Exposure Therapy vs Lifestyle Intervention to Reduce Food Cue Reactivity and Binge Eating in Obesity

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Exposure therapy vs lifestyle intervention to reduce food cue reactivity and binge eating in obesity: A pilot study

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ABSTRACT

Background and objectives: Learning models of overeating predict that exposure therapy is effective in reducing food cue reactivity and overeating. This pilot study tested an eight-session exposure therapy aimed at inhibitory learning vs. an active control condition aimed at lifestyle improvement for obesity (treatment-as-usual). Main outcomes are snacking behavior, eating psychopathology, food cue reactivity, and weight loss. Change in overeating expectancies was assessed as mediator for outcomes, and the associations between habituation of eating desires and outcomes were investigated in the exposure condition. Sleep quality was investigated as moderator for outcomes.

Methods: 45 overweight women were randomly assigned to the exposure intervention or control condition. The main outcomes, overeating expectancies and sleep quality were re-assessed at post-treatment and three-month follow-up. Habituation of eating desires was measured during exposure sessions.

Results: Compared to the control intervention, exposure led to a significantly stronger reduction in snacking behavior of exposed foods, though this effect did not generalize to non-exposed foods, and stronger binge eating frequency. The exposure condition lost significantly more weight at post-treatment and follow-up than the lifestyle condition. Changes of expectancies mediated the effect of condition on kcal consumption of exposed foods, while habituation during exposure was not related to better treatment outcome. Sleep quality did not moderate the effect of condition on treatment outcome.

Limitations: Small sample size and limited follow-up period.

Conclusions: This short exposure therapy reduced snacking behavior, binge eating and weight more than a lifestyle intervention and is therefore a recommendable intervention for obesity and overeating disorders.

1. Introduction

While a large proportion of overweight and obese individuals engage in weight loss attempts (Yaemsiri, Slining, & Agarwal, 2011), successful weight loss seems difficult to achieve (Fildes et al., 2015). Eating for hedonic purposes instead of physiological needs is a major cause of weight gain (Lowe & Butryn, 2007). Food-associated cues in the environment (e.g., sight of Italian ice-cream, or smell of fresh waffles) may play an important role in hedonic eating, by initiating food cue reactivity (Jansen, Houben, & Roefs, 2015). This food cue reactivity includes psychological (i.e., craving) and physiological (e.g., salivation) responses, and promotes (over)eating. Indeed, cue reactivity was found to be prospectively related to eating and weight gain (Boswell & Kober, 2016), while found to be reduced in successful weight loss maintainers (Jansen, Stegerman, Roefs, Nederkoorn, & Havermans, 2010). These findings suggest that successfully refraining from consuming high-caloric foods is associated with decreased food cue reactivity, which in turn might make it easier to resist tempting foods and hence promotes weight loss (Jansen, Schyns, Bongers, & van den Akker, 2016).

Models of overeating state that food cue reactivity is at least partly learned (e.g., Jansen, 1998; Jansen et al., 2016): Food cues (conditioned stimuli; CSs) can become associated with eating (unconditioned stimulus; US) through repeated pairings, and these CSs can elicit cue reactivity. Similarly, learning models predict that extinction of food cue reactivity can be achieved through repeated exposure to CSs without the US (eating), thereby lowering cue-elicited motivation to eat (e.g., Jansen, 1998; van den Akker, Havermans, Bouton, & Jansen, 2014). A clinical translation of this learning model of overeating is food cue exposure therapy, in which individuals who overeat and/or binge eat are repeatedly exposed to personalized food cues (CSs) without eating (US). Pilot studies in bulimia nervosa patients show substantial

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reductions in cue-elicited cravings and binge eating after cue exposure therapy (Jansen, Broekmate, & Heymans, 1992; Jansen, Van den Hout, De Loof, Zandbergen, & Griez, 1989; Martinez-mallen et al., 2007; Toro et al., 2003). In overweight children, cue exposure reduced overeating of snack foods compared to control interventions (Boutelle et al., 2014, 2011; Schyns, Roefs, Smulders, & Jansen, 2018). In overweight adult females, it was found that cue exposure specifically leads to less overeating of food items included in exposure therapy (exposed foods), but not of food items *not* included in exposure (generalization) (Schyns, Roefs, Mulkens, & Jansen, 2016; Schyns, van den Akker, Roefs, Hilberath, & Jansen, 2018). While cue exposure has been found to prevent weight regain after successful weight loss (Mount, Neziroglu, & Taylor, 1990), it is unclear whether pure cue exposure therapy also effectively facilitates weight loss.

It is also not clear what the working mechanisms of food cue exposure therapy are. In contrast to the limited research on exposure therapy for overeating, great advances have been made on workings mechanisms of exposure therapy for anxiety disorders. It has long been proposed that exposure outcomes depend on sufficient reductions, or habituation, of anxiety (or cravings) during treatment. Specifically, according to the Emotional Processing Theory, habituation of fear is imperative to change pathological fear structures and therefore serves as an index of emotional processing during exposure therapy and thus treatment outcome (Foa & Kozak, 1986; Foa & McNally, 1996). This approach is also widely used in cue exposure studies, as cue exposure sessions are usually continued until arousal (e.g., Mount et al., 1990) or cravings (e.g., Boutelle et al., 2011, 2014) have sufficiently declined. However, Craske et al. (2008) showed that stronger habituation during exposure therapy is not consistently related to better treatment outcome in anxiety disorders. Interestingly, two recent cue exposure studies also found that stronger habituation of cue reactivity during exposure was not significantly associated with better treatment outcome in overweight adolescents and adult women (Schyns et al., 2016; Schyns, Roefs, et al., 2018). Instead of targeting habituation, Craske, Treanor, Conway, Zbozinek, and Vervliet (2014) proposed that the aim of exposure should be to strengthen *inhibitory learning* between a fear-associated stimulus (CS; e.g., a dog) and the *non*-occurrence of an aversive outcome (US; e.g., being bitten; Craske et al., 2014). Research has indicated that CS-US associations are not destroyed during exposure therapy, but that novel inhibitory CS-noUS associations are formed during exposure, as extinguished responses can return (relapse) after therapy (Bouton, 1993; Bouton & King, 1983). Exposure should therefore aim at strengthening the new CS-noUS association as much as possible (Craske et al., 2014). Although CS-US associations involved in anxiety are *content*-wise distinct from CS-US associations involved in overeating and obesity, learning principles in the acquisition and extinction of fear and reward associations might essentially be the same. In fact, Peters, Kalivas, & Quirk (2009) demonstrated an overlapping neural circuitry involved in extinction in fear and reward memories, suggesting a common mechanism and transdiagnostic treatment possibility for anxiety and reward-related disorders.

