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| 7 | Beating uncontrolled eating: |
| 8 | Training inhibitory control to reduce food intake and food cue sensitivity |
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Abstract

24 In our food-rich environment we must constantly resist appealing food in order to maintain a 25 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 26 27 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 28 targeted training interventions increasing the potential efficacy of such interventions. In the 29 current study, we investigated directly whether training-induced effects on inhibitory control 30 might underlie the predicted change in eating behaviour. Healthy individuals who scored high 31 on uncontrolled eating were randomly assigned to receive six online training sessions over six 32 33 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-34 and post-training inhibitory control in the context of food and food cue sensitivity, as well as 35 food consumption in a bogus taste test. As expected, food-specific inhibition training 36 decreased snack consumption in the bogus taste test relative to control training. However, the 37 38 active training did not improve inhibitory control towards food, nor did it reduce food cue 39 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 40 41 what drives the reliable effect on eating behaviour.

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Keywords: Cognitive training, response inhibition, food cue sensitivity, overeating, selfcontrol, go/no-go

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We are living in an obesogenic environment where we are constantly confronted with 46 advertisement for foods, and overeating of unhealthy foods is an important contributor to the 47 rising levels of obesity (Hill, Wyatt, Reed, & Peters, 2003). Although almost everyone 48 overeats on occasion, some people overeat on a more regular basis, despite efforts to resist 49 50 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 51 is a characteristic of various eating disorders, such as bulimia nervosa and binge eating 52 disorder (American Psychiatric Association, 2013) as well as obesity (Cornelis et al., 2014). 53 An important factor in regulating eating behaviour and resisting palatable food is 54 inhibitory control (i.e. response inhibition): an executive function that is required to inhibit 55 impulsive responses so that behaviour can be selected that is consistent with one's standards 56 and (long-term) goals (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). Individuals 57 58 with weaker inhibitory control are more often overweight or obese (Guerrieri, Nederkoorn, & Jansen, 2008; Nederkoorn, Breat, Van Eijs, Tanghe, & Jansen, 2006; Nederkoorn, Guerrieri, 59 Havermans, Roefs, & Jansen, 2009; Nederkoorn, Jansen, Mulkens, & Jansen, 2007) and their 60 61 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 62 food intake in the lab (Guerrieri et al., 2007), especially in non-dieters (Guerrieri, 63 Nederkoorn, Schrooten, Martijn, & Jansen, 2009). Although the link between behaviourally 64 65 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, 66 67 Nederkoorn, & Jansen, 2007; Guerrieri et al., 2008). Another important factor in regulating eating behaviour and resisting palatable food is 68

69 food reward sensitivity: the degree to which neurological reward responses to food cues elicit

70 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, 71 Heartherton, & Kelley, 2012), obesity (Stice, Spoor, Bohon, Veldhuizen, & Small, 2008) and 72 bulimia nervosa (Brooks et al., 2011). However, findings by Lawrence et al. (2012) suggest 73 74 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 75 76 sensitivity was associated with increased BMI in individuals reporting low inhibitory control. Interestingly, food reward sensitivity was negatively correlated with BMI in individuals 77 reporting high inhibitory control. 78

These findings of previous studies are in line with traditional dual process models. 79 This theoretical model emphasizes the role of inhibitory control whenever there is conflict 80 between two different systems – an impulsive system and reflective system – that operate in 81 82 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 83 significance, and based on that evaluation predisposes one to either approach or avoid. Unlike 84 85 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 86 cognitive control (Strack & Deutsch, 2014). Without inhibitory control the reflective system 87 88 would not be able to overrule the initial response of the more fast-acting impulsive system. 89 Although a dual-process model might serve as a useful way to describe impulsive and

