory learning seems especially important for overweight and obese individuals (A. Jansen, Schyns, Bongers, & van den Akker, 2016), as several studies found associations between overeating and obesity and weak inhibitory skills (e.g., Guerrieri, Nederkoorn, & Jansen, 2012; Nederkoorn, Houben, Hofmann, Roefs, & Jansen, 2010; Nederkoorn, Smulders, Havermans, Roefs, & Jansen, 2006), also in children and adolescents (e.g., Batterink, Yokum, & Stice, 2010; Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006). To improve inhibitory learning, the expectancy violation approach could be translated to food cue exposure by exposure to specific overeating cues that are linked to strong eating expectancies (e.g., "If I feel exhausted and palatable food is available [CS], then I will have a binge [US]") while testing whether the US indeed takes place as expected. Although evidence on the role of habituation and expectancy violation in cue exposure research is scarce, one study established that, in line with the findings of anxiety studies, WSH of cue reactivity (salivation and self-reported eating desires) did not predict EAH. Instead, self-reported change in CS-US expectancy was significantly related to EAH: lower US expectancies predicted less EAH of an exposed food item (Schyns et al., 2016). However, no generalisation to non-exposed foods was found. Generalisation of the exposure effect remains very important for the usability of exposure in clinical practice; it is plausible that more sessions are required for generalisation to occur, and/or better consolidation of the new CS-noUS memory, for example by sleeping after the session (Pace-Schott, Verga, Bennett, & Spencer, 2012). Further, even when the new CS-noUS association has been well-consolidated, the original CS-US association remains intact and forms a risk of return of responses in another context or later in time. One possible way to enhance accessibility of the CS-noUS association are retrieval cues: mental or physical cues that help to remember the extinction memory (Craske et al., 2014). Adding a retrieval cue has been shown to attenuate renewed responding after a context switch in conditioning studies (Dibbets, Havermans, & Arntz, 2008; Vansteenwegen et al., 2006), though results in more clinically applied exposure studies are mixed (Culver, Stoyanova, & Craske, 2011; Dibbets, Moor, & Voncken, 2013).

The primary aim of the present experiment is to investigate whether two sessions of food cue exposure reduce EAH of the exposed food item and EAH of non-exposed food items in overweight and obese adolescents. It is hypothesised that EAH is less in the exposure condition relative to a control condition, both for exposed and non-exposed foods (generalisation). Further, the role of habituation of cue reactivity (WSH and BSH of salivation and self-reported eating desires) and the violation of US expectancies in the prediction of EAH are tested, hypothesising that WSH and BSH of habituation are positively associated with EAH if habituation is critical for extinction, whereas CS-US expectancies are related to EAH if inhibitory learning is critical. As a secondary outcome measure, the effects of cue exposure on eating psychopathology and the added benefit of a retrieval cue herein are examined at one-month follow-up.

2. Material and methods

2.1. Participants

41 participants, aged 12–18 years were recruited from the local Area Health Authority and the paediatric obesity outpatient clinic of Zuyderland in Kerkrade, the Netherlands (see Fig. 1). Participants were eligible when having sufficient Dutch speaking skills and being overweight according to criteria for children, as defined by scoring above the age and sex-specific BMI cut-off (BMI of 25 in adults; Cole, Bellizzi, Flegal, & Dietz, 2000). After being informed by the researcher, children and parents received one week to consider participation. After written consent was given by the child and parents, the participant was put on a waiting list for a group of participants. As soon as six participants could make it one the same two intervention dates, the group was set by the experimenter. The

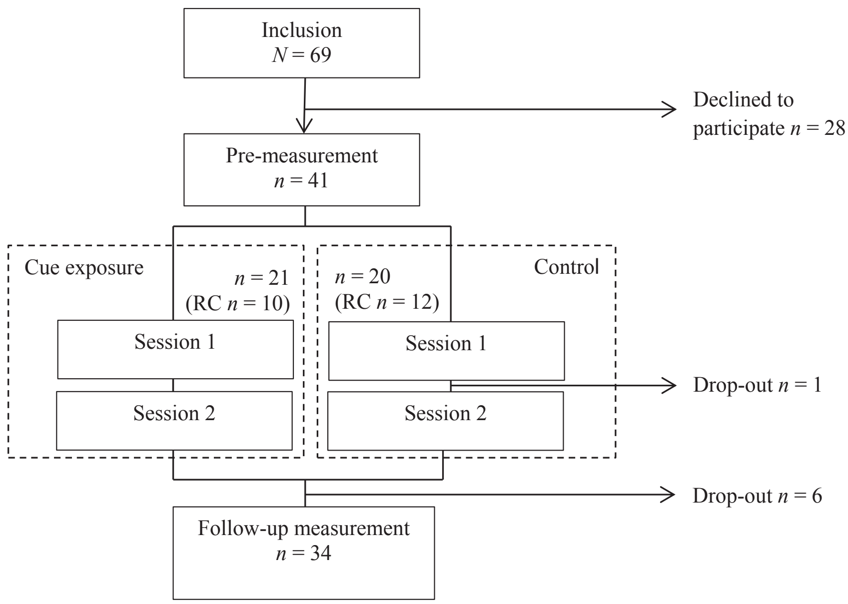

Fig. 1. Inclusion, randomisation and study completion of participants. RC = the number of participants within each condition that received a retrieval cue.

condition that was assigned to each group of participants was alternated (cue exposure – lifestyle control – cue exposure, etc.). Participants had an equal chance of being randomized to one or the other condition: the dates on which the participant was available for the intervention determined in which group and thus which condition (s)he was assigned to, assignment to conditions was not based on personal characteristics, and groups were not pre-existing. Half of the groups in each condition were further randomized to a retrieval cue and no retrieval cue condition. At the moment of inclusion, neither the experimenter nor the participant had information about which group and condition the next participant would be assigned to. In addition, participants did not know between inclusion and the first session to which condition they would be assigned to; conditions were concealed until all baseline measures were completed on the first session. The study was approved by the Ethical Committee of the Faculty of Psychology and Neuroscience of Maastricht University.

2.2. Intervention

2.2.1. Food cue exposure intervention

Participants were exposed to large portions of their personalised four favourite foods that were continuously available right in front of the participant on a tray. In addition, every participant was exposed to chocolate mousse, being the standard exposure food item. Participants were coached in getting their desire to eat the food as high as possible, by smelling, touching, licking and imagining eating the food, but consumption was not allowed. During the breaks, tea towels were placed over the food trays. After the exposure in session one, one personalised food item of choice was selected for the home-work assignment, and put in a plastic container with the instruction to repeat the exposure exercise each day during the next week for at least 15 min. All other foods were thrown away by the participant in a garbage bin. In order to keep the homework foods appealing and unspoilt during the entire week, only non-perishable foods were selected as exposure foods.

2.2.2. Lifestyle control intervention

The lifestyle control intervention focussed on providing psycho-education about healthy eating, physical exercise, body satisfaction and weight loss. Dutch guidelines for healthy eating were provided (Voedingscentrum, 2011), and the discussed themes were regularly coupled with interactive exercises. Participants also received a home-work assignment, in which they were instructed to make a poster containing pictures and drawings of foods, categorised as healthy and unhealthy. The poster was presented for the other group members during the second session.

