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The impulsive brain: Neural underpinnings of binge eating behavior in normal-weight adults

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ABSTRACT

Converging evidence suggests that dysfunctional inhibitory control might be at the roots of overeating and binge eating disorder (BED). The majority of these results stems from studies on obese populations, however we hypothesized that potential prodromes might be evident also in non-clinical conditions, when binge eating episodes are present (without a diagnosis of BED) and a normal Body Mass Index is preserved.

To explore this issue, brain activity of 42 normal weight individuals with and without binge eating episodes (21 binge eaters and 21 non-binge eaters, BE and non-BE respectively) was assessed by means of functional magnetic resonance imaging (fMRI) during response inhibition tasks. We adopted a food-modified version of a go/no-go (GNG) and stop signal task (SST): these tasks investigate different aspects of inhibitory control (action restraint and cancellation) that have been rarely studied in the same individuals but that are known to involve different neural networks. In addition, impulsivity traits were assessed with self-report instruments.

Despite similar behavioral performances, the two groups differed in trait impulsivity and brain activity. The fMRI results revealed differential engagement of fronto-striatal regions between the groups during the tasks. The BE group, compared to non-BE, showed lower activation of the right middle frontal gyrus (MFG) and Putamen during the GNG task, and higher activation of the left MFG during the SST.

These findings provide evidence of a dissociation of the neural underpinnings of action restraint and cancellation in impulsive individuals. Moreover, they add support to the hypothesis that impulsivity may be a possible hallmark of binge eating behavior (in the absence of weight or full-blown eating disorders) and yield new insights on the role of regions typically involved in response inhibition and selection as possible substrates of impulsive eating.

1. Introduction

Due to the wide availability of highly palatable and calorically dense foods, an alarming number of people worldwide is becoming overweight and obese (World Health Organization - WHO, 2016). For some individuals the ability to control their eating habits and resist tempting foods represents a challenge, leading them to experience episodes of binge eating. This term refers to the consumption of a large amount of food within discrete time intervals, often accompanied by a perceived loss of control (Bulik, Trace, & Mazzeo, 2013). Recurrent binge eating episodes are common in the normal-weight (NW) general population (Lowe, van Steenburgh, Ochner, & Coletta, 2009) and, according to some authors, they tend to become more frequent and compulsive over time (Davis, 2013) increasing the risk for the

Abbreviations: AR, Art Repair; BE, binge eaters; BED, Binge Eating Disorder; BES, Binge Eating Scale; BIS/BAS, Behavioral Inhibition/Behavioral Activation Systems; BIS-11, Barratt Impulsiveness Scale; BMI, Body Mass Index; BN, Bulimia Nervosa; BOLD, Blood Oxygenation Level-Dependent; DLPFC, Dorsolateral Prefrontal Cortex; EAT, Eating Attitude Test; EHI, Edinburgh Handedness Inventory; fMRI, Functional Magnetic Resonance Imaging; FOV, Field of View; FWE, Family Wise Error; FWHM, Full-Width at Half-Maximum; GLM, General Linear Model; GLME, Generalized Mixed Effects; IPL, Inferior Parietal Lobule; ITI, Inter-Trial-Interval; LME, Linear Mixed Effects; M1, Primary Motor Cortex; MFG, Middle Frontal Gyrus; MNI, Montreal Neurological Institute; NAc, Nucleus Accumbens; Non-BE, non binge eaters; OFC, Orbitofrontal Cortex; PFC, Prefrontal Cortex; REML, Restricted Maximum Likelihood; RFT, Gaussian Random Field Theory; RFX, Random Effects Analysis; ROI, Region of Interest; RTs, Reaction Times; SSD, Stop Signal Delay; SSRT, Stop Signal Reaction Time; SST, Stop Signal Task; TR, Repetition Time; WHO, World Health Organization; YFAS, Yale Food Addiction Scale

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development of clinical eating disturbances (e.g., Binge Eating Disorder, BED or obesity). Therefore, a better understanding of the underlying mechanisms of binge eating might help prevent these escalation mechanisms. Within this context, an essential question is what makes some individuals more vulnerable than others to engage in this behavior.

One potential answer to this question can be found in the role played by impulsivity (Wonderlich, Connolly, & Stice, 2004). Converging evidence on BED individuals suggests that dysfunctional inhibitory control and heightened impulsivity might be at the roots of overeating behavior (Stice, Marti, & Rohde, 2013). Indeed, both high trait general impulsivity (as assessed by Barratt Impulsiveness Scale, BIS-11 – Patton, Stanford, & Barratt, 1995) and response inhibition impairments (measures by tasks such as the Go/No-Go, GNG and Stop Signal Task, SST) have been described in obese individuals with BED (Balodis et al., 2015; Kessler, Hutson, Herman, & Potenza, 2016; Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016; Nasser, Gluck, & Geliebter, 2004; Schag et al., 2013; Ural et al., 2017). In addition, functional Magnetic Resonance Imaging (fMRI) studies reported that obese participants with BED, compared to obese non-BED, showed a consistent pattern of reduced activation in inhibitory-control areas, such as inferior frontal gyrus, ventromedial and dorsolateral prefrontal cortex, during response inhibition tasks (e.g., GNG, SST, Stroop Task; Balodis et al., 2015; Lavagnino et al., 2016).

Taken together, these pieces of evidence point towards a role of impulsivity – with specific regard to self-reported impulsivity and response inhibition abilities – in the characterization of BED. However, the majority of these findings are derived from studies that focused on obese population, often considering heterogeneous samples of obese individuals with and without BED (Lavagnino et al., 2016; Lowe et al., 2009). Albeit very informative, the documentation of such characteristics cannot provide definitive information about the mechanisms underlying binge eating and cannot reveal the brain or behavior-based predispositions to this condition (Lowe et al., 2009). Given that individuals with binge eating, those high in impulsivity traits and with strong cravings for food may represent particularly at-risk populations for full-blown BED (Lowe et al., 2009), the investigation of binge eating mechanisms, independently of weight status, could provide the unique opportunity for a deeper understanding of the role of impulsivity as a potential risk factor for the development of clinically-relevant conditions (such as, BED and obesity) in the general population. The identification of possible prodromal factors for eating disturbances could provide new and valuable insights into the most effective approaches toward weight gain and obesity prevention.

With this in mind, our study aimed to develop an understanding of impulsivity, response inhibition abilities and their neural correlates in NW binge eaters (BE) compared to individuals without binge eating episodes (non-binge eaters, non-BE). To this purpose, we recruited healthy participants, characterized by a Body Mass Index (BMI) falling within the normal range, and divided them into two groups according to the presence or the lack of binge eating episodes in the previous months (BE and non-BE, respectively). Given the multi-dimensional nature of the impulsivity construct (Bari & Robbins, 2013), the current study sought to combine different measures: (i) self-reported measures to assess general-trait impulsivity; (ii) reaction times and accuracy in performing a food-specific GNG and SST task to examine response inhibition abilities; (iii) task-related brain activity measured by fMRI during the execution of both the GNG and SST.

In order to assess response inhibition and its neural underpinnings, we adopted two different paradigms: GNG and SST. Performances on these two tasks are usually weakly correlated (Reynolds, Ortengren, Richards, &, de Wit, 2008), suggesting that they assess different aspects of response inhibition (Price, Lee, & Higgs, 2016; Eagle, Bari & Robbins, 2008). More specifically, the GNG is thought to measure the ability to restrain a non-initiated action (*action restraint*), while the SST measures the ability to cancel an already-initiated action (*action cancellation*). Given that the two tasks appear to differ in the gradation of inhibition that is required, many neuroimaging studies explored whether a successful inhibition mechanism in Stop and No-Go signal – in the SST and GNG, respectively – also engage different brain regions and networks (Aron and Poldrack, 2005; McNab et al., 2008; Schachar et al., 2007; Swick, Ashley, & Turken, 2011; Zhang & Li, 2012; Zheng, Oka, Bokura, & Yamaguchi, 2008). A couple of these studies has shown that successful inhibition processes engaged in both the SST and GNG seem to share a set of overlapping right-lateralized brain regions (McNab et al., 2008; Zheng et al., 2008). In particular, Zheng et al. (2008) hypothesize that the right middle prefrontal cortex (PFC) could be crucial for inhibition mechanism engaged in both tasks, supported by the evidence of a significant correlation between activity in this region and the performance level observed in both GNG and SST. More recently, a meta-analysis of Swick et al. (2011) directly compared results from studies adopting the GNG and SST, highlighting that the most significant overlap between brain activity associated with the two tasks was observed in the right anterior insula and medial PFC. Nonetheless, results also showed that the GNG engaged the fronto-parietal control network to a greater extent than SST, with a strong ***right*** lateralization in the middle frontal gyrus (MFG) and Inferior Parietal Lobule (IPL). Whereas, the SST engaged the cingulo-opercular network, with prominent foci in the ***left*** anterior insula and ***bilateral*** thalamus. According to Swick et al. (2011) the differences between the two tasks suggest that different aspects of response inhibition are involved, consistently with the notion of action restraint and cancellation (Eagle et al., 2008). In line with this conclusion, further support to the idea of a right-lateralization of the fronto-parietal network for action restraint, and a left-lateralization of the same circuit for the cancellation of an ongoing action has been provided by several functional studies (Aron and Poldrack, 2005; Schachar et al., 2007; Swick et al., 2011; Zhang & Li, 2012).

Within this context, the use of both paradigms in the same individual might provide valuable insights to better characterize the behavioral and neural underpinnings of the diverse aspects of response inhibition. Moreover, since previous studies have revealed that low inhibitory control, combined with a strong approach tendency toward food, may lead to overeating and an enhanced consumption of palatable food (Kakoschke, Kemps, & Tiggemann, 2015), both paradigms have been modified to assess the ability to manage impulses towards food and neutral stimuli, and to investigate the neural correlates of successful inhibition toward these two categories of cues. To this end, images depicting both common use objects (neutral; e.g., household items) and palatable food (e.g., ice-cream, pizza, etc.) have been included to disentangle possible differences in the ability to inhibit prepotent responses according to the nature of the stimulus and to assess the effect of a possible bias for food on inhibitory control.

Despite the majority of the evidence on this topic is derived from studies that focused on already-obese or BED individuals (Donnelly et al., 2018; Kessler et al., 2016), we hypothesized that impulsivity and inhibitory control deficits might also play a role in the characterization of binge eating, regardless of the presence of weight or eating disorders. Hence, we expected (i) to find greater trait impulsivity and lower inhibitory control abilities in the BE group compared to non-BE; (ii) this

difference to be particularly pronounced when inhibition toward food stimuli was required, in both the GNG and SST. Besides, (iii) we expected to reveal corresponding between-group differences in terms of brain activity supporting inhibition in both tasks (action restraint and action cancellation), especially in the regions involved in inhibitory control (i.e., fronto-parietal and subcortical regions; Swick et al., 2011; Zheng et al., 2008; Zhang and Li, 2012).