Several exposure techniques have recently been proposed that should maximize inhibitory learning and hence, improve the effectiveness of exposure therapy for anxiety disorders (Craske et al., 2014). A food cue exposure therapy developed for eating disorders (Jansen, 1998) and obesity (Jansen et al., 2016) was used in the present study and, based on recent extinction insights, we included the new exposure techniques in the protocol. One recommendation by Craske et al. (2014) is to design exposure sessions to maximize the mismatch between the expectancy of what will happen during exposure and what *actually* happens (i.e., the expected catastrophic event does not occur). This violation of CS-US expectancies could be achieved in cue exposure by identifying patient's specific overeating cues that are linked to strong overeating expectancies (e.g., “If I feel exhausted and chocolate is available [CS], then I will lose control and eat the entire chocolate bar [US]”), and exposing that patient to these exact cues while testing whether the US indeed takes place as expected. Exposure therapy targeting expectancy violation was found superior to exposure targeting habituation of anxiety for panic disorder patients (Salkovskis, Hackmann, Wells, Gelder, & Clark, 2006), and lower expectancies at the end of exposure were associated with better treatment outcome in panic disorder patients (Deacon et al., 2013). In cue exposure research, lower overeating expectancies were also found to be significantly associated with reduced kcal intake (Schyns et al., 2016).

In addition to aiming sessions at expectancy violation, Craske et al. (2014) and Jansen et al. (1998; Jansen et al., 2016) recommended more techniques to enhance inhibitory learning in exposure therapy. Given that the original CS-US association remains intact, there are several risk factors that can cause this old association to pop-up (Bouton, 1988, 2011). One risk factor is a change of context (‘renewal’). Solutions to limit renewal are to extend extinction learning to other relevant contexts, or to include mental ‘reminders’ of extinction, so that the participant actively remembers extinction in different contexts. Another important risk of relapse is reinforcement of the original CS-US association, referred to as rapid reacquisition (Bouton, 2011). A solution to rapid reacquisition is to include occasional US reinforcements during extinction, which can be translated to practice by occasionally instructing participants to take a small bite during cue exposure (Bouton, Woods, & Pineño, 2004; van den Akker, Havermans, & Jansen, 2015). Other techniques have been proposed as well, including deepened extinction (i.e., extinction of CSs separately before combining multiple CSs during extinction), removal of safety signals, and variability of various therapy-related factors (Craske et al., 2014).

While inhibitory learning can be enhanced using the above-mentioned techniques, it can also be enhanced by sleep. For example, an exposure study for spider phobia showed that sleeping (as opposed to staying awake) following an exposure session with one spider, led to reduced anxiety responses to that same spider at test (extinction retention), as well as reduced responses to a novel spider, suggesting generalization (Pace-Schott, Verga, Bennett, & Spencer, 2012). Studies have indeed shown that both the quality and quantity of sleep are associated with consolidation and generalization of extinction memories (Pace-Schott, Rubin, et al., 2015; Pace-Schott, Germain, & Milad, 2015). Studying the influence of sleep on exposure therapy seems especially relevant for the obese population, as obesity is associated with decreased sleep quality and quantity (Bidulescu et al., 2010; Patel & Hu, 2008).

The current study investigates whether cue exposure therapy targeted at inhibitory learning is beneficial for treatment outcomes in overweight adult females (Body Mass Index [BMI] > 27). Cue exposure therapy is compared to an active control condition, consisting of treatment aimed at improving lifestyle to achieve weight loss. We hypothesize that: 1) the food cue exposure treatment will produce greater reductions in weight, food cue reactivity, snacking behavior, and eating psychopathology (i.e., binge eating frequency and compensatory behaviors) than the control condition, 2) reduction of CS-US expectancies will mediate the effect of treatment on outcome variables, and stronger habituation of cue reactivity will be associated with better outcomes in the cue exposure condition, and 3) sleep quality will moderate the effect of treatment on outcome variables.

2. Material and methods

The method of the study and an extensive description of cue exposure intervention (including clinical examples) can be found in (van den Akker, Schyns, & Jansen, 2016).

2.1. Participants

A total of 45 females who were motivated to lose weight were included with a mean BMI of 33.68 (SD = 4.32; range = 27.14–49.12) and a mean age of 44.26 years (*SD* = 10.42; range = 18–59). After

completing pre-measurements, participants were randomly assigned to the food cue exposure condition ($n = 23$) or the lifestyle control condition ($n = 22$). Age and BMI were not significantly different between conditions ($ps > .29$). Six participants dropped out during the course of the study (cue exposure $n = 4$; control $n = 2$), these missing data were imputed. The study was approved by the Ethical Committee of … (the Faculty of Psychology and Neuroscience of Maastricht University).