90 reflective processes, more recent articles argue for a unitary model of action control (Hommel
91 & Wiers, 2017). This unitary model considers all behaviours to be goal-directed. Goals can
92 act as selection criteria that under certain conditions may promote actions that are simple, fast,
93 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 94 reluctance to indulge in unhealthy foods in a motivationally 'neutral' situation, this intention 95 can weaken when being primed with palatable foods or when hungry. Individual preferences 96 for fast-acting decision making versus slow and controlled decision-making could then 97 98 translate into individual differences regarding 'acceptable' behaviour, such as overeating. Reduced inhibitory control may not imply an inability to translate intentions into action but 99 may relate to a preference for fast-acting (impulsive) decisions based on salient cues 100 (Hommel & Wiers, 2017). Overeating could thus depend on an interaction between individual 101 differences in food reward sensitivity (sensitivity to salient cues) and inhibitory control. For a 102 full discussion of the unitary model, see Hommel and Wiers (2017). 103

Considering the findings of previous studies that indicate a relationship between 104 inhibitory control and overeating behaviour it should come as no surprise that there has been a 105 growing interest in targeting inhibitory control to help people refrain from overeating. A task 106 that is repeatedly used to measure inhibitory control is the go/no-go task, in which people are 107 instructed to respond as fast as possible to 'go' items, and to withhold their response to 'no-108 109 go' items (Donders, 1969). Researchers have developed food-specific go/no-go training tasks 110 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 111 112 training has been found to reduce food intake (Adams, Lawrence, Verbruggen & Chambers, 113 2017; Houben, 2011; Houben & Jansen, 2011, 2015; Lawrence, Verbruggen, Morisson, Adams, & Chambers, 2015b; Van Koningsbruggen, Veling, Stroebe, & Aarts, 2014; Veling, 114 115 Aarts, & Papies, 2011; Veling, Aarts, & Stroebe, 2013a, 2013b), facilitate weight-loss (Veling, Van Koningsbruggen, Aarts, & Stroebe, 2014; Lawrence et al., 2015b), and reduce 116 self-served food portion sizes (Van Koningsbruggen et al., 2014). Recent meta-analyses found 117

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 121 122 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-123 related responses. Besides strengthening top-down inhibitory control, two alternative 124 explanations have been since postulated for how food response inhibition training may reduce 125 food consumption (Veling, Lawrence, Chen, Van Koningsbruggen, & Holland, 2017): 1. 126 training could create automatic 'bottom-up' associations between no-go food items and 127 stopping responses (automatic inhibition); 2. training leads to devaluation of food items. This 128 second alternative mechanism is based on Behaviour Stimulus Interaction (BSI) theory. The 129 BSI theory proposes that devaluation of appetitive food stimuli takes place when an initial 130 approach response to appetitive food stimuli is inhibited in order to prevent continuous 131 oscillation between approach and inhibition (Chen, Veling, Dijksterhuis, Holland, 2016). 132 133 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. 134 (2017) have argued that this is unlikely as the training task is very easy and a type of training 135 136 that is considered more demanding for top-down control, the stop-signal training, is generally 137 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 138 139 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 140 in eating behaviour. This proposed mechanism is thus yet to be experimentally demonstrated. 141

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 147 food go/no-go training is a devaluation of food items. Although some studies indeed found 148 that training led to food devaluation when measured with explicit rating scales of food items 149 (Chen et al., 2016), other studies using the Implicit Association Task found no evidence for 150 devaluation of appetitive stimuli (Jones et al., 2016). One study did find that devaluation of 151 no-go food stimuli was related to weight loss after training, but devaluation did not mediate 152 the effect of go/no-go training (Lawrence et al., 2015a). The evidence that food go/no-go 153 training leads to a devaluation of food and underlies the effects of training on food intake thus 154 remain mixed. Therefore, we were interested to test if food specific go/no-go training would 155 reduce food cue induced craving. Unlike explicitly asking individuals to rate food items on a 156 157 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 158 incentive value of food. 159

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

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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.

170 Current Study

The current study aimed to investigate the effect of 6 sessions of food-specific 171 inhibition training on eating behaviour in a sample of healthy individuals who scored high on 172 uncontrolled eating. Participants either received an active training, that is, the food-specific 173 inhibition training or a control training that was equal to the active condition except that only 174 non-food stimuli were used. By adopting an 'active' control condition we aimed to equalize 175 training elements such as cognitive effort over conditions. Furthermore, this control condition 176 allowed us to investigate whether training needs to include behaviour-specific stimuli in order 177 to achieve behaviour change or whether training of general inhibitory control is sufficient to 178 improve outcomes in a specific domain. During a baseline and post-training test session we 179 measured inhibitory control in the context of food and food cue sensitivity. Additionally, in 180 181 the post-training session we measured food consumption with a bogus taste test.