2. Materials and methods

2.1. Participants

We recruited normal-weight (NW) male and females ranging from 20 to 35 years old and divided them in two groups according to the presence or the absence of BE episodes in the previous 3 months (BE and non-BE, respectively). We enrolled 21 participants for the BE group and 21 for the non-BE group. The NW sample was defined by a Body Mass Index (BMI; kg/m^2) ranging from 18.5 to 25. The BE status was certified by means of the behavioral questions of the Eating Attitude Test (EAT 26 – Garner, Olmsted, Bohr, & Garfinkel, 1982), assessing the presence of BE episodes and the absence of compensatory behaviors (i.e., excessive physical activity, purging etc.). In more detail, a specific item was considered: "I have gone on eating binges where I feel that I may not be able to stop" which was scored on a six-point scale ranging from 1 (never) to 6 (once a day or more). The following items assessed the absence of purging behavior in both groups: (i) "Ever made yourself sick (vomited) to control your weight or shape?"; (ii) "Ever used laxatives, diet pills or diuretics to control your weight or shape?". Further, the absence of a history of eating disorders was assessed by one item: "Have you ever been treated for an eating disorder?". Participants reporting at least one BE episode per month in the last three months (i. e. at least three episodes in the last three months) constituted the BE group, while participants declaring to have never had a BE within the same time window constituted the non-BE group. To further confirm the surmised BE status we used the Binge Eating Scale (BES – Gormally, Black, Daston, & Rardin, 1982): participants who reported no episodes of overeating were expected to score lower than 8 in the BES to be included in the non-BE group (Filbey, Myers, & DeWitt, 2012). Every participant of both groups scored as expected in the BES and was included in the group to which they belonged.

All participants were right-handed according to the Edinburgh Handedness Inventory (EHI – Oldfield, 1971). In addition, we assessed the absence of specific exclusion criteria with a screening questionnaire. In particular, both groups were required to have no history of psychiatric, neurological disorders or head injuries, to be free of medical illnesses and not be treated with any psychoactive medication or psychotherapy. Participants of both groups were excluded if they reported one or more exclusion criteria for magnetic resonance (MR) examination (e.g., metal implants, pacemaker, claustrophobia, etc.). 10 participants were excluded after the screening (rejection was mainly due to MR exclusion criteria, treatment with psychoactive medication, history of eating disorder, psychotherapy and high degree myopia). The finale sample involved 21 participants (17 females; age: M = 23.9, SD = 3.19) for the BE group and 21 participants (16 females; age: M = 25.23, SD = 3.08) for the non-BE. The study was conducted according to the guidelines provided by the Declaration of Helsinki and the ethical requirements of the University of Padua (protocol n. 2025).

2.2. General procedure

Participants were recruited through local advertisements at the University of Padua. The screening for exclusion criteria (paragraph 2.1) and a complete description of study procedures was carried out during the first appointment. During the same appointment they were asked to fill out the written informed consent, to complete self-report assessment related to eating behavior and impulsivity, and to report their height and weight (in order to compute BMI). If they meet inclusions criteria (NW range, BE status as assessed by the BES and EAT-26, MRI criteria), the MR measurement occurred in a subsequent appointment (approximately one week after the screening). Before the fMRI measurement, participants were asked to refrain from drinking caffeinated beverages and from smoking for 3 h preceding their imaging session. Since hunger might be an additional factor to consider in the assessment of response inhibition toward food, we ensured comparable hunger states of participants by instructing them not to come hungry to the imaging session and to consume a small meal right before their appointment (Loeber, Grosshans, Herpertz, Kiefer, & Herpertz, 2013; Price et al., 2016). Before the imaging session, participants were familiarized with the behavioral paradigms through a practice session with additional stimulus material (stimuli that were not used in the paradigms during the fMRI acquisition). All fMRI scanning sessions occurred between 2 p.m. and 6 p.m. These experiments belong to a broader research project aiming at characterizing BE-related differences in both brain structure and function. All participants underwent functional and structural MRI acquisition. In this report, we analyzed the task-based functional data.

2.3. Measures

2.3.1. Questionnaires

During the screening, all participants completed self-reported assessment related to impulsivity, eating behavior and the feelings and thoughts associated with such behavior. Impulsivity measurements included: (1) Barratt Impulsiveness Scale (BIS-11 – Patton et al., 1995), and (2) Behavioral Inhibition/Behavioral Activation Scale (BIS/BAS – Carver & White, 1994). In line with the definition of impulsivity as a multidimensional construct, the BIS-11 investigates distinct forms of impulsivity: attentional impulsivity (the inability to concentrate or focus attention), motor impulsivity (the tendency to act without thinking), and non-planning impulsivity (lack of future orientation or forethought). On the other hand, the BIS/BAS scale refers to two complementary motivational systems controlling behavior. The BIS represents the aversive motivational system, sensitive to cues of punishment/non-reward and supposed to inhibit behavior that may lead to negative outcomes; the BAS represents the appetitive motivational system which is sensitive to cues of reward and instrumental in activating goal directed behavior (Gray, 1991). Responsiveness of both systems (BIS and BAS) is thought to play a substantial role in body weight regulation (Dietrich, Federbusch, Grellmann, Villringer, & Horstmann, 2014).

Eating behavior assessment included: EAT-26 (Garner et al., 1982); BES (Gormally et al., 1982) and Yale Food Addiction Scale (YFAS – Gearhardt, Corbin, & Brownell, 2009). The EAT-26 is a screening questionnaire that allows to assess the possible presence of an eating disorder, by measuring the symptoms and concerns that are characteristic of eating disorders. In the present research, we were interested in assessing the presence of BE episodes and the absence of a history of eating disorders or compensative behaviors, therefore we specifically focused on the behavioral questions of the questionnaires (see paragraph 2.1). The BES is a 16-item questionnaire used to assess the presence of binge eating behavior with questions based upon both behavioral characteristics (e.g., amount of food consumed) and the

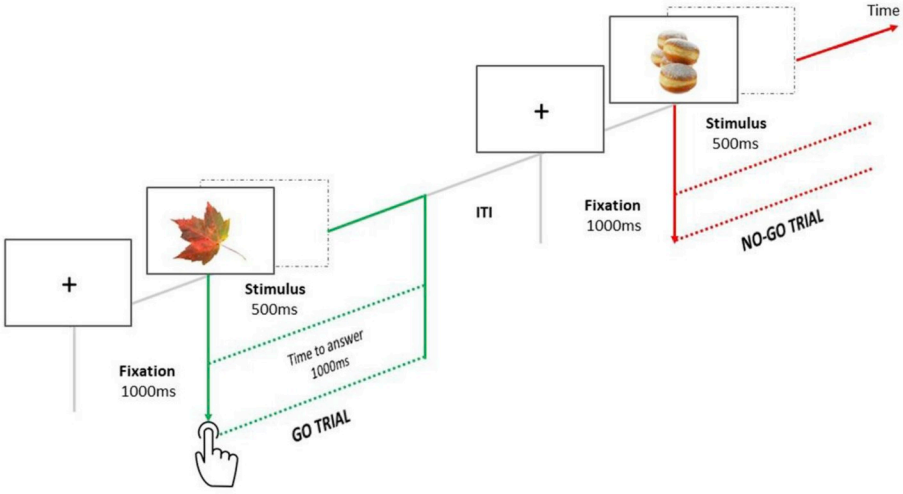

Fig. 1. GNG task. Example of 'go non-food run': participants had to respond to neutral (i.e., non-food) stimuli (75%) and withhold the response to food stimuli (25%). Stimuli appeared (for 500 ms) after a random Inter-Trial interval, ITI (2000–5000 ms) followed by the appearance of a fixation cross (1000 ms). The instructional set of the other run ('go food') was the opposite. ITI: Inter-Trial Interval.

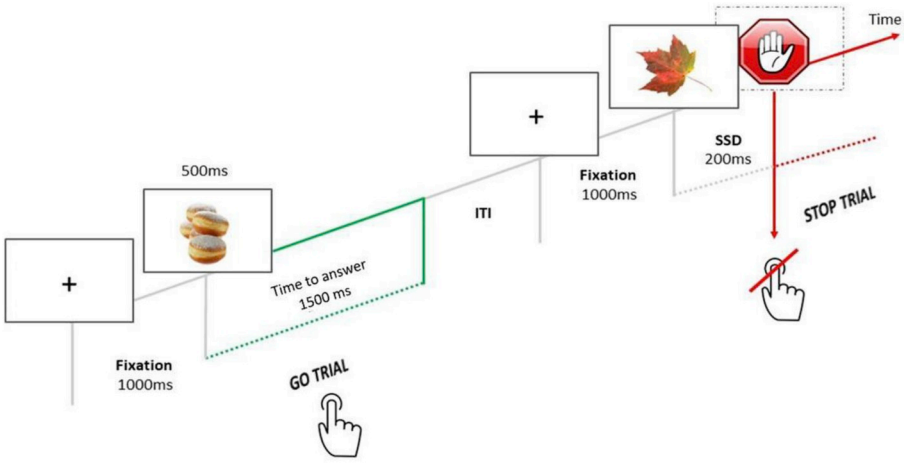

Fig. 2. SST paradigm. Participants had to respond to both food and neutral (i.e., non-food) stimuli (go trials: 75%) and withhold the response if they saw a stop signal (25%). Stimuli appeared after a random Inter-Trial interval, ITI (2000–5000 ms) following by the appearance of a fixation cross (1000 ms). The stop signal appeared after a fixed delay (stop signal delay, SSD) of 200 ms, following either food or non-food (i.e., neutral) stimuli.

emotional, cognitive response (e.g., guilt/shame, preoccupation with food and eating). Given that an increasing number of perspectives conceptualizes overeating as a 'food addiction', involving changes in brain regions implicated in executive functions, reward and interoceptive processes (Barry, Clarke, & Petry, 2009; Volkow, Wang, Tomasi, & Baler, 2013), we decided to further investigate the potential differences between the two groups in their eating behavior with the YFAS. The YFAS is a 25-item self-reported measure used to identify those who are most likely to be exhibiting markers of substance dependence with the consumption of high fat/high sugar foods. The answers to the items (both dichotomous and Likert-type format) are used to obtain a food-addiction symptoms count (e.g., loss of control, tolerance) based on the criteria for substance dependence of the DSM IV-TR (American Psychiatric Association, 2000).

2.3.2. Behavioral paradigms

We assessed response inhibition by means of two paradigms: the GNG and SST. Even though these tasks are often treated as equivalent under the assumption that they reflect a common process and a common neural substrate, they are thought to reflect different aspects of inhibitory control (Schacher et al., 2007; Swick et al., 2011). In general, the main difference between the two paradigms is represented by the timing of presentation of the no-go cues relative to the go cues (Bari & Robbins, 2013). In the GNG the no-go stimulus is presented unexpectedly instead of the go signal; whereas, in the SST the no-go signal is always presented after a go stimulus, so that the response is already in the process of completion (Bari & Robbins, 2013). Thus, by means of both tasks we were able to investigate two different aspects of response inhibition: (i) the inhibition of a planned response, namely '*action restraint*', with the GNG and (ii) the inhibition of an already initiated action, '*action cancellation*', with the SST. In addition, for both tasks we used food and neutral stimuli to assess a possible bias of food-cue reactivity on response inhibition abilities. The order of the two tasks during the fMRI acquisition was counterbalanced across subjects, and at the beginning of each task an instruction slide was presented as a reminder.

