Uncontrolled Eating in Healthy Women Has Limited Influence on Food Reward Sensitivity and Food-Related Inhibitory Control

Maud Grol¹, Luis Cásedas², Danna Oomen¹, Desiree B. Spronk³, Elaine Fox

Department of Experimental Psychology, University of Oxford, UK

Author Note

Maud Grol <https://orcid.org/0000-0001-9933-4569>

Luis Cásedas <https://orcid.org/0000-0001-5650-6332>

Danna Oomen <https://orcid.org/0000-0003-2638-1975>

Desiree B. Spronk <https://orcid.org/0000-0002-9334-3971>

Elaine Fox <https://orcid.org/0000-0002-2464-0462>

Correspondence concerning this article should be addressed to Maud Grol

e-mail: maudgrol@gmail.com, Department of Experimental Clinical and Health Psychology, Ghent

University, Henri Dunantlaan 2, 9000 Gent, Belgium

¹ Maud Grol and Danna Oomen have since moved to the Department of Experimental Clinical and Health Psychology at Ghent University, Belgium

² Luis Cásedas has since moved to the Mind, Brain, and Behavior Research Center, Department of Experimental Psychology, Universidad de Granada, Spain

³ Desiree Spronk has since moved to the Trimbos institute, Utrecht, The Netherlands

Abstract

Uncontrolled eating—in the general population—is characterized by overeating, hedonic hunger and being drawn towards palatable foods. Theoretically, it is the result of a strong food reward signal in relation to a poor ability to exert inhibitory control. How food consumption influences inhibitory control and food reward sensitivity, and how this relates to the continued urge to eat, remains unclear though. We used fMRI ($N=40$) in order to investigate the neural mechanism underlying food reward sensitivity and food-specific response inhibition (go-nogo task), by comparing women reporting high versus low/average uncontrolled eating across two sessions: during an inter-meal hunger state and after consumption of a high-caloric snack. We found no effects of individual differences in uncontrolled eating, food consumption, nor their interaction on food reward sensitivity. Differences in uncontrolled eating and food consumption did interact in modulating activity in the left superior occipital gyrus during response inhibition of non-food stimuli, an area previously associated with successful nogo- vs. go-trials. Yet, behavioural performance on the go-nogo task was not modulated by uncontrolled eating nor food consumption. Women with a low/average tendency for uncontrolled eating may need more cognitive resources to support successful response inhibition of non-food stimuli during food 'go' blocks in an inter-meal hunger state, whereas women with a high tendency for uncontrolled eating showed this after food consumption. Considering current and previous findings, it seems that individual differences in uncontrolled eating in healthy women have only limited influence on food reward sensitivity and food-related inhibitory control, whereas differences in weight status (e.g., obesity) may have more impact.

Keywords: uncontrolled eating, food reward sensitivity, inhibitory control, superior occipital gyrus, food consumption, hedonic hunger

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In an ever more food-rich environment, overeating of energy-dense foods is an important contributor to rising levels of obesity (Hill et al., 2000). Uncontrolled eating is characterized by a tendency to overeat with a sense of being out of control and finding it hard to stop (Anglé et al., 2009). Higher levels of uncontrolled eating—in the general population—are associated with higher intake of energy-dense foods (De Lauzon et al., 2004) and increased weight (Mustelin et al., 2017). Only when uncontrolled eating is regular and accompanied by additional features such as distress, guilt, eating alone due to embarrassment, or eating much more rapidly than normal, it is considered a binge eating disorder (American Psychiatric Association, 2000). Given the potential role of (non-clinical) uncontrolled eating in weight gain and obesity, it is important to increase our understanding of what factors contribute to individual differences in uncontrolled eating. We utilized functional Magnetic Resonance Imaging (fMRI) to investigate individual variability in uncontrolled eating with a particular focus on the role of food reward sensitivity and inhibitory control. fMRI is useful in that it provides a measure of (reward) responsivity to food cues that cannot be observed at the behavioural level, and is less prone to the demand effects that can influence self-report measures.

1.1 Food Reward Sensitivity and Inhibitory Control

Uncontrolled eating is a tendency to overeat accompanied by a sense of being out of control or finding it hard to stop, and has been operationalized as a combination of features that tap into overeating, hedonic hunger and craving for palatable foods (Three Factor Eating Questionnaire - R18; Anglé et al., 2009).⁴ Overeating behaviour has been proposed to arise from an imbalance between food reward sensitivity and inhibitory control (Appelhans et al., 2011). Food reward sensitivity reflects cue-elicited reward responses that contribute to the motivation to eat. It is likely a composite process that contains both the hedonic reaction (“liking”), incentive salience and

⁴ Clinical instances of binge eating are also characterized by overeating with a loss of control and could be a way to cope with negative feelings, but these lead to feelings of excessive guilt and distress.

cognitive desire (“wanting”), as well as associative and predictive learning components (Berridge et al., 2010). On the other hand, inhibitory control is an executive function that permits an individual to inhibit impulses and dominant behavioural responses to stimuli. As an example, food-cue elicited craving can be controlled, so that behaviours (e.g. eating less, eating healthy) can be selected that are in line with one’s (long-term) goals (Miyake et al., 2000) such as losing excess weight. Inhibitory control contributes to successful self-regulation of eating behaviour (Dohle et al., 2018) and is relevant for goal-directed behaviour such as following a healthy diet and lifestyle. In support of this view, despite some failures to replicate (Guerrieri et al., 2008), behavioural studies have shown that poorer performance on inhibitory control tasks is associated with higher trait food craving (Meule & Kübler, 2014) and increased food intake in laboratory settings (Allom & Mullan, 2014; Guerrieri et al., 2007; Price et al., 2016).

There is growing evidence that body weight, food selection and intake vary as a function of individual differences in food reward sensitivity (for a meta-analysis see Boswell & Kober, 2016). Obesity and individual differences in (trait) food craving have also been shown to be associated with differences in the neural response to food cues in brain regions that are part of the reward system, such as striatal areas, anterior cingulate cortex (ACC), insula, orbitofrontal cortex (OFC), and amygdala (García-García et al., 2013; Stice et al., 2008; Ulrich et al., 2016; Verdejo-Román et al., 2017). Interestingly, higher food reward sensitivity has been associated with greater palatable food intake in a laboratory setting (Appelhans et al., 2011) or greater weight gain across a year (Nederkoorn et al., 2010) only for individuals with lower levels of inhibitory control. Inhibitory control thus seems to be important in that it can moderate the relationship between food reward sensitivity and differences in weight status.

The interaction between inhibitory control and food reward sensitivity is also clear when considering the underlying neural network that supports inhibitory control. Food-specific inhibitory control is associated with activity in a wide network of frontal and parietal regions (Batterink et al., 2010; He et al., 2014; Sebastian et al., 2013). Explicit cognitive regulation during reward cue

exposure has also been associated with increased activity in similar areas such as middle frontal gyrus, postcentral gyrus, inferior parietal lobule, temporal gyrus and superior occipital gyrus, which was accompanied by relatively reduced activity in areas of the reward system mentioned earlier (Delgado et al., 2008; Kober et al., 2010). Inhibitory control can thus influence the neural response to rewarding cues, such as visual food cues, and thereby potentially influence food intake, but this ability may differ across individuals.