To investigate possible underlying working mechanisms of the food-specific inhibition 182 training, we first examined whether inhibition training modifies response inhibition for food, 183 184 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 185 inhibition towards unhealthy food. We expected that the active training group would show a 186 187 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 188 transfer), using a cue reactivity paradigm previously used by Brockmeyer, Hahn, Reetz, 189

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 193 194 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 195 196 consume less food as compared to the control group. In order to take into account interindividual differences in training effectiveness, and to directly test whether changes in 197 inhibitory control for food and food cue sensitivity were related to a change in eating 198 behaviour, we also explored whether these changes across training were related to food 199 200 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 201 202 control group; 2) the active group will show a greater reduction in cue-induced food craving 203 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. 204

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Participants

Method

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

| 214 | score for the TFEQ-R18 was determined based on a large community sample from Oxford by |
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| 215 | taking the upper tertial (unpublished data). Participants were excluded if they revealed any of |
| 216 | the following: specific diets (vegan, vegetarian, and/or diabetes); food allergies for foods used |
| 217 | in the taste test (chocolate and crisps); $BMI < 18.5$ or > 30.03 ; currently being on a diet with |
| 218 | the aim to lose weight; having sought professional help in the last six months for an eating |
| 219 | disorder or other mental health problems. Participants were randomly assigned to either the |
| 220 | active (N = 21) or the control group (N = 20). Participants were reimbursed for their time. |
| 221 | This study was approved by the University of Oxford Central University Research Ethics |
| 222 | Committee (R25997/RE005). |
| 223 | Measurements |
| 224 | Pre-screening questionnaire. The pre-screening questionnaire assessed the |
| 225 | inclusion/exclusion criteria by asking about specific diets, food allergies, being on a diet with |
| 226 | the aim to lose weight, and whether individuals sought professional help in the last six months |
| 227 | for an eating disorder or other mental health problems. To calculate BMI, height and weight |
| 228 | were asked. Additionally, participants completed the 9 items belonging to the uncontrolled |
| 229 | eating subscale from the TFEQ-R18. |
| 230 | 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-

³ 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.

236 24; uncontrolled 9-36; emotional 3-12) measuring the frequency of a certain behaviour or 237 how true a statement is for the participant. Higher scores indicate more of the unhealthy 238 eating behaviour. Psychometric properties indicate that the measure is valid and reliable in a 239 sample with a varying range of body weights (Anglé et al., 2009). The internal consistencies 240 of the TFEQ-R18 subscales in the current sample were acceptable (restrained eating: $\alpha = .77$) 241 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-249 P Impulsive Behaviour Scale (Lynam, 2013). The SUPPS-P contains 20 items, separated into 250 251 five subscales that each contain four items: negative urgency, (lack of) premeditation, (lack 252 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), 253 254 with higher scores indicating more impulsive behaviour. Psychometric properties indicate that 255 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 256 257 urgency: $\alpha = .64$; perseverance: $\alpha = .68$), acceptable (sensation seeking: $\alpha = .73$), and good (premeditation: $\alpha = .84$; positive urgency: $\alpha = .81$). 258

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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 266 measured with the FCQ-S (FCQ-S; Cepeda-Benito, Gleaves, Williams, & Erath, 2000). The 267 FCQ-S contains 15 items, which are divided across five dimensions (intense desire to eat, 268 anticipation of positive reinforcement, anticipation of relief from negative states, lack of 269 270 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 271 17-75), with higher scores indicating higher state food craving. Psychometric properties 272 indicate that the measure is valid and reliable (Cepeda-Benito et al., 2000). The internal 273 274 consistency of the FCQ-S in the current sample as measured on the different time points was 275 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