2.3.2.1. Go/No-Go Task (GNG). The GNG is a measure of response inhibition that requires participants to perform speeded responses on go trials and to inhibit responding on no-go trials - *action restraint* (Schachar et al., 2007). It involves a high load on response selection since the participant has *a priori* knowledge about whether or not to

respond to a certain stimulus. In this context, the food-specific GNG paradigm was designed to examine inhibition of prepotent responses to food stimuli compared to neutral stimuli. The task was programmed and administered using E-Prime 2.0 presentation software (Psychology Software Tools, Inc. Pittsburgh, PA) and consisted of two runs, in which pictures of food (i.e. hamburger, ice-cream, sandwich etc.) or neutral (i.e. tools, books etc.) were presented (for further details on the stimuli used see Section 2.3.3). Participants had to either press a button with their right hand or inhibit their response to each picture, according to the instructions at the beginning of each run. The role of food and neutral images was different according to the run: in the "**GO FOOD**" run, food pictures served as target stimuli, therefore participants were told to press the button with the right index finger to food pictures (GO) and withhold their response to Neutral pictures (NO-GO). Conversely, in the "**GO NEUTRAL**" run, neutral pictures served as target stimuli, therefore participants were told to press the button with Neutral stimuli (GO) and to withhold their response to food stimuli (NO-GO). The order of the runs was counterbalanced. In either case, participants were instructed to respond as quickly and accurately as possible. In order to develop a prepotent response pattern, the GO stimuli appeared in 75% of the trials of each run (n = 75 trials/run) and NOGO stimuli appeared 25% of the time (n = 25 trials/run).

At the beginning of each run, an instruction slide was presented as a reminder. In each run, the trials began with a fixation cross (1000 ms), followed by a neutral or a food stimulus presented for 500 ms. The time window to respond lasted 1000 ms. Within a given run, trials were separated by a random inter-trial interval (ITI) ranging from 2000 to 5000 ms (Fig. 1). The order of the stimuli was randomized across participants and the order of the runs was counter-balanced across the groups to further optimize the efficiency of the design. Each run lasted 9 min and 15 s (18 min and 30 s scanning in total).

2.3.2.2. Stop signal task (SST). The SST requires to withhold an already initiated response - *action cancellation* (Logan, 1994; Schachar et al., 2007), triggered by a stop signal shortly following the go signal. In comparison to GNG, SST has a high load on response inhibition processes rather than response selection. Like for GNG, the SST adopted in the present study included food and neutral stimuli and consisted of two functional runs of 112 trials each. At the beginning of each run, an instruction slide was presented as a reminder. During the go trials (75%; n = 84 trials/run), a 1000 ms fixation point preceded the stimuli, and participants were instructed to respond as fast and accurate as possible to both food and neutral stimuli using the left or the right button of the response pad (e.g., press the left button for food with the index finger and right button for Neutral stimuli with the middle finger). During the stop trials (25%; n = 28 trials/run), participants were instructed to inhibit their response if after a fixed delay of 200 ms (Stop Signal Delay, SSD) from the presentation of either a food or neutral stimulus a visual stop signal would appear (Fig. 2). The two runs were counterbalanced across subjects and differed only for the instructions regarding which button to press to which stimulus (left for food and right for neutral in one run; left for neutral and right for food in the other run). Within a given run, trials were separated by a jittered inter-trial interval (ITI) ranging from 2000 to 5000 ms. Each run last approximately 11 min. The order of the trials was randomized to optimize the efficiency of the design. Within each experimental set, SSD remained fixed at 200 ms to yield a low inhibitory rate (Logan, 1994) and make the task more demanding, compared to the GNG.

Table 1
Go/No-Go and Stop Signal Task: contrasts for successful trials. List of the contrast images entered into the second-level full factorial model for each participant in the Go/No-Go Task (1A) and Stop Signal Tasks (1B). Non-BE: non-binge eaters; BE: Binge Eaters.

1A	Non-BE		BE	
	GO/NO-GO TASK			
	FOOD	NEUTRAL	FOOD	NEUTRAL
GO	Non-BE_GO Food	Non-BE_GO Neutral	BE_GO Food	BE_GO Neutral
NO-GO	Non-BE_NOGO Food	Non-BE_NOGO Neutral	BE_NOGO Food	BE_NOGO Neutral
1B	STOP SIGNAL TASK			
	FOOD	**NEUTRAL**	**FOOD**	**NEUTRAL**
GO	Non-BE_GO Food	Non-BE_GO Neutral	BE_GO Food	BE_GO Neutral
STOP	Non-BE_STOP Food	Non-BE_STOP Neutral	BE_STOP Food	BE_STOP Neutral

2.3.3. Stimuli

Pictures were selected from the *food.pics database-extended* (www.food-pics.sbg.ac.at) which contains information on calorie content, subjectively rated palatability and physical features of the food pictures (Blechert, Meule, Busch, & Ohla, 2014). Stimuli had previously been independently rated (Blechert et al., 2014), with food and neutral images matched as closely as possible for size, colors and visual complexity. Both GNG and SST food images included different kinds of foods/meals, whereas neutral images included everyday life objects that had no association with eating (e.g., books, cars or household items - see Supplementary Materials, Figs. S1 and S2). In more detail, food items included pictures of sweet (e.g., ice cream, chocolate), savory (e.g., sandwiches), and processed (e.g., hamburger, French fries, chips, chocolate bars) foods. Given that the considered database includes a vast variety of foods – derived from different cultures around the world – we selected only those foods that are usually consumed in Italy. Both food and neutral images were items with simple figure-ground composition. In both tasks, images were presented in the center of the

Table 2
Descriptive characteristics: between-group comparison. The following details are reported: M = mean; SD= Standard Deviation; t score and p-value. BMI: Body Mass Index; BES: Binge Eating Scale; YFAS: Yale Food Addiction Scale; BIS-11: Barratt Impulsiveness Scale; BAS: Behavioral Activation System; BIS: Behavioral Inhibition System; Non-BE: non-binge eaters; BE: Binge Eaters.

Characteristics	BE (n = 21)	Non-BE (n = 21)	Two-sample *t*-test	
	M ± SD	M ± SD	T	P
AGE	23.9 ± 3.19	25.23 ± 3.08	2.05	.191
BMI (kg/m^2)	22.3 ± 2.1	21.29 ± 2.02	1.73	.074
BES	17.7 ± 3.8	3.8 ± 2.6	17.1	.000*
YFAS	3.05 ± 1.43	0.29 ± 0.56	8.23	.000*
BIS-11				
Attentional subscale	17.05 ± 3.7	15 ± 3.3	1.8	.075
Motor subscale	20.73 ± 4.2	17.75 ± 3.3	2.5	.015*
Non-planning subscale	26.32 ± 5.1	22.25 ± 4.1	2.8	.007*
Total score	63.4 ± 8.8	56 ± 7.5	2.7	.011*
BIS/BAS				
BAS Reward responsiveness	7.3 ± 1.8	7.6 ± 2.1	0.43	.075
BAS Drive	7.8 ± 1.7	9.3 ± 1.9	2.51	.017*
BAS Fun seeking	8.7 ± 2.1	9.4 ± 2.4	1.03	.13
BIS	13.3 ± 2.3	16.2 ± 3.6	2.88	.007*

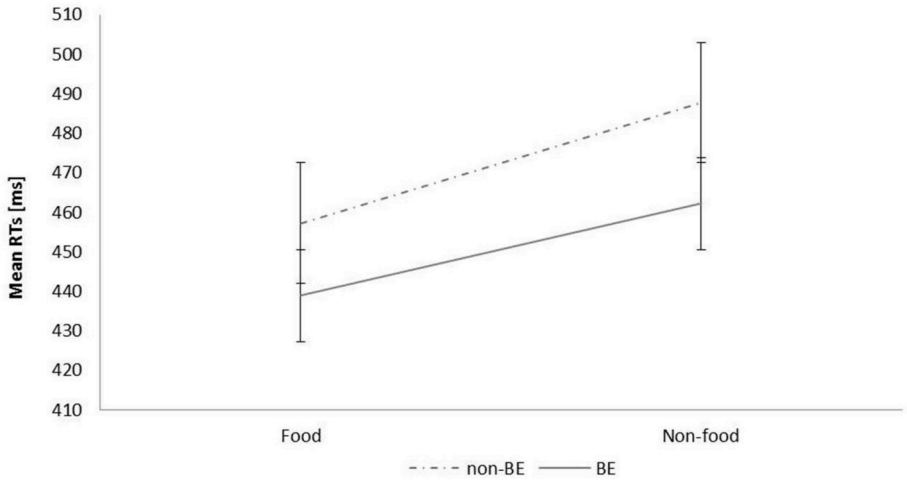

Fig. 3. Go/No-Go Task: Mean reaction times (RTs) for correct GO trials (food and non-food).
BE: Binge eaters, non-BE: non-binge eaters; RTs: Reaction Times. Error bars are representative of Standard Errors (SE).

screen. In the GNG, each image was presented only once in the whole task. Whereas in the SST, each image was presented four times within a run (three times (75%) it served as a GO stimulus and for one time (25%) it was followed by a STOP signal). We decided to have the same GO/STOP ratio for each stimulus so that we could control for a possible effect of the stimulus type on response inhibition. We used different images for the GNG and the SST.

2.4. Behavioral analysis

Analyses of behavioral data were conducted with R (R Core Team, 2018), lme4 (Bates, Mächler, Bolker, & Walker, 2015), and lmerTest (Kuznetsova, Brockhoff, & Christensen, 2014). Statistical analyses were carried out by means of linear mixed-effect model (LME) for reaction times (RTs) and generalized mixed-effect model (GLME) with binomial link function for commission errors (Pinheiro & Bates, 2000). These models provide greater statistical power compared to traditional repeated measures ANOVA, and a robust method for the analyses of repeated and unbalanced measures, such as RTs (Baayen, Davidson, & Bates, 2008). LME and GLME allow taking into consideration both the standard and the random-effect factors. In this study, fixed effects consisted of group (BE and non-BE) and condition (food and neutral); whereas, random effects consisted of experimental blocks and participants. Models were fitted using the Restricted Maximum Likelihood (REML) and p-values were estimated by likelihood ratio tests of the full model with the effect in question against the model without the effect in question.