2.2. Procedure

Every participant engaged in a pre-measurement, a post-measurement directly after the intervention, and a follow-up measurement three months after completing the intervention. Measurements were conducted by experimenters blinded to conditions. Both interventions consisted of eight individual therapy sessions of roughly one hour that took place during approximately one month (two sessions per week). In addition, participants in both conditions received psycho-education for ten minutes in the first session on the importance of sleep for weight loss and unhealthy snacking behavior was strongly discouraged to achieve weight loss. BMI, the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), CS-US expectancies, food cue reactivity and the binge eating interview were included in all three measurements. The CS-US interview was done at pre-measurement to compose individual CS-US expectancy items, to identify personally relevant favorite snack foods associated with overeating, the cues that predict overeating (e.g., time of the day, feelings) and the contexts in which the overeating took place for the exposure intervention. After the CS-US interview, the therapist received a report of the participant's favorite foods (USs) and food cues (CSs) to compose the exposure treatment. In addition, the CS-US interview was used to identify the two personal most favorite snack foods, of which one was included in exposure therapy (personal-exposed food item) and the other one would not be included (personal-non-exposed food item). Snacking behavior was measured at post-measurement and follow-up. The Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 1994) was administered at pre- and follow-up measurements. The participant also rated treatment expectation before therapy and evaluation after therapy.

2.3. Intervention

2.3.1. Cue exposure intervention

The cue exposure intervention consisted of eight face-to-face sessions. The participant's favorite foods and individual expectancies were used for the exposure sessions, including one most favorite food item (personal-exposed food item). In addition, the most palatable food item was selected by the participant from a list of ten snack items and also included in exposure therapy (general-exposed food item). Exposures were aimed at violating personal CS-US expectancies by optimally increasing the likelihood of the US, while testing whether the US indeed takes place as expected. The first two exposure sessions took place at the university, followed by exposure sessions in various relevant contexts. Occasional reinforcements were included by occasionally instructing the participant to take very small bites (a few grams) from the foods during sessions. Further, variability of hunger, time of day, and length of exposure sessions were taken into account in scheduling exposure sessions for each participant, and hunger was measured at the start of each session. Mental rehearsal was included as a form of mental retrieval cues, and safety signals were excluded as much as possible (e.g., therapists leaving the room during exposures). Participants were instructed to do daily homework exposure exercises.

2.3.2. Control intervention

The 'Lifestyle+' control intervention consisted of eight sessions, including four face-to-face sessions (two at the university and two at the participant's home) that were alternated with four sessions via telephone. The basic components of the Lifestyle + treatment included (dietary) advice on a healthy lifestyle, mindfulness, power posing and psycho-education on body image. Daily homework exercises consisted of mindfulness and exercises related to the content of the previous session. During telephone sessions, the homework exercises were evaluated.

2.4. General assessments

2.4.1. Intervention acceptability

Prior to and after the intervention, the participant rated expectations/evaluations of the intervention on how appropriate, helpful and recommendable the intervention will be/was on a 9-point scale from 1 (not at all) to 9 (very much).

2.4.2. BMI

Height and weight were measured in the laboratory to calculate BMI (kg/m^2).

2.4.3. Cue exposure characteristics

The number of different foods included in exposure exercises and the number of exposures to the personal-exposed food item and general-exposed food item were recorded, including homework exposures. Furthermore, the level of hunger of each exposure session was measured on a visual analogue scale (VAS; 'How hungry are you right now?'), ranging from 0 (not hungry at all) to 100 (very hungry), and the time of day and length of each session was recorded. The number of homework exercises performed between sessions was also recorded.

2.5. Primary outcome measures

2.5.1. Weight loss

Weight loss at post-measurement and follow-up was calculated by the change in weight percentage relative to pre-measurement: a larger negative score reflecting more weight loss.

2.5.2. Food cue reactivity

To measure food cue reactivity, the general-exposed food item was used. The participant was exposed to this food item for three minutes. Desire to eat, salivation and prospective portion size (PPS) were measured before and after exposure. Desire to eat was measured on a 100 mm VAS ('How strongly do you desire palatable food right now?'), ranging from 0 (no desire at all) to 100 (very strong desire). Salivation was measured using dental rolls (Hartmann, nr 2, 10 × 35 mm), placed between the cheek and lower gum on the left and right side, for exactly one minute. The dental rolls were kept in a sealed plastic bag and weighed before and after saliva collection using a weighing scale accurate to 0.01 g (Mettler Toledo, PB3002). The computerized PPS task is a validated measure to assess the desired food quantity at the present moment (van den Akker, Bongers, Hanssen, & Jansen, 2017). In this task, participants indicated how much she would currently like to eat by selecting an amount of food on a computer screen.

2.5.3. Snacking behavior

A 10-min bogus taste test was completed, which was personalized for each participant, consisting of three snack foods: the personal-exposed food item, the personal-non-exposed food item (to test generalization) and the general-exposed food item. Foods were weighed before and after the taste test, and the consumed kcal were calculated.

2.5.4. Eating psychopathology

The diagnostic items of the EDE-Q (Fairburn & Beglin, 1994) measuring compensatory behaviors were administered to assess eating psychopathology, specified in frequency during the past 28 days. The EDE-Q has an acceptable test-retest reliability regarding the assessment of the diagnostic eating disorder features (Luce & Crowther, 1997).

Table 1
Mean and standard deviations (SD) of expectations and evaluations of intervention acceptability, separated by condition (cue exposure, control).