2.3. Procedure

An overview of study measurements is displayed in Fig. 2. Prior to the intervention sessions, participants filled out their personal favourite foods to determine exposure foods and the Eating Disorder Examination-Questionnaire online. In addition to the description of the favourite food, participants also rated palatability and difficulty of not eating this food. When participants were assigned to the exposure condition, the researcher selected and bought four foods that were most palatable and difficult to refrain from. After completing the online survey, participants were planned for two group sessions with exactly one week in between the two sessions. Blinding of participants and experimenters was not possible. On average, 4.1 children (range 2–7) participated in one group, and 10 groups took place in total; five of each intervention. Group sessions took place from 3 p.m. to 6.45 p.m., including a sandwich meal in the final part of each session, and a bogus taste test after the meal in session two. Participants were instructed to eat a regular lunch before the intervention, but no snacks between lunch and the intervention. Eating and drinking (except water) were not allowed during the intervention.

During each session, baseline measurements were done (saliva, CS-US expectancy and VASs on desire to eat and hunger), as well as questions on homework compliance at session two only. Then in session one, participants were given a short rationale for the intervention they were assigned to (cue exposure or lifestyle). Furthermore, participants in the retrieval cue condition received a ribbon in a colour of choice that was explicitly linked to what would be learned during the intervention, and instructed to wear the ribbon until the online follow-up measurement. They rated their expectation of the intervention, after which the intervention started. Both interventions lasted exactly 60 min, excluding breaks. In

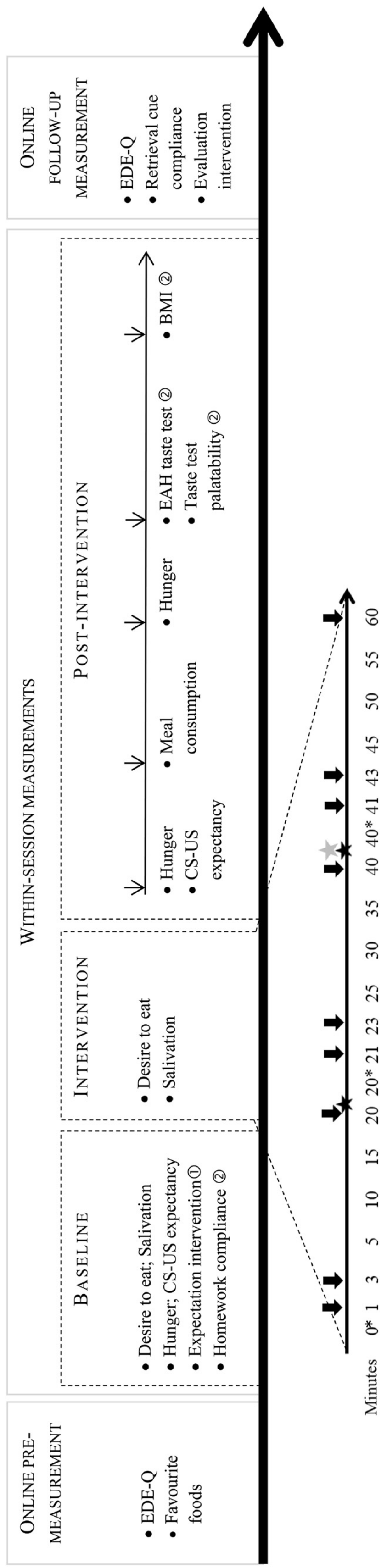

Fig. 2. Schematic overview of timing of study assessments during online measurements and within-session measurement during both sessions. ① reflect measurements that were done only in session 1, and ② measurements only in session 2. Numbers on timeline reflect minutes of 60-min intervention on which measurements took place: minutes marked with ➡ reflect desire to eat and salivation measurements that were measured in both conditions, minutes without ➡ represent additional desire to eat measurements in the cue exposure condition, measurements that were taken when exposure foods were not visible are displayed with *. ★ represent the 10-min breaks of the cue exposure condition, ☆ represents the 20-min break of the control condition.

the cue exposure condition, there were two 10-min breaks after every 20 min of exposure, while there was one 20-min break after 40 min in the control condition. Saliva and desire to eat were measured at 10 time-points (at baseline and nine times during the intervention). In the cue exposure condition, 12 additional desire to eat measurements were taken throughout the intervention, including three measurements (minutes 0, 20, 40) when exposure foods were not visible. The timer was stopped for interruptions due to the measurements, resulting in 60 min of pure interventions. After the intervention, participants rated current hunger and CS-US expectancy. Then, all participants received the meal after which hunger was again rated. Finally, in session one, participants received information about their homework assignment, whereas in session two, the meal was followed by the bogus taste test to measure EAH. Weight and height were measured after the bogus taste test in session two.

One month after the intervention (follow-up), participants filled out the Eating Disorder Examination-Questionnaire online, as well as the questions intervention evaluation and compliance on the retrieval cue, after which they received a compensation of €20 for participation and were thanked and debriefed about the nature of the study.

2.4. Assessment

2.4.1. EAH behavioural paradigm

The EAH paradigm was adapted from the paradigm described by Birch and Fisher (2000) and Schyns et al. (2016). To induce a state of satiation, participants received a sandwich meal at the end of both sessions, and they were verbally instructed to eat until satiation. Sandwiches were freely available on a food cart, with a maximum of three sandwiches per participant. The number of consumed chicken (per sandwich 160 gr; 295.6 kcal) and cheese (per sandwich 175 gr; 300.1 kcal) sandwiches was secretly recorded by the experimenters, in order to check whether intake before the taste test was equal for both conditions. Water and diet soda were served with the sandwiches. After finishing the sandwiches, participant's current hunger (2.4.5) was measured. Only after the post meal hunger rating at session two, a 10-min bogus taste test of six desserts started. Participants were instructed to rate the taste of each of the desserts (2.4.8), while being allowed to eat as much as they wanted from the generous portions: chocolate mousse (~80 g/~142 kcal; Almhof), whipped cream (~90 g/~165 kcal; Campina), muffin (~80 g/~262 kcal; Jumbo supermarket), custard (~190 g/~173 kcal; Campina), strawberry mousse (~120 g/~156 kcal; Dr. Oetker), chocolate cake (~80 g/~330 kcal; Coolmore), in total approximately 1230 kcal. Items on the questionnaire were presented in the same order for every participant. The chocolate mousse that was used during the taste test was the same chocolate mousse as used during the exposure exercises. The taste test was done individually; tables were separated by large screens or testing occurred in different rooms. After the bogus taste test, foods were removed. Each food item was weighed before and after the test, and the number of consumed kcal was calculated. Consumed kcals for each participant, both for sandwich consumption and taste test consumption, were converted into a percentage of the participant's daily energy requirement, using the Human Energy Requirements Report (FAO/WHO/UNU, 2001). Specifically, the daily energy requirement (i.e., kcal per kg body weight per day; specific for sex and age and assuming light physical activity) was calculated per participant and the percentage of consumed kcal during the meal and taste test relative to the daily energy requirement was calculated. Regarding taste test consumption, separate percentages for exposed (i.e. chocolate mousse) vs. non-exposed food items were calculated in order to address the hypothesis on generalisation.

2.4.2. CS-US expectancy

Participants rated how much they believed in the following 'if CS then US' statement for overeating: 'If I have tasty food in front of me, then I can … resist to eat it' by indicating what word was most appropriate at the present moment, ranging from 'not at all' (1) to 'very well' (5).