For both GNG and SST, we compared the two groups in both (1) RTs in milliseconds during correct GO trials and (2) percentage of commission errors (a 'GO' response for NO-GO trials). The main effect of

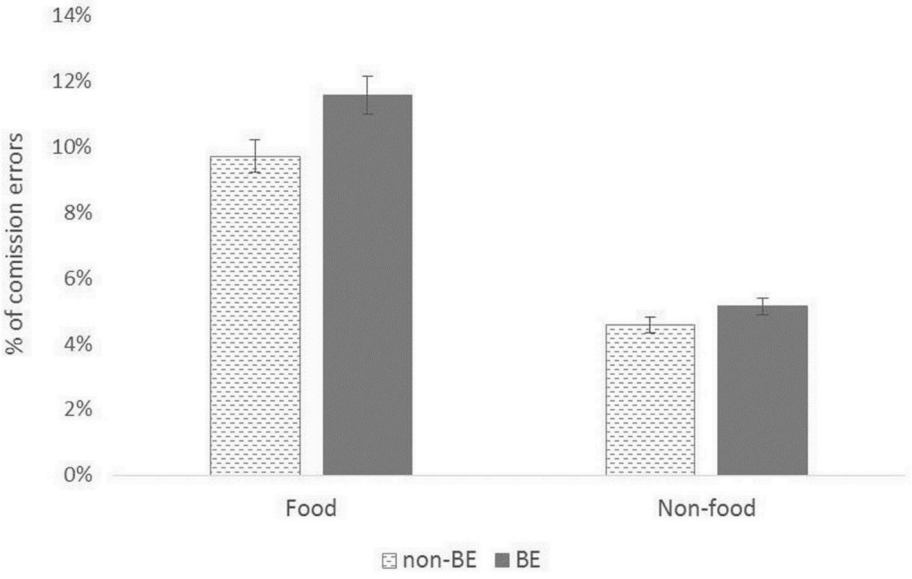

Fig. 4. Go/No-Go Task: Percentage of commission errors.
BE: Binge eaters, non-BE: non-binge eaters. Error bars are representative of Standard Errors (SE).

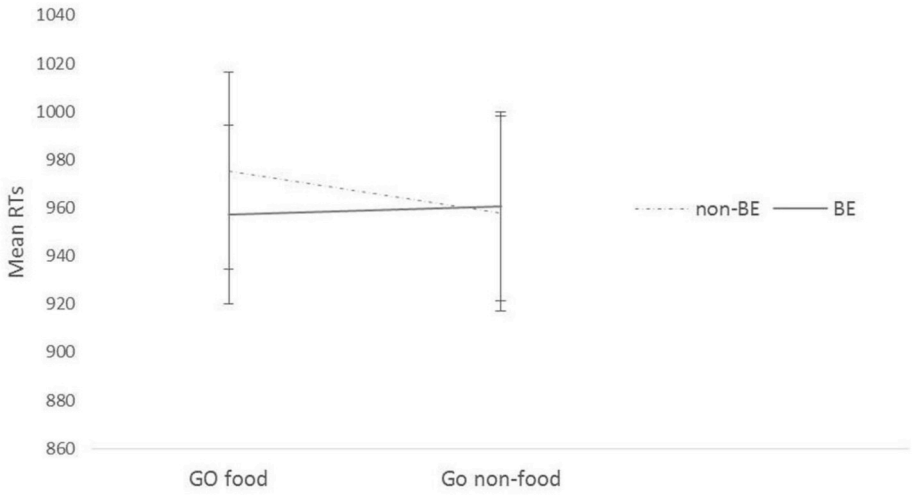

Fig. 5. Stop Signal Task: Mean reaction times (RTs) for correct GO trials (food and non-food).
BE: Binge eaters, non-BE: non-binge eaters; RTs: Reaction Times. Error bars are representative of Standard Errors (SE).

condition and group and the interaction group-by-condition were considered in the analysis. If participants failed to stop their response to every stimulus within one run, they were excluded from the fMRI analysis.

Usually the SST allows computing a measure of the latency of the stopping process, namely the Stop Signal Reaction Time (SSRT). The SSRT calculation is based on the horse-race model (Logan, 1994) which assumes that the stop process starts when the stop signal is presented. The estimation is thus based on the mean (or the median) of the different SSDs used during the task (Verbruggen & Logan, 2009). Since in our paradigm we used only one fixed SSD, we decided to consider exclusively commission errors and RTs to correct go trials as variables of interest of this study.

2.5. MRI acquisition

Whole-brain functional Magnetic Resonance Imaging (fMRI) data were obtained using a 1.5 T S Avanto MRI scanner (Siemens Medical Systems, Erlangen, Germany) equipped with a standard Siemens eight-channel coils. Functional images were acquired with a gradient-echo, echo-planar (EPI) T2*-weighted sequence in order to measure blood oxygenation level-dependent (BOLD) contrast throughout the whole brain (37 contiguous axial slices acquired with ascending interleaved sequence, 56 × 64 voxels, 3.5 mm × 3.5 mm × 4.0 mm resolution, Field of View, FOV = 196 mm × 224 mm, flip angle = 90°, TE = 49 ms). Volumes were acquired continuously for each run with a repetition time (TR) of 3 s. During the fMRI measurement, participants

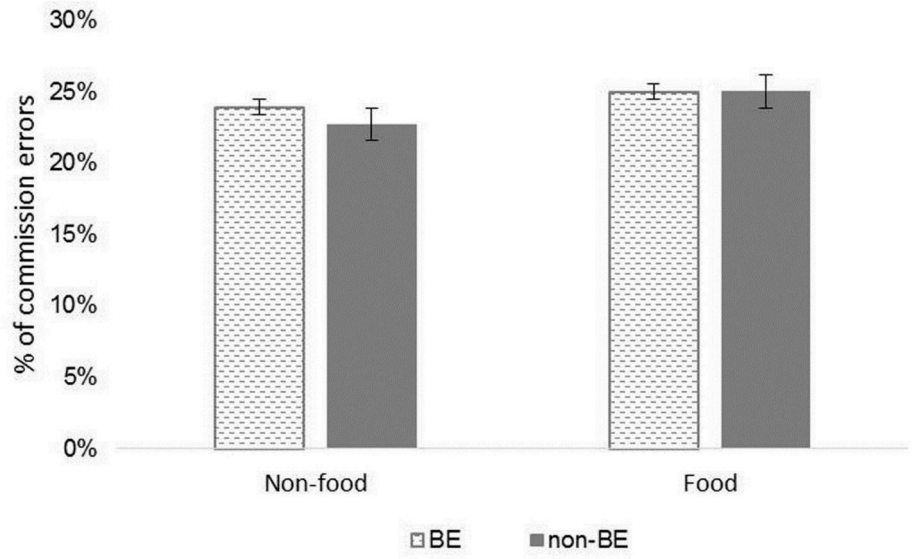

Fig. 6. Stop Signal Task: Percentage of commission errors.
BE: Binge eaters, non-BE: non-binge eaters. Error bars are representative of Standard Errors (SE).

were lying down in the scanner and wore MR-compatible LCD video goggles (VisuaStim XGA, Resonance Technology Inc.) with a resolution of 800x600 and 60 Hz refresh rate. Responses to the task (see section 2.3.2) were given with the index and middle fingers of the right (dominant) hand using an MR-compatible response box (Evoke Response Pad, Resonance Technology Inc.). For the GNG, functional data were collected in two runs of 188 vol (9 min and 15 s each; 18 min and 30 s total scanning time). For the SST, data were collected in two functional runs of 229 vol each (11 min each; 22 min total scanning time). Structural scans were collected using T1-weighted 3D MPRAGE sequence in the same orientation as the functional sequences to provide detailed anatomic images aligned to the functional scans. High-resolution structural MRI sequences were acquired (176 axial slices, FOV = 256 × 256 mm^2, 256 × 256 matrix, 0.7 mm isotropic voxels, TR = 1900 ms, TE = 2.91 ms).

2.6. MRI preprocessing and analysis

Data were preprocessed and analyzed using SPM12 (www.fil.ion.ucl.ac.uk/spm) working in Matlab environment (MathWorks, Natick, MA, USA). The ArtRepair (AR) toolbox was used to detect slices corrupted by motion artifacts and/or signal spikes at both slice and entire volume levels (Mazaika, Whitfield-Gabrieli, & Reiss, 2007). Then standard preprocessing including realignment, coregistration to the anatomical T1-weighted image, normalization into Montreal Neurological Institute (MNI) space and smoothing with a 7 × 7 × 8 mm full-width-at half-maximum (FWHM) Gaussian Kernel (twice the native voxel size) was performed. Statistical analysis of fMRI data was performed using a General Linear Model (GLM; Friston et al., 1994) approach. Analysis was conducted on the whole brain and statistical inference was performed by using a cluster-wise control of Family-Wise Error (FWE). Statistical images were first assessed for cluster-wise significance with a primary cluster-defining threshold of p = 0.001, then the thresholded cluster was considered significant at a FWE rate of 0.05.

2.6.1. Go/No-Go Task

At the first-level, a GLM was applied to identify activations in relation to separate event types:

(i) correct GO FOOD trials;
(ii) correct GO NEUTRAL trials;
(iii) correct NO-GO FOOD trials;
(iv) correct NO-GO NEUTRAL trials;
(v) unsuccessful NO-GO trials.

This resulted in five task-related regressors (one for each condition) for each participant. The onset of each event was set according to the onset of the appearance of the stimuli (see Figs. 1 and 2) and it was modeled using a stick function (duration = 0) convolved with the canonical hemodynamic response function (HRF, Henson and Friston, 2007). RTs for GO conditions were included in the model as parametric modulators (Grinband, Wager, Lindquist, Ferrera, & Hirsch, 2008). To account for head movement, the six movement parameters of the rigid body transformation applied by the realignment procedure were also introduced as regressors in the first-level analysis. We used a 128-second high-pass filter (SPM12 convention) to remove low-frequency noise and slow drifts in the signal.

At the second level, between-group differences were examined for successful trials (see Table 1): the four individual contrast images (GO to FOOD trials, GO NEUTRAL trials, NO-GO FOOD trials, NO-GO NEUTRAL) were entered into a full factorial design, with Group (BE; non-BE); Type of stimulus (Food; Neutral) and Response (Go; No-Go) as factors. Further, the BES score was added as covariate to control for their possible effect on the results. Given that BES score was the criterion used to confirm the assignment to one of the two groups (BE and non-BE), the inclusion of this regressor in the model and the interaction with the factor 'Group' allowed us to maintain the differences between the groups in this variable but to control for within-group differences in the interpretation of the results.

2.6.2. Stop signal task

For the SST, the analyses at both the first and the second level (including parameters, regressors and covariates chosen) adopted the same conventions as for GNG. At the first level, the following conditions were created for each participant: correct GO to FOOD trials;

(i) correct GO NEUTRAL trials;
(ii) correct GO FOOD trials;
(iii) correct STOP FOOD trials;
(iv) correct STOP NEUTRAL trials;
(v) unsuccessful STOP trials.

The four contrast images of successful trials (Table 1B) were then entered in the second level full-factorial model, with the three factors (Group, Stimulus, Response) and BES as covariate (Interaction with Group).

3. Results

For the Go/No-Go (GNG) the images of one participant of the non-binge eaters (non-BE) group had to be excluded from the analysis due to artifacts in the fMRI acquisition, therefore the resulting number for total sample for the GNG was 41 (BE = 21; non-BE = 20). For the SST, three participants of the Binge Eaters (BE) group and three participants from the non-BE group were excluded from the analysis because no correct responses were provided in one or more 'Stop' conditions (either to food or Neutral stimuli) per run. Thus, the resulting sample for the Stop Signal Task (SST) was of 36 participants in total: 18 for the BE group and 18 for the non-BE group. The complete sample (BE = 21; non-BE = 21) was considered for descriptive characteristics.

