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Beating uncontrolled eating:  
Training inhibitory control to reduce food intake and food cue sensitivity

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## Abstract

In our food-rich environment we must constantly resist appealing food in order to maintain a healthy lifestyle. Previous studies have found that food-specific inhibition training can produce changes in eating behaviour, such as a reduction in snack consumption. However, the mechanisms that drive the effect of inhibition training on eating behaviour remain unknown. Identifying the mechanism underlying food-specific inhibition training could lead to more targeted training interventions increasing the potential efficacy of such interventions. In the current study, we investigated directly whether training-induced effects on inhibitory control might underlie the predicted change in eating behaviour. Healthy individuals who scored high on uncontrolled eating were randomly assigned to receive six online training sessions over six consecutive days of either food-specific response inhibition training (active group;  $n = 21$ ) or response inhibition training without food stimuli (control group;  $n = 20$ ). We measured pre- and post-training inhibitory control in the context of food and food cue sensitivity, as well as food consumption in a bogus taste test. As expected, food-specific inhibition training decreased snack consumption in the bogus taste test relative to control training. However, the active training did not improve inhibitory control towards food, nor did it reduce food cue sensitivity above and beyond the control training. Future studies are needed to investigate the potential underlying mechanism of food-specific inhibition training, as it remains unclear what drives the reliable effect on eating behaviour.

**Keywords:** Cognitive training, response inhibition, food cue sensitivity, overeating, self-control, go/no-go

We are living in an obesogenic environment where we are constantly confronted with advertisement for foods, and overeating of unhealthy foods is an important contributor to the rising levels of obesity (Hill, Wyatt, Reed, & Peters, 2003). Although almost everyone overeats on occasion, some people overeat on a more regular basis, despite efforts to resist overeating or attempts to make healthier food choices. Uncontrolled eating refers to a tendency to overeat, accompanied by feelings of being out of control (Anglé et al., 2009), and is a characteristic of various eating disorders, such as bulimia nervosa and binge eating disorder (American Psychiatric Association, 2013) as well as obesity (Cornelis et al., 2014).

An important factor in regulating eating behaviour and resisting palatable food is inhibitory control (i.e. response inhibition): an executive function that is required to inhibit impulsive responses so that behaviour can be selected that is consistent with one's standards and (long-term) goals (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). Individuals with weaker inhibitory control are more often overweight or obese (Guerrieri, Nederkoorn, & Jansen, 2008; Nederkoorn, Breat, Van Eijs, Tanghe, & Jansen, 2006; Nederkoorn, Guerrieri, Havermans, Roefs, & Jansen, 2009; Nederkoorn, Jansen, Mulken, & Jansen, 2007) and their dieting is more often unsuccessful compared to individuals with stronger inhibitory control (Jansen et al., 2009). Reduced inhibitory control has also been directly related with increased food intake in the lab (Guerrieri et al., 2007), especially in non-dieters (Guerrieri, Nederkoorn, Schrooten, Martijn, & Jansen, 2009). Although the link between behaviourally measured inhibitory control and food intake (in the lab) is not always replicated, there is more consistent evidence for self-reported increased impulsivity and food intake (Guerrieri, Nederkoorn, & Jansen, 2007; Guerrieri et al., 2008).

Another important factor in regulating eating behaviour and resisting palatable food is food reward sensitivity: the degree to which neurological reward responses to food cues elicit

the motivation to eat (Berridge, Ho, Richard, & DiFeliceantonio, 2010). Food reward sensitivity has been found to predict food intake (Lawrence et al., 2012), weight gain (Demos, Heartherton, & Kelley, 2012), obesity (Stice, Spoor, Bohon, Veldhuizen, & Small, 2008) and bulimia nervosa (Brooks et al., 2011). However, findings by Lawrence et al. (2012) suggest that individual differences in inhibitory control may moderate the impact of food reward sensitivity on body mass index (BMI). Lawrence et al. (2012) found that food reward sensitivity was associated with increased BMI in individuals reporting low inhibitory control. Interestingly, food reward sensitivity was negatively correlated with BMI in individuals reporting high inhibitory control.

These findings of previous studies are in line with traditional dual process models. This theoretical model emphasizes the role of inhibitory control whenever there is conflict between two different systems – an impulsive system and reflective system – that operate in parallel and compete for action control (e.g. Kahneman & Frederick, 2002; Strack & Deutsch, 2004). The impulsive system evaluates stimuli in terms of affective and motivational significance, and based on that evaluation predisposes one to either approach or avoid. Unlike the impulsive system, the reflective system is flexible, slow and controlled, and enables personal standards and (long-term) goals to influence decisions and actions via top-down cognitive control (Strack & Deutsch, 2014). Without inhibitory control the reflective system would not be able to overrule the initial response of the more fast-acting impulsive system.

Although a dual-process model might serve as a useful way to describe impulsive and reflective processes, more recent articles argue for a unitary model of action control (Hommel & Wiers, 2017). This unitary model considers all behaviours to be goal-directed. Goals can act as selection criteria that under certain conditions may promote actions that are simple, fast, and overlearned (stimulus-driven actions), or actions that are slow, complex and more

controlled (value-driven actions). As an example, although most individuals would report reluctance to indulge in unhealthy foods in a motivationally ‘neutral’ situation, this intention can weaken when being primed with palatable foods or when hungry. Individual preferences for fast-acting decision making versus slow and controlled decision-making could then translate into individual differences regarding ‘acceptable’ behaviour, such as overeating. Reduced inhibitory control may not imply an inability to translate intentions into action but may relate to a preference for fast-acting (impulsive) decisions based on salient cues (Hommel & Wiers, 2017). Overeating could thus depend on an interaction between individual differences in food reward sensitivity (sensitivity to salient cues) and inhibitory control. For a full discussion of the unitary model, see Hommel and Wiers (2017).

Considering the findings of previous studies that indicate a relationship between inhibitory control and overeating behaviour it should come as no surprise that there has been a growing interest in targeting inhibitory control to help people refrain from overeating. A task that is repeatedly used to measure inhibitory control is the go/no-go task, in which people are instructed to respond as fast as possible to ‘go’ items, and to withhold their response to ‘no-go’ items (Donders, 1969). Researchers have developed food-specific go/no-go training tasks in which unhealthy food items are consistently paired with a no-go cue aiming to improve response inhibition for food stimuli (Houben & Jansen, 2011). Such food-specific go/no-go training has been found to reduce food intake (Adams, Lawrence, Verbruggen & Chambers, 2017; Houben, 2011; Houben & Jansen, 2011, 2015; Lawrence, Verbruggen, Morisson, Adams, & Chambers, 2015b; Van Koningsbruggen, Veling, Stroebe, & Aarts, 2014; Veling, Aarts, & Papies, 2011; Veling, Aarts, & Stroebe, 2013a, 2013b), facilitate weight-loss (Veling, Van Koningsbruggen, Aarts, & Stroebe, 2014; Lawrence et al., 2015b), and reduce self-served food portion sizes (Van Koningsbruggen et al., 2014). Recent meta-analyses found

that inhibitory control training using the go/no-go paradigm has a moderate effect on reducing appetitive behaviours in healthy samples (Allom, Mullan, & Hagger, 2016; Jones et al., 2016; Turton, Bruidegom, Cardi, Hirsch, & Treasure, 2016).

Although these studies and meta-analyses show promising effects, the underlying mechanisms of change for the food-specific go/no-go training remain unclear. Houben and Jansen (2011) postulated that the training strengthens top-down inhibitory control over food-related responses. Besides strengthening top-down inhibitory control, two alternative explanations have been since postulated for how food response inhibition training may reduce food consumption (Veling, Lawrence, Chen, Van Koningsbruggen, & Holland, 2017): 1. training could create automatic ‘bottom-up’ associations between no-go food items and stopping responses (automatic inhibition); 2. training leads to devaluation of food items. This second alternative mechanism is based on Behaviour Stimulus Interaction (BSI) theory. The BSI theory proposes that devaluation of appetitive food stimuli takes place when an initial approach response to appetitive food stimuli is inhibited in order to prevent continuous oscillation between approach and inhibition (Chen, Veling, Dijksterhuis, Holland, 2016).

Given the aim of the training, the most obvious mechanism of change would be that the training strengthens top-down inhibitory control over food-related responses. Veling et al. (2017) have argued that this is unlikely as the training task is very easy and a type of training that is considered more demanding for top-down control, the stop-signal training, is generally less effective (Allom et al., 2016; Jones et al., 2016). However, we cannot rule out this proposed mechanism, because none of the aforementioned training studies measured transfer from the training task to an inhibitory control task to determine if inhibitory control (for food) improved. Nor did they test whether a change in inhibitory control was underlying the change in eating behaviour. This proposed mechanism is thus yet to be experimentally demonstrated.

The alternative mechanism that training increases ‘automatic inhibition’ is often hard to disentangle from the suggestion that training increases top-down inhibitory control. It is possible to look at a slowing of reaction times for responding to trained no-go items when these are presented on go trials but this may only occur when attention to these no-go items is increased during training (Veling et al., 2017).