284 the control group received a control version of the task only containing non-food stimuli 285 (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. 286 On each trial a rectangle frame was presented in which a stimulus would appear for 1250 ms 287 288 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 289 290 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 291 had to withhold their response (no-go trial). Feedback on accuracy and mean RT for go trials 292 were presented at the end of each block to increase motivation. Each training session 293 consisted of six blocks of 32 trials and took approximately 10 minutes to complete. 294

Stimuli in the active training condition consisted of 16 food and 16 non-food filler 295 296 stimuli of clothes. Of the food stimuli eight depicted healthy food items (fruit, vegetables, and 297 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 298 299 consisted of 16 household objects (furniture, home tools, gardening tools), and the same 16 300 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 301 302 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 303 equally associated with go and no-go trials (48 go and 48 no-go trials per training session), 304 305 resulting in 50% no-go trials overall. The different trial types appeared in equal numbers 306 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 307

stimuli of electrical items, furniture, and buckets were always paired with go-trials (48 trials). 308 309 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 310 their correct response less obvious, to enhance learning in the automatic, associative system, 311 312 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 313 and non-food stimuli were selected from the food.pics database (see www.food-pics.sbg.ac.at; 314 Blechert, Meule, Busch, & Ohla, 2014). The healthy food stimuli and the unhealthy food 315 stimuli in the active training were matched for valence and arousal. In the control version, the 316 household stimuli paired with no-go trials and the household stimuli paired with go-trials 317 were matched for valence and arousal. The filler stimuli of clothes and household objects 318 were the same stimuli as those previously used in a study by Lawrence et al. (2015a). The 319 320 training task was administered online using the Inquisit 4 Millisecond Web player (Inquisit, Millisecond Software Seattle, WA). 321



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.

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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 foodrelated 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 339 and non-food stimuli presented in the centre of the screen. The 25 food stimuli and 25 non-340 food stimuli were selected from the food.pics database (Blechert et al., 2014), but none of the 341 stimuli were identical to the ones used in the training task to increase internal and external 342 validity (Batterink, Yokum, Stice, 2010; Kullman et al., 2014). Food and non-food stimuli 343 were matched on brightness, complexity, valence, and arousal. For the food stimuli we 344 selected only high calorie foods (e.g. donuts, chocolate, crisps, cakes, and fast food). For the 345 346 non-food stimuli we selected a variety of objects (e.g. clock, book, bag, and candle). The task 347 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. 348

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 355 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 356 357 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 358 359 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 360 (i.e. which category of stimuli was the go stimulus). Before the start of the task, participants 361 first completed two practice blocks (8 trials of each block version) to ensure they understood 362 the task. 363



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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

- al., 2010). Participants first rated their current state level of food craving with the FCQ-S
- 372 (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
- 374 state food craving was assessed again, which allowed us to assess the change in state craving

³⁷⁰ was assessed with the Food Challenge task (e.g. Brockmeyer et al., 2015; Van den Eynde et

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in response to food cues.

376 **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 377 ready salted crisps) and 200gr. of chocolate (Cadbury milk chocolate buttons). These 378 379 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. 380 They were instructed that they could consume as much or little as they wished to complete the 381 taste test. The participants were given 10 minutes to complete the taste test, during which the 382 researcher left the room. The rating scales consisted of 9-point Likert scales about the 383 sweetness, sourness, saltiness, bitterness, and palatability of the snacks. Separate Likert scales 384 were completed for the crisps and chocolate. It also included open-ended questions measuring 385 the usual frequency of consumption of the snacks per week (i.e. for crisps and chocolate 386 387 separately). After the test session, when the participant had left, snack consumption was measured in grams. 388

389 **Procedure**

390 Participants were recruited via online and printed advertisements distributed around 391 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 392 393 attended two test sessions of 1.5 hrs each and completed six computer-based training sessions 394 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 395 396 arrival at the first session, participants completed the consent form and measurements of 397 height, weight, waist, and hip circumference. Subsequently, participants completed the preassessment questionnaires, the food-based go/no-go task and thereafter the food cue 398

sensitivity task⁴. At the end of the first test session, participants received information 399 regarding the online training, but were kept naïve as to the purpose of the training. All 400 401 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, 402 403 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 404 task (e.g. when the rectangle frame is normal they have to respond as quickly and accurately 405 as possible to indicate the side of the stimulus presentation, etc.) and completed one practice 406 block of 32 trials to ensure that they understood the task. Daily training reminders were sent 407 to the participants via e-mail to promote compliance. Participants were blind to intervention 408 condition. 409

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.