	Before intervention			After intervention		
	Cue exposure *M* (*SD*)	Control*M* (*SD*)	Comparison of conditions	Cue exposure *M* (*SD*)	Control *M* (*SD*)	Comparison of conditions
Appropriate for complaints	7.24 (1.51)	7.80 (1.55)	$t(43) = 0.97$, $p = .337$, $d = 0.37$	7.24 (1.41)	6.26 (2.45)	$t(33.18) = 1.64$, $p = .110$, $d = 0.49$
Helpful for complaints	6.88 (1.60)	7.18 (1.47)	$t(43) = 0.67$, $p = .508$, $d = 0.20$	7.49 (1.64)	5.63 (2.66)	$t(34.64) = 2.85$, $p = .008$, $d = 0.84$
Recommendable to friend	7.04 (1.77)	7.63 (1.59)	$t(43) = 1.12$, $p = .240$, $d = 0.35$	8.03 (1.38)	6.50 (2.77)	$t(30.46) = 2.33$, $p = .027$, $d = 0.70$

However, as self-reports of binge eating appear to be rather unreliable (Fairburn & Beglin, 1994), objective binge eating (i.e., consumption of objective large amounts of food while losing control) during the last week was assessed during a semi-structured interview that was created for this study, using the DSM-V criteria (American Psychiatric Association, 2013). Participants were not diagnosed with an eating disorder.

2.6. Mechanisms of change

2.6.1. Habituation of cue reactivity

Self-reported desire to eat was measured on a 100 mm VAS ('How strongly do you desire palatable food right now?'), ranging from 0 (no desire at all) to 100 (very strong desire) at every minute during cue exposure sessions to assess within (WSH) and between-session habituation (BSH). WSH was operationalised by subtracting the individual end-level of eating desires from the individual peak desire during each session and averaging the scores for all sessions. BSH was calculated by subtracting the individual peak desire from session eight from the individual peak desire of session one (Craske et al., 2008).

2.6.2. CS-US expectancies

During the CS-US interview, individual overeating situations were identified to formulate four personalized CS-US expectancy items in 'If CS then US' statements to construct the CS-US expectancy scale. CS-US expectancies were rated on expectancy of a CS to be followed by the US using a 100 mm VAS, a higher score reflecting a greater expectancy. In addition to four personalized items, four standard CS-US expectancies were assessed (see Table 3 for overview of the eight items). The Cronbach's alpha of the eight-item scale ranged between 0.86 and 0.95, indicating good to excellent internal consistency. CS-US expectancy change at post-measurement and follow-up was calculated by percentage change relative to pre-measurement: a larger negative percentage reflecting stronger change.

2.6.3. Sleep quality

The PSQI (Buysse et al., 1989) is a validated sleep quality questionnaire with higher scores indicating poorer sleep quality during the last month. The Cronbach's alpha ranged between .70 and .78, indicating acceptable internal consistency that is comparable to the alpha's found in other studies (Beaudreau et al., 2012; Hinz et al., 2017).

2.7. Statistical analyses

To enhance statistical power, we used multiple imputations to replace missing values (5 data sets were imputed). Analyses on the comparison of conditions on continuous variables included independent samples t-tests, and mixed model analyses of variance (ANOVAs) for between-subjects and within-subjects variables. Paired-samples t-tests were used to compare the frequency of exposures to the personal-exposed food item and general-exposed food item, and the degree habituation within the cue exposure condition. Further, to examine the association between habituation and treatment outcomes in the cue exposure condition, Pearson correlations were conducted. The bootstrapping method as described by Preacher and Hayes (2008) was used to test expectancy change as mediator, using the INDIRECT macro with a 95% confidence interval of the indirect effect and using 5000 samples. Indirect mediation effects are considered significant when the 95% confidence interval does not contain zero. The moderating effect of sleep quality was investigated using condition*sleep quality interactions (ANOVA).

3. Results

3.1. Intervention acceptability and evaluation

As shown in Table 1, expectations of participants were not significantly different between conditions. However, after treatment, cue exposure was rated as more helpful and recommendable to friends than the control intervention.

3.2. Cue exposure characteristics

On average, 6.87 (SD = 2.38) different foods were included in exposure exercises during the course of therapy. The number of exposures was not significantly different between the personal-exposed food (M = 7.14, SD = 6.39) and the general-exposed food (M = 8.05, SD = 8.76), $t(22) = 0.98$, $p = .340$, $d = 0.20$. Exposure sessions were scheduled between 8 a.m. and 7 p.m. and hunger levels at the start of exposure sessions ranged between 0 and 98 mm. The mean length of exposure sessions was 8.61 min (SD = 1.45) and ranged between five and 22 min across sessions. Participants did on average a total number of 25.39 (SD = 11.53) homework exposures during therapy.

3.3. Primary outcomes

3.3.1. Weight loss

As displayed in Fig. 1, participants in the cue exposure intervention lost a significantly larger percentage of weight compared to the control condition at post-measurement, $t(43) = 2.31$, $p = .026$, $d = 0.69$. Larger weight loss was also found at follow-up measurement for the cue exposure versus control condition, $t(43) = 2.19$, $p = .034$, $d = 0.65$.

3.3.2. Food cue reactivity

A detailed description of the food cue reactivity data analyses is presented in the supplementary table. Cue-induced eating desires and PPS were not successfully increased at pre-measurement during the food cue reactivity task, neither were any significant main effects of condition nor condition*time (baseline, during the cue reactivity task) interaction effects found. In contrast, salivation was significantly induced in both conditions at pre-measurement. At post-measurement, cue reactivity was not induced in any of the measures, nor were any main effects of condition or time*condition interaction effects found. At follow-up, cue reactivity was successfully induced in all measures, indicated by significant main effects of time on salivation, eating desires and pps. No time*condition interaction effects were found at follow-up, and neither were main effects of condition.