1.2 Effects of Satiation

Individual differences in both food reward sensitivity and inhibitory control are thus likely to be important drivers for differences in uncontrolled eating. However, attention for food (Stamatakis et al., 2019) and activity in response to food cues in brain regions that are part of the reward system (Führer et al., 2008; Goldstone et al., 2009; LaBar et al., 2001; Morris & Dolan, 2001), have also been shown to change with acute states of energy balance, i.e., a fasted versus satiated state. Palatable (high-caloric) foods have been shown to elicit greater responses in striatal regions, amygdala, insula, OFC, ACC, dorsolateral prefrontal gyrus, anterior fusiform and (para-)hippocampal gyrus when in a hungry state as compared to a satiated state (Führer et al., 2008; Goldstone et al., 2009; LaBar et al., 2001). A study comparing a natural inter-meal hunger state with a satiated state showed that satiation attenuated activity to food cues in the ventrolateral and ventromedial PFC, nucleus accumbens, OFC, hypothalamus, and (left) insula, while activity increased in the dorsolateral PFC and (right) insula (Thomas et al., 2015). These satiation-related changes in food reward sensitivity could reflect a (relative) decrease in the rewarding value and incentive salience of foods, and reduced motivation to obtain food when homeostatic needs are met, thereby regulating eating behaviour. However, it has recently been shown that weight status can modulate such satiation effects on food reward sensitivity, with normal weight individuals showing attenuated activity in response to food cues in ACC, dorsolateral PFC and posterior cingulate cortex after eating, while obese individuals did not (Puzziferri et al., 2016). It is possible that individual differences in effects of food consumption on the response to food cues, could also underlie differences in uncontrolled eating.

Most studies (for exceptions see, Dimitropoulos et al., 2012; Thomas et al., 2015) that have investigated effects of satiation, have compared more ‘extreme’ states: a satiated and fasted state (> 8hrs of fasting). These findings therefore mostly relate to effects of homeostatic hunger and eating to remove an energy deficit. Instead, uncontrolled eating has been characterized by problems with stopping eating once people start and also by hedonic hunger. This is not to say that uncontrolled eating cannot start from a drive to remove an energy deficit, but individuals have difficulties stopping even after homeostatic needs are met or eat in the absence of homeostatic hunger. Given that uncontrolled eating does not necessarily happen in the context of fasting, but reflects an excessive appetite to consume (palatable) food also in the absence of an energy deficit, it is important to examine food consumption-related differences in food reward sensitivity and inhibitory control when comparing to a normal inter-meal hunger state. Moreover, previous studies have examined how obesity modulates effects of food consumption on food reward sensitivity, but whether potential food consumption-related differences in food reward sensitivity or inhibitory control depend on the tendency for uncontrolled eating in a normal-weight population remains unclear.

1.3 Current Study

We will therefore investigate 1) if individual differences in uncontrolled eating influence food reward sensitivity and food-related inhibitory control in healthy women; 2) whether food consumption influences food reward sensitivity and food-related inhibitory control; and 3) whether effects of food consumption are further modulated by individual differences in uncontrolled eating, that is, whether there is an interaction between hunger state and individual differences in uncontrolled eating. We compared healthy women who report low to average levels of uncontrolled eating behaviour (i.e., low UE group) to women who report higher levels of uncontrolled eating (i.e., high UE group). We focussed on women because in the general population, women (13.7%) more often than men (5.5%), report features such as uncontrolled eating (Mustelin et al., 2017). First, this will allow us to examine if individual differences in both food reward sensitivity and inhibitory

control are relevant for understanding differences in non-clinical uncontrolled eating. Second, we can investigate if food consumption also changes sensitivity to food cues and inhibitory control when comparing to an inter-meal hunger state rather than a fasted state. Finally, by investigating whether potential effects of food consumption on food reward sensitivity and inhibitory control depend on differences in uncontrolled eating, we could present a possible explanation for why some people find it harder to stop (over)eating than others.

Based on previous work on obesity and individual differences in (trait) food craving (García-García et al., 2013; Ulrich et al., 2016; Verdejo-Román et al., 2017), we expected to find greater food cue related activity in areas of the reward system, including striatal areas, ACC, insula, OFC, lateral and medial PFC, and amygdala, in individuals with higher levels of uncontrolled eating, relative to individuals with low to average levels of uncontrolled eating. In terms of food reward sensitivity we also expected that during the post food consumption state, as compared to the inter-meal hunger state, there would be attenuation of food cue related activity in these areas of the reward system (García-García et al., 2013; Stice et al., 2008; Verdejo-Román et al., 2017). Furthermore, we expected these effects of eating to be more pronounced in individuals with low to average levels of uncontrolled eating as compared to individuals who report higher levels of uncontrolled eating.

Our hypotheses regarding effects of food consumption on control-related processes are more explorative, as to date there is very little (behavioural and imaging) research on this and results are mixed. One behavioural study has shown that hungry, healthy participants made more commission errors in a go-nogo task when responses had to be inhibited to food-associated words as compared to when non-food-associated words were the nogo-category, which was not observed in satiated participants (Loeber et al., 2013). On the other hand, a behavioural study that compared healthy women who had not consumed any food for at least 3 hrs to those who had consumed a bowl of kefir, found no effects of food consumption on go-nogo performance in a task with food and neutral images (Meule, Lutz, et al., 2014). When considering food reward sensitivity and inhibitory control not to be independent of each other, we would expect that inhibitory control is higher—

operationalized as less commission errors—1) in individuals with low to average levels of uncontrolled eating as compared to individuals with higher levels of uncontrolled eating, and 2) after snack consumption as compared to an inter-meal hunger state, especially for food stimuli. Finally, we would expect that this change after food consumption is more pronounced in individuals who report low to average levels of uncontrolled eating as compared to individuals with higher levels of uncontrolled eating.

To our knowledge, no imaging studies to date have directly investigated the effects of food consumption on inhibitory control. Similarly, imaging studies on individual differences in food-related inhibitory control are scarce. One study in adolescents found that more overweight adolescents showed reduced activation of frontal inhibitory regions, including frontal gyrus, ventrolateral and medial prefrontal cortex, and OFC, relative to leaner individuals (Batterink et al., 2010). Although this question is thus more exploratory, we expected greater control-related activity in individuals with low to average levels of uncontrolled eating, as compared to individuals with higher levels of uncontrolled eating, in a network that has been associated with response inhibition, including frontal gyrus, postcentral gyrus, inferior parietal lobule, temporal gyrus and middle/superior occipital gyrus (Batterink et al., 2010; Sebastian et al., 2013). Similarly, we expected greater control-related activity after food consumption, as compared to an inter-meal hunger state, in this fronto-parietal network, and that these effects of eating would be more pronounced in individuals with lower/average levels of uncontrolled eating, as compared to individuals who report higher levels of uncontrolled eating.

2. Method

2.1 Participants

A screening survey was completed by 208 individuals. Forty-six participants met the eligibility criteria according to the screening survey and were invited to participate in the study. Individuals were screened using the uncontrolled eating (UE) subscale of the Three-Factor Eating Questionnaire-R18 (Karlsson et al., 2000) and considered eligible for the study if they either scored

≤ 18 (low UE group) or ≥ 20 (high UE group). These cut-off scores for the TFEQ-R18 were determined based on a large community sample from the same area ($n = 237$; unpublished data) and reflected the lower half (i.e., 50th percentile and below) and upper third. A study that included a large sample ($n = 919$) of British women (Keskitalo et al., 2008) and had no BMI restrictions (the average BMI in that sample—25.8—was slightly higher than in our sample), reported an average score of 16.1 on the uncontrolled eating subscale. We thus compared individuals who report lower to middle levels of uncontrolled eating behaviour to individuals who report a higher tendency for uncontrolled eating behaviour. Additional inclusion criteria were right-handedness, female and aged 18-40 years old. Participants were excluded based on the following criteria: specific dietary needs or preferences (i.e., vegetarian, vegan, or diabetic), self-reported glucose intolerance, being on a diet with the aim to lose weight, food allergies for the snack provided, BMI below 18.5 or above 30 (i.e., being underweight or obese), having sought professional help for any mental health issue during the six months prior to participation, and contraindication for MRI scanning. We excluded individuals with a vegetarian or vegan diet as they may have aversive responses to certain food stimuli that contain meat. We included women who are overweight (BMI 25-29.9), because the average BMI for women in the United Kingdom is 27.2 (Conolly & Saunders, 2017).