⁴ Participants also completed a probabilistic monetary reward task during the first and the second testsession. However, this task is not used to answer our research question and is therefore not reported here.

| #45 le 1 | |
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| Participant characteristics. | |

| | Control group | Active group | Range Control | Range Active | F-value ^a (p) |
|--|---------------|---------------|---------------|--------------|------------------------------|
| | M(SD) | M(SD) | | | |
| 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 ²) | 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 | 7.10 (2.99) | 7.67 (2.37) | 4-14 | 4-12 | 0.45 (.504) |
| urgency | | | | | |
| Impulsivity - Negative | 9.10 (2.75) | 10.00 (2.17) | 5-15 | 6-16 | 1.36 (.250) |
| urgency | | | | | |
| 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 | 25.00 (3.33) | 25.14 (3.55) | 20-31 | 20-34 | 0.02 (.895) |
| eating | , . | | | | . , |
| TFEQ - Emotional eating | 8.05 (2.09) | 8.38 (2.50) | 5-12 | 3-12 | 0.21 (.649) |
| State hunger 1 st session | 3.70 (1.13) | 3.67 (1.28) | 2-6 | 1-5 | 0.01 (.930) |
| State hunger 2 nd session | 3.35 (1.14) | 3.00 (1.67) | 1-6 | 1-6 | 0.51 (.440) |
| Hours since last | 3.31 (2.72) | 3.17 (2.02) | 2-12 | 2-12 | 0.04 (.837) |
| meal/snack 1st session | | | | | |
| Hours since last | 3.70 (3.44) | 4.38 (4.25) | 1-14 | 2-16 | 0.32 (.577) |
| meal/snack 2 nd session | | | | | |

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

^a Group difference in sex is a chi-square value.

- 416
- 417

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.

427 Training Performance

428 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 429 condition), resulting in a compliance rate of 98% who completed all six sessions. To assess 430 changes in training performance across the training period we performed mixed design 431 ANOVAs on the proportion of commission errors (i.e. number of false alarms divided by total 432 number of no-go trials) with condition (control, active) as between-subject factor, and time 433 (first, last training session), and stimulus type (filler, non-filler) as within-subject factors. See 434 435 supplemental material for additional analysis performed on other outcome measures of the training task (e.g. omission errors). 436

For the commission error analysis, we excluded three cases due to outlying data (i.e. 437 standardized residuals >3 SDs from the mean). The ANOVA yielded a main effect of time, 438 $F(1, 36) = 4.75, p = .036, \eta_p^2 = .12$, a marginally significant main effect of stimulus type, $F(1, 36) = 4.75, p = .036, \eta_p^2 = .12$ 439 36) = 3.87, p = .057, η_p^2 = .10, a Time x Stimulus type interaction, F(1, 36) = 5.50, p = .025, 440 $\eta_p^2 = .13$, a Time x Condition interaction, F(1, 36) = 6.46, p = .016, $\eta_p^2 = .15$, and a Stimulus 441 type x Condition interaction, F(1, 36) = 8.59, p = .006, $\eta_p^2 = .19$. There was no main effect of 442 condition or a Time x Stimulus type x Condition interaction effect, p > .05. Figure 3 443 graphically displays the change in performance of commission error rate across the training 444 period for the two groups separately. 445

To follow-up the Time x Stimulus type interaction we performed paired t-tests to 446 compare commission errors at first and last training session for the two stimulus types 447 separately (i.e. regardless of condition). The commission error rate did not significantly 448 decrease for filler stimuli from first session (M = 0.04; SD = 0.03) to last session (M = 0.04; 449 SD = 0.03, t(37) = -0.37, p = .711, d = 0.06, while it did significantly decrease for non-filler 450 stimuli (i.e. food items in the active training, household items in the control training) from 451 first session (M = 0.04; SD = 0.03) to last session (M = 0.02; SD = 0.03), t(37) = 3.34, p =452 .002, d = 0.54. Additionally, a paired t-test was done to compare filler and non-filler stimuli at 453 454 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. 455 However, during the final training session there were more commission errors for filler 456 stimuli than for non-filler stimuli, t(37) = -2.77, p = .009, d = 0.45. 457