Veling et al. (2017) have argued that so far the most supported mechanism underlying food go/no-go training is a devaluation of food items. Although some studies indeed found that training led to food devaluation when measured with explicit rating scales of food items (Chen et al., 2016), other studies using the Implicit Association Task found no evidence for devaluation of appetitive stimuli (Jones et al., 2016). One study did find that devaluation of no-go food stimuli was related to weight loss after training, but devaluation did not mediate the effect of go/no-go training (Lawrence et al., 2015a). The evidence that food go/no-go training leads to a devaluation of food and underlies the effects of training on food intake thus remain mixed. Therefore, we were interested to test if food specific go/no-go training would reduce food cue induced craving. Unlike explicitly asking individuals to rate food items on a visual analogue scale (Chen et al., 2016), this measures individual’s craving response to food items and could thus be seen as a physical equivalent of food evaluation or the evaluated incentive value of food.

Investigating the underlying working mechanism of the food-specific go/no-go training is theoretically valuable as it will allow us to increase our understanding of the cognitive processes that contribute to food intake and overeating. This could further support models of uncontrolled eating (and binge eating) that propose a central role for inhibitory control or suggest the need for fine-tuning such models by incorporating other processes (e.g. food cue sensitivity or food evaluation). Moreover, improving our understanding of the

working mechanisms of food-specific go/no-go training will ultimately have clinical benefits as it allows for development of more sophisticated cognitive training protocols (e.g. as add-on to other treatment of binge eating or obesity) targeting specific processes to increase effectiveness on reducing unhealthy food intake.

### **Current Study**

The current study aimed to investigate the effect of 6 sessions of food-specific inhibition training on eating behaviour in a sample of healthy individuals who scored high on uncontrolled eating. Participants either received an active training, that is, the food-specific inhibition training or a control training that was equal to the active condition except that only non-food stimuli were used. By adopting an ‘active’ control condition we aimed to equalize training elements such as cognitive effort over conditions. Furthermore, this control condition allowed us to investigate whether training needs to include behaviour-specific stimuli in order to achieve behaviour change or whether training of general inhibitory control is sufficient to improve outcomes in a specific domain. During a baseline and post-training test session we measured inhibitory control in the context of food and food cue sensitivity. Additionally, in the post-training session we measured food consumption with a bogus taste test.

To investigate possible underlying working mechanisms of the food-specific inhibition training, we first examined whether inhibition training modifies response inhibition for food, by measuring near transfer of training effects to a food go/no-go measure of inhibitory control. This was done to test the underlying assumption that training strengthens response inhibition towards unhealthy food. We expected that the active training group would show a greater improvement in response inhibition towards food items compared to the control group. Next, we examined whether inhibition training influenced food cue sensitivity (i.e. far transfer), using a cue reactivity paradigm previously used by Brockmeyer, Hahn, Reetz,



Schmidt, and Friederich (2015). We expected that the active training group would show a greater reduction in cue-induced food craving from baseline to post-training session compared to the control group.

Finally, to investigate the effect of response inhibition training on eating behaviour we assessed snack consumption in a bogus taste test (Houben, 2011; Lawrence et al., 2015a, 2015b). Based on previous findings, we expected that the active training group would consume less food as compared to the control group. In order to take into account inter-individual differences in training effectiveness, and to directly test whether changes in inhibitory control for food and food cue sensitivity were related to a change in eating behaviour, we also explored whether these changes across training were related to food consumption in the taste test. To summarize, we tested three hypotheses: 1) the active group will show a greater improvement in response inhibition for food items compared to the control group; 2) the active group will show a greater reduction in cue-induced food craving from baseline to post-training session compared to the control group and, 3) the active group will consume less food in the bogus taste test as compared to the control group.

## Method

### Participants

An online pre-screening questionnaire was completed by 221 participants, 50 of whom met the eligibility criteria and were invited to participate. We selected a sample that scored high ( $\geq 20$ ) on the uncontrolled eating subscale of the Three Factor Eating Questionnaire-R18 (TFEQ-R18; Karlsson, Persson, Sjöström, & Sullivan, 2000). Nine participants no longer met this inclusion criterion when measured at baseline and therefore did not continue with the study beyond the first test session. The final sample therefore consisted of 41 participants of which 31 were female. Age ranged from 18 to 34 years ( $M = 22.59$ ;  $SD = 3.98$ ). The cut-off

score for the TFEQ-R18 was determined based on a large community sample from Oxford by taking the upper tertial (unpublished data). Participants were excluded if they revealed any of the following: specific diets (vegan, vegetarian, and/or diabetes); food allergies for foods used in the taste test (chocolate and crisps); BMI < 18.5 or > 30.03; currently being on a diet with the aim to lose weight; having sought professional help in the last six months for an eating disorder or other mental health problems. Participants were randomly assigned to either the active (N = 21) or the control group (N = 20). Participants were reimbursed for their time. This study was approved by the University of Oxford Central University Research Ethics Committee (R25997/RE005).

## Measurements

**Pre-screening questionnaire.** The pre-screening questionnaire assessed the inclusion/exclusion criteria by asking about specific diets, food allergies, being on a diet with the aim to lose weight, and whether individuals sought professional help in the last six months for an eating disorder or other mental health problems. To calculate BMI, height and weight were asked. Additionally, participants completed the 9 items belonging to the uncontrolled eating subscale from the TFEQ-R18.

## Pre- and post-assessment questionnaires.

**Three Factor Eating Questionnaire-R18 (TFEQ-R18).** Three dimensions of human eating behaviour were measured using the TFEQ-R18, namely, restrained eating, uncontrolled eating, and emotional eating (Karlsson et al., 2000). The TFEQ-R18 contains 18 items. An example item of the uncontrolled eating subscale is “Sometimes when I start eating, I just can’t seem to stop”. Responses are given on a 4-point Likert scale (score range: restrained 6-

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<sup>3</sup> When we measured participants’ height and weight ourselves in the baseline session, one participant had a BMI under and another participant above our BMI inclusion range (BMI’s of 18.3 and 31.30). We decided not to exclude these participants due to their BMI’s being extremely close to the cut-off scores.

24; uncontrolled 9-36; emotional 3-12) measuring the frequency of a certain behaviour or how true a statement is for the participant. Higher scores indicate more of the unhealthy eating behaviour. Psychometric properties indicate that the measure is valid and reliable in a sample with a varying range of body weights (Anglé et al., 2009). The internal consistencies of the TFEQ-R18 subscales in the current sample were acceptable (restrained eating:  $\alpha = .77$ ) and good (uncontrolled eating:  $\alpha = .81$ ; emotional eating:  $\alpha = .88$ ).

***Binge Eating Scale (BES)***. Binge eating behaviour was measured with the BES (Gormally, Black, Daston, & Rardin, 1982). The BES contains 16 items reflecting key behavioural and cognitive/affective symptoms of binge eating. Each item contains three to four numbered statements with the numerical value reflecting the severity (total score range 0-32), with higher scores indicating more severe binge eating symptoms. Psychometric properties indicate that the measure is valid and reliable (Gormally et al., 1982). The internal consistency of the BES in the current sample was good ( $\alpha = .83$ ).

***Short version of the UPPS-P (SUPPS-P)***. Impulsivity was measured with the SUPPS-P Impulsive Behaviour Scale (Lynam, 2013). The SUPPS-P contains 20 items, separated into five subscales that each contain four items: negative urgency, (lack of) premeditation, (lack of) perseverance, sensation seeking, positive urgency. Items are rated on a 4-point scale ranging from 1 ('strongly agree') to 4 ('strongly disagree'; total score ranging from 20-80), with higher scores indicating more impulsive behaviour. Psychometric properties indicate that the measure is valid and reliable (Cyders, Littlefield, Coffey, & Karyadi, 2014). The internal consistency of the UPPS-P subscales in the current sample were questionable (negative urgency:  $\alpha = .64$ ; perseverance:  $\alpha = .68$ ), acceptable (sensation seeking:  $\alpha = .73$ ), and good (premeditation:  $\alpha = .84$ ; positive urgency:  $\alpha = .81$ ).

***Food Craving Questionnaire – Trait Version Revised (FCQ-T-r)***. Trait food craving

was measured with the FCQ-T-r (Meule, Hermann, & Kübler, 2014). The FCQ-T-r contains 15 items. Responses are given on a 6-point Likert scale ranging from 1 ('never') to 6 ('always'; total score ranging from 15-90), with higher scores indicating more severe trait food craving. The FCQ-T-r constitutes a valid and reliable self-report measure (Meule et al., 2014), and the internal reliability of the FCQ-T-r was excellent in the present sample ( $\alpha = .94$ ).

***Food Craving Questionnaire – State version (FCQ-S).*** State food craving was measured with the FCQ-S (FCQ-S; Cepeda-Benito, Gleaves, Williams, & Erath, 2000). The FCQ-S contains 15 items, which are divided across five dimensions (intense desire to eat, anticipation of positive reinforcement, anticipation of relief from negative states, lack of control over eating, and craving as a physiological state). Participants respond on a 5-point Likert scale ranging from 1 ('strongly disagree') to 5 ('strongly agree'; total score range of 17-75), with higher scores indicating higher state food craving. Psychometric properties indicate that the measure is valid and reliable (Cepeda-Benito et al., 2000). The internal consistency of the FCQ-S in the current sample as measured on the different time points was excellent ( $\alpha = .91 - .93$ ).