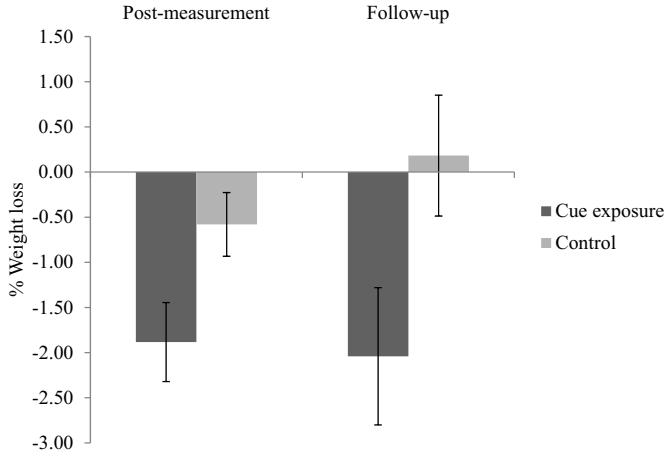

Fig. 1. Percentage of weight loss per condition (cue exposure, control) per time point (post and follow-up measurement). Error bars represent standard errors of means.

3.3.3. Snacking behavior

As shown in Table 2, the cue exposure condition consumed significantly less kcal of the personal-exposed food compared to the control condition at post-measurement, and marginally less at follow-up. Kcal consumption of the personal-non-exposed food and the general-exposed food was not significantly different between conditions at post-measurement or at follow-up.

3.3.4. Eating psychopathology

As displayed in Fig. 2, binge eating frequency at pre-measurement was significantly higher in the cue exposure than the control condition, $t(37.29) = 2.21$, $p = .033$, $d = 0.66$. Interestingly, the number of binges was reduced to zero in all measured participants in the exposure condition at post-measurement, while this was not the case in the control condition. Over these three time points, the main effect of condition was not significant, $F(1,43) = 3.15$, $p = .083$, $\eta_p^2 = 0.068$, while a significant main effect of time, $F(2,86) = 20.30$, $p < .001$, $\eta_p^2 = 0.321$, and a significant time*condition interaction was found, $F(2,86) = 5.09$, $p = .021$, $\eta_p^2 = 0.106$. Follow-up analyses revealed that the reduction of binge eating frequency from pre-measurement to post-measurement was marginally stronger in the cue exposure versus control condition, $t(38.82) = 1.97$, $p = .056$, $d = 0.59$, and significantly stronger from pre-measurement to follow-up, $t(43) = 2.25$, $p = .030$, $d = 0.67$. Vomiting was reported by one participant at pre-measurement and follow-up, while none of the participants reported using laxatives at either measurement. At pre-measurement, 28.9% of participants reported excessive exercise (control: M = 3.41, SD = 6.16; cue exposure: M = 1.13, SD = 2.75), not significantly different between conditions, $t(28.78) = 1.59$, $p = .123$, $d = 0.48$. At follow-up, participants in both conditions did not report less occasions of excessive exercise (control: M = 1.88, SD = 3.14; cue exposure: M = 0.67, SD = 2.11), as the main effect of time was not significant, $F(1,43) = 1.84$, $p = .182$, $\eta_p^2 = 0.041$, and neither was the condition*time interaction, $F(1,43) = 0.53$, $p = .471$, $\eta_p^2 = 0.012$.

3.4. Mechanisms of change

To investigate the findings of significant differential weight change (post-measurement and follow-up), kcal consumption of the personal-exposed food (post-measurement) and binge eating frequency reduction (follow-up) between conditions, the mediating and moderating roles of change of expectancies and sleep quality, respectively, were examined. In addition, the association between habituation of eating desires during exposure sessions and outcomes in the cue exposure condition was investigated.

3.4.1. Habituation of cue reactivity

Habituation of eating desires during the cue exposure sessions is presented in Fig. 3, as reflected by the mean peak and end-levels per session. Averaged over sessions, participants in the cue exposure condition experienced a significant within-session decrease (WSH) from peak desire to eat (M = 57.19, SD = 24.03) to end level (M = 43.86, SD = 25.58), $t(22) = 5.05$, $p < .001$, $d = 1.05$. In addition, the average peak desire to eat on session one was significantly higher than peak desire to eat on session eight, indicating that on average, participants experienced BSH, $t(22) = 5.43$, $p < .001$, $d = 1.13$. When investigating correlations between habituation and treatment outcome within the exposure condition, WSH did significantly positively correlate with weight change at post-measurement, $r(23) = 0.46$, $p = .029$, indicating that more WSH was related to more weight gain. However, WSH was neither correlated to weight change at follow-up, $r(23) = 0.22$, $p = .304$, nor related to kcal intake of the exposure food item at post-measurement, $r(23) = -0.25$, $p = .250$, nor the reduction of binge eating at follow-up, $r(23) = -0.04$, $p = .858$. BSH did not correlate with percentage weight loss at post-treatment, $r(23) = 0.01$, $p = .959$, or at follow-up, $r(23) = -0.05$, $p = .829$. BSH also did not

Table 2
Mean and standard deviations (SD) of kcal intake during the bogus taste test at post and follow-up measurement, separated by condition (cue exposure, control).

Kcal intake	Cue exposure		Control		Comparison of conditions
	M	*SD*	*M*	*SD*	
Post-measurement					
Personal-exposed food item[a]	68.90	36.23	115.90	91.46	$t(27.19) = 2.25$, $p = .033$, $d = 0.70$
Personal-non-exposed food item[b]	92.85	83.39	85.86	46.03	$t(43) = 0.35$, $p = .731$, $d = 0.10$
General-exposed food item[c]	148.57	231.59	132.50	116.53	$t(43) = 0.29$, $p = .772$, $d = 0.09$
Follow-up*					
Personal-exposed food item[a]	73.22	48.77	109.11	85.66	$t(43) = 1.74$, $p = .090$, $d = 0.51$
Personal-non-exposed food item[b]	80.37	54.62	85.88	52.34	$t(43) = 0.35$, $p = .732$, $d = 0.10$
General-exposed food item[c]	97.11	76.75	119.82	117.26	$t(43) = 0.77$, $p = .444$, $d = 0.23$

[a] Personal-exposed food item represents the individually selected item that was included in the exposure intervention.
[b] Personal-non-exposed food item represents the individually selected item that was not included in the exposure intervention.
[c] General-exposed food item represents the food item selected from a list of ten food items, also included in the exposure intervention.