To follow-up the Time x Condition interaction we performed a paired t-test to 458 investigate the effect of time on rate of commission errors for the two groups separately (i.e. 459 regardless of stimulus type). The commission error rate did not significantly differ between 460 461 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 462 decrease from first session (M = 0.04; SD = 0.02) to last session (M = 0.02; SD = 0.02) in the 463 464 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 465 commission error rate did not significantly differ between the control and active group at first 466 467 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 =468 0.96. 469

470 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 471 separately (i.e. regardless of training session). The commission error rate did not significantly 472 differ between filler stimuli (M = 0.03; SD = 0.02) and non-filler stimuli (M = 0.03; SD =473 0.03) in the active group, t(19) = 0.16, p = .876, d = 0.04. However, significantly more 474 commission errors were made to filler stimuli (M = 0.05; SD = 0.03) than to non-filler stimuli 475 (M = 0.02; SD = 0.02) in the control group, t(17) = 3.47, p = .003, d = 0.82. Additionally, an 476 independent t-test was performed to compare the control group and the active group on rate of 477 commission errors for the stimuli types separately. More commission errors were made in the 478 control group compared to the active group for filler stimuli, t(36) = 3.00, p = .005, d = 1.00. 479 Rate of commission errors did not differ significantly between the control group and the 480 active group for non-filler stimuli, t(36) = -0.23, p = .816, d = 0.04. 481

Training awareness. During the debriefing procedure, 38% of the active participants 482 reported to have noticed that no-go signals were associated with stimuli of high calorie food, 483 whereas 15% of the control participants noticed that specific categories of objects were 484 485 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 486 the training task had influenced their taste test. Only two active participants thought that the 487 488 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 489 during eating"). 490



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.

494

495 Near Transfer: Go/no-go task

To assess training-related changes in food-related inhibitory control on the go/no-go task, we 496 497 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 498 factors on commission error rate (see Figure 4). For the commission errors analyses, we 499 excluded two cases due to outlying data (i.e. standardized residuals > 3 SDs from the mean). 500 The ANOVA on the number of commission errors yielded a main effect of time, F(1, 37) =501 7.11, p = .011, $\eta_p^2 = .16$ revealing that overall more commission errors were made at baseline 502 (M = 0.19; SD = 0.09) than at post-training session (M = 0.16; SD = 0.08). Furthermore, there 503 was a main effect of stimulus type, F(1, 37) = 24.15, p < .001, $\eta_p^2 = .40$, as overall less 504 505 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 506 significant interaction effects, all ps > .05. See supplemental material for additional analysis 507 performed on other outcome measures of the go/no-go task (e.g. omission errors). 508





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.

513

514 Far Transfer: Cue reactivity

To assess cue-induced food craving, we performed a mixed design ANOVA with 515 condition (control, active) as between-subject factor, and reactivity (before, after craving 516 517 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 518 baseline data. Three participants each had a missing value on the FCQ-S which were replaced 519 520 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 521 <.001, $\eta_p^2 = .58$. There was a significant increase in state food craving from pre- to post-cue 522 exposure across the sessions and groups (see Table 2 for means and standard deviations). 523 There was also a main effect of time as state craving (average across pre- and post-craving 524 525 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 526 did not influence the effect of reactivity, time, nor their interaction, all ps > .10. 527

| Table 2 |
|----------------|
| Cue reactivity |

| Cue reactivity | • | | | |
|----------------|---------------|---------------|---------------|---------------|
| | T1-1 | T1-2 | T2-1 | T2-2 |
| | M(SD) | M (SD) | M (SD) | M(SD) |
| 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.