***Grand Hunger Scale.*** State hunger was measured using the Grand Hunger Scale (Grand, 1968). The self-report measure contains two open-ended questions recording length of time since the participant ate, and length of time until they expect to eat again. It also records how hungry participants are at the moment on a 7-point Likert scale (1 'not hungry at all' – 7 'Extremely hungry'), and how much of their favourite food they could eat at the moment on a 6-point Likert scale (1 'none at all' – 6 'as much as I could get').

**Training task.** The online training was a modified go/no-go task to train response inhibition to high calorie food-related information in the active training group, while

the control group received a control version of the task only containing non-food stimuli (based on Lawrence et al., 2015a). No food stimuli were used in the control task since this may inadvertently increase approach behaviour towards food in control participants. On each trial a rectangle frame was presented in which a stimulus would appear for 1250 ms on the left or right-hand side within the frame, followed by a 1250 ms inter-stimulus interval (see Figure 1). When the rectangle frame was normal (go-trial), participants had to press a button as quickly and accurately as possible to indicate the side of the stimulus presentation (left arrow for left and right arrow for right). When the rectangle frame was thick, participants had to withhold their response (no-go trial). Feedback on accuracy and mean RT for go trials were presented at the end of each block to increase motivation. Each training session consisted of six blocks of 32 trials and took approximately 10 minutes to complete.

Stimuli in the active training condition consisted of 16 food and 16 non-food filler stimuli of clothes. Of the food stimuli eight depicted healthy food items (fruit, vegetables, and rice cakes) and eight depicted frequently consumed high calorie food items (greater than 4 kcal/g; biscuits, chocolate, crisps, and cakes). Stimuli in the control training condition consisted of 16 household objects (furniture, home tools, gardening tools), and the same 16 filler stimuli of clothes as in the active training condition. In the active training condition, high calorie food stimuli were always paired with no-go trials (48 high calorie food no-go trials per training session), whereas healthy foods were always paired with go-trials (48 healthy food go-trials per training session). The filler stimuli of non-food items (clothes) were equally associated with go and no-go trials (48 go and 48 no-go trials per training session), resulting in 50% no-go trials overall. The different trial types appeared in equal numbers within each block. Similarly, in the control task, the household stimuli of DIY tools, gardening tools and stationery were always paired with no-go trials (48 trials), whereas

stimuli of electrical items, furniture, and buckets were always paired with go-trials (48 trials). The filler stimuli of clothes were equally associated with go and no-go trials (48 go and 48 no-go trials per training session), and aimed to make the association between the stimuli and their correct response less obvious, to enhance learning in the automatic, associative system, instead of the explicit, rule-based system (Lawrence et al., 2015a). Adding filler stimuli with unpredictable correct responses also makes the task more challenging and engaging. The food and non-food stimuli were selected from the food.pics database (see [www.food-pics.sbg.ac.at](http://www.food-pics.sbg.ac.at); Blechert, Meule, Busch, & Ohla, 2014). The healthy food stimuli and the unhealthy food stimuli in the active training were matched for valence and arousal. In the control version, the household stimuli paired with no-go trials and the household stimuli paired with go-trials were matched for valence and arousal. The filler stimuli of clothes and household objects were the same stimuli as those previously used in a study by Lawrence et al. (2015a). The training task was administered online using the Inquisit 4 Millisecond Web player (Inquisit, Millisecond Software Seattle, WA).



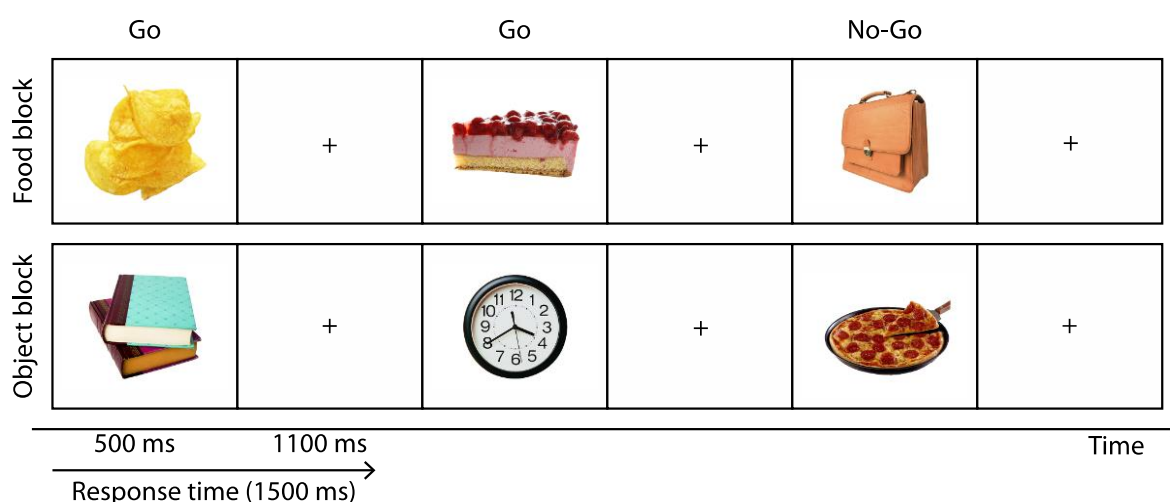
*Figure 1.* [Two columns] Schematic illustration of the active and control training task. Participants had to indicate the side of the stimulus presentation when the stimulus was depicted in a normal frame (left arrow for left and right arrow for right). When the stimulus was depicted in a thick frame, participants had to withhold their response. Healthy food (active task) and electrical items, furniture, and buckets (control task) were always presented on go trials. Unhealthy food (active task) and DIY tools, gardening tools and stationery (control task) were always presented on no-go trials. Filler images of clothes (active and control task) were associated with no-go signals 50% of the time.

**Go/no-go task.** Before and after the 6 training sessions, training-related changes in food-related inhibitory control were assessed using a food-specific go/no-go task (based on Batterink et al., 2010; Kullman et al., 2014), see also Figure 2. To evaluate changes in food-related inhibitory control we looked at the commission error rate (i.e. number of responses to no-go trials divided by total number of no-go trials) as a reduction in commission errors over time is thought to reflect an improvement in response inhibition for no-go stimuli (Veling et al., 2017).

In the food-specific go/no-go task participants had to pay attention to a series of food and non-food stimuli presented in the centre of the screen. The 25 food stimuli and 25 non-food stimuli were selected from the food.pics database (Blechert et al., 2014), but none of the stimuli were identical to the ones used in the training task to increase internal and external validity (Batterink, Yokum, Stice, 2010; Kullman et al., 2014). Food and non-food stimuli were matched on brightness, complexity, valence, and arousal. For the food stimuli we selected only high calorie foods (e.g. donuts, chocolate, crisps, cakes, and fast food). For the non-food stimuli we selected a variety of objects (e.g. clock, book, bag, and candle). The task was presented using E-Prime 2.0 software (Psychology Software Tools, Pittsburgh, PA, USA) on a 21-inch monitor, viewed from a distance of approximately 65 cm.

In each trial the stimulus was presented for 500 ms and was then replaced by a fixation cross for 1100 ms. While the stimulus disappeared after 500 ms, participants had 1500 ms to give a response. Participants were instructed to press the space bar as quickly as possible in response to ‘go’ stimuli and withhold their response to ‘no-go’ stimuli. The task consisted of four blocks of 100 trials (400 trials in total). In each block there were 75% go-trials and 25% no-go-trials in order to develop a pre-potent response pattern. The go and no-go trials were

presented in a pseudo randomized order with a no-go trial appearing equally often after 1, 2, 3, 4, or 5 go trials. There were two types of task blocks, one in which the food stimuli were assigned to go trials and non-food stimuli to no-go trials, and one in which the stimuli had the reverse status. The different type of task blocks alternated in order (e.g. ABAB). The type of block with which participants started was counterbalanced across participants. Before the start of each block, an instruction indicated which type of stimuli the participant had to respond to (i.e. which category of stimuli was the go stimulus). Before the start of the task, participants first completed two practice blocks (8 trials of each block version) to ensure they understood the task.



*Figure 2.* [Two columns] Schematic illustration of the go/no-go task and the two different types of blocks. In the food block, food stimuli were assigned to go trials and non-food stimuli to no-go trials. In the object block, the stimuli had the reverse status.

**Food cue sensitivity.** Before and after the training sessions, cue-induced food craving was assessed with the Food Challenge task (e.g. Brockmeyer et al., 2015; Van den Eynde et al., 2010). Participants first rated their current state level of food craving with the FCQ-S (Cepeda-Benito et al., 2000), after which they were presented with a 5-minute video clip of highly appetite-inducing foods (Brockmeyer et al., 2015). Directly after watching the video state food craving was assessed again, which allowed us to assess the change in state craving



in response to food cues.