Table 3
Mean and standard deviations (SD) of CS-US expectancies at pre, post and follow-up measurement, separated by condition (cue exposure, control).

		Cue exposure			Control		
		Pre	Post	Follow-up	Pre	Post	Follow-up
Average of eight items below		72.16 (17.41)	25.36 (14.94)	30.15 (15.60)	68.71 (17.60)	53.12 (27.17)	44.08 (24.79)
#1	If palatable food is in front of me, then I cannot refrain from eating it	72.33 (23.73)	24.78 (14.45)	34.10 (22.42)	72.86 (18.21)	50.55 (32.47)	46.69 (27.08)
#2	If general-exposed food[b] is in front of me, then I cannot refrain from eating it	73.43 (28.12)	23.16 (18.76)	31.03 (22.56)	75.52 (21.73)	49.20 (32.33)	46.32 (27.17)
#3	If I eat a small amount of general-exposed food[b], then I cannot stop eating	70.65 (23.15)	25.16 (17.60)	26.44 (19.46)	64.81 (25.47)	49.09 (32.46)	39.74 (28.39)
#4	If I eat a small amount of general-exposed food[b], then I cannot stop eating[a]	59.24 (34.88)	22.06 (16.33)	19.99 (18.07)	58.48 (28.13)	53.71 (32.17)	37.04 (28.89)
#5 & 6[d]	Personalized CS-US expectancy	79.73 (14.23)	29.34 (17.39)	39.59 (17.41)	75.16 (16.28)	54.09 (24.16)	51.80 (23.59)
#7	If I eat a small amount of personal-exposed food[c], then I cannot stop eating	71.36 (17.73)	22.71 (20.99)	26.80 (21.38)	64.33 (30.05)	50.91 (31.72)	38.69 (31.04)
#8	If I eat a small amount of personal-exposed food[c], then I cannot stop eating[a]	71.57 (19.86)	24.12 (21.91)	25.06 (23.74)	62.04 (33.05)	57.03 (32.97)	36.92 (33.68)

[a] On statements marked with participants took a small bite of the specific food before rating their expectancy.
[b] General-exposed food refers to the food item chosen from a list of 10 palatable food items and was used during exposure sessions.
[c] Personal-exposed food refers to the personal favorite food item that was used during exposure sessions.
[d] As items 5 and 6 were personalized and randomly assessed in order, the mean of both items is provided.

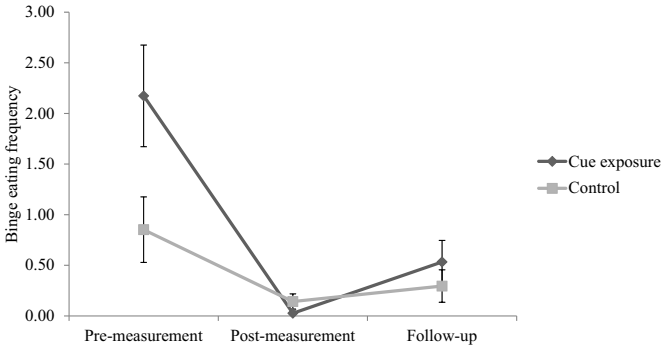

Fig. 2. Binge eating frequency during the last 7 days per condition (cue exposure, control) per time point (pre, post and follow-up measurement). Error bars represent standard errors of means.

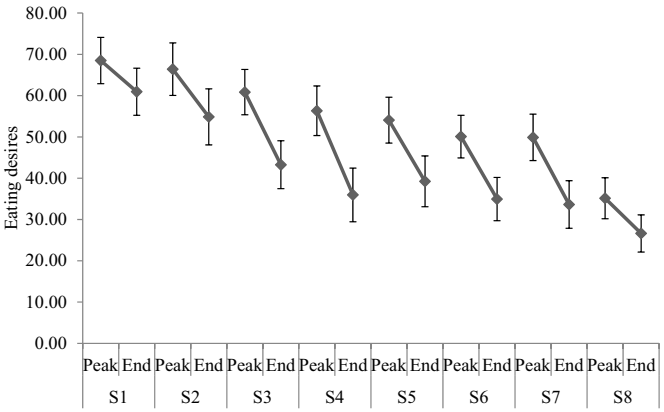

Fig. 3. Mean peak and end-level desire to eat scores per session in the cue exposure condition. Error bars represent standard errors of means.

correlate with kcal intake of the personal-exposed food item at post-measurement, $r(23) = -0.23$, $p = .295$, or the reduction of binge eating at follow-up, $r(23) = 0.13$, $p = .560$.

3.4.2. CS-US expectancy change

Mean CS-US expectancies are shown in Table 3. Average CS-US expectancy of all eight items at pre-measurement was not significantly different between conditions, $t(43) = 0.66$, $p = .513$, $d = 0.20$. As can be seen in Fig. 4, the change in expectancies of all items at post-measurement was significantly larger in the cue exposure versus control condition, $t(31.00) = 4.03$, $p < .001$, $d = 1.21$. At follow-up, the change in expectancies was marginally stronger in cue exposure versus control condition, $t(43) = 1.98$, $p = .055$, $d = 0.59$.