2.4.3. Desire to eat

Desire to eat was measured on a Visual Analogue Scale (VAS): 'At the present moment, I have … ' ranging from 0 (no desire at all to eat tasty food) to 100 mm (an extreme desire to eat tasty food).

2.4.4. Salivation

Salivation was measured by placing two dental cotton rolls (Hartmann, nr2, 10 × 35 mm) in the mouth between the cheek and left and right lower gums. The cotton rolls stayed in the mouth for exactly one minute. They were placed and removed by the participant and kept in a sealed plastic bag that was weighed before and after the measurement on a 0.01 g accurate weighing scale (Mettler Toledo, PB3002).

2.4.5. Hunger

Current hunger was measured on a VAS: 'At the present moment, I am … ', ranging from 0 (not hungry at all) to 100 mm (very hungry).

2.4.6. Intervention credibility

Each participant rated how much (s)he expected to learn/had learned from the intervention on a 5-point scale from 1 (not at all) to 5 (very much), and whether (s)he expected the intervention to help gaining control over eating, rated from 1 (much less control) to 5 (much more control).

2.4.7. BMI

To calculate body mass index (BMI; kg/m2), height and weight (0.1 kg accurate; My Weigh, XL-550), were measured in the laboratory by the experimenter. Participants were further defined as overweight or obese using the BMI criteria for children (Cole et al., 2000).

2.4.8. Taste test palatability

The palatability of each of the six taste test items was scored on a 0 (not palatable at all) to 100 (very palatable) VAS.

2.4.9. Eating psychopathology

The Eating Disorder Examination-Questionnaire (Fairburn & Beglin, 1994) was administered to assess eating psychopathology. The Dutch version for children was used (E. Jansen, Mulkens, Hamers, & Jansen, 2007). 30 items target the frequency and severity of eating psychopathology, and are answered on a 7-point scale, ranging from 0 (not present) to 6 (very frequent or severe). A global score is calculated, indicating the severity of eating psychopathology, and binge eating is assessed.

2.4.10. Retrieval cue compliance

To assess instruction adherence concerning the retrieval cue, a question was asked to what extent the ribbon that served as retrieval cue was worn, from 1 'not wearing the ribbon at all' to 8 'wearing the ribbon continuously'.

2.4.11. Homework compliance

To check for homework compliance, three questions were asked on the number of days, amount of time and intensity to which homework exercise were performed and completed on a 5-point Likert scale, ranging from 1 (no homework done at all) to 5 (7 days/at least 15 min each day/super-intensive).

2.5. Statistical analyses

Analyses on the comparison of conditions on continuous variables included independent samples t-tests, analysis of variance (ANOVA) and mixed model ANOVAs for between-subjects and within-subjects variables, while Chi-square and Fisher's exact (in case of violation of Chi-square's assumptions) tests were used for comparisons for binary outcomes. If the Levene's test for homogeneity of variances was found to be violated, a *t*-test not assuming homogeneous variances was reported. The data were normally distributed. WSH and BSH were calculated for both cue reactivity measures. WSH was operationalised by subtracting the individual end-level of cue reactivity from the individual peak response during each session and averaging the scores for the two sessions. BSH was calculated by subtracting the individual peak response from session two from the individual peak response of session one (Craske et al., 2008). Mediational effects of habituation (condition → habituation [mediator] → EAH) were tested by applying the bootstrapping method with a 95% confidence interval of the indirect effect using 5000 samples as described by Preacher and Hayes (2008). Indirect mediation effects are considered significant when the 95% confidence interval does not contain zero. Per-protocol analyses were applied to investigate the true effects and working mechanisms of the exposure intervention for participants who completed both exposure sessions. No data were estimated for participants ($n = 6$) who were lost to follow-up after the intervention, as applying the recommended last value carried forward strategy (Shah, 2011) would lead to a serious underestimation of the time effect, because the only available EDE-Q data are pretreatment scores. To correct for the possibility of type I errors for two primary outcomes (i.e., intake of exposed and non-exposed foods) and two secondary outcomes (i.e., EDE-Q global score and binge eating), obtained p-values were adjusted for multiple comparisons using the sequentially rejective Bonferroni-Holm correction (Holm, 1979).

3. Results

3.1. Descriptives

As presented in Table 1, (unadjusted) BMI, age, weight status, gender and binge eating status did not differ between the two conditions.

3.2. Credibility and compliance to homework and retrieval cue instructions

As displayed in Table 1, the mean interventions' expectations and evaluations were equal for both conditions. Retrieval cue compliance was poor; only four of 22 participants kept the ribbon on until the follow-up measurement, data on retrieval cue effects were therefore not further analysed. Compliance of homework assignments was poor as well: while participants in the cue exposure condition where instructed to do the exposure assignment each day, only one of 21 participants followed this instruction, and four participants practiced five to six days. The mean number of days, intensity and minutes spent on homework were not-significantly different between conditions, as can be seen in Table 1. Although not significant, the mean number of days spent on homework were marginally higher in the cue exposure condition vs control condition.

Table 1
Comparison baseline characteristics, intervention credibility and homework compliance scores between the cue exposure and control condition.

		Cue exposure $n = 21$	Control $n = 19$	Comparison of conditions
Baseline characteristics				
BMI	*M (SD)*	32.17 (3.38)	31.87 (5.88)	t (28.13) = 0.20, $p = 0.84$, $d = 0.06$
	95% CI	[30.63, 33.71]	[29.03, 34.70]	
Age	*M (SD)*	14.24 (1.87)	14.53 (1.81)	t (38) = 0.50, $p = 0.62$, $d = 0.16$
	95% CI	[13.39, 15.09]	[13.66, 15.40]	
Obese participants	*n* (%)	18 (85.7)	16 (84.2)	$P = 1.00$, Fisher's exact test, $\varphi = -0.21$
Females	*n* (%)	11 (52.4)	13 (68.4)	$\chi2$ (1) = 1.07, $p = 0.25$, $\varphi = 0.16$
Binge eaters	*n (%)*	8 (38.1)	4 (21.1)	$\chi2$ (1) = 1.38, $p = 0.24$, $\varphi = -0.19$
Intervention credibility – learning during intervention				
Expectation	*M (SD)*	3.81 (0.93)	3.42 (0.96)	t(38) = 1.30, $p = 0.20$, $d = 0.42$
	95% CI	[3.39, 4.23]	[2.96, 3.88]	
Evaluation	*M (SD)*	3.06 (1.14)	2.82 (0.88)	t (32)[a] = 0.67, $p = 0.51$, $d = 0.24$
	95% CI	[2.47, 3.65]	[2.37, 3.28]	
Intervention credibility - control over eating				
Expectation	*M (SD)*	4.24 (0.83)	4.00 (0.47)	t (32.23) = 1.13, $p = 0.27$, $d = 0.36$
	95% CI	[3.86, 4.62]	[3.77, 4.23]	
Evaluation	*M (SD)*	2.35 (1.06)	2.94 (0.97)	t (32)[a] = 1.69, $p = 0.10$, $d = 0.60$
	95% CI	[1.81, 2.90]	[2.44, 3.44]	
Homework compliance				
Number of days spent	*M (SD)*	2.48 (1.21)	1.93 (0.27)	t (22.85) = 2.00, $p = 0.06$, $d = 0.63$
	95% CI	[1.93, 3.03]	[1.77, 2.08]	
Minutes each day spent	*M (SD)*	3.43 (1.54)	3.93 (1.27)	t (33) = 1.01, $p = 0.32$, $d = 0.35$
	95% CI	[2.73, 4.13]	[3.20, 4.66]	
Intensity performance	*M (SD)*	3.10 (1.45)	3.21 (0.97)	t (32.98) = 0.29, $p = 0.77$, $d = 0.09$
	95% CI	[2.44, 3.75]	[2.65, 3.78]	

[a] Degrees of freedom are different for this measure, due to drop-out at follow-up. *M* = mean; *SD* = standard deviation; CI = confidence interval.