**Taste test.** To covertly measure snack consumption, a bogus taste test was given at the end of the post-training test session. Participants were presented with 100gr. crisps (Tesco ready salted crisps) and 200gr. of chocolate (Cadbury milk chocolate buttons). These quantities were selected because they appeared as similar portions when presented in plastic bowls. Participants were asked to taste the products and answer rating scales about them. They were instructed that they could consume as much or little as they wished to complete the taste test. The participants were given 10 minutes to complete the taste test, during which the researcher left the room. The rating scales consisted of 9-point Likert scales about the sweetness, sourness, saltiness, bitterness, and palatability of the snacks. Separate Likert scales were completed for the crisps and chocolate. It also included open-ended questions measuring the usual frequency of consumption of the snacks per week (i.e. for crisps and chocolate separately). After the test session, when the participant had left, snack consumption was measured in grams.

### **Procedure**

Participants were recruited via online and printed advertisements distributed around the University of Oxford. To assess eligibility all participants completed an online screening questionnaire. Those who met the eligibility criteria were invited to participate. Participants attended two test sessions of 1.5 hrs each and completed six computer-based training sessions at home, between the two test sessions. Participants were asked not to eat food for at least 2 hrs before the start of the two test sessions in the department to ensure a pre-meal state. On arrival at the first session, participants completed the consent form and measurements of height, weight, waist, and hip circumference. Subsequently, participants completed the pre-assessment questionnaires, the food-based go/no-go task and thereafter the food cue

sensitivity task<sup>4</sup>. At the end of the first test session, participants received information regarding the online training, but were kept naïve as to the purpose of the training. All participants were asked to complete six training sessions across six consecutive days, and to come back for the second test session on the seventh day. Participants were also asked to, when possible, complete each training session every day at the same time in a place where they would not be disturbed. Furthermore, participants received instructions on the training task (e.g. when the rectangle frame is normal they have to respond as quickly and accurately as possible to indicate the side of the stimulus presentation, etc.) and completed one practice block of 32 trials to ensure that they understood the task. Daily training reminders were sent to the participants via e-mail to promote compliance. Participants were blind to intervention condition.

During the second test session, measurements of height, weight, waist and hip circumference were taken again. Participants also completed the Grand Hunger Scale. They then repeated the go/no-go task and the food cue sensitivity task. The test session ended with the bogus taste test, an awareness check about the training task (see supplementary material for the task awareness questions), and a debriefing.

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<sup>4</sup> Participants also completed a probabilistic monetary reward task during the first and the second test-session. However, this task is not used to answer our research question and is therefore not reported here.

Table 1

*Participant characteristics.*

|   | Control group<br><i>M (SD)</i> | Active group<br><i>M (SD)</i> | Range Control | Range Active | <i>F</i> -value <sup>a</sup> ( <i>p</i> ) |
|---|--------------------------------|-------------------------------|---------------|--------------|---|
| Age   | 22.20 (4.05)                   | 22.95 (3.97)                  | 18-34         | 18-32        | 0.36 (.551)                               |
| Sex (% female)                                      | 70                             | 81                            | N/A           | N/A          | 0.67 (.484)                               |
| Years of education                                  | 15.90 (2.32)                   | 16.62 (2.77)                  | 12-20         | 11-22        | 0.81 (.373)                               |
| Baseline BMI (kg/m <sup>2</sup> )                   | 22.75 (2.50)                   | 22.26 (3.55)                  | 19.65-28.92   | 18.34-31.31  | 0.32 (.577)                               |
| Baseline waist-hip ratio                            | 0.71 (0.16)                    | 0.74 (0.05)                   | 0.67-0.84     | 0.66-0.88    | 0.53 (.472)                               |
| Binge eating  | 10.60 (5.95)                   | 11.62 (5.98)                  | 4-23          | 3-23         | 0.30 (.588)                               |
| Trait food craving                                  | 48.40 (10.84)                  | 51.43 (12.05)                 | 30-67         | 27-72        | 0.71 (.403)                               |
| Impulsivity - (Lack of) perseverance                | 6.80 (1.80)                    | 7.14 (2.08)                   | 4-10          | 4-13         | 0.32 (.576)                               |
| Impulsivity - (Lack of) premeditation               | 7.25 (2.51)                    | 7.71 (2.69)                   | 4-12          | 4-14         | 0.33 (.571)                               |
| Impulsivity - Positive urgency                      | 7.10 (2.99)                    | 7.67 (2.37)                   | 4-14          | 4-12         | 0.45 (.504)                               |
| Impulsivity - Negative urgency                      | 9.10 (2.75)                    | 10.00 (2.17)                  | 5-15          | 6-16         | 1.36 (.250)                               |
| Impulsivity - Sensation seeking                     | 12.10 (3.02)                   | 10.29 (2.47)                  | 5-16          | 6-16         | 4.44 (.042)                               |
| TFEQ - Restraint eating                             | 14.60 (4.12)                   | 14.33 (3.60)                  | 8-22          | 7-20         | 0.05 (.826)                               |
| TFEQ - Uncontrolled eating                          | 25.00 (3.33)                   | 25.14 (3.55)                  | 20-31         | 20-34        | 0.02 (.895)                               |
| TFEQ - Emotional eating                             | 8.05 (2.09)                    | 8.38 (2.50)                   | 5-12          | 3-12         | 0.21 (.649)                               |
| State hunger 1 <sup>st</sup> session                | 3.70 (1.13)                    | 3.67 (1.28)                   | 2-6           | 1-5          | 0.01 (.930)                               |
| State hunger 2 <sup>nd</sup> session                | 3.35 (1.14)                    | 3.00 (1.67)                   | 1-6           | 1-6          | 0.51 (.440)                               |
| Hours since last meal/snack 1 <sup>st</sup> session | 3.31 (2.72)                    | 3.17 (2.02)                   | 2-12          | 2-12         | 0.04 (.837)                               |
| Hours since last meal/snack 2 <sup>nd</sup> session | 3.70 (3.44)                    | 4.38 (4.25)                   | 1-14          | 2-16         | 0.32 (.577)                               |

*Note.* Control group: *n* = 20, Active group: *n* = 21; BMI = Body Mass Index; TFEQ = Three Factor Eating Questionnaire-R18.

<sup>a</sup> Group difference in sex is a chi-square value.

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**Results****418 Participants**

419 The means and standard deviations of the demographics and questionnaires are  
 420 presented in Table 1. For correlations between all the questionnaires, see supplementary Table  
 421 2. To test for pre-existing group differences, univariate ANOVAs with training group as

between-subject factor were performed (see Table 1). The control group scored higher on sensation seeking at baseline, compared to the active group,  $F(1, 39) = 4.44, p = .042, \eta_p^2 = .10$ . This trait correlates negatively with emotional eating, but did not correlate with any of the other measures (see supplementary Table 1). The groups did not differ significantly on any other baseline traits.

### Training Performance

The final training session was the sixth training session for all participants but two: for one participant it was the fifth (active condition) and for one the seventh session (control condition), resulting in a compliance rate of 98% who completed all six sessions. To assess changes in training performance across the training period we performed mixed design ANOVAs on the proportion of commission errors (i.e. number of false alarms divided by total number of no-go trials) with condition (control, active) as between-subject factor, and time (first, last training session), and stimulus type (filler, non-filler) as within-subject factors. See supplemental material for additional analysis performed on other outcome measures of the training task (e.g. omission errors).

For the commission error analysis, we excluded three cases due to outlying data (i.e. standardized residuals  $>3$  SDs from the mean). The ANOVA yielded a main effect of time,  $F(1, 36) = 4.75, p = .036, \eta_p^2 = .12$ , a marginally significant main effect of stimulus type,  $F(1, 36) = 3.87, p = .057, \eta_p^2 = .10$ , a Time x Stimulus type interaction,  $F(1, 36) = 5.50, p = .025, \eta_p^2 = .13$ , a Time x Condition interaction,  $F(1, 36) = 6.46, p = .016, \eta_p^2 = .15$ , and a Stimulus type x Condition interaction,  $F(1, 36) = 8.59, p = .006, \eta_p^2 = .19$ . There was no main effect of condition or a Time x Stimulus type x Condition interaction effect,  $p > .05$ . Figure 3 graphically displays the change in performance of commission error rate across the training period for the two groups separately.

To follow-up the Time x Stimulus type interaction we performed paired t-tests to compare commission errors at first and last training session for the two stimulus types separately (i.e. regardless of condition). The commission error rate did not significantly decrease for filler stimuli from first session ( $M = 0.04$ ;  $SD = 0.03$ ) to last session ( $M = 0.04$ ;  $SD = 0.03$ ),  $t(37) = -0.37$ ,  $p = .711$ ,  $d = 0.06$ , while it did significantly decrease for non-filler stimuli (i.e. food items in the active training, household items in the control training) from first session ( $M = 0.04$ ;  $SD = 0.03$ ) to last session ( $M = 0.02$ ;  $SD = 0.03$ ),  $t(37) = 3.34$ ,  $p = .002$ ,  $d = 0.54$ . Additionally, a paired t-test was done to compare filler and non-filler stimuli at the two time points separately. There was no significant difference in the rate of commission errors for filler stimuli and non-filler stimuli at first session,  $t(37) = 0.46$ ,  $p = .650$ ,  $d = 0.07$ . However, during the final training session there were more commission errors for filler stimuli than for non-filler stimuli,  $t(37) = -2.77$ ,  $p = .009$ ,  $d = 0.45$ .