3.1. Descriptive characteristics

Results of the self-reported questionnaires and descriptive characteristics are shown in Table 2. The two groups did not differ for age, sex (~30% males) and Body Mass Index (BMI) parameters. In line with our hypotheses, the groups differed in most of the total and subscales' scores of the questionnaires. The BE group was characterized by higher scores in the Binge Eating Scale (BES), Yale Food Addiction Scale (YFAS) and Barratt Impulsiveness Scale (BIS-11), whereas, Non-BE had higher scores for the Behavioral Inhibition System (BIS) and the Behavioral Activation System (BAS) drive subscales of the BIS/BAS questionnaire. Contrary to expectations, no significant differences between the groups were found in the reward responsiveness subscale of the BIS/BAS questionnaire.

3.2. Behavioral results

3.2.1. Go/No-Go Task

3.2.1.1. Reaction times (RTs). Fig. 3 shows that the analysis on RTs yielded a significant main effect of condition ($\chi 2(1) = 120.61$, $p < 0.001$) but no main effect of group ($\chi 2(1) = 0.34$, $p = 0.56$). The interaction (Group X Condition) was not significant ($\chi 2(1) = 3.69$,

p = 0.06). The statistically significant result for the main effect of condition indicates that all participants were faster (lower RTs) when they had to respond to Food compared to Neutral (i.e., non-food) stimuli. The main effect of group as well as the interaction did not reach significance indicating that the two groups did not differ in RTs neither within the overall task nor for a specific condition (Food or Neutral). For a summary of mean RTs and percentage of commission errors see Supplementary Materials (Tables S1–S2).

3.2.1.2. Commission errors. Fig. 4 summarizes the percentage of commission errors for each condition and for both groups. In line with the RTs results, also for commission errors, the main effect of Condition was significant ($\chi2(1) = 26.116$, $p < 0.001$), indicating that all participants tended to make more errors when they were asked to inhibit their responses to Food compared to Neutral (i.e., non-food) stimuli. No differences between the groups across all conditions (main effect of Group, $\chi2(1) = 0.3381$, $p = 0.561$) and for specific conditions (interaction Group X Condition, $\chi2(1) = 1.3949$, $p = 0.24$).

3.2.2. Stop signal task

3.2.2.1. Reaction times. The analysis on RTs revealed no significant results for main effect of Condition ($\chi2(1) = 1.611$, $p = 0.204$); main effect of Group ($\chi2(1) = 0.184$, $p = 0.668$). Whereas, the interaction Group X Condition was statistically significant ($\chi2(1) = 4.57$, $p = 0.032$). For a summary of mean RTs see Supplementary Materials (Table S3). The significant interaction indicates that the type of stimuli used (Food or Neutral) had an effect on RTs in the non-BE group: they tended to respond faster (lower RTs) with Neutral (i.e., non-food) stimuli compared to Food stimuli. An effect was not observed in the BE group: the type of stimuli used did not affect RTs (Fig. 5).

3.2.2.2. Commission errors. Fig. 6 shows the percentage of commission errors for each condition and for both groups. This analysis revealed no significant results for any of the factors considered: main effect of Condition ($\chi2(1) = 1.1214$, $p = 0.29$); main effect of Group ($\chi2(1) = 0.472$, $p = 0.49$); interaction Group X Condition ($\chi2(1) = 0.3065$, $p = 0.58$). This indicates that there were no differences between the two groups neither in the overall percentage of commission errors nor within each specific condition. For a summary of the percentage of commission errors see Supplementary Materials (Table S4).

3.3. fMRI results

In this section the between-group comparisons within each condition for both the GNG and the SST are reported. First, we focused on the between-group comparison within GO and NO-GO conditions. Subsequently, we investigated the between-group differences within NO-GO conditions (food and neutral) to highlight possible differences in brain activity during response inhibition toward Food and Neutral stimuli. For a summary of main effects for both tasks see Supplementary Materials (Tables S5–S8).

3.3.1. Go/No-Go Task

Between-group comparisons within NO-GO and GO trials (food and neutral images combined).

NO-GO trials: The contrast *BE > non-BE* revealed two clusters in the right Superior and Inferior Occipital Gyrus, while for the contrast *Non-BE > BE* differences were located in the right Middle Frontal Gyrus (MFG), left Cerebellum, right Precuneus and right Caudate/Putamen (Fig. 7; Table 3).

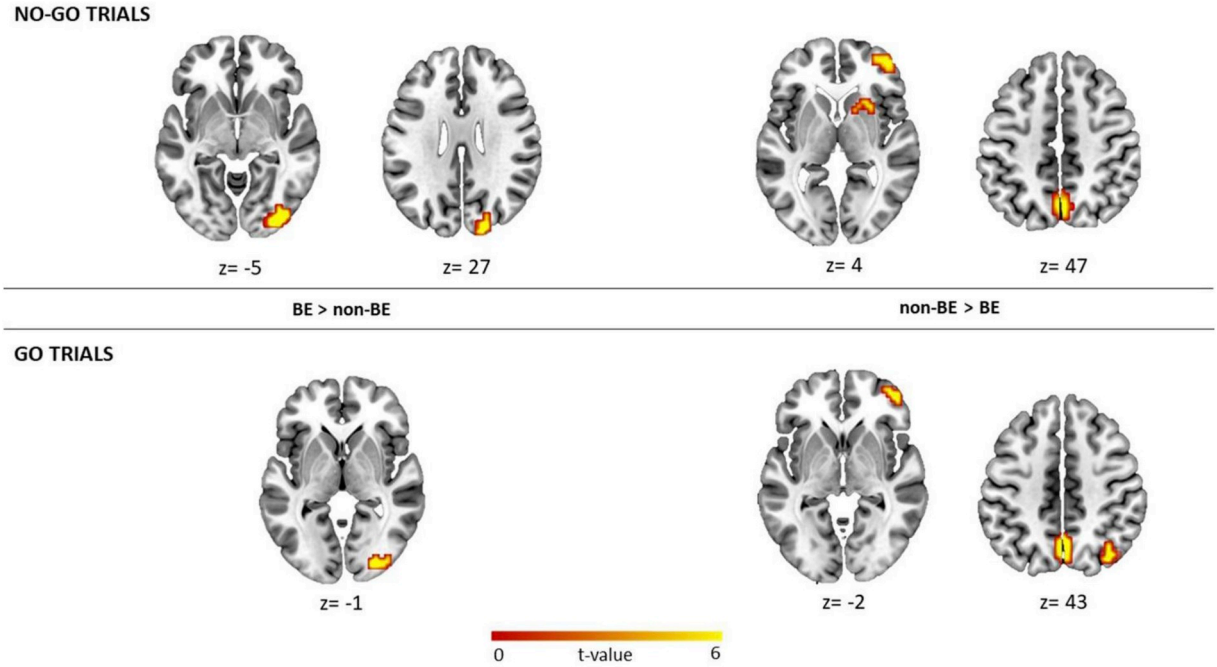

Fig. 7. Between-group comparisons for successful no-go trials (top) and go trials (down). Figures on the left part show results for the contrast BE > non-BE. Figures on right show results for contrast non-BE > BE. Statistical parametric maps were overlaid onto a T1-weighted canonical image, provided by the MRIcroGL software. The color bar is representative of the t-scores given in the table below. Images are shown in neurological convention and with axial slice coordinates as defined in Montreal Neurological Institute (MNI) 152 space. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

Table 3
Between-group comparisons for successful no-go trials and go trials. The following details are reported: k = number of voxels; t and z scores; stereotaxic coordinates according to the Montréal Neurological Institute (MNI); brain side and region. Statistic threshold: Results were considered significant at p < 0.001 that additionally met a FWE correction at cluster level (p < 0.05). Non-BE: non-binge eaters; BE: binge eaters; FWE= Family Wise Error; L = left; R = right.

cluster		peak		MNI			Side	Region
k	p(FWE-corr)	t	z	x	y	z		
NO-GO TRIALS								
BE > non-BE								
41	0.004	5.45	5.21	36	−88	−6	R	Inferior Occipital Gyrus
24	0.048	5.27	5.04	19	−91	30	R	Superior Occipital Gyrus
non-BE > BE								
67	0.0003	5.45	5.21	−45	−67	−22	L	Cerebellum
		4.62	4.46	−27	−84	−26	L	Cerebellum
59	0.001	4.83	4.65	1	−70	50	R	Precuneus
		4.83	4.65	1	−63	42	R	Precuneus
30	0.020	4.61	4.45	43	53	−2	R	Middle Frontal Gyrus
24	0.048	4.19	4.07	12	7	−2	R	Caudate
		4.13	4.02	26	14	−2	R	Putamen
GO TRIALS								
BE > non-BE								
40	0.005	5.50	5.24	36	−88	−6	R	Inferior Occipital Gyrus
non-BE > BE								
66	0.000	5.46	5.22	−45	−67	−22	L	Cerebellum
		4.74	4.58	−34	−84	−38	L	Cerebellum
29	0.023	4.78	4.61	36	−74	46	R	Angular Gyrus
		4.49	4.34	29	−67	58	R	Angular Gyrus
45	0.003	4.67	4.51	1	−63	42	R	Precuneus
		4.61	4.46	5	−70	50	R	Precuneus
26	0.035	4.45	4.31	43	53	−2	R	Middle Frontal Gyrus
		3.63	3.55	33	56	2	R	Middle Frontal Gyrus
32	0.015	4.23	4.11	−13	−70	−46	L	Cerebellum
		4.09	3.98	−13	−53	−46	L	Cerebellum

GO trials: The contrast *BE > non-BE* revealed significant differences in one cluster in the right Inferior Occipital Gyrus. For the contrast *Non-BE > BE* differences between the groups were located in the right MFG, left Cerebellum, right Precuneus and right Angular Gyrus (Fig. 7; Table 3).

3.3.1.1. Between-group comparisons within NO-GO food and NO-GO neutral trials. **NO-GO food**: The contrast *BE > non-BE* revealed differences in the right Inferior Occipital Gyrus; while for the contrast *Non-BE > BE* differences were located in the right Putamen, Precuneus, left Cerebellum and Precentral gyrus (Fig. 8; Table 4).