3.3. EAH

3.3.1. Hunger

As shown in Table 2, participants in both conditions experienced an equal increase in hunger during the intervention in both sessions, followed by an equal decrease in hunger after meal consumption. This was confirmed by significant main effects of time in a 2 (condition: cue exposure, control) × 3 (time: baseline, pre-meal, post-meal) mixed model ANOVA for session one, $F(2, 37) = 57.99$, $p < 0.001$, $\eta_p^2 = 0.76$, and session two, $F(2, 37) = 30.18$, $p < 0.001$, $\eta_p^2 = 0.62$. The effects of the time × condition interactions were not significant for either session one, $F(2,37) = 0.13$, $p = 0.878$, $\eta_p^2 = 0.01$, nor session two, $F(2,37) = 1.25$, $p = 0.299$, $\eta_p^2 = 0.06$. No significant main effect of condition was found for session one, F (1, 38) = 0.09, $p = 0.762$, $\eta_p^2 < 0.01$, or session two, F (1,38) = 3.76, $p = 0.060$, $\eta_p^2 = 0.09$, although the mean hunger rating over the three time-points on session two were marginally higher in the control condition than the cue exposure condition. However, post-meal hunger ratings before starting the taste test in session two were not significantly different between both conditions, $t(38) = 1.61$, $p = 0.116$, $d = 0.51$. All participants reported not being hungry anymore after the meal in session two (all hunger scores < 25 on a 100 scale), indicating that the absence of hunger manipulation was successful and not significantly different between both conditions before starting the EAH taste test.

3.3.2. Meal

Average energy intake of sandwiches was 726.33 kcal ($SD = 344.40$) in the first session and 652.63 kcal ($SD = 256.38$) in the second session. The percentage of energy intake during the meal relative to the individually daily required amount of energy did not differ significantly between conditions, for both session one (cue exposure: $M = 18.84$, $SD = 6.30$; control: $M = 20.59$, $SD = 12.51$; t (38) = 0.55, $p = 0.588$, $d = 0.18$), and session two (cue exposure: $M = 17.36$, $SD = 5.59$; control: $M = 18.52$, $SD = 10.06$; t (38) = 0.44, $p = 0.661$, $d = 0.14$).

3.3.3. Taste test

Average palatability ratings of the taste test items did not differ significantly between conditions, neither for the exposed food item chocolate mousse t (38) = 0.56, $p = 0.580$, $d = 0.18$, nor for the non-exposed food items, t (38) = 1.26, $p = 0.215$, $d = 0.41$. Further, to test the influence of hunger before the taste test on EAH, hunger was included as a covariate in an ANOVA with percentage intake of all foods (exposed and non-exposed) as dependent variable and condition as fixed factor. The results of this analysis showed that

Table 2
Means (M), standard deviations (*SD*) and 95% confidence intervals (CI) of hunger ratings at baseline, before and after meal consumption in each session, separated by condition (cue exposure, control).

	Session 1			Session 2		
	Baseline	Pre-meal	Post-meal	Baseline	Pre- meal	Post-meal
Cue exposure ($n = 21$)						
M (SD)	18.24 (18.59)	47.86 (27.75)	5.95 (11.62)	10.05 (12.22)	32.55 (30.05)	3.19 (4.99)
95% CI	[9.78, 26.70]	[35.23, 60.49]	[0.66, 11.24]	[4.48, 15.61]	[18.87, 46.23]	[0.92, 5,46]
Control ($n = 19$)						
M (SD)	20.08 (20.74)	50.63 (24.03)	5.47 (7.72)	21.40 (26.42)	48.03 (31.63)	6.24 (6.90)
95% CI	[10.08, 30.07]	[39.05, 62,21]	[1.75, 9.20]	[8.66, 34.13]	[32.78, 63.27]	[2.91, 9.56]

condition was a significant predictor of food intake, $F(1,37) = 6.08$, $p = 0.018$, $\eta_p^2 = 0.141$, while hunger in included in the model did not have a significant influence on intake, $F(1,37) = 1.21$, $p = 0.279$, $\eta_p^2 = 0.032$, suggesting that hunger did not play a role in differences in percentage food intake below (3.3.3.1. and 3.3.3.2).

3.3.3.1. Exposed food: chocolate mousse. As presented in Table 3, in line with the hypothesis, the percentage exposure food item consumption (i.e., chocolate mousse) relative to the daily required energy was significantly lower in the cue exposure condition than in the control condition, $t(25.32) = 2.52$, $p = 0.018$, $d = 0.80$.

3.3.3.2. Non-exposed food items. Also in line with the hypothesis, the percentage non-exposed food items consumption relative to the daily required energy was also significantly lower in the cue exposure condition than in the control condition, $t(28.58) = 2.32$, $p = 0.028$, $d = 0.76$, see Table 3.

Both effects of condition on intake of exposed and non-exposed foods remained significant according to the sequentially rejective Bonferroni-Holm correction (Holm, 1979).

3.4. Predictors of EAH

3.4.1. Habituation of cue reactivity

Figs 3 and 4 show the mean patterns of cue reactivity, desire to eat and salivation, during both intervention sessions per condition. Results of the mediation analyses suggest that neither WSH (desire to eat: $\beta < -0.01$, 95% *CI* [-0.05, 0.06]; salivation: $\beta = 0.02$, 95% CI [-0.04, 0.14]), nor BSH (desire to eat: $\beta = -0.05$, 95% CI [-0.27, 0.13]; salivation: $\beta < -0.01$, 95% CI [-0.05, 0.06) were significant mediators between condition and EAH of the exposed food item. Regarding EAH of non-exposed food items, the indirect effect between condition and EAH via WSH of desire to eat did not include zero, $\beta = -1.01$, 95% CI [-2.65, -0.04], indicating that the amount of WSH of desire to eat mediated the relationship between condition and EAH. In this model, condition was significantly associated with EAH (c-path, consistent with 3.3.3.2; $\beta = 4.49$, $t(38) = 2.38$, $p = 0.022$), the relationship between condition and habituation (a-path) was not significant, $\beta = -0.77$, $t(38) = 1.57$, $p = 0.124$, indicating that WSH of desire to eat was not significantly different between conditions, while the relationship between habituation and EAH (b-path) of this model was statistically significant with a positive coefficient, $\beta = 1.37$, $t(38) = 2.32$, $p = 0.026$, indicating that more WSH of desire to eat was related to more EAH of the non-exposed food items. The other habituation measures were not found to be significant mediators in the relationship between condition and EAH of the non-exposed food items (BSH of desire to eat: $\beta = 0.53$, 95% CI [-0.60, 2.57]; WSH of salivation: $\beta = -0.69$, 95% CI [-2.31, 0.18]; BSH of salivation: $\beta = -0.21$, 95% CI [-1.12, 0.28]).