To follow-up the Time x Condition interaction we performed a paired t-test to investigate the effect of time on rate of commission errors for the two groups separately (i.e. regardless of stimulus type). The commission error rate did not significantly differ between first session ( $M = 0.04$ ;  $SD = 0.03$ ) and last session ( $M = 0.04$ ;  $SD = 0.02$ ) in the control group,  $t(17) = -0.20$ ,  $p = .842$ ,  $d = 0.05$ . However, the commission error rate did significantly decrease from first session ( $M = 0.04$ ;  $SD = 0.02$ ) to last session ( $M = 0.02$ ;  $SD = 0.02$ ) in the active group,  $t(19) = 4.65$ ,  $p < .001$ ,  $d = 1.04$ . Additionally, an independent t-test was done to compare the control group and the active group at the two time points separately. The commission error rate did not significantly differ between the control and active group at first session,  $t(36) = 0.55$ ,  $p = .584$ ,  $d = 0.18$ . However, the control group made significantly more commission errors than the active group in the last training session,  $t(36) = 2.87$ ,  $p = .007$ ,  $d = 0.96$ .

To follow-up the Stimulus type x Condition interaction we performed a paired t-test to compare rate of commission errors on filler stimuli and non-filler stimuli for the two groups separately (i.e. regardless of training session). The commission error rate did not significantly differ between filler stimuli ( $M = 0.03$ ;  $SD = 0.02$ ) and non-filler stimuli ( $M = 0.03$ ;  $SD = 0.03$ ) in the active group,  $t(19) = 0.16$ ,  $p = .876$ ,  $d = 0.04$ . However, significantly more commission errors were made to filler stimuli ( $M = 0.05$ ;  $SD = 0.03$ ) than to non-filler stimuli ( $M = 0.02$ ;  $SD = 0.02$ ) in the control group,  $t(17) = 3.47$ ,  $p = .003$ ,  $d = 0.82$ . Additionally, an independent t-test was performed to compare the control group and the active group on rate of commission errors for the stimuli types separately. More commission errors were made in the control group compared to the active group for filler stimuli,  $t(36) = 3.00$ ,  $p = .005$ ,  $d = 1.00$ . Rate of commission errors did not differ significantly between the control group and the active group for non-filler stimuli,  $t(36) = -0.23$ ,  $p = .816$ ,  $d = 0.04$ .

**Training awareness.** During the debriefing procedure, 38% of the active participants reported to have noticed that no-go signals were associated with stimuli of high calorie food, whereas 15% of the control participants noticed that specific categories of objects were associated with no-go signals. This proportion of ‘aware’ participants was not significantly higher in the active than control group,  $\chi^2(1) = 2.78$ ,  $p = .159$ . No control participants felt that the training task had influenced their taste test. Only two active participants thought that the task could have possibly influenced the taste test (“I told my friends that I disliked unhealthy foods more in the last few days”; “Maybe, I was thinking about how unhealthy the food was during eating”).

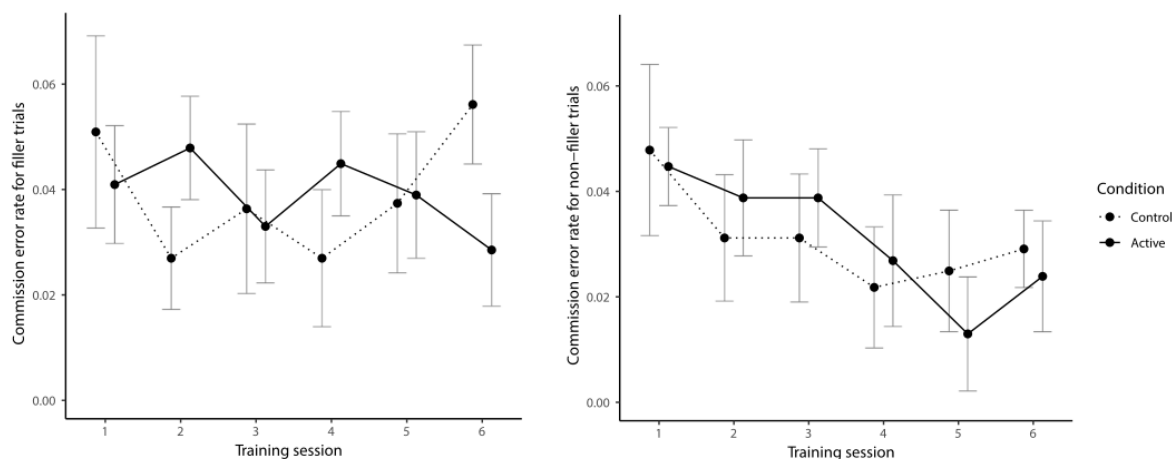
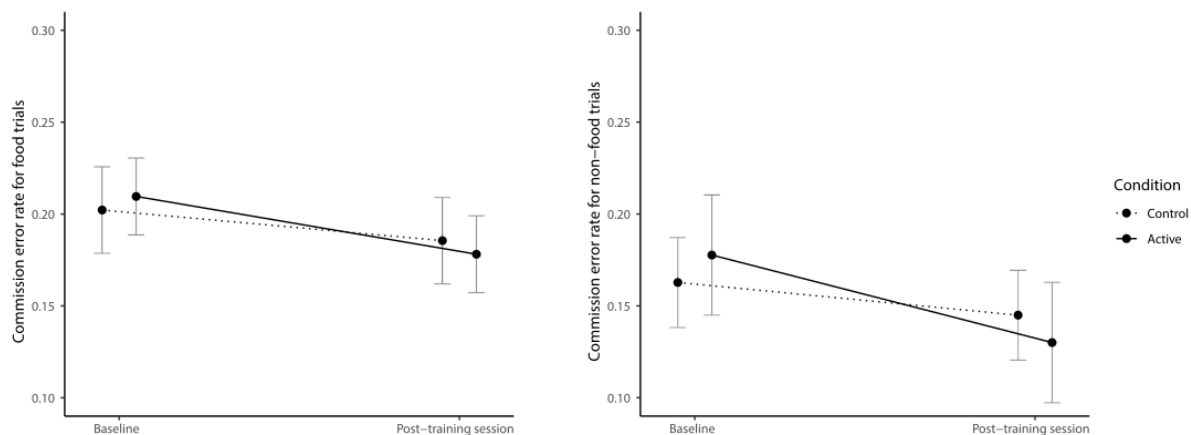


Figure 3. [2 columns] Training performance over time. Change in training performance of commission error rate across the training period per condition for filler trials and non-filler trials separately (left and right panel, respectively). Error bars represent the 95% CI of the mean.

### Near Transfer: Go/no-go task

To assess training-related changes in food-related inhibitory control on the go/no-go task, we performed mixed design ANOVAs with condition (control, active) as between-subject factor, and time (baseline, post-training session) and stimulus type (food, non-food) as within-subject factors on commission error rate (see Figure 4). For the commission errors analyses, we excluded two cases due to outlying data (i.e. standardized residuals  $> 3$  SDs from the mean). The ANOVA on the number of commission errors yielded a main effect of time,  $F(1, 37) = 7.11, p = .011, \eta_p^2 = .16$  revealing that overall more commission errors were made at baseline ( $M = 0.19; SD = 0.09$ ) than at post-training session ( $M = 0.16; SD = 0.08$ ). Furthermore, there was a main effect of stimulus type,  $F(1, 37) = 24.15, p < .001, \eta_p^2 = .40$ , as overall less commission errors were made to non-food stimuli ( $M = 0.16; SD = 0.08$ ) than to food stimuli ( $M = 0.19; SD = 0.09$ ). There was no significant main effect of condition and there were no significant interaction effects, all  $ps > .05$ . See supplemental material for additional analysis performed on other outcome measures of the go/no-go task (e.g. omission errors).



*Figure 4.* [2 columns] Performance at baseline and post-training session. Training-related change of commission error rate from baseline to post-training session per condition for food and non-food trials separately (left and right panel, respectively). Error bars represent the 95% CI of the mean.

### Far Transfer: Cue reactivity

To assess cue-induced food craving, we performed a mixed design ANOVA with condition (control, active) as between-subject factor, and reactivity (before, after craving induction) and time (baseline, post-training session) as within-subject factors. One participant was excluded from the cue reactivity analysis due to a procedure failure that led to missing baseline data. Three participants each had a missing value on the FCQ-S which were replaced with the persons' mean score on the questionnaire. State food craving was effectively manipulated by the cue exposure as shown by a main effect of reactivity,  $F(1, 38) = 53.06$ ,  $p < .001$ ,  $\eta_p^2 = .58$ . There was a significant increase in state food craving from pre- to post-cue exposure across the sessions and groups (see Table 2 for means and standard deviations). There was also a main effect of time as state craving (average across pre- and post-craving induction) was lower in the post-training session, compared to state craving as measured before the training, at baseline session,  $F(1, 38) = 6.23$ ,  $p = .017$ ,  $\eta_p^2 = .14$ . However, training did not influence the effect of reactivity, time, nor their interaction, all  $ps > .10$ .