The TFEQ-R18 was re-administered during the test session to check whether participants still met the selection criterion. Based on UE subscale scores from this assessment, one participant was excluded who did not meet the cut-off score. We also measured height and weight during the first test session to calculate BMI. One participant was excluded based on a BMI > 30 . One participant decided to stop the MRI scan during the first session and did not continue participation. Functional MRI data from three participants could not be analysed due to substantial movement. Data for these participants was excluded from further behavioural analyses as well. The final sample therefore consisted of 21 participants in the high UE group (age $M = 22.67$; $SD = 3.31$) and 19 in the low UE group (age $M = 23.84$; $SD = 4.43$). Participants were reimbursed for their time. The experiment was

approved by the Central University Research Ethics Committee of the University of Oxford (R47826/RE007).

2.2 Measurements

2.2.1 Screening Survey

The screening survey assessed the inclusion and exclusion criteria: age, gender, handedness, following a special diet, glucose intolerance, food allergies, restriction of food intake to control weight, and having sought professional help for an eating disorder and/or other mental health problems in the last six months. To calculate BMI for initial screening, we asked individuals to indicate their height and weight. Additionally, participants completed the items of the uncontrolled eating subscale from the TFEQ-R18 and we assessed MRI safety criteria.

2.2.2 Questionnaires.⁵

2.2.2.1 Three Factor Eating Questionnaire – R18. The Three Factor Eating Questionnaire (Karlsson et al., 2000) was used to measure three different eating styles: cognitive restraint eating, uncontrolled eating, and emotional eating. Participants indicate on a 4-point scale (one item is answered using an 8-point scale) either how true a statement is (e.g., ranging from ‘*definitely true*’ to ‘*definitely false*’) or the frequency of a certain behaviour (e.g., ranging from ‘*never true*’ to ‘*at least once a week*’). Scores for the cognitive restraint factor range from 6-28, for the uncontrolled eating factor from 9-36, and for the emotional eating factor from 3-12. The internal consistency of the TFEQ-R18 subscales in the current sample were good (restrained eating: $\alpha = 0.79$; uncontrolled eating: $\alpha = 0.90$; emotional eating: $\alpha = 0.88$).

2.2.2.2 Binge Eating Scale. Binge eating was assessed with the Binge Eating Scale (Gormally et al., 1982), which assesses behavioural, cognitive, and affective symptoms of binge eating. For each item participants select 1 of 3 or 4 statements that best describes the way they feel about problems controlling their eating behaviour. The total score ranges from 0-32, with higher scores

⁵ We also assessed mood at the start of each session using Visual Analogue Scales but this data will not be used to test the hypotheses of the current study.

indicating more severe binge eating symptoms. Internal consistency of the BES in the current sample was good ($\alpha = 0.85$).

2.2.2.3 Food Craving Questionnaire – Trait. Individual differences in food craving were measured with the short version of the Food Craving Questionnaire – Trait (Meule, Hermann, et al., 2014). Participants indicated to what extent items apply on a 6-point Likert scale ranging from 1 ‘*never*’ to 6 ‘*always*’. Total scores can range from 15-90. Internal consistency of the FCQ-T in the current sample was very good ($\alpha = 0.95$).

2.2.2.4 Food Craving Questionnaire – State. To assess state food craving we used the Food Craving Questionnaire – State (Cepeda-Benito et al., 2000). Participants indicate to which extent items apply to them at that moment on a 5-point scale ranging from 1 ‘*strongly disagree*’ to 5 ‘*strongly agree*’, resulting in a total score ranging from 15-75. The internal consistency of the FCQ-S in the present study was excellent (eating session first assessment: $\alpha = 0.93$; eating session second assessment: $\alpha = 0.92$; non-eating session: $\alpha = 0.93$).

2.2.2.5 Short UPPS-P Impulsive Behaviour Scale. Individual differences in impulsivity were assessed using the short version of the UPPS-P Impulsive Behavior Scale (Cyders et al., 2014; Lynam, 2013). Cyders et al. (2014) confirmed 5 factors of impulsivity, each consisting of 4 items: negative urgency, (lack of) premeditation, (lack of) perseverance, sensation seeking, and positive urgency. Participants rated the items on a 4-point scale ranging from 1 ‘*strongly agree*’ to 4 ‘*strongly disagree*’, resulting in a total score ranging from 20-80 with higher scores indicating more impulsive behaviour. For this study we only looked at the total score. Internal consistency of the SUPPS-P total score in the present study was good ($\alpha = 0.77$).

2.2.2.6 Grand Hunger Scale. During both test sessions we measured state hunger using the Grand Hunger Scale (Grand, 1968). The scale contains two open-ended items asking participants how many hours it has been since their last meal and how many hours it will be until their next planned meal. Additionally, two items ask participants to indicate how hungry they are at that moment on a scale ranging from 1 ‘*not hungry at all*’ to 7 ‘*extremely hungry*’ and how much of their

favourite food they could eat at that moment on a scale from 1 '*none at all*' to 6 '*as much as I could get*'.

2.2.2.7 Snack Rating. Following consumption of the snack, participants rated two 9-point Likert scales ranging from '*not at all*' to '*very much*' on how much they liked the snack and how full they feel at that moment.

2.2.3 Blood Glucose Level Measurement

We tested for differences in blood glucose between the high UE and low UE group before and after food consumption, because blood glucose regulation is part of metabolic homeostasis and measurement is relatively non-invasive. Blood glucose levels were measured with a finger prick test. Participants washed their hands before the measurement and gently massaged the finger to stimulate the blood flow. We used single-use lancets from Accu-Check Safe-T-Pro Plus (Roche, Mannheim, Germany) to obtain a drop of blood from the finger. The drop of blood was then applied onto a single-use test strip for a MediTouch2 blood glucose monitor (Medisana, Neuss, Germany). Blood glucose levels were measured in units of mmol/L.

2.2.4 Functional MRI Tasks

Tasks were programmed using Presentation® software v.18.3 (Neurobehavioral Systems, Berkeley, California). Pictures of food and non-food items for both tasks were selected from the Food-pics database (Blechert et al., 2014). None of the pictures in the food reward sensitivity paradigm overlapped with pictures in the go-nogo task. For a list of used pictures see Supplemental Table S1.

2.2.4.1 Food Reward Sensitivity. Food reward sensitivity was assessed with a passive viewing task in which participants were presented with blocks of food and non-food items. Thirty food pictures depicted appetizing food items (e.g., chocolate bar, hamburger, or ice-cream) and 30 non-food pictures depicted items that can be found around the house (e.g., stationary, furniture, a clock). The food pictures depicted high-caloric items that on average contain 421.7 kcal/100 grams ($SD = 120.77$). Food and non-food pictures were selected such that these categories were balanced

for valence, $t(58) = 0.83$, $p = .409$, arousal, $t(58) = 1.79$, $p = .078$, complexity, $t(58) = 1.07$, $p = .290$, and brightness, $t(58) = 0.11$, $p = .916$. In each block 10 pictures of either food or non-food items were presented in the centre of the screen on a white background. Each picture was presented for 3 s resulting in a block duration of 30 s. The task always started with a food block, and food and non-food blocks were presented in an ABAB order. Picture blocks were interleaved with the central presentation of a fixation cross which was displayed for 15 s. Participants were not required to press any buttons during the task but were instructed to pay attention to the pictures. The task finished with a bogus memory task (no fMRI data was collected during this memory task) in which participants were presented with eight food and non-food pictures for which they had to indicate whether the pictures had appeared during the task blocks or not. Participants were informed about this memory task before going into the scanner. The sole aim of this memory task was to encourage participants to pay attention to the presented pictures.