Mediation analyses showed that change in expectancies mediated the relationship between condition and kcal intake at post-measurement, $ab = -33.92$, $SE = 16.72$, 95% CI[-71.50, −6.06]. The findings of this model are summarized in Fig. 5: the cue exposure condition reported stronger expectancy change scores (a-path) and consumed less kcal (c-path), while a stronger expectancy change was also related to

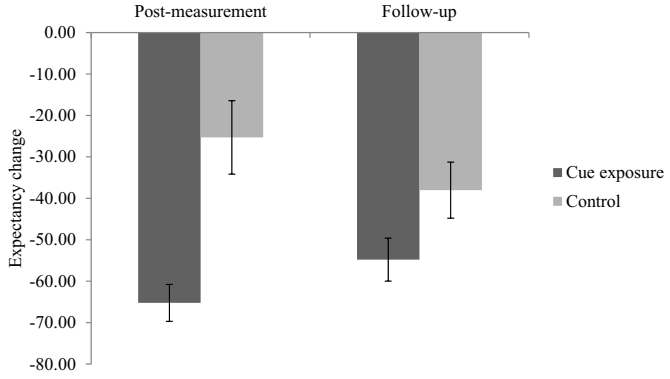

Fig. 4. Percentage of change of overeating expectancies relative to pre-measurement per condition (cue exposure, control) per time point (post and follow-up measurement). Error bars represent standard errors of means.

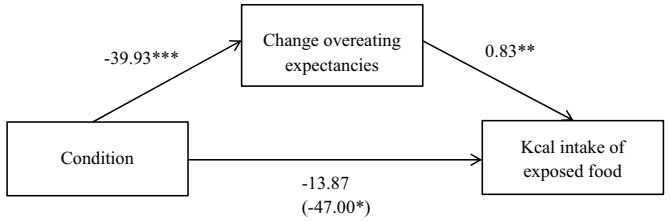

Fig. 5. Indirect of condition (cue exposure scored as 1, control scored as 0) on kcal intake of exposed food on post-measurement via change of overeating expectancies. Note: $*p < .05$, $**p < .01$, $***p < .001$.

less kcal intake (b-path). The direct effect between condition and kcal became non-significant when including expectancy change as a mediator, suggesting full mediation. Expectancy change did not mediate the relationship between condition and weight loss at post-measurement, $ab = 0.06$, $SE = 0.35$, 95% CI[-0.55, 0.87], nor at follow-up, $ab = 0.41$, $SE = 0.35$, 95% CI[-0.02, 1.49]. Expectancy change also did not mediate the relationship between condition and binge eating reduction at follow-up, $ab = 0.21$, SE = 0.44, 95% CI[-0.41, 1.45].

3.4.3. Sleep quality

Sleep quality improved, demonstrated by a significant decrease in mean PSQI score from pre-measurement (cue exposure: $M = 6.26$, $SD = 3.22$; control: $M = 5.77$, $SD = 3.22$) to post-measurement (cue exposure: $M = 5.35$, $SD = 2.85$; control: $M = 4.26$, $SD = 2.51$) and follow-up (cue exposure: $M = 5.72$, $SD = 3.57$; control: $M = 4.86$, $SD = 3.06$) in both conditions, as reflected by a significant main effect of time, $F(2,75.33) = 4.21$, $p = .023$, $\eta_p^2 = 0.089$, and the absence of a main effect of condition, $F(1,43) = 1.07$, $p = .307$, $\eta_p^2 = 0.024$ or a condition*time interaction, $F(2,75.33) = 0.27$, $p = .739$, $\eta_p^2 = 0.006$. Sleep quality at post-treatment, reflecting sleep during the month of the intervention, did not significantly moderate the relationship between condition and kcal intake (personal-exposed food) or weight loss at post-measurement, since both condition*sleep interactions were not significant (weight loss: $F(1,41) = 2.54$, $p = .118$, $\eta_p^2 = 0.058$; kcal personal-exposed food: $F(1,41) = 0.83$, $p = .367$, $\eta_p^2 = .020$). Sleep quality also had no main effect on weight loss, $F(1,41) = 0.38$, $p = .541$, $\eta_p^2 = 0.009$, or kcal intake of the personal-exposed food, $F(1,41) = 0.18$, $p = .674$, $\eta_p^2 = 0.004$.

4. Discussion

This pilot study tested the effects of an eight-session cue exposure therapy aimed at inhibitory learning (Craske et al., 2014) on several outcomes in overweight and obese females, and was compared to an active control intervention. The importance of changing overeating expectancies and habituation of eating desires as mechanisms of change were also investigated. Results showed that participants in the cue exposure condition lost significantly more weight at post-measurement and follow-up than participants in the control condition. Further, participants in the cue exposure condition had a marginally larger binge eating reduction at post-measurement and significantly larger binge eating reduction at follow-up. The cue exposure condition also consumed significantly less kcal of the personal-exposed food at post-treatment and marginally less kcal at follow-up compared to the control condition, though no differences were found between conditions for the personal-non-exposed food or the general-exposed food. The change in expectancies was significantly larger in the cue exposure condition compared to the control condition, and was a significant (full) mediator for kcal consumption of the personal-exposed food, but no mediator for weight loss or binge eating reduction. Habituation of eating desires was not associated with better treatment outcome.

Although participants in the control condition received a lifestyle intervention to achieve weight loss, participants in the cue exposure intervention lost significantly more weight at post-measurement and at follow-up. While previous studies have reported prevention of weight regain after weight loss (Mount et al., 1990), and weight loss in a program containing cue exposure elements (Boutelle, Knatz, Carlson, Bergmann, & Peterson, 2017), this is the first study that found benefits of pure exposure therapy for weight loss. It should be noted that weight loss of two percent is limited in terms of clinical relevance, and the long-term effects are unknown. Given the focus of exposure on relapse prevention, interesting findings on weight loss might actually emerge more on longer term than on short-term. A second important finding is that binge eating frequency strongly decreased in the cue exposure condition. While participants in the exposure condition had a marginally higher binge eating frequency at pre-measurement compared to the control condition, binge eating frequency was reduced to zero in all participants in the exposure condition. Three months after the end of the intervention, participants in the cue exposure condition still had a larger reduction of binge eating frequency compared to control participants. However, it should be noted that these findings could be confounded by the pre-measurement differences, despite randomization; limited improvements in the control condition could be due to a floor-effect. The improvements in the cue exposure condition do indicate that exposure is also promising in reducing binge eating, which is a prevalent problem in obese samples and a central problem in Binge Eating Disorder (BED). Finding beneficial effects of exposure on binge eating is in line with studies in bulimia nervosa patients and overweight children (Boutelle et al., 2011; Jansen et al., 1989, 1992; Martinez-mallen et al., 2007; Toro et al., 2003). While replication of the current results is necessary, a next interesting step would be to test exposure therapy in eating disorder patients, such as BED. It would also be useful to compare exposure therapy to other treatments such as cognitive therapy, as Cognitive Behavioural Therapy (CBT) is the current standard for eating disorders, including exposure elements in the behavioural part (NICE, 2017).