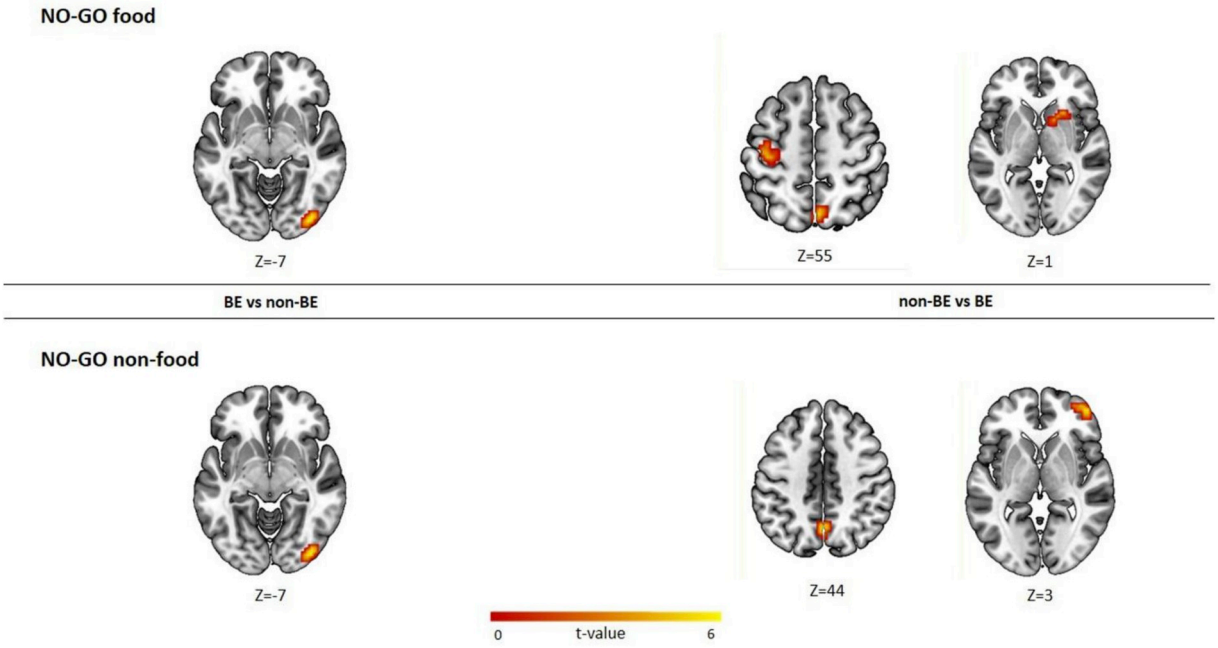

Fig. 8. Between-group comparisons in no-go food trials (top) and no-go neutral (i.e., non-food) trials (down). Figures on the left part show results for the contrast BE > non-BE. Figures on right show results for contrast non-BE > BE. Statistical parametric maps were overlaid onto a T1-weighted canonical image, provided by the MRIcroGL software. The color bar is representative of the *t*-scores given in the table below. Images are shown in neurological convention and with axial slice coordinates as defined in Montreal Neurological Institute (MNI) 152 space. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

Table 4
Between-group comparisons in no-go food trials and no-go neutral (i.e., non-food) trials. The following details are reported: k = number of voxels; t and z scores; stereotaxic coordinates according to the Montréal Neurological Institute (MNI); brain side and region. Statistic threshold: Results were considered significant at $p < 0.001$ that additionally met a FWE correction at cluster level ($p < 0.05$). Non-BE: non-binge eaters; BE: binge eaters; FWE= Family Wise Error; L = left; R = right.

cluster		peak		MNI			Side	Region
k	p(FWE-corr)	t	z	x	y	z		
NOGO FOOD								
BE > non-BE								
25	0.0410	4.90	4.71	36	−88	−6	R	Inferior Occipital Gyrus
non-BE > BE								
56	0.0007	5.29	5.07	−45	−67	−22	L	Cerebellum
		4.40	4.26	−24	−81	−26	L	Cerebellum
63	0.0003	4.95	4.76	5	−70	50	R	Precuneus
		4.72	4.55	5	−63	42	R	Precuneus
56	0.0007	4.70	4.53	−13	−70	−46	L	Cerebellum
		4.18	4.07	−13	−39	−38	L	Cerebellum
25	0.0410	3.89	3.79	26	14	−2	R	Putamen
		3.86	3.77	12	7	−2	R	Putamen
24	0.0477	3.78	3.69	−38	−21	58	L	Precentral Gyrus
NO-GO NEUTRAL								
BE > non-BE								
38	0.0070	5.04	4.84	36	−88	−6	R	Inferior Occipital Gyrus
non-BE > BE								
45	0.003	4.67	4.51	−34	−84	−38	L	Cerebellum
		4.63	4.47	−45	−67	−22	L	Cerebellum
24	0.048	4.40	4.26	43	53	2	R	Middle Frontal Gyrus
		3.71	3.63	33	56	2	R	Middle Frontal Gyrus
28	0.026	4.17	4.05	1	−63	42	R	Precuneus
		4.11	3.99	5	−67	54	R	Precuneus

NO-GO Neutral (i.e., non-food): The contrast *BE > non-BE* showed differences in the right Inferior Occipital Gyrus, whereas the contrast *Non-BE > BE* revealed differences located in the right Precuneus, right MFG and left Cerebellum (Fig. 8; Table 4).

3.3.2. Stop signal task

Between-group comparisons within STOP and GO trials (food and neutral images combined).

STOP trials: The contrast *BE > non-BE* revealed two clusters in the left MFG and left Cerebellum. For the contrast *Non-BE > BE,* differences between the groups were located in one cluster in the right postcentral gyrus (Fig. 9; Table 5).

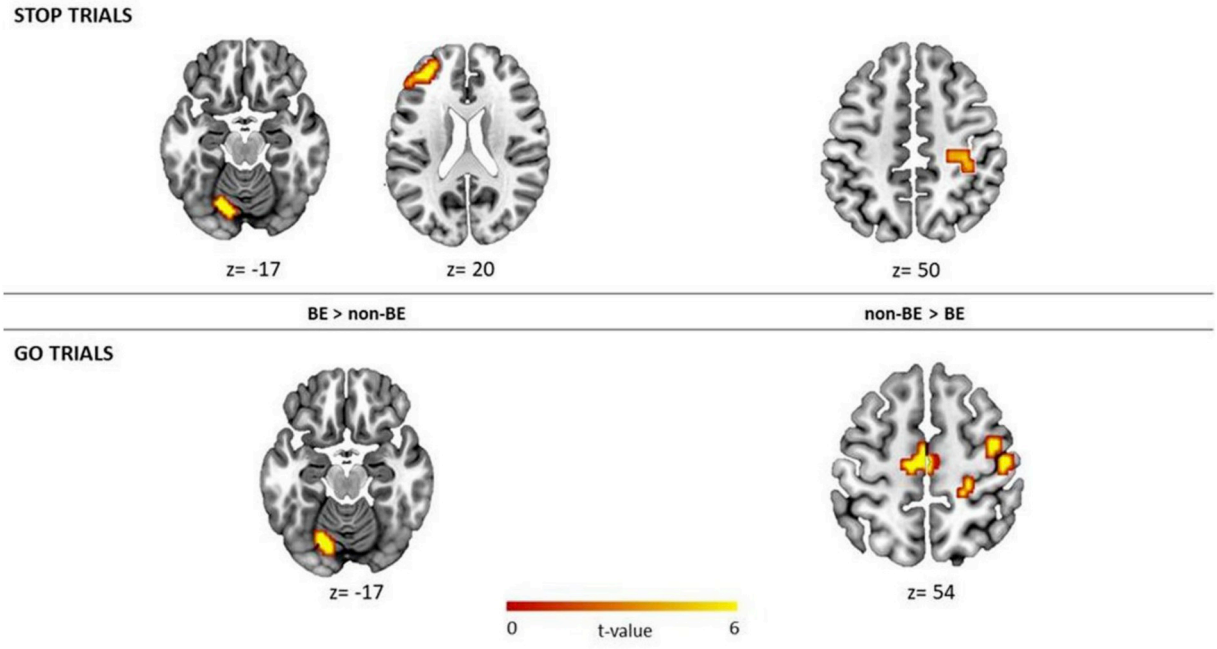

Fig. 9. Between-group comparisons for successful stop (top) and go (down) trials. Figures on the left show results for the contrast BE > non-BE. Figures on the right show results for contrast non-BE > BE. Statistical parametric maps were overlaid onton a T1-weighted canonical image, provided by the MRIcroGL software. The color bar is representative of the *t*-scores given in the table below. Images are shown in neurological convention and with axial slice coordinates as defined in Montreal Neurological Institute (MNI) 152 space. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

Table 5
Between-group comparisons for successful stop and go trials. The following details are reported: k = number of voxels; t and z scores; stereotaxic coordinates according to the Montréal Neurological Institute (MNI); brain side and region. Statistic threshold: Results were considered significant at $p < 0.001$ that additionally met a FWE correction at cluster level ($p < 0.05$). BE= Binge Eaters; non-BE = non-binge eaters; FWE= Family Wise Error; L = left; R = right.

cluster		peak		MNI			Side	Region
k	p(FWE-corr)	t	z	x	y	z		
STOP TRIALS								
BE > non-BE								
37	0.004	5.18	4.94	−20	−70	−22	L	Cerebellum
42	0.002	4.87	4.66	−31	49	26	L	Middle Frontal Gyrus
		4.83	4.62	−48	−39	18	L	Middle Frontal Gyrus
		4.46	4.30	−34	42	22	L	Middle Frontal Gyrus
non-BE > BE								
23	0.038	4.78	4.58	36	−28	46	R	Postcentral Gyrus
		3.96	3.84	29	−25	42	R	Postcentral Gyrus
GO TRIALS								
BE > non-BE								
48	0.001	5.64	5.33	−17	−74	−18	L	Cerebellum
non-BE > BE								
95	0.000	5.31	5.05	36	−28	46	R	Postcentral Gyrus
		4.65	4.47	19	−25	46	R	Precentral Gyrus
		4.62	4.44	36	−11	46	R	Postcentral Gyrus
46	0.001	4.32	4.18	−20	−21	70	L	Precentral Gyrus
		4.23	4.09	−10	−18	54	L	Precentral Gyrus

GO trials: The contrast *BE > non-BE* revealed one cluster in the left Cerebellum, whereas the contrast *Non-BE > BE* revealed differences between the groups located in two clusters: one in the right postcentral gyrus and the left precentral gyrus (Fig. 9; Table 5).

3.3.2.1. Between-group comparisons within STOP food and STOP neutral trials. **STOP food**: The contrast *BE > non-BE* revealed differences located in the left MFG, while the contrast *Non-BE > BE* did not yield any significant results (Fig. 10; Table 6).