3.4.2. CS-US expectancy violation

CS-US expectancy change scores were calculated (baseline session one minus post-intervention session two). Due to practical errors, the CS-US expectancies were not assessed in eight participants (cue exposure $n = 3$; control $n = 5$). As the assumptions on expected cell frequencies of the Chi-square test were violated, CS-US expectancy change scores were merged into two categories: expectancy violation (change score ≥ 1) vs no expectancy violation (change score ≤ 0). No significant differences were found between conditions regarding expectancy violation, $\chi^2(1, n = 32) = 3.35$, $p = 0.067$, $\varphi = -0.32$, although the proportion of individuals experiencing expectancy violations was marginally higher in the cue exposure condition (11 of 18 participants) compared to the control condition (four of 14 participants). With regard to EAH, no significant interactions were found in a 2 (condition) × 2 (expectancy violation: yes vs no) ANOVA for the exposed food item, $F(1,28) = 0.06$, $p = 0.814$, $\eta_p^2 < 0.01$, and non-exposed food items $F(1,28) = 0.27$, $p = 0.605$, $\eta_p^2 = 0.01$, nor were any main effects found of expectancy violation on EAH of the exposed food item, $F(1,30) = 0.17$, $p = 0.680$, $\eta_p^2 < 0.01$, or non-exposed food items, $F(1,30) = 1.06$, $p = 0.311$, $\eta_p^2 = 0.03$.

3.5. Eating psychopathology

The Eating Disorder Examination-Questionnaire global score decreased significantly from pre-measurement (cue exposure: $M = 2.35$, $SD = 1.17$; control: $M = 2.21$, $SD = 0.98$) to follow-up (cue exposure: $M = 2.03$, $SD = 1.14$; control: $M = 1.91$, $SD = 0.97$) in both conditions, as reflected by a significant main effect of time in a 2 (condition) × 2 (time: pre vs. follow-up measurement) mixed model ANOVA, $F(1, 32) = 5.26$, $p = 0.029$, $\eta_p^2 = 0.14$, and no significant condition × time interaction $F(1,32) = 0.11$, $p = 0.744$, $\eta_p^2 < 0.01$. No main effect of condition was found, $F(1,32) = 0.11$, $p = 0.744$, $\eta_p^2 < 0.01$. At follow-up, four participants still reported binge eating relative to 12 participants at pre-measurement, the change of binge eating status was not significantly different between conditions, $P = 0.398$, Fisher's exact test. Results remain unchanged when using the sequentially rejective Bonferroni-Holm correction (Holm, 1979).

4. Discussion

The main aim of the present study was two-fold: (1) to investigate whether two sessions of cue exposure lead to less EAH of the exposed food item and less EAH of non-exposed food items (generalisation), and (2) to investigate the predictive value of cue reactivity habituation and CS-US expectancy violation for EAH. Consistent with the a-priori hypothesis, it was found that cue exposure led to less EAH of the exposed and non-exposed food

Table 3
Means (*M*), standard deviations (*SD*) and 95% confidence intervals (CI) of absolute kcal consumption and percentage of consumed kcal relative to the individual daily amount of required energy during the EAH paradigm, separated by condition (cue exposure, control).

	Exposed food item[a]		Non-exposed food items[b]	
	Absolute kcal	Percentage of daily required kcal[c]	Absolute kcal	Percentage of daily required kcal[c]
Cue exposure ($n = 21$)				
M (*SD*)	23.05 (28.24)	0.57 (0.68)	257.68 (184.39)	6.37 (4.34)
95% CI	[10.19, 35.90]	[0.26 0.88]	[173.74, 341, 61]	[4.39, 8,35]
Control ($n = 19$)				
M (*SD*)	47.40 (43.72)	1.46 (1.41)	357.72 (232.55)	10.86 (7.36)
95% CI	[26.33, 68.47]	[0.78, 2.15]	[245.63, 469.80]	[7.32, 14.41]

[a] Kcal intake of the item that was used during cue exposure: chocolate mousse.
[b] Total kcal intake of items not used during cue exposure (whipped cream, muffin, custard, strawberry mousse, chocolate cake).
[c] Percentage eaten of individual daily required amount of energy, adjusted for sex, age and weight (FAO/WHO/UNU, 2001).

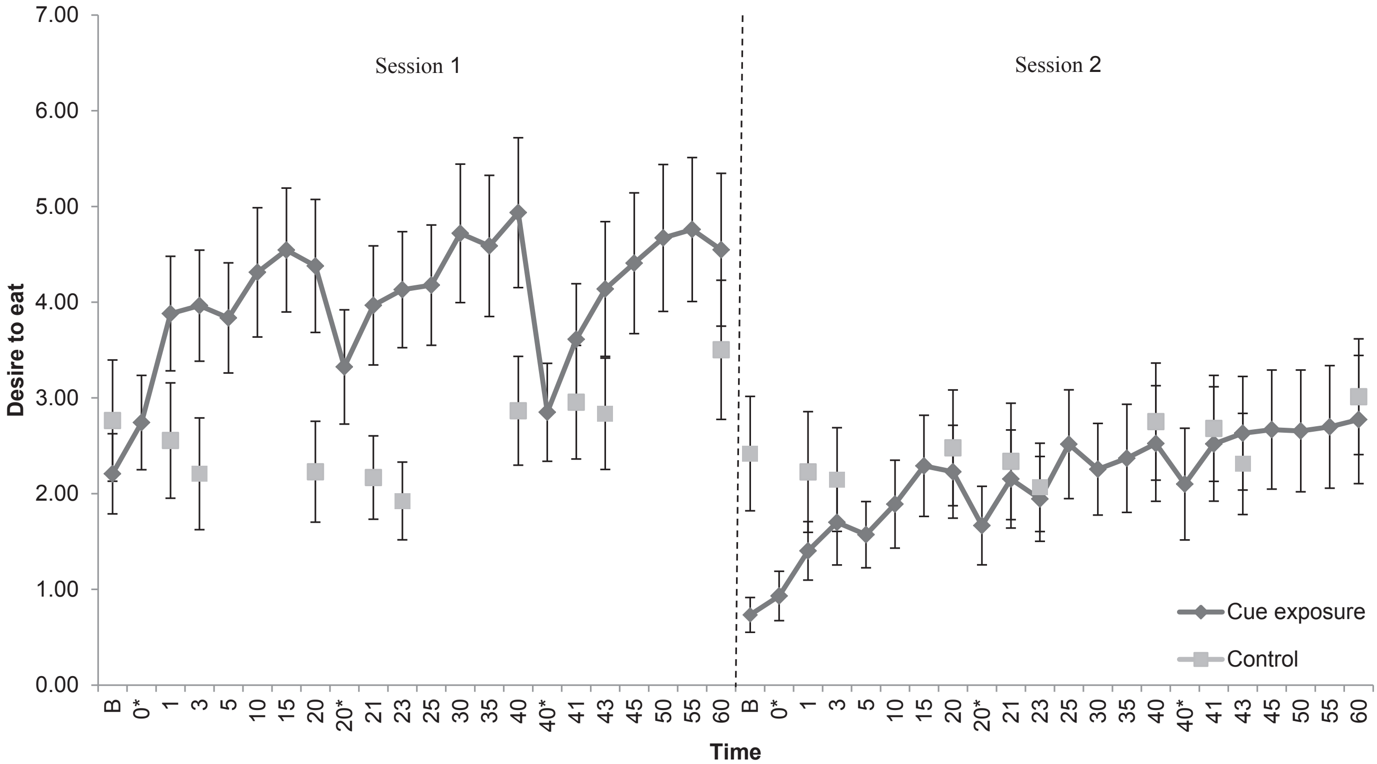

Fig. 3. Mean desire to eat scores per condition (cue exposure, control) per time point at baseline (B) and during the intervention (minutes 0–60) on session one and two. Error bars represent standard errors of means. Additional measurements in the cue exposure condition that were taken when exposure foods were not visible are displayed with *.