Considerably less kcal consumption of the personal-exposed food after exposure replicates findings of previous cue exposure studies (Schyns et al., 2016; Schyns, Roefs, et al., 2018; Schyns, van den Akker et al., 2018). However, no significant differences were found on kcal consumption of the personal-non-exposed food, or the general-exposed food. Regarding the general-exposed food, a possible explanation is that although the food item was selected as most palatable from a list of ten items, it rarely occurred that participants also mentioned this specific food during the CS-US interview. In other words, although participants rated these items as palatable; it is possible that these were not their 'problematic' foods in daily life and, therefore, did not lead to important learning experiences. So it seems to be important to include *personalized* foods in exposure sessions that are associated with loss of control and not snack foods in general. We did not find generalization; participants did not eat less of the tasty food item they were not exposed to. So even

after eight exposure sessions and an average of 25 homework exposure exercises, participants do not transfer the inhibitory learning experiences to non-exposed foods. Therapists should therefore include as many problematic foods during exposure sessions as possible.

A surprising finding was that we generally failed to induce cue reactivity during the reactivity task in the measurements, which was in sharp contrast with the cue-induced eating desires during exposure sessions. It is possible that participants suppressed their hedonic responses during the measurements. Recently, attention has been paid to palatable versus health 'mindsets', as these might explain inconsistent findings concerning reward responses to food cues in obese individuals (Roefs, Houben, & Werthmann, 2015). Specifically, certain contexts/cues induce a health mindset, for example being in a hospital might facilitate thinking about the health effects of eating the food instead of thinking about its tastiness, thereby decreasing eating desires. Other contexts might induce a hedonic mindset, for example thinking about the delightful taste of chocolate, and increase eating desires. The current measurement context might have triggered a health mindset, causing cue reactivity responses to be dampened during the measurements.

Habituation of cue reactivity and change in overeating expectancies were investigated as mechanisms of change during therapy. Although eating desires indeed habituated within (WSH) and between (BSH) exposure sessions, no correlations were found between WSH/BSH and significant treatment outcomes, with one exception: WSH did significantly correlate with weight loss at post-measurement, in the direction that more WSH was related to *less* weight loss. Overall, it can be concluded that larger habituation was not predictive of better treatment outcome, which is a replication of previous cue exposure studies (Schyns et al., 2016; Schyns, Roefs, et al., 2018) and in line with findings in anxiety exposure studies (Craske et al., 2008). With regard to the change in overeating expectancies, it was found that reductions of CS-US expectancies after the intervention were stronger in the exposure versus control condition. Expectancy change was found to be a significant and full mediator between condition and kcal intake of the exposed food item, but did not serve as a mediator for weight loss and binge eating reduction. The finding that expectancy change was important for kcal intake is interesting and in line with previous research (Schyns et al., 2016), suggesting that the violation of expectancies indeed is an important target for cue exposure sessions. While it is indeed possible that reductions of expectancies in the present study were due to expectancy violation during sessions, expectancy violation was not actually measured: violation of overeating expectancies should be measured during exposure sessions instead of pre and post-treatment. Although a recent cue exposure study suggested that targeting expectancy violation was not superior to targeting habituation of eating desires (Schyns, van den Akker et al., 2018), further well-powered studies with sufficient within-session measurements are needed on the importance of expectancy violation as exposure's treatment target. Moreover, while several techniques were included in the present exposure protocol to enhance inhibitory learning, no separate investigations were performed to study to importance of every separate technique, which is an interesting and important challenge for future studies. One specific technique that might deserve more attention is the implementation of occasional US reinforcements during exposure. In the present study, small bites were used as occasional reinforcements, while it can be argued that these did not represent the entire USs of overeating and losing control. For future studies, it would be highly interesting and important to investigate larger food portions as occasional reinforcements (i.e., portions representative of an actual binge or overeating episode), although it might be a challenge to also induce a sense of loss of control. And although sleep quantity and quality has shown to be important for consolidation of CS–noUS memories (Pace-Schott, Rubin, et al., 2015; Pace-Schott, Germain, et al., 2015), sleep quality did not moderate outcomes in the present study.

Limitations of this pilot study should be noted. First, the sample size was small, posing a general problem for the reliability of study findings and the risk of type II errors. Interpretations of the study effects should be done with caution and replication of the findings is necessary. Further, the three-month follow-up period was limited: studying long-term exposure effects on overeating and weight (cycling) after one to several years is warranted for the utility for clinical practice. While including the lifestyle control condition is a strength of the study, the face-to-face contact frequency was not equal in both interventions due to feasibility constraints. Additionally, females were investigated in the present study, limiting the generalizability to males.

5. Conclusions

Cue exposure therapy targeting inhibitory learning effectively reduced body weight, binge eating frequency and snacking behavior of exposed foods in overweight and obese females. Although habituation of eating desires did occur during cue exposure, habituation was not related to better treatment outcome. Instead, the current data suggest that exposure sessions should target individual CS-US expectancies, as the change in expectancies was related to better treatment outcome.

Conflict of interest

The authors report no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jbtep.2019.01.005.

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