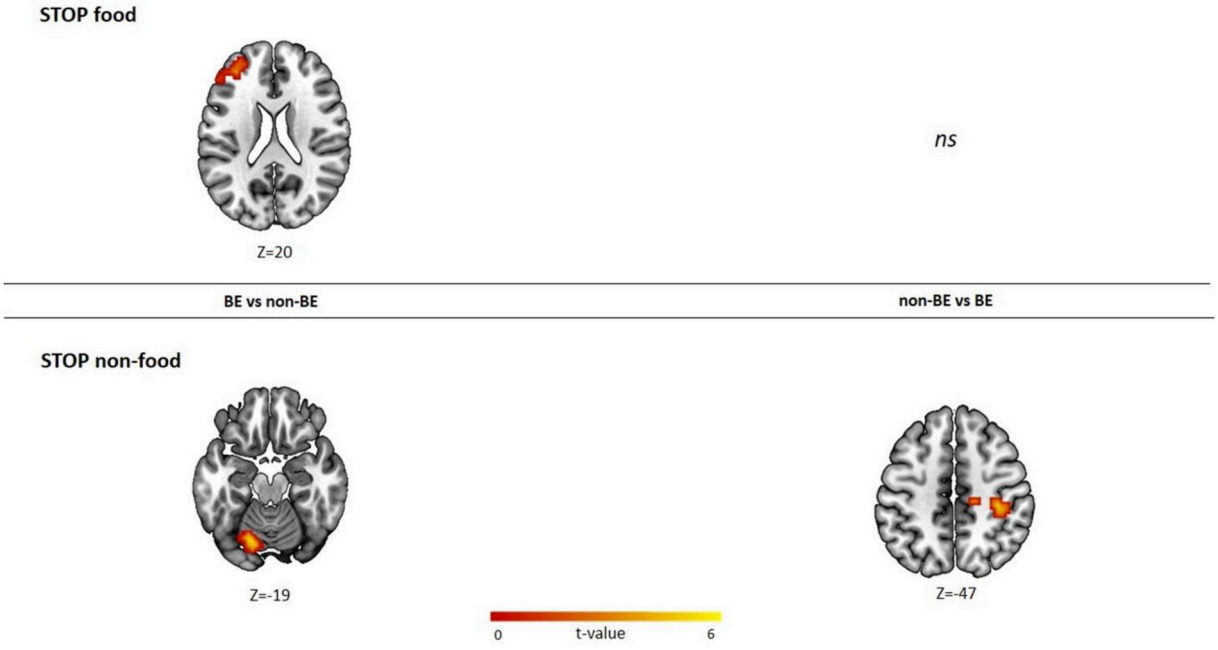

Fig. 10. Between-group comparisons in stop food trials (top) and stop neutral (i.e., non-food) trials (down). Figures on the left part show results for the contrast BE > non-BE. Figures on right show results for contrast non-BE > BE. Statistical parametric maps were overlaid onto a T1-weighted canonical image, provided by the MRIcroGL software. The color bar is representative of the *t*-scores given in the table below. Images are shown in neurological convention and with axial slice coordinates as defined in Montreal Neurological Institute (MNI) 152 space. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

Table 6
Between-group comparisons in stop food trials (top) and stop neutral (i.e., non-food) trials (down). The following details are reported: k = number of voxels; t and z scores; stereotaxic coordinates according to the Montréal Neurological Institute (MNI); brain side and region. Statistic threshold: Results were considered significant at $p < 0.001$ that additionally met a FWE correction at cluster level ($p < 0.05$). Non-BE: non-binge eaters; BE: binge eaters; FWE= Family Wise Error; L = left; R = right; ns: non-significant.

cluster		peak		MNI			Side	Region
k	p(FWE-corr)	t	z	x	y	z		
STOP FOOD								
BE > non-BE								
26	0.024	4.21	4.07	−31	49	26	L	Middle Frontal Gyrus
		3.67	3.57	−45	42	14	L	Middle Frontal Gyrus
non-BE > BE								
				ns				
STOP NEUTRAL								
BE > non-BE								
4	0.003	5.17	4.93	−17	−74	−22	L	Cerebellum
non-BE > BE								
39	0.003	5.44	5.17	36	−28	46	R	Postcentral Gyrus
		3.88	3.77	15	−25	46	R	Precentral Gyrus
		3.71	3.61	22	−25	42	R	Precentral Gyrus

STOP Neutral (i.e., non-food): In the contrast *BE > non-BE* differences were located in the left Cerebellum, while the contrast *Non-BE > BE* revealed differences in the right Precentral Gyrus (Fig. 10; Table 6).

4. Discussion

In recent years, an increased attention has been paid to the possible mechanisms underlying binge eating behavior. This issue is of great interest given that binge eating episodes are common in the general population and, when becoming frequent and persistent, they might be possible risk factors for the development of overweight and overeating disorders (Lyu, Zheng, Chen, & Jackson, 2017; Stice et al., 2013). In this context, impulsivity seems to play a role in the maintenance of overeating behavior. Mounting evidence has indeed underscored a clear connection between higher impulsivity trait and overeating in overweight and obese individuals, with and without Binge Eating Disorder (BED); Chamberlain, Derbyshire, Leppink, & Grant, 2015; Galanti, Gluck, & Geliebter, 2007; Kessler et al., 2016; Micanti et al., 2017; Schag et al., 2013). However, to understand if impulsivity can be considered a hallmark and trait of BE behavior, regardless of weight status, more attention should be paid to normal-weight (NW) individuals with overeating episodes. In our study we aimed to characterize impulsivity and its neural correlates in a non-clinical population of NW individuals with binge eating episodes (Binge Eaters, BE), using self-reported, behavioral and brain imaging measures.

Overall, our results showed that, despite comparable inhibitory efficiency in behavioral terms, BE and non-BE individuals differed in self-reported general impulsivity measures and in brain activity engagement during tasks requiring the ability to inhibit an already planned/ongoing response.

4.1. Self-reported impulsivity

In line with previous studies (Guerrieri, Nederkoorn, Schrooten, Martijn, & Jansen, 2009; Meule & Platte, 2015; Waxman, 2009), we found that BE group scored higher on self-reported measures of impulsivity compared to non-BE. In particular, the Barratt Impulsiveness Scale (BIS-11) motor, non-planning subscales and total scores were higher in BE, indicating a greater degree of impulsivity in this group. This result confirms the close relationship between self-reported impulsivity and disinhibition in eating behavior, as already reported both in clinical (Nasser et al., 2004) and non-clinical samples (Lyke & Spinella, 2004). In addition, BE showed lower scores compared to non-BE on the Behavioral Inhibition System (BIS) subscale of the Behavioral Inhibition/Activation System (BIS/BAS) questionnaire. This subscale investigates the regulation of aversive motives (in which the goal is to move away from something unpleasant) and higher scores usually indicates a tendency to avoid aversive or new stimuli (Avila, 2001; Carver & White, 1994). Since there is evidence of higher scores of the BIS as a function of restraint (Nederkoorn, Van Eijs, & Jansen, 2004; Yeomans & Brace, 2015), lower scores in the BE group might indicate a lower tendency in these individuals to avoid or inhibit behavior with a greater propensity to respond. Contrary to expectations, the BE group did not score higher than the non-BE in the 'reward responsiveness' subscale of the BIS/BAS questionnaire. This result is in accordance with the assumption that the impulsivity trait (rash spontaneous behavior) plays a clearer role in the characterization of BE behavior compared to reward sensitivity, for which evidence on BE population is still mixed (Giel, Teufel, Junne, Zipfel, & Schag, 2017). In addition, since the small number of questions of the subscale refer to general rewards (either internal, such as expectancies of goal attainment, or external, such as presence of a desired goal) but not specifically to food, our results could indicate that the BE group did not have a generalized heightened sensitivity toward any type of reward-relevant stimuli.

Overall, our findings support the relationship between disinhibited eating behavior and impulsivity facets, already revealed in obesity and BED (Giel et al., 2017; Meule, 2013) and further extend this concept showing that higher scores in measures of impulsivity characterize also NW individuals with BE, without a diagnosis of eating or weight disorder. This evidence together with the assumption that self-reported measures of impulsivity investigate stable personality traits (Meule, 2013) underscores the relevance of trait impulsivity as a possible general hallmark of BE, evident even in the absence of obesity or clinically significant eating disturbances (Lyu et al., 2017).

4.2. Behavioral impulsivity

Both GNG and the SST were considered to assess between-group differences in two aspects of response inhibition (action restraint and action cancellation, respectively) and to highlight possible differences in inhibitory control abilities accordingly to the type of stimuli used (Food or Neutral). Contrary to expectations, the two groups were characterized by comparable performances in terms of reaction times (RTs) and commission errors in both tasks. In the GNG, a general main effect of condition (Food/Neutral) indicated that both groups tended to respond faster and less accurately to food stimuli. This result, already reported in the literature, is consistent with the assumption that food-cues – probably due to the high relevance of food for survival – elicit automatic actions and approach tendencies regardless of dieting success, self-reported impulsivity, or hunger levels (Meule, 2013). The SST highlighted a significant Group x Condition interaction, indicating that RTs in the specific conditions (GO Food; GO Neutral) differed between the groups. The non-BE tended to have slower RTs when asked to respond to food stimuli, compared to Neutral stimuli, whereas, for the BE group, the nature of the stimuli (Food and Neutral) did not affect RTs. Even if unexpected, the lack of effects of stimulus' category on RTs in BE was consistent with the results of a recent study (Mühlberg, Mathar, Villringer, Horstmann, & Neumann, 2016) where authors found that in a food-specific SST, the GO RTs of the obese sample did not differ across picture categories (Food and Neutral). On the other hand, the different RTs in non-BE could be associated to different hedonic values of the presented visual cues; however, since we did not rate them within the group, this result needs to be confirmed with further investigations.

In general, the lack of strong between-group differences in both tasks might have several possible explanations. First, even if self-reported impulsivity is usually positively correlated with behavioral measures, these correlations are often weak and inconsistent (Cyders & Coskunpinar, 2012). It is assumed that while self-reported measures represent impulsivity as a stable trait, behavioral tasks are subject to state-dependent variations (Meule, 2013). Second, tasks' design and difficulty might have influenced the groups' performances. In fact, both the GNG and the SST should elicit prepotent responses to make response inhibition toward no-go stimuli difficult to achieve. However, especially in our GNG paradigm, to better capture the hemodynamic response at the basis of the fMRI signal, for methodological reasons single events had to be separated by long inter-trial interval (ITI), making response inhibition easier. Third, since we decided to use a fixed SSD, the difficulty of the SST task was not adjusted according to the performance of each participant, and this might have heightened the variability within the group. Nevertheless, the sample-specifics must be considered very carefully for results' interpretation: most of the evidence on the deficits in response inhibition toward food and neutral stimuli derives from studies that focused on obese population. And, even when inhibitory control deficits are present in this population compared to NW individuals, the impairment seems to be independent of the presence of a BED, thus more linked to obesity itself (Lavagnino et al., 2016). The impact of obesity on cognitive performance may be attributable to obesity-related central effects, such as systematic inflammation or insulin resistance (Smith, Hay, Campbell, & Trollor, 2011; van den Akker, Stewart, Antoniou, Palmberg, & Jansen, 2014). Therefore, the association between obesity and executive functions is likely to be bi-directional (Smith et al., 2011), not solely linked to eating behavior but more to the weight disorder itself. In sum, given that tasks seem to be more exposed to state-dependent variations (Meule, 2013), the possibility that they could be less reliable than self-reported measures in capturing stable personality traits and that some of the above-mentioned aspects might have played a role in the lack of clear behavioral differences between the groups should be considered.

4.3. fMRI results

Contrary to behavioral data, the fMRI results showed between-group differences in brain activity during the execution of both tasks, with diverse patterns of activation characterizing action restraint (GNG) and action cancellation (SST). The co-occurrence of comparable behavioral performances on one side and differences in brain activity on the other side has already been reported in the literature. Several task-based fMRI studies on substance users (Roberts & Garavan, 2010), gambling addicted (Ding et al., 2014) and obese individuals (Hendrick, Luo, Zhang, & Li, 2011) have revealed that, compared to healthy controls, participants' brain activity differed when completing response inhibition tasks (such as GNG and SST), despite similar behavioral performances. In the light of this evidence, our results might thus indicate that the two groups differed in the neural recruitment of specific brain regions to adequately perform the tasks.