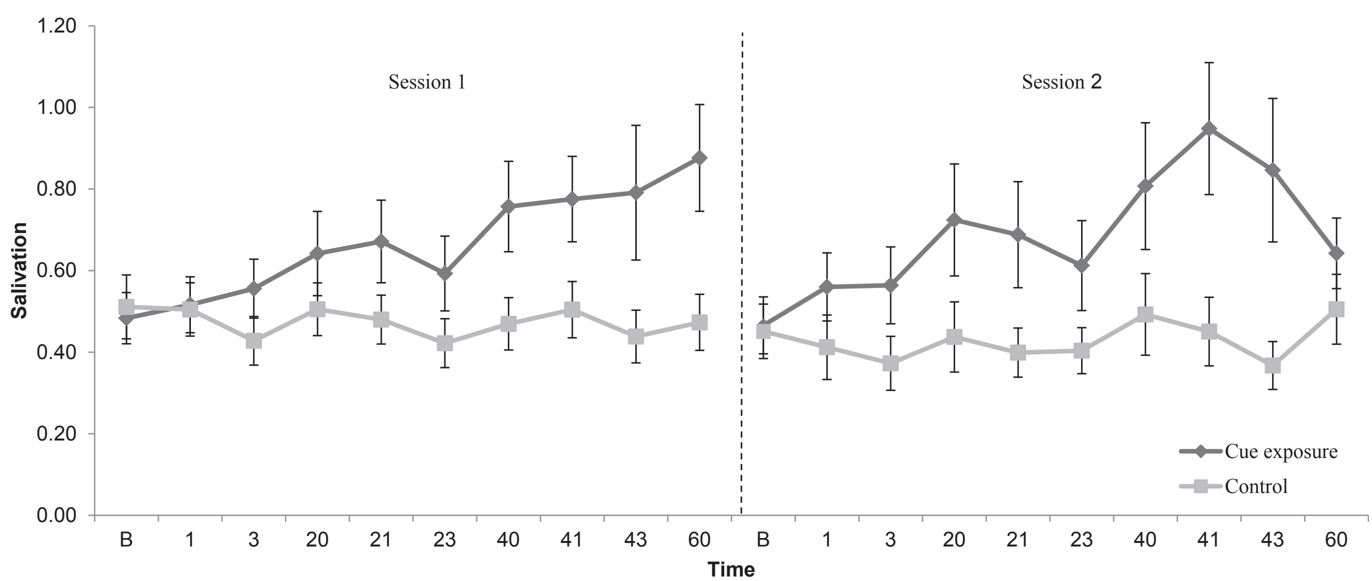

Fig. 4. Mean salivation per condition (cue exposure, control) per time point at baseline (B) and during the intervention (minutes 0–60) on session one and two. Error bars represent standard errors of means.

items, relative to the control condition. Habituation of cue reactivity either within or between sessions did - as hypothesised - not mediate the relationship between condition and EAH, except for WSH of desire to eat. WSH of desire to eat was found to be a significant mediator, but unbeneficial for treatment outcome: larger habituation was associated with *more* EAH. Unexpectedly, CS-US expectancy violation was not predictive of EAH. With regard to the secondary outcome measure, eating psychopathology improved over time, but not significantly different between conditions.

The finding of less EAH of the exposed food item after two exposure sessions in the present study replicates the finding that was previously found with one exposure session in overweight adult women (Schyns et al., 2016). However, no significant differences were found in the one-session study on EAH of the non-exposed food items whereas a difference *was* found in the current two-session study with a younger sample. The current finding of less EAH of non-exposed food items indicates generalisability of the exposure effect: the exposure was not only effective for the items that were present during the exposure session, the learning transferred to non-exposed highly palatable foods. This could potentially be an important notion for clinical practice; the current data might indicate that doing multiple exposure sessions makes it not essential to include all ‘problematic’ foods during exposure sessions. Moreover, the significant differences in EAH of non-exposed food items of approximately 100 kcal during one meal are not only statistically significant, but also clinically relevant for weight loss, indicating the need to study long-term exposure effects on weight loss in the future. Generalisation of the exposure effect does however not automatically imply that randomly selected palatable foods can be used for exposure sessions, it seems plausible to use individually selected favourite foods in order to maximise learning experiences during exposure sessions.

The differences on EAH between both conditions could not be explained by potentially differential hunger levels between conditions before starting the taste test. The finding of generalisation in the present study where participants had sufficient time to consolidate the new CS-noUS memory fits well with the findings of Pace-Schott et al. (2012), who suggest that sleeping between sessions improves generalisation of exposure effects. An alternative, or additive, explanation for finding generalisation in the present study could be the sample of adolescents who might be better learners than adults and therefore constitute a good target group for interventions. Note that these explanations are speculative, as we did not manipulate sleep or selectively study age within the present experiment.

It was further studied whether habituation of food cue reactivity and expectancy violation served as predictors of EAH. Regarding cue reactivity, neither habituation within exposure sessions (WSH) or between sessions (BSH), for both self-reported eating desires and salivation, mediated the relationship between condition and EAH, except for WSH of desire to eat. WSH of desire to eat was a significant mediator between condition and EAH of non-exposed food items. However, further inspection indicated that larger WSH was associated with more EAH, suggesting that aiming at increasing habituation of eating desires during exposure might have a negative impact. However, this could be a coincidental finding and needs to be replicated. From the current results, it can be concluded that habituation of cue reactivity, including salivation and eating desires, did not have beneficial effects on EAH, which is in line with previous findings (Schyns et al., 2016). Moreover, these findings indicate that the non-promoting role of habituation for exposure therapy outcome in anxiety disorders (Craske et al., 2008) might also hold for food cue exposure therapy. As suggested in the anxiety literature, exposure should aim at increasing associative strength of the inhibitory CS-noUS association, for example by violating CS-US expectancies (Craske et al., 2014). However, current data did not provide support for the importance of CS-US expectancy violation for EAH, which contrasts previous research (Schyns et al., 2016). Our findings suggest that expectancy violation does not play an important role in overeating, which is in line with a recent conditioning study in which expectancy violation did not lead to lower conditioned desires at test compared to a control condition (van den Akker, van den Broek, Havermans, & Jansen, 2016). Alternatively, finding no relationship between expectancy violation and EAH can be explained by the fact that the exposure sessions were not designed to explicitly tackle CS-US expectancies, instead a standard exposure procedure was used that might not have been sufficient to violate expectancies in all participants. More research on the importance of expectancy violation for food cue exposure therapy outcomes is clearly warranted. An interesting and challenging next step would be to manipulate expectancy violation during cue exposure sessions.