529 Far Transfer: Taste test

To compare training groups in the bogus taste test (i.e. average consumption of crisps 530 and chocolate in grams), while controlling for state craving, we performed a one-way 531 ANCOVA. State craving, as measured by the FCQ-S administered prior to the bogus taste 532 test, was added as a covariate to control for differences in state craving after the food cue 533 sensitivity task. One participant was excluded from the taste test analysis due to outlying data 534 (i.e. standardized residuals > 3 SDs from the mean). The ANCOVA revealed that the control 535 536 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 537 were pre-existing group differences in the usual frequency of crisps and chocolate 538 539 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 540 two snacks separately. The usual frequency of crisps consumption per week did not 541 significantly differ between the two groups (control: M = 1.95; SD = 1.64; active: M = 2.35; 542 SD = 1.42), F(1, 38) = 0.68, p = .415, $\eta_{p}^{2} = .01$, nor was there a significant difference of 543 544 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, $\eta_p^2 = .04$. 545

⁵²⁸

Additionally, we explored whether the change in inhibitory control and food cue 546 sensitivity, across groups, was related to food consumption across groups. We focused on the 547 change in inhibitory control for food stimuli only. We performed a simple linear regression 548 analysis on the consumption of snacks. For the change in inhibitory control, we first entered 549 state craving, as measured by the FCQ-S administered prior to the bogus taste test. In a 550 second step we entered the change in inhibitory control across the training period, calculated 551 as a difference score (i.e. rate of the first session subtracted from the rate of the second 552 session). The regression analyses revealed that changes in commission errors over time on the 553 go/no-go task were not directly related to the amount of consumed snacks in the taste test, $\beta =$ 554 -0.22, t=-1.51, p = .139, $\Delta R^2 = 0.05$. See supplemental material for additional simple linear 555 556 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 557 change in reactivity. That is, the reactivity in state craving from before the food video to after 558 the food video and how that changed from pre-training to post-training. One participant was 559 excluded from the taste test analysis due to outlying data (i.e. standardized residuals > 3 SDs 560 561 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, $\beta = -0.10$, 562 $t=-0.62, p=.541, R^2=0.01.$ 563

564

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 573 574 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 575 taste test; Houben, 2011; Houben & Jansen, 2011, 2015), as well as studies that have used 576 other behavioural measures of eating (Lawrence et al., 2015b; Veling et al., 2011) and shows 577 that food-specific inhibition training can influence eating in a sample with a tendency for 578 uncontrolled eating. The consistent findings from a variety of studies gives some confidence 579 that the food response inhibition training can indeed reduce snack consumption and influence 580 eating behaviour. In contrast to previous studies we measured inhibitory control pre- and post-581 training with a go/no-go task that included blocks in which participants had to respond rapidly 582 to food stimuli. Previous studies have not attempted to measure inhibitory control after 583 training because of a concern that assessing inhibitory control with food-related stimuli after 584 585 training would reduce any effects of training on a measure of eating behaviour (Veling et al., 586 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 587 588 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 589 effect (d = 0.31) of training on snack consumption, indicating that the training had a rather 590 591 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

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594 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 595 thus enhance inhibitory control for the relevant training stimuli. There was however no 596 specific (near) transfer of active training to the food go/no-go task, as training groups showed 597 598 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 599 commission errors at post-training but this was regardless of stimulus type. Generally, 600 inhibitory control was worse for food stimuli, likely because these stimuli are more salient, 601 but this did not change across training nor was this different between training groups. 602

We compared an active inhibition training with food stimuli to a control condition that 603 also required participants to inhibit their responses but not in the context of food stimuli. 604 Although the advantage of such a control condition is that it equalizes general training effects 605 (e.g. cognitive effort, adhering to a training schedule) and allowed us to investigate whether 606 training needs to include behaviour-specific stimuli, it is also a more conservative test of 607 whether food-specific go/no-go training can increase inhibitory control for food stimuli. The 608 609 use of our specific control condition could therefore have contributed to this lack of difference 610 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 611 612 task did not relate to individual differences in snack consumption. Improving inhibitory 613 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-614 615 driven actions) as described in the unitary model of action selection (Hommel & Wiers, 616 2017). However, based on our findings we suggest that enhanced inhibitory control is most

617 likely *not* the mechanism underlying the effect of food response inhibition training on eating618 behaviour, at least not in the current study.