Table 2  
Cue reactivity.

|         | T1-1<br><i>M (SD)</i> | T1-2<br><i>M (SD)</i> | T2-1<br><i>M (SD)</i> | T2-2<br><i>M (SD)</i> |
|---------|-----------------------|-----------------------|-----------------------|-----------------------|
| Control | 41.61 (10.79)         | 49.95 (11.67)         | 38.21 (10.90)         | 45.61 (10.66)         |
| Active  | 44.48 (10.60)         | 53.57 (8.08)          | 40.19 (12.36)         | 49.14 (13.34)         |

*Note.* Control group:  $n = 19$ , Active group:  $n = 21$ ; T1-1 as measured at baseline before craving induction, T1-2 as measured at baseline after craving induction; T2-1 as measured at post-training session before craving induction; T2-2 as measured at post-training session after craving induction.

### Far Transfer: Taste test

To compare training groups in the bogus taste test (i.e. average consumption of crisps and chocolate in grams), while controlling for state craving, we performed a one-way ANCOVA. State craving, as measured by the FCQ-S administered prior to the bogus taste test, was added as a covariate to control for differences in state craving after the food cue sensitivity task. One participant was excluded from the taste test analysis due to outlying data (i.e. standardized residuals  $> 3$  SDs from the mean). The ANCOVA revealed that the control group consumed significantly more snacks ( $M = 52.90$ ;  $SD = 29.30$ ) than the active group ( $M = 38.40$ ;  $SD = 22.61$ ),  $F(1, 37) = 4.58$ ,  $p = .039$ ,  $\eta_p^2 = .11$ . We further examined whether there were pre-existing group differences in the usual frequency of crisps and chocolate consumption that could have influenced the results of the taste test. An ANOVA was done to compare the control group and the active group on the usual consumption frequency of the two snacks separately. The usual frequency of crisps consumption per week did not significantly differ between the two groups (control:  $M = 1.95$ ;  $SD = 1.64$ ; active:  $M = 2.35$ ;  $SD = 1.42$ ),  $F(1, 38) = 0.68$ ,  $p = .415$ ,  $\eta_p^2 = .01$ , nor was there a significant difference of chocolate consumption between the two groups (control:  $M = 3.80$ ;  $SD = 2.80$ ; active:  $M = 4.95$ ;  $SD = 2.96$ ),  $F(1, 38) = 0.68$ ,  $p = .415$ ,  $\eta_p^2 = .04$ .

Additionally, we explored whether the change in inhibitory control and food cue sensitivity, across groups, was related to food consumption across groups. We focused on the change in inhibitory control for food stimuli only. We performed a simple linear regression analysis on the consumption of snacks. For the change in inhibitory control, we first entered state craving, as measured by the FCQ-S administered prior to the bogus taste test. In a second step we entered the change in inhibitory control across the training period, calculated as a difference score (i.e. rate of the first session subtracted from the rate of the second session). The regression analyses revealed that changes in commission errors over time on the go/no-go task were not directly related to the amount of consumed snacks in the taste test,  $\beta = -0.22$ ,  $t = -1.51$ ,  $p = .139$ ,  $\Delta R^2 = 0.05$ . See supplemental material for additional simple linear regression analyses to check whether the other behavioural measures of the go/no-go task are related to food intake in the taste test. For the change in food cue sensitivity we calculated the change in reactivity. That is, the reactivity in state craving from before the food video to after the food video and how that changed from pre-training to post-training. One participant was excluded from the taste test analysis due to outlying data (i.e. standardized residuals  $> 3$  SDs from the mean). The regression analyses revealed that changes in food cue reactivity over time were not directly related to the amount of consumed snacks in the taste test,  $\beta = -0.10$ ,  $t = -0.62$ ,  $p = .541$ ,  $R^2 = 0.01$ .

## Discussion

This study examined the effect of food response inhibition training on eating behaviour in a sample of individuals scoring high on uncontrolled eating. Specifically, we tested whether food-specific go/no-go training modified response inhibition for food, influenced food cue sensitivity or modified snack consumption. Although we found that food response inhibition training decreased snack consumption in a bogus taste test, the active

training did not improve inhibitory control towards food more than control training, nor did it reduce food cue sensitivity (i.e. cue induced craving). Therefore, the mechanisms of change in reducing food consumption remain unclear.

Our main finding is that food response inhibition training decreased snack consumption in a sample of (healthy) individuals who are relatively high in uncontrolled eating. This is consistent with previous studies that have used the same paradigm (i.e. bogus taste test; Houben, 2011; Houben & Jansen, 2011, 2015), as well as studies that have used other behavioural measures of eating (Lawrence et al., 2015b; Veling et al., 2011) and shows that food-specific inhibition training can influence eating in a sample with a tendency for uncontrolled eating. The consistent findings from a variety of studies gives some confidence that the food response inhibition training can indeed reduce snack consumption and influence eating behaviour. In contrast to previous studies we measured inhibitory control pre- and post-training with a go/no-go task that included blocks in which participants had to respond rapidly to food stimuli. Previous studies have not attempted to measure inhibitory control after training because of a concern that assessing inhibitory control with food-related stimuli after training would reduce any effects of training on a measure of eating behaviour (Veling et al., 2017). If this is the case, however, one could question whether the training is truly valuable if it is so easily influenced by this external factor. Moreover, including the go/no-go task allowed us to measure whether food response inhibition training actually enhanced inhibitory control over food related responses. Despite including a go/no-go task we still found a small effect ( $d = 0.31$ ) of training on snack consumption, indicating that the training had a rather robust effect on snack consumption.

Both training conditions (i.e. active and control) effectively improved participants' performance over time: commission errors for non-filler stimuli decreased significantly in

both groups from first to last training session, and the active group also showed a significant decrease in commission errors over time regardless of stimulus type. Both training conditions thus enhance inhibitory control for the relevant training stimuli. There was however no specific (near) transfer of active training to the food go/no-go task, as training groups showed no difference in change in commission errors for food stimuli. That is, both training groups improved in inhibitory control across the training period, as reflected by a decrease in commission errors at post-training but this was regardless of stimulus type. Generally, inhibitory control was worse for food stimuli, likely because these stimuli are more salient, but this did not change across training nor was this different between training groups.

We compared an active inhibition training with food stimuli to a control condition that also required participants to inhibit their responses but not in the context of food stimuli. Although the advantage of such a control condition is that it equalizes general training effects (e.g. cognitive effort, adhering to a training schedule) and allowed us to investigate whether training needs to include behaviour-specific stimuli, it is also a more conservative test of whether food-specific go/no-go training can increase inhibitory control for food stimuli. The use of our specific control condition could therefore have contributed to this lack of difference between training conditions in terms of change in commission errors for food stimuli. More importantly though, individual differences in the change in performance on the food go/no-go task did not relate to individual differences in snack consumption. Improving inhibitory control would seem the most obvious mechanism underlying effects of food inhibition training on food intake, by promoting more slow and controlled decision-making (value-driven actions) as described in the unitary model of action selection (Hommel & Wiers, 2017). However, based on our findings we suggest that enhanced inhibitory control is most

likely *not* the mechanism underlying the effect of food response inhibition training on eating behaviour, at least not in the current study.

Two alternative explanations for how food response inhibition training may reduce snack consumption have previously been described by Veling et al. (2017): 1. the training might create an automatic bottom-up association between no-go food items and stopping responses; 2. the training might lead to food devaluation. If the first explanation were true, we should have found a decrease in commission errors towards food (similarly as for the top-down inhibitory control account) and a slowing in RT of hits for food stimuli. As we did not find this (see also supplementary material), our data do not support the first alternative explanation. Regarding the second alternative mechanism, although we did not directly test modification of valuation of food with explicit ratings of food items, we did measure individuals' craving reactivity towards food items. Cue induced craving could reflect a physical equivalent of food evaluation. From the perspective of the unitary model of action control (Hommel & Wiers, 2017) this could mean that training works through reducing the tendency to use fast-acting, stimulus-driven decision making based on salient cues.

Both food inhibition training and control training conditions resulted in a reduction in food cue sensitivity after the training. The cause of this effect across both conditions remains unclear. It may simply be due to a testing effect: familiarity with the video may have led to weaker reactivity effects, rather than a training effect. It may also be due to demand characteristics caused by the somewhat obvious manipulation of the food challenge task. Another possible explanation as to why we did not find a training effect on food cue sensitivity might be that the foods depicted in the video were too different from the no-go food items. The video did not only include high calorie foods but showed a variety of foods including normal everyday ingredients (e.g. pasta, bread, potatoes) and healthy foods (e.g.

vegetables, fruit). Perhaps the effect of food inhibition training does not generalize to the extent that it reduces food craving (or food evaluation) in the context of all types of food cues, but only reduces food cue sensitivity for high calorie foods like those used in the training. Additionally, individual differences in the change in food cue sensitivity did not relate to individual differences in snack consumption.

Although our study does not show a different effect of active and control training on food cue sensitivity, nor does the change in food cue sensitivity relate to snack consumption, we suggest that it is worthwhile for future research to investigate whether this is food type dependent, before drawing firm conclusions. We agree with Veling et al. (2017) that devaluation of unhealthy foods is a plausible mechanism underlying the effects of food go/no-go training, but the evidence so far is not satisfactory. Furthermore, it is important to establish to what extent the effects of food inhibition training generalize to other types of food (not used in the training task) as this could limit the efficacy of training and/or indicate that training should be personalized to certain food categories depending on the individual. Such research would be especially relevant if food inhibition training indeed leads to food devaluation and thereby influences eating behaviour.