Looking at eating disorder psychopathology one month after the intervention, both conditions improved significantly on the Eating Disorder Examination-Questionnaire global score; cue exposure was not superior in this respect. No changes were found for binge eating. The effects of retrieval cues on eating psychopathology could not be studied due to poor compliance to the use of the retrieval cue and posits a challenge for studies and clinical practice to effectively apply exposure in adolescents, for example by including parents to facilitate adherence to all intervention components (including retrieval cues and homework assignments).

It should be noted that the present study was underpowered. A larger effect size was expected for kcal intake of exposed food in the current study compared to a recent single-session cue exposure study (Schyns et al., 2016), as adolescent participants were expected to be faster learners, and participants received two sessions of cue exposure instead of one session. Although no a priori power analysis was conducted, a post hoc power analysis indicates that 68 participants would have been required in total ($n = 34$ per condition) to detect an effect size of 0.80 with the desired power of 90% and alpha of 0.05. Because 40 participants were included, interpretations of the study results should be done with caution, and replication of the study effects is necessary. Further, the CS-US expectancy item has not been validated and might not have been applicable to every single participant to capture individual learning processes. Also, adherence to the homework instructions during the week between both sessions was poor; intervention effects might have even been larger when participants would have done daily homework exercises. Finally, it should be noted that a taste test was not included at pre-test to prevent pre-test sensitization (i.e., allowing participants to formulate hypotheses about the study's goals and realize what is being measured in the taste test, potentially causing them to respond differently to the treatment and at post-test; Bordens & Abbott, 2011). Because participants were randomized to one of either conditions, it is justified and sufficient to interpret post-intervention EAH scores (Gruijters, 2016).

5. Conclusions

Food cue exposure was effective to reduce EAH of exposed and non-exposed food items, indicating that exposure not only works for foods that were present during exposure sessions, but that inhibitory learning transfers to other foods as well. Consistent with exposure research in anxiety disorders, habituation within and between sessions was not found consistently related, or in any case not beneficial, to EAH: though eating desires might be an important motivation to start cue exposure therapy, the present data suggest that habituation of eating desires is not necessary during exposure sessions. However, the present data also suggest that expectancy violation is not necessary for reducing EAH. More research is clearly needed to understand the working mechanisms in cue exposure therapy for overeating and obesity.

Conflict of interest

The authors report no conflict of interest.

Acknowledgements

This work was supported by the Netherlands Organisation for Scientific Research: Vici Grant 453.10.006, awarded to Anita Jansen. The authors would like to thank Anastacia de Graaff for her help in collecting data. Further, the authors would like to thank the Area

Health Authority (Gemeenschappelijke Gezondheidsdienst; GGD) in Geleen, the Netherlands, and the paediatric obesity outpatient clinic of Zuyderland in Kerkrade, the Netherlands, for notifying clients about the study.

References

van den Akker, K., Havermans, R. C., Bouton, M. E., & Jansen, A. (2014). How partial reinforcement of food cues affects the extinction and reacquisition of appetitive responses. A new model for dieting success? *Appetite, 81*, 242–252. https://doi.org/10.1016/j.appet.2014.06.024.

van den Akker, K., van den Broek, M., Havermans, R. C., & Jansen, A. (2016). Violation of eating expectancies does not reduce conditioned desires for chocolate. *Appetite, 100*, 10–17. https://doi.org/10.1016/j.appet.2016.02.004.

Batterink, L., Yokum, S., & Stice, E. (2010). Body mass correlates inversely with inhibitory control in response to food among adolescent girls: An fMRI study. *NeuroImage, 52*(4), 1696–1703. https://doi.org/10.1016/j.neuroimage.2010.05.059.

Birch, L. L., & Fisher, J. O. (2000). Mothers' child-feeding practices influence daughters' eating and weight. *The American Journal of Clinical Nutrition, 71*(5), 1054–1061.

Bongers, P., van den Akker, K., Havermans, R., & Jansen, A. (2015). Emotional eating and Pavlovian learning: Does negative mood facilitate appetitive conditioning? *Appetite, 89*, 226–236. https://doi.org/10.1016/j.appet.2015.02.018.

Bordens, K. S., & Abbott, B. B. (2011). *Research designs and methods. A process approach* (8th ed.). New York, NY: McGraw-Hill.

Boswell, R. G., & Kober, H. (2016). Food cue reactivity and craving predict eating and weight gain: A meta-analytic review. *Obesity Reviews, 17*, 159–177. https://doi.org/10.1111/obr.12354.

Boutelle, K. N., Zucker, N. L., Peterson, C. B., Rydell, S. a, Cafri, G., & Harnack, L. (2011). Two novel treatments to reduce overeating in overweight children: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 79*(6), 759–771. https://doi.org/10.1037/a0025713.

Boutelle, K. N., Zucker, N., Peterson, C. B., Rydell, S., Carlson, J., & Harnack, L. J. (2014). An intervention based on Schachter's externality theory for overweight children: The regulation of cues pilot. *Journal of Pediatric Psychology, 39*(4), 405–417. https://doi.org/10.1093/jpepsy/jst142.

Bouton, M. E. (1993). Context, time, and memory retrieval in the interference paradigms of pavlovian learning. *Psychological Bulletin, 114*(1), 80–99. https://doi.org/10.1037/0033-2909.114.1.80.

Bouton, M. E., & King, D. A. (1983). Contextual control of the extinction of conditioned fear: Tests for the associative value of the context. *Journal of Experimental Psychology: Animal Behavior Processes, 9*(3), 248–265. https://doi.org/10.1037/0097-7403.9.3.248.

Butte, N. F., Cai, G., Cole, S. A., Wilson, T. A., Fisher, J. O., Zakeri, I. F., … Comuzzie, A. G. (2007). Metabolic and behavioral predictors of weight gain in Hispanic children: The viva la familia study. *American Journal of Clinical Nutrition, 85*(6), 1478–1485. https://doi.org/85/6/1478 [pii].

Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: International survey. *BMJ, 320*, 1–6.

Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy, 46*(1), 5–27. https://doi.org/10.1016/j.brat.2007.10.003.

Craske, M. G., Treanor, M., Conway, C. C., Zbozinek, T., & Vervliet, B. (2014). Maximizing exposure therapy: An inhibitory learning approach. *Behaviour Research and Therapy, 58*, 10–23. https://doi.org/10.1016/j.brat.2014.04.006.

Culver, N. C., Stoyanova, M., & Craske, M. G. (2011). Clinical relevance of retrieval cues for attenuating context renewal of fear. *Journal of Anxiety Disorders, 25*(2), 284–292. https://doi.org/10.1016/j.janxdis.2010.10.002.

Deacon, B. J., & Abramowitz, J. S. (2004). Cognitive and behavioral treatments for anxiety disorders: A review of meta-analytic findings. *Journal of Clinical Psychology, 60*(4), 429–441. https://doi.org/10.1002/jclp.10255.

Dibbets, P., Havermans, R., & Arntz, A. (2008). All we need is a cue to remember: The effect of an extinction cue on renewal. *Behaviour Research and Therapy, 46*(9), 1070–1077. https://doi.org/10.1016/j.brat.2008.05.007.

Dibbets, P., Moor, C., & Voncken, M. J. (2013). The effect of a retrieval cue on the return of spider fear. *Journal of Behavior Therapy and Experimental Psychiatry, 44*(4), 361–367. https://doi.org/10.1016/j.jbtep.2013.03.005.