2.2.4.2 Food Go-Nogo Task. Inhibitory control was measured with a rapid event-related go-nogo task in which participants were presented with pictures of food and non-food items. Thirty-three food pictures depicted appetizing food items (e.g., chocolate cake, pastry, or ice-cream) and 33 non-food pictures depicted flowers and leaves. The food pictures depicted high-caloric items that on average contain 365.7 kcal/100 grams ($SD = 155.6$). Food and non-food pictures were selected such that these categories were balanced for valence, $t(64) = 1.68$, $p = .097$, arousal, $t(64) = 1.10$, $p = .275$, complexity, $t(64) = 0.30$, $p = .764$, and brightness, $t(64) = 0.32$, $p = .754$. The task consisted of five food go blocks and five non-food go blocks. During food go blocks, participants were required to respond as fast and accurately as possible, by pressing a button with their right index finger, when a food item was presented and to withhold their response when a non-food item was presented. Conversely, in the non-food go blocks participants were instructed to respond when a non-food item was presented and to withhold their response when a food item was presented. In each trial (see Figure 1) a picture was presented on screen for 500 ms and was then replaced by a fixation cross which was presented with a jittered duration ranging between 1100 and 2900 ms in steps of 100 ms

($M = 2003.25$ ms). Participants had a maximum of 1500 ms to respond from the onset of the trial. During each block, 40 trials were presented of which 75% were go trials and 25% nogo trials. Specific block instructions were presented before the onset of each block for 10 s. Food go blocks and non-food go blocks were presented in an ABAB order. The initial block was either a food go block or a non-food go block, which was counterbalanced across groups and session order.

Figure 1

Task design illustrating two consecutive trials of the food go block.

2.3 Procedure

2.3.1 Recruitment

Potential participants were recruited through flyers and online message boards from the university. Women who were interested in taking part emailed the main researcher and completed the screening survey. If inclusion criteria were met, a short follow-up phone call was arranged to double check MRI safety criteria. If all requirements were met, two test sessions were planned on separate days: the non-eat session and eat session. Session order (i.e., eat session first vs. non-eat session first) was counterbalanced across participants in each group. We planned the test sessions no more than one week apart, but for five participants (three from the low- and two from the high UE group) this period was longer (10-39 days apart) because participants required a rescheduling or because the scanner was out of use for a longer period due to technical problems.

2.3.2 Test sessions

For both test sessions participants were instructed to eat something 2 hrs prior to the test session and then refrain from eating. At the start of the first test session participants signed informed consent and received an elaborate explanation about the MRI scanning procedure and different scans/tasks to be performed. Before the scan, participants practiced the go-nogo task. Each practice block consisted of 12 trials, 9 go trials and 3 nogo trials. Additionally, height and weight were measured during the first session, which were used to calculate BMI. The MRI scanning procedure was identical across sessions: first the food reward sensitivity paradigm, then a structural scan, followed by the go-nogo task, and ending with acquiring fieldmaps. The procedure for each of the different sessions is described in more detail below. See Supplemental Figure S1 for a visual depiction of the procedure of the two sessions.

2.3.2.1 Non-Eat Session. Participants first completed a demographic survey (i.e., age, gender, and handedness) and baseline questionnaires: BES, FCQ-T-r, TFEQ-R18, SUPPS-P, and the Grand Hunger scale. Additionally, they filled out the survey checking MRI safety criteria. Participants then completed the FCQ-S, followed by a measurement of their blood glucose level. Afterwards, participants were prepared for the scanner and were fitted with earplugs before going into the scanner. Upon ending of the imaging procedure, the session was completed.

2.3.2.2 Eat Session. Participants started the session by completing the Grand Hunger Scale. This was followed by the first FCQ-S assessment and measurement of blood glucose level. Following this, participants consumed a triple chocolate muffin (~90 grams, 400 kcal). We looked at the effects of consumption of a high-caloric snack as binge-type meals are often palatable, high-caloric foods rich in fat and sugar (Yanovski et al., 1992). The amount of calories was comparable to what is advised for a breakfast meal (400 kcal) by the National Health Service (NHS, n.d.). Participants were asked to consume the muffin within 10 min and then complete the two snack rating scales afterwards. Thirty min after snack consumption (approx. 40 min after start of consumption) we measured state craving (FCQ-S) and blood glucose level once more. This time frame was chosen because in healthy individuals blood glucose typically begins to rise 10 min and peaks about 30-60

min after the start of a meal (American Diabetes Association, 2001; Rozendaal et al., 2018). During the 30 min wait time, participants completed the survey checking MRI safety criteria. Participants were prepared for the scanner in the same way as during the non-eat session. Upon ending of the imaging procedure, the session was completed.

2.4 fMRI Data Acquisition

Scanning was performed with a Siemens Magnetom Prisma 3T scanner (Siemens Medical Systems, Erlangen, Germany) and 32-channel head coil. After manual shimming, we started the food reward sensitivity paradigm. We used an echo-planar imaging (EPI) sequence (72 interleaved slices, voxel size = 2 x 2 x 2 mm, TR = 1570ms, TE = 30ms, GRAPPA acceleration factor = 2, multi-band acceleration factor = 3, FoV = 216mm, flip angle = 70°) orienting the field-of-view at approximately 30° with respect to the AC-PC line to minimize signal distortions in orbitofrontal areas. For the food reward sensitivity paradigm we acquired 583 volumes. During each session we then acquired T1-weighted 3D high resolution MPAGE images in the transversal plane (192 slices, voxel size = 1 x 1 x 1 mm, TR = 1900 ms, TE = 3.97 ms, TI = 904 ms, FoV = 192 mm, flip angle = 8°). Following the structural scan we started the go-nogo paradigm. We used the same EPI sequence as for the food reward sensitivity paradigm but collected 715 volumes. Finally, fieldmap images (phase difference and magnitude images) were acquired for B0 unwarping to aid registration of functional EPI images.

2.5 Data Analysis

2.5.1 Behavioural Data

Data was analysed using R (R Core Team, 2019). Measures of eating style, binge eating, trait food craving and impulsive behaviour were used to describe the participant sample, and we report correlations amongst these measures in the current sample. We used one-way ANOVAs to test whether the low and high UE groups differed on the baseline measures, how much they liked the snack and how full they felt after eating. Where parametric assumptions were violated we report the Wilcoxon Rank-Sum test.

Linear mixed effect models were tested with the nlme package (Pinheiro et al., 2018) to examine effects of snack consumption on state craving and blood glucose levels, and to investigate whether this differed across UE groups. We used maximum likelihood estimation to compare nested models with different fixed effects. Planned contrasts were set to compare levels before and after snack consumption in the eat session, and to compare levels before snack consumption in the eat session to levels in the non-eat session. We first built a model with a fixed effect of time on state food craving or blood glucose levels and included random intercepts (model 1). Building on this model, we included random slopes for time (model 2). Following this we included the main effect of UE group and its interaction with time (model 3).

To investigate behavioural changes in food-related inhibitory control, we examined the commission error rate (i.e., number of responses to nogo trials divided by total number of nogo trials), as commission errors are thought to reflect response inhibition for nogo stimuli (Veling et al., 2017). Linear mixed effects models were used to test whether the commission error rate was different depending on stimuli (non-food vs. food), session (eat vs. non-eat session), UE group (low vs. high), and their interactions. We first built a model with a fixed effect of stimulus and included random intercepts (model 1). Building on that model we included the effect of session and its interaction with stimulus, and added random slopes for session (model 2). Finally, we added the effect of UE group and all interactions between stimulus, session and UE group (model 3). Although our hypotheses were specific to commission errors, we also explored similar associations with reaction times (RT) for correct go-trials and omission errors (i.e., number of missed responses to go trials divided by total number of go trials) to determine if results were specific to inhibitory control or related to overall task performance (see supplemental material).