A further possibility is that the effect of food inhibition training on eating behaviour is due to an exposure mechanism that diminishes learned responses to food in the context of a food cue. During the active training, participants are repeatedly exposed to food items to which they withhold their response. Hence, food cues that were once associated with approach and subsequent intake of food are repeatedly unreinforced during the training. In other words, the approach behaviour towards food becomes detached from the food cue (see also, Jansen, Schyns, Bongers, & Van den Akker, 2016). Although speculative, this account provides a potential direction for future research to explore. For example, investigating the

effects of food inhibition training on approach/avoidance tasks for food could be utilized to test this hypothesis.

Strengths of the present study include the well-controlled design and the fact that we directly investigated the hypothesis that food-specific go/no-go training strengthens inhibitory control. However, the current study also had a number of limitations. First, it has been argued (Veling et al., 2017) that the training is too easy to enhance inhibitory control over food related responses, evident by the low error rate that is associated with the training. Using a more challenging food response inhibition training (e.g. with a lower percentage of no-go trials) might be necessary to consistently modify inhibitory control over approach tendencies towards food. That said, the far transfer effect of inhibitory control training on snack consumption has been repeatedly found with this relatively easy training task. Thus, a relatively easy training task may be effective in influencing eating behaviour due to a mechanism other than inhibitory control. Second, we measured food cue sensitivity with the food challenge task that is susceptible to demand effects, by measuring craving just before and after presentation of a video displaying food stimuli. Most studies investigating food cue sensitivity rely on physiological methodologies such as fMRI with which it is possible to covertly monitor processing of food information. Research efforts should be directed at the development of a more implicit, behavioural measure of food cue sensitivity or cue induced craving that is robust against demand characteristics.

Food-specific go/no-go training has the potential to help people that overeat to (re)gain control over their eating behaviour. However, before we can evaluate the clinical relevance of the training it is important to explore the working mechanism. This will require study designs that directly test whether training effects transfer to other tasks/processes. Although proposed mechanisms like increasing inhibitory control and food devaluation are plausible, the

evidence so far is inconclusive. Future research could test interventions that combine training of potential working mechanisms to influence food consumption, for example to increase inhibitory control combined with more directly targeting devaluation of unhealthy foods. It is possible that single processes cannot fully explain changes in food intake but that multiple processes have an additive or interacting effect increasing the impact on eating behaviour. Furthermore, research should investigate whether response inhibition training also has an influence on eating behaviour in clinical samples of people with binge eating disorder or bulimia nervosa. Additionally, longitudinal studies, preferably with a double-blind design and a large sample, should determine whether there is a long-term effect of training on eating behaviour, and under which conditions such an effect is present. For example, it may be necessary for participants to repeat training at certain intervals for an enduring effect on eating behaviour to develop.

To summarize, we found that food response inhibition training can reduce snack consumption even though the active and control training conditions did not differ in training effects on inhibitory control for food and food cue sensitivity. Based on our findings it seems unlikely that increased inhibitory control or decreased food cue sensitivity explains the effect on eating behaviour (on their own). Thus, it remains unclear what underlying mechanism is responsible for the effect of this form of cognitive training on eating behaviour and further research is needed. Ultimately, that would entail further development of a training protocol that fully utilizes the working mechanism to increase effects of training on uncontrolled eating, which could increase the potential clinical value of such an intervention.



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## References

- Adams, R. C., Lawrence, N. S., Verbruggen, F., & Chambers, C. D. (2017). Training response inhibition to reduce food consumption: Mechanisms, stimulus specificity and appropriate training protocols. *Appetite*, *109*, 11-23. doi:10.1016/j.appet.2016.11.014.
- Allom, V., Mullan, B., & Hagger, M. (2016). Does inhibitory control training improve health behavior? A meta-analysis. *Health Psychology Review*, *10*(2), 168-186. doi:10.1080/17437199.2015.1051078.
- American Psychiatric Association. (2013). Cautionary statement for forensic use of *DSM-5*. In *Diagnostic and statistical manual of mental disorders* (5<sup>th</sup> ed.). Washington, DC: Author. doi:10.1176/appi.books.9780890425596.dsm10.
- Anglé, S., Engblom, J., Eriksson, T., Kautiainen, S., Saha, M-T., Lindfors, P., . . . Rimpelä, A. (2009). Three factor eating questionnaire-R18 as a measure of cognitive restraint, uncontrolled eating and emotional eating in a sample of young Finnish females. *International Journal of Behavioral Nutrition and Physical Activity*, *6*(1), 41. doi:10.1186/1479-5868-6-41.
- 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. doi:10.1016/j.neuroimage.2010.05.059.
- Berridge, K. C., Ho, C. Y., Richard, J. M., & DeFeliceantonio, A. G. (2010). The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Research*, *1350*, 43-64. doi:10.1016/j.brainres.2010.04.003.
- Blechert, J., Meule, A., Busch, N. A., & Ohla, K. (2014). Food-pics: an image database for experimental research on eating and appetite. *Frontiers in Psychology*, *5*. doi:10.3389/fpsyg.2014.00617.

- 741 Brockmeyer, T., Hahn, C., Reetz, C., Schmidt, U., & Friederich, H-C. (2015). Approach bias  
 742 and cue reactivity towards food in people with high versus low levels of food craving.  
 743 *Appetite*, 95, 197-202. doi:10.1016/j.appet.2015.07.013.
- 744 Brooks, S. J., O'Daly, O. G., Uher, R., Friederich, H-C., Giampietro, V., Brammer, M., . . .  
 745 Campbell, L. C. (2011). Differential neural responses to food images in women with  
 746 bulimia versus anorexia nervosa. *PLoS ONE*, 6(7), e22259.  
 747 doi:10.1371/journal.pone.0022259.
- 748 Cepeda-Benito, A., Gleaves, D. H., Williams, T. L., & Erath, S. A. (2000). The development  
 749 and validation of the state and trait Food-Cravings Questionnaires. *Behavior Therapy*,  
 750 31(1), 151-173. doi:10.1016/s0005-7894(00)80009-x.
- 751 Chen, Z., Veling, H., Dijksterhuis, A., & Holland, R. W. (2016). How does not responding to  
 752 appetitive stimuli cause devaluation: Evaluative conditioning or response inhibition?  
 753 *Journal of Experimental psychology*, 145(12), 1387-1701. doi:10.1037/xge0000236.
- 754 Cornelis, M. C., Rimm, E. B., curhan, G. C., Kraft, P., Hunter, D. J., Hu, F. B., & van Dam.  
 755 R. M. (2014) Obesity susceptibility loci and uncontrolled eating, emotional eating and  
 756 cognitive restraint behaviors in men and women. *Obesity*, 22(5), E135-E141. doi:  
 757 10.1002/oby.20592.
- 758 Cyders, M. A., Littlefield, A. K., Coffey, S., & Karyadi, K. A. (2014). Examination of a short  
 759 English version of the UPPS-P Impulsivity Behavior Scale. *Addictive Behaviors*,  
 760 39(9), 1372-1376. doi:10.1016/j.addbeh.2014.02.013.
- 761 Demos, K. E., Heatherton, T. F., & Kelley, W. M. (2012). Individual differences in nucleus  
 762 accumbens activity to food and sexual images predict weight gain and sexual  
 763 behavior. *Journal of Neuroscience*, 32(16), 5549-5552. doi:10.1523/jneurosci.595  
 764 11.2012.

- 765 Donders, F. C. (1969; original work published in 1868). On the speed of mental processes.  
 766 *Acta Psychologica*, 30, 412–431. doi:10.1016/0001–6918 (69)90065–1.
- 767 Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating  
 768 severity among obese persons. *Addictive Behavior*, 7(1), 47-55.  
 769 doi:10.1016/03064603(82)90024-7.
- 770 Grand, S. (1968). Color-word interference: An investigation of the role of vocal conflict and  
 771 hunger in associative priming. *Journal of Experimental Psychology*, 77(1), 31-40.  
 772 doi:10.1037/h0025759.
- 773 Guerrieri, R., Nederkoorn, C., & Jansen, A. (2007). How impulsiveness and variety influence  
 774 food intake in a sample of healthy women. *Appetite*, 48, 119-122. doi:  
 775 10.1016/j.appet.2006.06.004.
- 776 Guerrieri, R., Nederkoorn, C., & Jansen, A. (2008). The interaction between impulsivity and a  
 777 varied food environment: Its influence on food intake and overweight. *International*  
 778 *Journal of Obesity*, 32(4), 708-714. doi:10.1038/sj.ijo.0803770.
- 779 Guerrieri, R., Nederkoorn, C., Schrooten, M., Martijn, C., & Jansen, A. (2009). Inducing  
 780 impulsivity leads high and low restrained eaters into overeating, whereas current  
 781 dieters stick to their diet. *Appetite*, 53, 93-100. doi: 10.1016/j.appet.2009.05.013.
- 782 Guerrieri, R., Nederkoorn, C., Stankiewicz, K., Alberts, H., Geschwind, N., Martijn, C., &  
 783 Jansen, A. (2007). The influence of trait and induced state impulsivity on food intake  
 784 in normal-weight healthy women. *Appetite*, 49, 66-73. doi:  
 785 10.1016/j.appet.2006.11.008.
- 786 Hill, J.O., Wyatt, H.R., Reed, G.W., & Peters, J.C. (2003). Obesity and the environment:  
 787 Where do we go from here? *Science*, 299, 83-855. doi:10.1126/science.1079857.