Two alternative explanations for how food response inhibition training may reduce 619 snack consumption have previously been described by Veling et al. (2017): 1. the training 620 621 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 622 should have found a decrease in commission errors towards food (similarly as for the top-623 down inhibitory control account) and a slowing in RT of hits for food stimuli. As we did not 624 find this (see also supplementary material), our data do not support the first alternative 625 explanation. Regarding the second alternative mechanism, although we did not directly test 626 modification of valuation of food with explicit ratings of food items, we did measure 627 individuals' craving reactivity towards food items. Cue induced craving could reflect a 628 physical equivalent of food evaluation. From the perspective of the unitary model of action 629 control (Hommel & Wiers, 2017) this could mean that training works through reducing the 630 tendency to use fast-acting, stimulus-driven decision making based on salient cues. 631

632 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 633 unclear. It may simply be due to a testing effect: familiarity with the video may have led to 634 635 weaker reactivity effects, rather than a training effect. It may also be due to demand 636 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 637 638 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 639 including normal everyday ingredients (e.g. pasta, bread, potatoes) and healthy foods (e.g. 640

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 646 food cue sensitivity, nor does the change in food cue sensitivity relate to snack consumption, 647 we suggest that it is worthwhile for future research to investigate whether this is food type 648 dependent, before drawing firm conclusions. We agree with Veling et al. (2017) that 649 devaluation of unhealthy foods is a plausible mechanism underlying the effects of food go/no-650 go training, but the evidence so far is not satisfactory. Furthermore, it is important to establish 651 to what extent the effects of food inhibition training generalize to other types of food (not 652 used in the training task) as this could limit the efficacy of training and/or indicate that 653 training should be personalized to certain food categories depending on the individual. Such 654 research would be especially relevant if food inhibition training indeed leads to food 655 656 devaluation and thereby influences eating behaviour.

A further possibility is that the effect of food inhibition training on eating behaviour is 657 due to an exposure mechanism that diminishes learned responses to food in the context of a 658 659 food cue. During the active training, participants are repeatedly exposed to food items to 660 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 661 662 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 663 provides a potential direction for future research to explore. For example, investigating the 664

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

Strengths of the present study include the well-controlled design and the fact that we 667 directly investigated the hypothesis that food-specific go/no-go training strengthens inhibitory 668 669 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 670 related responses, evident by the low error rate that is associated with the training. Using a 671 more challenging food response inhibition training (e.g. with a lower percentage of no-go 672 trials) might be necessary to consistently modify inhibitory control over approach tendencies 673 towards food. That said, the far transfer effect of inhibitory control training on snack 674 consumption has been repeatedly found with this relatively easy training task. Thus, a 675 relatively easy training task may be effective in influencing eating behaviour due to a 676 mechanism other than inhibitory control. Second, we measured food cue sensitivity with the 677 food challenge task that is susceptible to demand effects, by measuring craving just before 678 and after presentation of a video displaying food stimuli. Most studies investigating food cue 679 680 sensitivity rely on physiological methodologies such as fMRI with which it is possible to 681 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 682 683 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

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689 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 690 inhibitory control combined with more directly targeting devaluation of unhealthy foods. It is 691 possible that single processes cannot fully explain changes in food intake but that multiple 692 693 processes have an additive or interacting effect increasing the impact on eating behaviour. Furthermore, research should investigate whether response inhibition training also has an 694 influence on eating behaviour in clinical samples of people with binge eating disorder or 695 bulimia nervosa. Additionally, longitudinal studies, preferably with a double-blind design and 696 a large sample, should determine whether there is a long-term effect of training on eating 697 behaviour, and under which conditions such an effect is present. For example, it may be 698 necessary for participants to repeat training at certain intervals for an enduring effect on 699 eating behaviour to develop. 700

To summarize, we found that food response inhibition training can reduce snack 701 consumption even though the active and control training conditions did not differ in training 702 703 effects on inhibitory control for food and food cue sensitivity. Based on our findings it seems 704 unlikely that increased inhibitory control or decreased food cue sensitivity explains the effect 705 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 706 707 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 708 709 eating, which could increase the potential clinical value of such an intervention.

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| 716 | |

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