2.5.2 Imaging Data

fMRI data were analysed using FSL (Smith et al., 2004). We used FEAT in FSL for image pre-processing and first-level analysis, keeping standard settings. Motion correction was applied using the MCFLIRT tool (Jenkinson et al., 2002), which provided six motion regressors that were included

in the fMRI analyses as confound regressors. We corrected for the effects of large motion using the FSL motion outliers tool. Functional images were spatially smoothed using a Gaussian kernel with 4mm full-width half-maximum, and temporally high-pass filtered. A high-pass filter cut-off for the food reward sensitivity paradigm was set to 90s and for the go-nogo task it was set to 128s. B0 unwarping was carried out using boundary-based registration (Greve & Fischl, 2009) in FMRIB's Linear Image Registration Tool (Jenkinson et al., 2002; Jenkinson & Smith, 2001) which performs registration to the structural image and simultaneously incorporates fieldmap correction. High resolution structural images were processed using the Brain Extraction Tool (Smith, 2002) to separate brain tissue from non-brain tissue and were then registered to Montreal Neurological Institute (MNI) space using FMRIB's Nonlinear Image Registration Tool (Andersson et al., 2010). Registration followed a two-step process: functional images were first registered to the high-resolution structural images using BBR with fieldmaps and then the structural image was registered to standard space. The data were pre-whitened before first-level analysis.

Individual protocol time files were created for first-level analysis (GLM), that represented the onset and duration of task events. For the food reward sensitivity paradigm we specified two regressors, indicating the onset and duration (i.e., 30 s) of the food blocks and non-food blocks. For the go-nogo task we specified 6 regressors, indicating the onset and duration of the different trial types (i.e., food go, non-food go, food nogo, and non-food nogo; duration 0.5 s), the onset and duration (i.e., 0.5 s) of incorrect trials, and the onset and duration of instructions throughout the task. Six motion regressors from MCFLIRT and the confound matrix from FSL motion outliers were added as (confound) regressors of no interest. The blood-oxygen-level-dependent (BOLD) response was modelled by convolving the neural functions with a double-gamma hemodynamic response function. Temporal derivatives were added, and the same temporal filtering was applied to the model as was applied to the data. For the food reward sensitivity paradigm, we set four contrasts: mean food, mean non-food, food > non-food, and non-food > food, with food > non-food being the main contrast of interest. For the go-nogo task we set eight contrasts: mean food go, mean non-food

go, mean food nogo, mean non-food nogo, food nogo > food go, non-food nogo > non-food go, food go > food nogo, and non-food go > non-food nogo. To explore the effects on inhibitory control we contrasted nogo activations to go activations, that is, the nogo > go contrasts were the main contrasts of interest. Results were cluster-corrected with cluster z threshold = 2.3 and $p < 0.05$.

First-level analyses were used as input for group-level whole-brain analyses. For inference on second-level analyses, we performed permutation tests with 5000 permutations and threshold free cluster enhancement TFCE using Randomise v2.9 (Winkler et al., 2014). We first performed one sample t-tests (i.e., group mean) on the contrasts of interest, comparing food to non-food stimuli from the food reward sensitivity paradigm, and on nogo > go trial contrasts from the go-nogo task. To test our research questions, we performed a two-sample unpaired t-test to compare the low and high UE group (main effect UE group), and a paired sample t-test for changes across the eating and non-eating session (main effect session) on the contrasts of interest. The Group x Session interaction was tested by performing two-sample unpaired t-tests on the session difference, to test if changes across the eating and non-eating session differed between the low and high UE group.

3. Results

3.1 Behavioural Data

3.1.1 Descriptive Statistics

See Table 1 for Spearman correlations (not all data was normally distributed) and descriptive statistics on baseline measures of binge eating, trait food craving, eating styles, impulsive behaviour, and BMI. We find positive correlations between uncontrolled eating, emotional eating, binge eating, trait food craving and impulsive behaviour. BMI was not significantly related to the other variables, possibly because we restricted the BMI range.

Table 1*Descriptive statistics and correlations between baseline variables.*

Variable	1	2	3	4	5	6	7
1. Binge Eating (BES)	-	0.84***	0.37*	0.68***	0.76***	0.41**	0.29
2. Food Craving trait (FCQ trait)		-	0.27	0.84***	0.79***	0.43**	0.21
3. Restraint Eating (TFEQ-R18)			-	0.30	0.19	-0.06	0.14
4. Uncontrolled Eating (TFEQ-R18)				-	0.66***	0.35*	0.10
5. Emotional Eating (TFEQ-R18)					-	0.40**	0.13
6. Impulsive Behaviour (SUPPSP total)						-	0.07
7. BMI							-
Low UE group <i>M (SD)</i>	4.05 (3.66)	27.79 (8.40)	13.47 (4.09)	14.84 (2.50)	5.11 (1.91)	37.21 (6.01)	22.17 (2.56)
High UE group <i>M (SD)</i>	10.67 (5.10)	44.62 (8.87)	15.14 (2.97)	24.24 (2.96)	7.90 (2.19)	41.14 (5.46)	21.97 (2.44)

* $p < .05$; ** $p < .01$; *** $p < .001$.**3.1.2 Group Differences**

The high UE group scored higher as compared to the low UE group not only on uncontrolled eating, $W = 0$, $p < .001$, $r = -0.86$, but also on emotional eating, $F(1,38) = 18.38$, $p < .001$, $\omega^2 = 0.30$, binge eating, $W = 52$, $p < .001$, $r = -0.63$, trait food craving, $W = 19$, $p < .001$, $r = -0.77$, and on impulsive behaviour, $F(1,38) = 4.71$, $p = .036$, $\omega^2 = 0.09$. Groups did not significantly differ in terms of cognitive restraint eating or BMI, $ps > .05$.

UE groups did not differ in the number of hours they ate something prior to the non-eating session (low: $M = 3.11$, $SD = 2.94$; high: $M = 2.69$, $SD = 1.76$), or eating session (low: $M = 2.26$, $SD = 0.71$; high: $M = 2.33$, $SD = 0.64$). The groups neither differed on self-reported hunger level at the start of the non-eating session (low: $M = 3.21$, $SD = 1.13$; high: $M = 3.57$, $SD = 1.21$) or eating session (low: $M = 2.79$, $SD = 1.51$; high: $M = 3.38$, $SD = 1.07$), all $ps > .10$. All participants indicated to have

eaten something at least 2 hours prior to both sessions. The low and high UE groups did not differ in how much they liked the consumed snack (low: $M = 7.47$, $SD = 0.96$; high: $M = 7.71$, $SD = 1.85$), nor in how full they felt afterwards (low: $M = 6.63$, $SD = 1.30$; high: $M = 6.57$, $SD = 1.33$), all $ps > .10$.

Figure 2

A) Levels of state food craving across the different sessions; B) Blood glucose levels across the different sessions. Error bars represent standard errors of the mean.

3.1.3 Effects of Snack Consumption

We tested the effect of snack consumption on state craving (FCQ-S) and blood glucose levels, and whether this differed across UE groups. We compared levels before and after snack consumption in the eat session, and levels before snack consumption in the eat session to levels in the non-eat session. Model comparison for effects on state craving showed that the final model had the best (relative) fit, $\chi^2(3) = 20.90$, $p < .001$. There was a main effect of time for the comparison before and after snack consumption, $b = -3.58$, $t(76) = -2.67$, $p = .009$, but not for the comparison of state craving before consumption in the eating session to the non-eating session, $b = 0.11$, $t(76) = 0.05$, $p = .963$. Additionally, there was a main effect of UE group, $b = 11.70$, $t(38) = 4.52$, $p < .001$, and UE group further modified the effect of time for the comparison before and after snack

consumption, $b = -5.94$, $t(76) = -3.21$, $p = .002$. Both groups showed a significant decrease in food craving after snack consumption, but this was more pronounced in the high UE group, possibly because the low UE group started off with lower levels of state food craving (see Figure 2A). After consumption, the high UE group reported greater state food craving, $t(37.84) = 2.38$, $p = .022$.