- 788 Hommel, B., & Wiers, R. W. (2017). Towards a unitary approach to human action control.  
789 *Trends in Cognitive Sciences*, 21(12), 940-949. doi:10.1016/j.tics.2017.09.009
- 790 Houben, K. (2011). Overcoming the urge to splurge: Influencing eating behavior by  
791 manipulating inhibitory control. *Journal of Behaviour Therapy and Experimental*  
792 *Psychiatry*, 42(3), 384-388. doi:10.1016/j.jbtep.2011.02.008.
- 793 Houben, K. & Jansen, A. (2011). Training inhibitory control: A recipe for resisting sweet  
794 temptations. *Appetite*, 56(2), 345-349. doi:10.1016/j.appet.2010.12.017.
- 795 Houben, K. & Jansen, A. (2015). Chocolate equals stop. Chocolate-specific inhibition training  
796 reduces chocolate intake and go associations with chocolate. *Appetite*, 87, 318-323.  
797 doi:10.1016/j.appet.2015.01.005.
- 798 Jansen, A., Schyns, G., Bongers, P., & Van den Akker, K (2016). From lab to clinic:  
799 Extinction of cued cravings to reduce overeating, *Physiology & Behavior*, 162, 174-  
800 180. doi:10.1016/j.physbeh.2016.03.018.
- 801 Jansen, A., Nederkoorn, C., Van Baak, L., Keirse, C., Guerrieri, R., & Havermans, R. (2009).  
802 High restrained eaters only overeat when they are also impulsive. *Behaviour Research*  
803 *and Therapy*, 47(2), 105-110. doi:10.1016/j.brat.2008.10.016.
- 804 Jones, A., Di Lemma, L C. G., Robinson, E., Christiansen, P., Nolan, S., Tudur-Smith, C.  
805 (2016). Inhibitory control training for appetitive behavior change: A meta-analytic  
806 investigation of mechanisms of action and moderators of effectiveness. *Appetite*, 97,  
807 16-28. doi:10.1016/j.appet.2015.11.013.
- 808 Kahneman, D. & Frederick, S. (2002). Representativeness revisited: Attribute substitution in  
809 intuitive judgement. In T. Gilovich, D. Griffin, & D., Kahneman (Eds.), *Heuristic and*  
810 *biases: The psychology of intuitive judgement* (pp. 49-81). New York: Cambridge  
811 University Press.

- 812 Karlsson, J., Persson, L-O., Sjöström, L., & Sullivan, M. (2000). Psychometric properties and  
 813 factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and  
 814 women: Results from the Swedish Obese Subjects (SOS) study. *International Journal*  
 815 *of Obesity and Related Metabolic Disorders*, 24(12), 1715-1725.  
 816 doi:10.1038/sj.ijo.0801442.
- 817 Kullman, S., Giel, K. R., Xiaochen, h., Bischoff, S. C., Teufel, M., Thiel, A., . . . Preissl, H.  
 818 (2014). Impaired inhibitory control in anorexia nervosa elicited by physical activity  
 819 stimuli. *Social Cognitive and Affective Neuroscience*, 9(7), 917-923.  
 820 doi:10.1093/scan/nst070.
- 821 Lawrence, N. S., Hinton, E. C., Parkinson, J. A., & Lawrence, A. D. (2012). Nucleus  
 822 accumbens response to food cues predicts subsequent snack consumption in women  
 823 and increased body mass index in those with reduced self-control. *NeuroImage*, 63(1),  
 824 415-422. doi:10.1016/j.neuroimage.2012.06.070.
- 825 Lawrence, N. S., Verbruggen, F., Morrison, S., Adams, R. C., & Chambers, C. D. (2015b).  
 826 Stopping to food can reduce intake. Effects of stimulus-specificity and individual  
 827 differences in dietary restraint. *Appetite*, 85, 91-203. doi:10.1016/j.appet.2014.11.006.
- 828 Lawrence, N. S., O'Sullivan, J., Parslow, D., Javaid, M., Adams, R. C, Chambers, C. D., . . .  
 829 Verbruggen, F. (2015a). Training response inhibition to food is associated with weight  
 830 loss and reduced energy intake. *Appetite*, 95, 17-28. doi:10.1016/j.appet.2015.06.009.
- 831 Lynam, D. R. (2013). Development of a short form of the UPPS-P impulsive behavior scale.  
 832 Unpublished Technical Report.
- 833 Meule, A., Hermann, T., & Kübler, A. (2014). A short version of the Food Cravings  
 834 Questionnaire – Trait: The FCQ-T-reduced. *Frontiers in Psychology*, 5, 190.  
 835 doi:10.3389/fpsyg.2014.00190.

- 836 Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The  
 837 unity and diversity of executive functions and their contributions to complex “frontal  
 838 lobe” tasks: A latent variable analysis. *Cognitive Psychology*, 41(1), 49-100.  
 839 doi:10.1006/cogp.1999.0734.
- 840 Nederkoorn, C., Braet, C., Van Eijs, Y., Tanghe, A., & Jansen, A. (2006). Why obese children  
 841 cannot resist food: The role of impulsivity. *Eating Behaviors*, 7(4), 315-322.  
 842 doi:10.1016/j.eatbeh.2005.11.005.
- 843 Nederkoorn, C., Guerrieri, R., Havermans, R. C., Roefs, A., & Jansen, A. (2009). The  
 844 interactive effect of hunger and impulsivity on food intake and purchase in a virtual  
 845 supermarket. *International Journal of Obesity*, 33(8), 905-912.  
 846 doi:10.1038/ijo.2009.98.
- 847 Nederkoorn, C., Jansen, E., Mulkens, S., & Jansen, A. (2007). Impulsivity predicts  
 848 overweight and treatment outcome in obese children. *Behaviour Research and*  
 849 *Therapy*, 45(5), 1071-2075. doi:10.1016/j.brat.2006.05.009.
- 850 Stice, E., Spoor, S., Bohon, C., Veldhuizen, M. G., & Small, D. M. (2008). Relation of reward  
 851 from food intake and anticipated food intake to obesity: A functional magnetic  
 852 resonance imaging study. *Journal of Abnormal Psychology*, 117(4), 924-935.  
 853 doi:10.1037/a0013600.
- 854 Strack, F. & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior.  
 855 *Personality and Social Psychology Review*, 8(3), 220-247.  
 856 doi:10.1207/s15327957pspr0803\_1.
- 857 Turton, R., Bruidegom, K., Cardi, V., Hirsch, C. R., Treasure, J. (2016). Novel methods to  
 858 help develop healthier eating habits for eating and weight disorders: A systematic

- 859 review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, 61, 132-155.  
 860 doi:10.1016/j.neubiorev.2015.12.008.
- 861 Van Koningsbruggen, G. M., Veling, H., Stroebe, W., & Aarts, H. (2014). Comparing two  
 862 psychological interventions in reducing impulsive processes of eating behavior:  
 863 Effects on self-selected portion size. *British Journal of Health Psychology*, 19(4), 767-  
 864 782. doi:10.1111/bjhp.12075.
- 865 Van den Eynde, F., Claudino, A. M., Mogg, A., Horrell, L., Stahl, D., Ribeiro, W., . . .  
 866 Schmidt, U. (2010). Repetitive transcranial magnetic stimulation reduces cue-induced  
 867 food craving in bulimic disorders. *Biological Psychiatry*, 67(8), 793-795.  
 868 doi:10.1016/j.biopsych.2009.11.023.
- 869 Veling, H., Aarts, H., & Papies, E. K. (2011). Using stop signals to inhibit chronic dieters'  
 870 responses towards palatable foods. *Behaviour Research & Therapy*, 49(11), 771-780.  
 871 doi:10.1016/j.brat.2011.08.005.
- 872 Veling, H., Aarts, H., & Stroebe, W. (2013a). Stop signals decrease choices for palatable  
 873 foods through decreased food evaluation. *Frontiers in Psychology*, 4.  
 874 doi:10.3389/fpsyg.2013.00875.
- 875 Veling, H., Aarts, H., & Stroebe, W. (2013b) Using stop signals to reduce impulsive choices  
 876 for palatable unhealthy foods. *British Journal of Health Psychology*, 18(2), 354-368.  
 877 doi:10.1111/j.2044-8287.2012.02092.x.
- 878 Veling, H., Van Koningsbruggen, G., Aarts, H., & Stroebe, W. (2014). Targeting impulsive  
 879 processes of eating behavior via the internet: Effects on body weight. *Appetite*, 78,  
 880 102-109. doi:10.1016/j.appet.2014.03.014.
- 881 Veling, H., Lawrence, N. S., Chen, Z., Van Koningsbruggen, G. M., & Holland, R. W.  
 882 (2017). What is trained during food go/no-go training? A review focusing on



- 883 mechanisms and research agenda. *Current Addiction Reports*, 4(1), 35-41.
- 884 doi:10.1007/s40429-017-0131-5.