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *The International Journal of Eating Disorders, 16*(4), 363–370. https://doi.org/10.1002/1098-108X(199412).

FAO/WHO/UNU. (2001). *Human energy requirements: Report of a joint FAO/WHO/UNU Expert consultation*. FAO Food and Nutrition Technical Report Series. https://doi.org/92 5 105212 3.

Gruijters, S. L. K. (2016). Baseline comparisons and covariate fishing: Bad statistical habits we should have broken yesterday. *European Health Psychologist, 18*(5), 205–209.

Guerrieri, R., Nederkoorn, C., & Jansen, A. (2012). Disinhibition is easier learned than inhibition. The effects of (dis)inhibition training on food intake. *Appetite, 59*(1), 96–99. https://doi.org/10.1016/j.appet.2012.04.006.

Holm, S. (1979). A simple sequentially rejective multiple test procedure. *Scandinavian Journal of Statistics, 6*(2), 65–70.

Jansen, A. (1998). A learning model of binge eating: Cue reactivity and cue exposure. *Behaviour Research and Therapy, 36*(3), 257–272. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9642846.

Jansen, A., Broekmate, J., & Heymans, M. (1992). Cue-exposure vs self-control in the treatment of binge eating: A pilot study. *Behaviour Research and Therapy, 30*(3), 235–241. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1586360.

Jansen, E., Mulkens, S., Hamers, H., & Jansen, A. (2007). Assessing eating disordered behaviour in overweight children and adolescents: Bridging the gap between a self-report questionnaire and a gold standard interview. *Netherlands Journal of Psychology, 63*(3), 93–97. https://doi.org/10.1007/BF03061070.

Jansen, A., Schyns, G., Bongers, P., & van den Akker, K. (2016). From lab to clinic: Extinction of cued cravings to reduce overeating. *Physiology and Behavior, 162*, 174–180. https://doi.org/10.1016/j.physbeh.2016.03.018.

Jansen, A., Stegerman, S., Roefs, A., Nederkoorn, C., & Havermans, R. (2010). Decreased salivation to food cues in formerly obese successful dieters. *Psychotherapy and Psychosomatics, 79*(4), 257–258. https://doi.org/10.1159/000315131.

Jansen, A., Van den Hout, M. A., De Loof, C., Zandbergen, J., & Griez, E. (1989). A case of bulimia successfully treated by cue exposure. *Journal of Behavior Therapy and Experimental Psychiatry, 20*(4), 327–332.

Kral, T. V., Allison, D. B., Birch, L. L., Stallings, V. A., Moore, R. H., & Faith, M. S. (2013). Caloric compensation and eating in the absence of hunger in 5- to 12-y-old weight-discordant siblings. *The American Journal of Clinical Nutrition, 96*, 574–583. https://doi.org/10.3945/ajcn.112.037952.

Martinez-mallen, E., Castro-fornieles, J., La, L., Moreno, E., Morer, A., Font, E., et al. (2007). Cue exposure in the treatment of resistant adolescent bulimia nervosa. *International Journal of Eating Disorders, 40*, 596–601. https://doi.org/10.1002/eat.

McIntosh, V. V. W., Carter, F. A., Bulik, C. M., Frampton, C. M. A., & Joyce, P. R. (2011). Five-year outcome of cognitive behavioral therapy and exposure with response prevention for bulimia nervosa. *Psychological Medicine, 41*(5), 1061–1071. https://doi.org/10.1017/S0033291710001583.

Mount, R., Neziroglu, F., & Taylor, C. J. (1990). An obsessive-compulsive view of obesity and its treatment. *Journal of Clinical Psychology, 46*(1), 68–78.

Nederkoorn, C., Braet, C., Van Eijs, Y., Tanghe, A., & Jansen, A. (2006). Why obese children cannot resist food: The role of impulsivity. *Eating Behaviors, 7*(4), 315–322. https://doi.org/10.1016/j.eatbeh.2005.11.005.

Nederkoorn, C., Houben, K., Hofmann, W., Roefs, A., & Jansen, A. (2010). Control yourself or just eat what you like? Weight gain over a year is predicted by an interactive effect of response inhibition and implicit preference for snack foods. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association, 29*(4), 389–393. https://doi.org/10.1037/a0019921.

Nederkoorn, C., Smulders, F. T. Y., Havermans, R. C., Roefs, A., & Jansen, A. (2006). Impulsivity in obese women. *Appetite, 47*(2), 253–256. https://doi.org/10.1016/j.appet.2006.05.008.

Ng, M., Fleming, T., Robinson, M., Thomson, B., Graetz, N., Margono, C., … Gakidou, E. (2014). Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: A systematic analysis for the global burden of disease study 2013. *The Lancet, 384*(9945), 766–781. https://doi.org/10.1016/S0140-6736(14)60460-8.

Pace-Schott, E. F., Verga, P. W., Bennett, T. S., & Spencer, R. M. C. (2012). Sleep promotes consolidation and generalization of extinction learning in simulated exposure therapy for spider fear. *Journal of Psychiatric Research, 46*(8), 1036–1044. https://doi.org/10.1016/j.jpsychires.2012.04.015.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*(3), 879–891. https://doi.org/10.3758/BRM.40.3.879.

Salkovskis, P. M., Hackmann, A., Wells, A., Gelder, M. G., & Clark, D. M. (2006). Belief disconfirmation versus habituation approaches to situational exposure in panic disorder with agoraphobia: A pilot study. *Behaviour Research and Therapy, 45*(5), 877–885. https://doi.org/10.1016/j.brat.2006.02.008.

Schyns, G., Roefs, A., Mulkens, S., & Jansen, A. (2016). Expectancy violation, reduction of food cue reactivity and less eating in the absence of hunger after one food cue exposure session for overweight and obese women. *Behaviour Research and Therapy, 76*, 57–64. https://doi.org/10.1016/j.brat.2015.11.007.

Serdula, M. K., Ivery, D., Coates, R. J., Freedman, D. S., Williamson, D. F., & Byers, T. (1993). Do obese children become obese adults? A review of the literature. *Preventive Medicine, 22*(2), 167–177. https://doi.org/10.1006/pmed.1993.1014.

Shah, P. B. (2011). Intention-to-treat and per-protocol analysis. *CMAJ, 183*(6), 695. https://doi.org/10.1503/cmaj.111-2031.

Toro, J., Cervera, M., Feliu, M. H., Garriga, N., Jou, M., Martinez, E., et al. (2003). Cue exposure in the treatment of resistant bulimia nervosa. *The International Journal of Eating Disorders, 34*(2), 227–234. https://doi.org/10.1002/eat.10186.

Vansteenwegen, D., Vervliet, B., Hermans, D., Beckers, T., Baeyens, F., & Eelen, P. (2006). Stronger renewal in human fear conditioning when tested with an acquisition retrieval cue than with an extinction retrieval cue. *Behaviour Research and Therapy, 44*(12), 1717–1725. https://doi.org/10.1016/j.brat.2005.10.014.

Voedingscentrum. (2011). *Guidelines food choices. De Gezondheidsraad*. Retrieved from www.voedingscentrum.nl.

Wilson, G. T. (1994). Behavioral treatment of childhood obesity: Theoretical and practical implications. *Health Psychology, 13*(5), 371–372.