Model comparison for tests on blood glucose level showed that adding random slopes to the baseline model which included random intercept and main effect of time did not improve fit, $\chi^2(5) = 5.10$, $p = .404$, nor did adding UE group further improve fit, $\chi^2(3) = 1.17$, $p = .761$. The baseline model showed a main effect of time for the comparison of blood glucose levels before consumption in the eating session to the non-eating session, $b = -0.45$, $t(74) = -2.92$, $p = .005$. Blood glucose levels did not significantly increase across 30 min after snack consumption, $b = 0.29$, $t(74) = -2.92$, $p = .060$, but the effect was in the expected direction. Blood glucose levels tended to be higher after snack consumption and blood glucose levels in the non-eating session were lower than baseline blood glucose levels during the eating session, but this was not different across groups (see Figure 2B).

3.1.4 Go-Nogo Task

Go-nogo data from one participant was missing because the MRI scan was stopped and data from three participants was excluded from further analyses due to the large amount of errors committed on nogo trials (i.e., more than 2.5SDs from the mean in the UE group), resulting in $n = 36$. We tested whether the commission error rate was different between food and non-food stimuli, across the two sessions, and whether this was modulated by UE group. Model comparison for tests on commission error rate showed that adding effects of session and random slopes to the baseline model which included random intercept and main effect of stimulus did not improve fit, $\chi^2(4) = 7.47$, $p = .113$, nor did adding UE group further improve fit, $\chi^2(4) = 3.61$, $p = .461$. The baseline model showed a main effect of stimulus, $b = -0.03$, $t(107) = -3.60$, $p < .001$, reflecting that overall less commission errors were made for food stimuli, but there was no effect of session, UE group nor their interactions (see Figure 3). Exploratory analyses on RT for correct go-trials and omission errors can be found in supplemental material.

Figure 3

Commission error rates across the different sessions and UE groups. Error bars represent standard errors of the mean.

3.2 Imaging Data

3.2.1 Food Reward Sensitivity

Overall (i.e., across all participants and during both sessions), exposure to food stimuli, as compared to non-food stimuli, was related to activity in areas including visual cortex, insula, amygdala, left inferior temporal gyrus/fusiform gyrus, and left OFC (see Figure 4, Supplemental Table S2). However, permutation tests did not reveal significant differences between the low and high UE group, no significant difference between the eating and non-eating session, nor was the effect of session further modulated by UE group.

Exploratory analyses on UE group differences *within* the eating and non-eating session, based on previous work in obese individuals (Dimitropoulos et al., 2012), are reported in supplemental material, including Supplemental Figure 2. In the eating session, the high UE group, as

compared to the low UE group, showed increased activity in response to food stimuli (vs. non-food stimuli) in a small cluster in the dorsal part of the left ACC. No group differences were found in the non-eating session.

Figure 4

Neural activation for food > non-food contrast (permutation tests TFCE-corrected).

3.2.2 Inhibitory Control

Across all participants and both sessions, response inhibition for food stimuli (food nogo > food go) activated a wide network of brain areas including the right pre- and postcentral gyrus extending to frontal gyrus, temporal occipital fusiform gyrus and inferior temporal gyrus, right frontal operculum, right dorsal anterior cingulate gyrus, and superior parietal lobule and anterior intra-parietal sulcus (see Figure 5, Supplemental Table S3). However, permutation tests did not reveal significant differences between UE groups, sessions, nor were effects of session modulated by UE group.

Figure 5

Neural activation for food nogo > food go contrast (permutation tests TFCE-corrected).

Across all participants and both sessions, response inhibition for non-food (non-food nogo > non-food go) also activated a wide network of brain areas including right precentral gyrus extending to postcentral gyrus and superior frontal gyrus, right inferior parietal lobule/ supramarginal gyrus and superior temporal gyrus, temporal-occipital/occipital fusiform gyrus and inferior temporal gyrus, frontal operculum/insula, right cuneal cortex, left amygdala, and right paracingulate gyrus (see Figure 6, Supplemental Table S4).

Figure 6

Neural activation for non-food nogo > non-food go contrast (permutation tests TFCE-corrected).

There were no differences between the UE groups that reached above threshold activation, nor any differences between sessions, but there was a significant Session (eating vs. non-eating) x Group (low vs. high UE group) interaction for response inhibition for non-food stimuli that was related to activity in a small cluster in the left lateral, superior occipital cortex. This interaction seemed driven by relatively more activity in the left lateral, superior occipital cortex for this contrast (i.e., non-food nogo > non-food go) during the non-eat session as compared to eat session in the low UE group, whereas in the high UE group there was relatively more activity during the eat session as compared to the non-eat session (see Figure 7).

Figure 7

Maximum percent BOLD signal change in the functionally defined ROI in the left superior parieto-occipital cortex, based on the Session x Group interaction effect on the non-food nogo > non-food go contrast. Error bars represent standard errors of the mean.

4. Discussion

In the current study we investigated effects of food consumption on food reward sensitivity and (food-specific) inhibitory control, and how this differed depending on non-clinical levels of uncontrolled eating. Our results on food reward sensitivity showed that generally the response to high-caloric foods (vs. non-food) was associated with activity in the visual cortex, insula, amygdala, left inferior temporal gyrus/fusiform gyrus, and left orbitofrontal cortex, consistent with previous fMRI research on visual food cues (for review see García-García et al., 2013). However, differences in the tendency for uncontrolled eating and snack consumption did not significantly change BOLD activity when viewing food cues. In terms of performance on the response inhibition task, we did not see any behavioural differences in food-related inhibitory control across sessions, nor based on levels of uncontrolled eating. Imaging data showed that generally response inhibition for both food and non-food stimuli activated a wide fronto-parietal-occipital network. This network is consistent with previous findings on the go-nogo task when nogo-trials are infrequent (Kolodny et al., 2017; Sebastian et al., 2013) and overlaps with brain areas previously associated with food-specific inhibitory control (Batterink et al., 2010). Our main finding showed differences in activity during response inhibition of non-food stimuli in the superior occipital gyrus, depending on an interaction between session and the tendency for uncontrolled eating.

Contrary to expectations, neither differences in uncontrolled eating behaviour nor food consumption seemed to influence food reward sensitivity. Previous work on food reward sensitivity has shown that individual differences in weight status can influence the response to food cues, showing greater activity in reward-related brain regions in response to food stimuli in individuals who are overweight or obese, as compared to healthy weight individuals (García-García et al., 2013; Stice et al., 2008; Verdejo-Román et al., 2017). Considering our results it is possible that individual differences in (non-clinical) uncontrolled eating—in the absence of obesity—do not have a strong influence on food reward reactivity. Similarly, research has shown that satiation can influence the response to visual food cues with greater activity in reward-related brain areas when exposed to

high-caloric foods (vs. low-caloric foods/non-foods) in a fasted state, than when in a satiated state (Führer et al., 2008; Goldstone et al., 2009; LaBar et al., 2001). However, in all of these studies individuals had fasted for at least 8 hrs or more. It is therefore hard to know if these effects of satiation can be generalized to understand what is underlying ‘hedonic eating’ and uncontrolled eating, which involve eating also in the absence of physiological hunger. One other study that compared inter-meal hunger to a satiated state showed attenuated activity to food stimuli (tasting fluids with pleasant, neutral and unpleasant taste) in reward related brain areas (Thomas et al., 2015), but the food intake was much higher with an average > 1000 kcal. It is thus possible that a much longer fasting period and/or more substantial food intake is necessary to see any significant *changes* in activity in reward related brain areas, but that such changes in the neural response are not apparent in the context of an inter-meal hunger state and consuming a snack with equivalent calories to a breakfast (NHS, n.d.).

The ability to self-regulate could be relevant to ‘overcome’ reward-related responses to food, reflected in individual differences in (food-specific) inhibitory control. However, behavioural performance in inhibitory control was not influenced by differences in the tendency for uncontrolled eating, nor different between an inter-meal hunger state and after snack consumption. We found that across both sessions participants made less commission errors for food stimuli as compared to non-food stimuli. Previous research has actually shown differences between commission errors for food and non-food stimuli in both directions across two studies (Meule, Lutz, et al., 2014). Exploratory analyses on reaction times for go-trials showed that participants were generally faster for non-food go-trials than for food go-trials. It thus seems that participants were generally faster in correctly responding in non-food go blocks and made less errors inhibiting their responses to food nogo-trials, i.e., overall better performance in dominant non-food ‘go’ blocks. Importantly though, behavioural effects on inhibitory control (i.e., commission errors) were not influenced by food consumption, nor differences in uncontrolled eating.

Whereas several studies have shown a link between individual differences in weight status and performance on food-specific response inhibition tasks (e.g., Batterink et al., 2010; Lavagnino et al., 2016; Nederkoorn et al., 2006; Price et al., 2016), it is possible that individual differences in levels of uncontrolled eating—in the absence of obesity—do not influence food-specific response inhibition. This is supported by previous findings that obesity was associated with reduced inhibitory control, but that the presence or absence of binge eating disorder did not impact this effect (Lavagnino et al., 2016). One other study that directly tested effects of food consumption on inhibitory control, compared healthy women who had not consumed food for at least 3 hrs to those who had consumed a bowl of kefir (lower in calories than our snack). They also found no effects of satiation on performance in a go-nogo task with food and neutral images (Meule, Lutz, et al., 2014).

We extend previous research, showing for the first time how food consumption can influence neural activation underlying (food-specific) inhibitory control. We found that individuals with a low/average tendency for uncontrolled eating showed relatively more activity in the left superior occipital gyrus when they had to inhibit responses to non-food stimuli in an inter-meal hunger state as compared to after snack consumption. On the other hand, individuals who reported a higher tendency for uncontrolled eating showed relatively more activity in this area after snack consumption (vs. an inter-meal hunger state). Activity in this area has previously been associated with successful nogo- vs. go-trials when using neutral stimuli (Kolodny et al., 2017). Yet, behavioural performance was similar across sessions. Although speculative, it is possible that individuals with a low/average tendency for uncontrolled eating needed greater cognitive resources to support successful response inhibition of non-food stimuli in blocks with a dominant food ‘go’ response, during an inter-meal hunger state when appetitive drive is increased (as compared to after food consumption). On the other hand, individuals with a higher tendency for uncontrolled eating showed such potential compensatory effects after eating, as if response inhibition during dominant food ‘go’ blocks becomes less efficient or more challenging after eating, requiring extra cognitive

resources. Such 'hidden costs' of inter-meal hunger on inhibitory control efficiency can only be observed when examining (differences in) neural activation during an inhibitory control task.

It is important to consider the limitations of the study when interpreting the effects. Our sample consisted of young, non-obese adult women who have not sought help for an eating disorder or other mental health issues in the last six months. Although we included individuals who report differing levels of uncontrolled eating behaviour, this restricts generalizability of our findings to people with obesity or binge eating disorder. However, it is important to understand if and what factors can contribute to individual differences in eating behaviour in the general healthy population if this could be a risk factor for increased weight or clinical binge eating. Second, self-reported uncontrolled eating was highly correlated with emotional eating, binge eating, and trait food craving. Although the overlap between these concepts is not surprising, it does mean that the current findings may not be specific to uncontrolled eating but could also be expected as a function of trait food craving or (non-clinical) binge eating behaviour more broadly. Third, participants consumed a high-caloric snack rather than a full meal which has mostly been used in previous research, but this is in keeping with binge-type foods that are often rich in fat and sugar (Yanovski et al., 1992). Moreover, the amount of calories is comparable to what is advised for a breakfast meal by the National Health Service (NHS, n.d.) and participants reported a significant decrease in state food craving after consumption. Additionally, we measured changes in blood glucose levels and food craving in response to food consumption as markers of satiety or appetite differences. Although blood glucose regulation is part of metabolic homeostasis we did not measure any levels of satiety or appetite-related peptides (e.g., ghrelin, cholecystokinin, glucagon-like peptide-1) and how these may differ across individuals, but measuring these peptides is difficult and it remains unclear whether it is a better marker of satiety than self-report (Gibbons et al., 2019). We thus had no physiological measure of appetite or satiety, but measured self-reported hunger levels at the start of each session and subjective food craving before and after snack consumption. Finally, due to planning constraints in MRI data collection we were unable to keep time between sessions constant,

but we tested most participants no more than one week apart and order of sessions was counterbalanced.

To conclude, we investigated effects of individual differences in uncontrolled eating and food consumption on food reward sensitivity and (food-specific) inhibitory control. We did not find any effects of differences in the tendency for uncontrolled eating, snack consumption, or their interaction on BOLD activity when viewing food and non-food cues. In terms of control-related processes, we did not see any differences in behavioural performance on the response inhibition task as a result of food consumption, nor based on levels of uncontrolled eating. We did find differences in BOLD activity during response inhibition of non-food stimuli associated with food consumption, that depended on the tendency for uncontrolled eating. It seems that individuals with a low/average tendency for uncontrolled eating needed greater cognitive resources to support response inhibition performance during dominant food 'go' blocks in an inter-meal hunger state, whereas individuals with a higher tendency for uncontrolled eating showed such potential compensatory effects after eating. We thus observed small differences depending on non-clinical levels of uncontrolled eating, modulated by food consumption, in inhibitory control which could contribute to finding it harder to stop once eating. However, based on our findings it does seem that non-clinical levels of uncontrolled eating have no, or only limited influence on food reward sensitivity and food-related inhibitory control, whereas previous research suggests differences in weight status (e.g., obesity) may have a bigger impact.

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Credit Author Statement

Maud Grol: Conceptualization, methodology, validation, formal analysis, investigation, data curation, writing – original draft, visualization, project administration; **Luis Cásedas:** methodology, investigation, writing – review & editing; **Danna Oomen:** methodology, resources, writing – review & editing; **Desiree B. Spronk:** conceptualization, methodology, writing – review & editing; **Elaine Fox:** Conceptualization, supervision, funding acquisition, writing - review & editing.

Data Availability

The neural datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request. Behavioural data and R scripts are available on the Open Science Framework: <https://osf.io/wn7ch/>

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Supplemental Material

Exploratory Analyses Behavioural Data Go-Nogo Task

We used linear mixed effects models to explore whether RTs on correct go-trials and omission error rate was different depending on stimuli (non-food vs. food), session (eat vs. non-eat session), UE group (low vs. high), and their interactions. We first built a model with a fixed effect of stimulus and included random intercepts (model 1). Building on that model we included the effect of session and its interaction with stimulus, and added random slopes for session (model 2). Finally, we added the effect of UE group and all interactions between stimulus, session and UE group (model 3).

Exploratory analyses on omission errors, showed that there were no effects of stimulus, session, UE group, or their interactions, all $ps > .10$. Model comparison for RTs on correct go-trials showed that the full model with effects of stimulus, session, UE group and their interactions showed the best fit, $\chi^2(4) = 15.96$, $p = .003$. There was a significant main effect of stimulus, $b = 39.76$, $t(102) = 7.17$, $p < .001$, reflecting overall higher RTs to food go-trials, but no significant effect of session, $b = 15.93$, $t(102) = 1.62$, $p = .108$, UE group, $b = -2.12$, $t(34) = -0.10$, $p = .922$, Stimulus x Session interaction, $b = -13.66$, $t(102) = -1.74$, $p = .085$, nor a Stimulus x UE group interaction, $b = -13.11$, $t(102) = -1.76$, $p = .081$. There was a significant Session x UE group interaction, $b = -40.08$, $t(102) = -3.04$, $p = .003$, and the full Stimulus x Session x UE group interaction was also significant, $b = 28.93$, $t(102) = 2.75$, $p = .007$. Follow-up analyses showed that the difference between RTs on food go-trials vs. non-food go-trials was smaller during the eat session than the non-eat session for the low UE group, $t(15) = -2.28$, $p = .038$, whereas the difference between RTs on food go-trials vs. non-food go-trials was greater during the eat session than the non-eat session for the high UE group, $t(19) = 2.96$, $p = .008$. This seems driven by faster RTs to non-food go-trials by the high UE group vs. low UE group, only during the eat session, $t(25.60) = -2.50$, $p = .019$.

Exploratory Analyses Imaging Data Food Cue Sensitivity

Main analyses on imaging data from the food cue sensitivity task showed no effect of UE group, nor a UE group x Session interaction; however, we explored UE group differences within each session (i.e. eating and non-eating session) based on previous work in obese individuals (Dimitropoulos et al., 2012). Whereas there was no difference between the low and high UE group during the non-eating session, the high UE group showed more activity in response to food stimuli, as compared to the low UE group, in a small cluster in the dorsal part of the left ACC during the eating session (cluster size = 29; t -value = 4.61, cluster p -value = .048; MNI $x = -5$, $y = 40$, $z = 22$). Further inspection of this effect, suggests it was likely driven by the low UE group showing more activity in left ACC in response to non-food stimuli, whereas the high UE group showed more activity in left ACC to food stimuli (see Supplemental Figure 2).

People with a higher tendency for uncontrolled eating thus seemed to show increased activity in left (dorsal) ACC in response to food cues after eating a high-caloric snack, as compared to individuals who report a low/average tendency for uncontrolled eating. Increased activity in ACC has been consistently shown in response to appealing foods (for review see García-García et al., 2013), with obese individuals showing relatively greater reactivity in ACC in response to high-palatable foods (Verdejo-Román et al., 2017). Moreover, obese individuals, as compared to normal weight individuals, have also showed more activity in (amongst other regions) left ACC in response to high-caloric foods vs. non-foods *after a meal* (Dimitropoulos et al., 2012). We extend these previous findings, showing that whilst during an inter-meal hunger state no differential activity was shown, individuals with a heightened tendency for uncontrolled eating showed a relatively increased response to food cues after eating, in an area implicated in food cue sensitivity and reward processing and reinforcement-guided decision making more broadly (Kennerley et al., 2006; Kolling et al., 2016; Walton et al., 2007). However, these results should be interpreted with caution given that the cluster of activity was very small.

Supplemental Figures

Figure S1

Visual depiction of the experimental procedure during the non-eat session and eat session.

Figure S2

Maximum percent BOLD signal change in the functionally defined ROI in the left ACC, based on the group modulation effect on the food > non-food contrast during the eating session. Error bars represent standard errors of the mean.

Supplemental Tables

Table S1

Images used from Food-pics database in food cue sensitivity paradigm and food go-nogo task.

Food cue sensitivity		Food go-nogo	
Picture nr	Description	Picture nr	Description
41	Donut with chocolate sprinkles	4	Chocolate cookie
63	Pretzels	7	Croissants
67	Ice cream	10	Spaghetti with tomato sauce
70	Cheese	18	German krapfen (pastry)
109	Peanuts	33	Spaghetti with tomato sauce
116	Chocolate popsicle with nuts	35	Coconut wafer balls
117	Crisps	38	Chocolate and strawberry ice cream
123	Lollipops	56	Chocolate and vanilla ice cream
135	Cake	66	Croissants
147	Marshmallows	83	Filled chocolates
148	Round shortbread cookies	107	Chocolate cake
180	Muesli bar	115	Sundae with raspberries
186	Pretzel sticks	137	Chocolate muffin
189	Slice of bread with chocolate cream	143	Pasta bake
286	Bar of chocolate	151	Lemon cake
297	M&m's peanut	156	Braided yeast bun with raisins
331	Marble cake	157	Gummi bears candy
351	Croissant	158	Cookies filled with chocolate cream
354	Blueberry and strawberry tart	178	German krapfen (pastry)
410	Peanuts	183	Peanuts
420	Raspberry cream cake	290	Bars of white chocolate
427	Walnuts	291	Chocolate truffle
485	Burger patty with French fries	294	Popcorn
488	Waffle with whipped cream	302	Fish sticks
494	Pretzel	304	Potato gratin
500	Chocolate bar	374	Doughnut with icing
503	Shortbread	377	Doughnut
504	Shortbread	416	Pistachios
506	Cookie	443	Croissant
533	Cookie	474	Croissant with butter and jam
1012	Cushion	539	Almonds
1029	Pocketknife	549	Bagel
1031	Telephone	551	Spaghetti with pesto
1035	Books	1280	Orchid
1036	Sponge	1281	Orchid
1147	Briefcase	1282	Leaf
1151	Pen, open	1283	Orchid
1156	Calculator	1284	Flower
1202	Suitcase	1285	Leaf
1218	Chair	1286	Leaf
1219	Chair	1287	Water lily
1223	Wooden case	1288	Aloe vera

1224	Tin can	1289	Leaf
1225	Pocket watch	1290	Leaf
1227	Wooden bowl	1291	Leaf
1232	Candle	1292	Leaf
1238	Flat iron	1293	Pine cone
1240	Umbrella	1294	Cloverleaf
1241	Fan	1295	Flower
1244	Pot	1296	Holly
1247	Keys	1297	Flower
1251	Paint brushes	1298	Anemone
1256	Flash light	1299	Leaf
1258	Wall clock	1300	Protea
1265	Pocket watch	1301	Flower
1266	Swiss pocket knife	1302	Cloverleaf
1273	Cabinet	1303	Ginko leaf
1275	Pocket watch	1304	Orchid
1279	Candle	1305	Leaf
1313	Bicycle	1306	Flower
		1307	Leaf
		1308	Leaf
		1309	Twig
		1310	Leaf
		1311	Leaf
		1312	Leaf

Session x Group	No above threshold activations
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Table S4

Cluster, peak voxel (incl. local maxima) and label information of non-food no-go > non-food go contrast in the go-nogo task. Permutation test TFCE-corrected, $p < 0.05$.

Effect	Cluster size	Cluster p-value	t-value	Label peak voxels	MNI coordinates		
					X	Y	Z
non-food nogo > non-food go	21462	.002	6.85	Right precentral gyrus, primary motor cortex	46	-9	47
			6.85	Right precentral gyrus, primary somatosensory/motor cortex	48	-10	49
			6.84	Right precentral gyrus, premotor cortex	50	3	49
			6.68	Right precentral gyrus, premotor cortex	47	2	46
			6.34	Right precentral gyrus, premotor cortex	44	1	43
			6.25	Right precentral gyrus, premotor cortex	52	-2	50
	11237	.008	6.5	Right inferior parietal lobule, supramarginal gyrus	65	-44	19
			5.79	Right inferior parietal lobule, supramarginal gyrus	64	-39	30
			5.74	Right central operculum	49	-6	2
			5.64	Right supramarginal gyrus, superior temporal gyrus	50	-36	10
			5.6	Right supramarginal gyrus, superior temporal gyrus	51	-38	13
			5.22	Right lateral occipital cortex	49	-65	5
	7324	.011	5.96	Right temporal occipital fusiform gyrus, inferior temporal gyrus	46	-54	-17
			5.54	Right temporal occipital fusiform gyrus	38	-39	-18
			5.52	Right occipital fusiform gyrus	25	-65	-5
			5.48	Right inferior temporal gyrus	45	-50	-12
			5.38	Right lingual gyrus	21	-46	-10
			5.05	Right occipital fusiform gyrus	28	-73	-6
	3083	.008	6.09	Right insula, frontal operculum	32	25	7
			6	Right frontal orbital cortex, insula	31	28	4
			5.51	Right frontal operculum	35	21	11
			5.5	Right insula	34	21	4
			5.45	Right frontal operculum	45	17	1
			5.43	Right insula	34	19	-7
	427	.042	4.32	Right cuneal cortex	15	-75	25
			4.17	Right cuneal cortex	20	-75	35
			4.02	Right lateral, superior occipital cortex	14	-84	38
			3.98	Right cuneal cortex	17	-72	23