4.3.1. GO/NO-GO TASK: between-group differences in action restrain

The between-group comparisons revealed that in conditions requiring the inhibition of responses (NO-GO trials), BE showed significantly lower activity in the right Middle Frontal Gyrus (MFG), Precuneus, Caudate/Putamen and bilateral Cerebellum, in comparison with non-BE. On the other hand, BE appeared to be characterized by higher involvement in occipital regions. When looking at the same contrasts (BE > non-BE; non-BE > BE) in GO trials, a similar picture of results was identified, except for the right Angular Gyrus and Caudate/Putamen.

Thus, in GO and NO-GO trials, the BE – when compared to non-BE – showed lower activity in regions that are typically engaged in the GNG tasks, namely prefrontal, parietal, temporal and striatal areas (Simmonds, Pekar, & Mostofsky, 2008). These regions are involved in stimulus recognition, maintenance, manipulation of stimulus-response (SR) associations and response selection (Braver, Barch, Gray, Molfese, & Snyder, 2001; Grafton, Mazziotta, Woods, & Phelps, 1992; Liddle, Kiehl, & Smith, 2001; Rubia et al., 2001), all of which are relevant aspects to perform the GNG task. In particular, the right fronto-parietal network is known to be involved in the GNG task (Stevens et al., 2009) and it is thought to play a role in attention to the no-go signals, hence to play a role in the restraining of the action (Aron and Poldrack, 2005; Schachar et al., 2007; Swick et al., 2011). In addition, the MFG together with temporo-parietal regions seem to be specifically involved in 'complex' GNG, namely those tasks with multiple GO cues (i.e., different types of objects within the same category) and thus requiring a frequent updating of SR association (Corbetta & Shulman, 2002; Simmonds et al., 2008). The common activation of these regions in both the GO and NO-GO conditions is consistent with the assumption that both GO and NO-GO involve response selection processes and should not be considered as opposite and independent events (Simmonds et al., 2008).

Nonetheless, one interesting finding was the between-group difference in the right dorsal striatum (Caudate/Putamen): this result was not only distinctive of NO-GO trials, but specifically of NO-GO trials when food stimuli were presented. The basal ganglia structures (especially the dorsal striatum consisting of putamen and caudate nuclei) are known to be involved in response inhibition and thus commonly recruited during stopping (Aron & Poldrack, 2005; Everitt & Robbins, 2016; Hampshire & Sharp, 2015). More importantly, the dorsal striatum together with prefrontal cortex is part of the mesocortical pathway, which is implicated in motor control (Toni & Passingham, 1999) and in the modulation of stimulus-response-reward associations (Ghahremani et al., 2012). Therefore, the lower activity in BE might indicate a different modulation of inhibitory control processes toward reward in this group compared to non-BE. Interestingly, a recent emphasis has been placed on the role of the fronto-striatal network in behavioral inhibition and more specifically, of the right striatum and prefrontal cortex (PFC) as core regions of impaired inhibitory control in individuals with bulimic symptoms (Berner & Marsh, 2014; Donnelly et al., 2018; Skunde et al., 2016). Since we did not find significant differences in inhibitory efficiency in behavioral terms between the groups, we cannot draw definitive conclusions on the directionality of these results. Still, the hypoactivation within the mesocortical pathway has been associated with impulsivity and increased vulnerability for compulsive behaviors (Atalayer et al., 2018; Geliebter et al., 2006; Kelley, Schiltz, & Landry, 2005; Wang et al., 2009). Thus, these findings may shed some light on the potential role of diminished fronto-striatal activity as a possible hallmark and susceptibility factor for loss of control eating in NW individuals.

Lastly, both between-group comparison in GO and NO-GO trials revealed higher activation in BE compared to non-BE in occipital regions. Although not strictly related to response inhibition, this result has been previously observed in Bulimia Nervosa (BN) patients engaged in both a visual task that involved emotional and food stimuli (Uher et al., 2004) and an attentional task (Seitz et al., 2016). According to Seitz et al. (2016), the hyperactivation of these regions, implicated in alerting functions (Fan, Mccandliss, Fossella, Flombaum, & Posner, 2005), might indicate a possible compensatory mechanism for the attentional network during the completion of the tasks. Moreover, a hypoactivation in fronto-striatal circuits paralleled by hyperactivation in occipital areas, might be linked to inattention and impulsivity features (Seitz et al., 2016).

4.3.2. STOP SIGNAL TASK: between-group differences in action cancellation

The between-group comparisons in the STOP conditions (action cancellation) revealed greater activation in the left MFG and cerebellum in the BE, while the opposite comparison revealed greater activation in precentral regions for the non-BE group. In comparison to the GO trials, results in the STOP trials are similar, with the exception of the left MFG. Therefore, the greater activity in the left MFG in the BE group seemed to be specific in conditions in which participants were

required to cancel an ongoing action. This region is part of a network believed to process low-probability stimuli, with higher activity linked to the presence of infrequent no-go stimuli and failed inhibition trials (Stevens, Kiehl, Pearlson, & Calhoun, 2009). In addition, higher activity in the left MFG was also found to be linked to efficiency during a response inhibition task (Hirose et al., 2012). Hirose et al. (2012) claimed that the neural substrate of response inhibition in the left hemisphere is a measure of efficiency and plays a supplementary role in inhibitory control when the right hemisphere is fully engaged or hypoactive (Hirose et al., 2012). In line with this premise, Zhang and Li (2012) reported different roles for the right and left fronto-parietal networks, with the right more linked to attention to the no-go signal and the left lateralized network implicated in motor inhibitory control during inhibition (Zhang and Li, 2012). Therefore, given that the inhibitory load is higher in the cancellation of an ongoing action than in withholding (Schachar et al., 2007), the task might place a greater demand on the system responsible for inhibitory control and require a supplementary engagement of this module, in addition to the regular involvement of the attentional monitoring system (Zhang and Li, 2012). In this context, a greater activity in the left MFG in the STOP conditions in BE compared to non-BE might mean that a supplementary engagement of this region for BE participants was required to successfully cancel the ongoing action at the sight of the stop signal. To further investigate this result, we separately looked at the STOP conditions toward Food and Neutral stimuli and interestingly, the heightened activity in the left MFG in the BE – compared to non-BE – was specific for STOP Food trials. Therefore, the hypothesized additional engagement of this region during stopping might be linked not only to response inhibition in general, but particularly to inhibition when food is involved. Hence, this result further supports the possibility that BE group engaged the left MFG to a greater extent compared to non-BE to successfully inhibit their response toward food stimuli. The specificity of this finding might say something about the mechanisms underlying response inhibition (and particularly, action cancellation) toward food in BE individuals.

In general, the common involvement of the MFG during the execution of the GNG (right MFG) and the SST (left MFG) in the BE group might be of extreme importance for different reasons. First, MFG has been defined as a key region involved in overeating and obesity (Alonso-Alonso & Pascual-Leone, 2007; Garçia-Garçia et al., 2015). Among others, Garçia-Garçia et al. (2015) observed an involvement of the MFG during both a resting-state condition and a visual task paradigm in obese participants compared to healthy controls. According to the authors, the consistency of this result, both at rest and during the task, may indicate that functional alterations in this region could reflect a stable (across conditions) feature of obesity. Given the role of the MFG in different cognitive processes (Fuster, 2002) and motor impulsivity (Ashai et al., 2004), the authors suggested to study this result with specific tasks targeting inhibitory control processes, such as SST and GNG (Garçia-Garçia et al., 2015). Further, our results can be read in light of the distinction between action restraint and action cancellation and the involvement of the right-hemispheric fronto-parietal network in attention to the no-go signal (when the action needs to be restrained) and the left fronto-parietal network in response inhibition itself (hence the canceling of the ongoing action; Zhang and Li, 2012). Therefore, these findings encourage further investigation not only of the role for the MFG in BE as a possible pre-morbid or pre-existing risk factor for obesity, but also of a possible functional lateralization of the MFG linked to differential aspects of response inhibition (action restraint and cancellation).

5. Conclusions

In sum, general trait impulsivity, as measured by self-reported questionnaires, characterized our BE group. Even with similar behavioral performances, the fMRI results showed between-group differences mainly located in the fronto-striatal and parietal areas, encompassing regions that are known to be involved in impulsive behavior. Moreover, some of these regions where specifically involved only in those conditions where response inhibition toward food stimuli was required. Therefore, these results warrant further investigation as possible underpinnings of response inhibition and stimulus-response-reward association in BE individuals. Overall, this combination of findings provides support for the compelling hypothesis that regions involved in response inhibition and selection might be the substrate of conditions characterized by impulsive behaviors, such as BE (Lubman, Yücel, & Pantelis, 2004). Moreover, given the recent assumption of the involvement of the MFG in the pathophysiology of obesity (Garçia-Garçia et al., 2015), our data suggest that this region may also be involved in the circuit modulating impulsivity and response inhibition even in individuals with binge eating, without a clinically-relevant disorder (such as, BED or obesity).

However, some limitations need to be acknowledged for the interpretation of the results. First, although we asked participants not to come hungry to the MRI session, we did not assess satiety levels or which foods were consumed as part of the small meal they ate prior to the scanning. Based on the possible contribution of the different states of satiety on regional brain activity in response to food cues (Führer et al., 2009), potential satiety differences between the groups might had an effect on our results. In addition, female individuals in both groups were not matched on stage of the menstrual cycle. We cannot exclude that this aspect may have added variability because reward-related brain activation may vary across the menstrual cycle (Dreher et al., 2007). Lastly, individual food preferences were not assessed: although results regarding inhibitory control abilities are in line with the previous literature, controlling for this aspect could be useful to magnify individual behavior responsiveness and inhibitory control toward food stimuli (Lyu et al., 2017). Nevertheless, to the best of our knowledge this is one of the first studies investigating self-reported impulsivity, response inhibition and their neural correlates in NW non-purging individuals with binge or loss of control eating.

Albeit preliminary, our findings yield valuable insights on the role of impulsivity in binge eating behavior, with impulsivity being a possible stable characteristic linked to this behavior even in the absence of a weight disorder. In addition, new hints on the role of frontal and striatal regions in response inhibition toward food in BE individuals are provided, highlighting their potential role as vulnerability factors for loss of control and impulsive eating behavior. Bearing in mind the cross-sectional nature of our study, the directionality of these effects can only be confirmed by further studies that incorporate longitudinal designs. In addition, based on our results there appears to be a failed convergent validity between self-reported questionnaires, behavioral performances, and neurobiological correlates of impulsivity, therefore a further investigation of the correlations among these measures might provide additional information for a more appropriate and complete assessment of impulsivity in the context of binge eating. Overall, a comprehensive investigation on the possible risk factors for weight gain should consider all the above mentioned aspects, in the perspective of possible interventions for binge eating prevention and treatment focusing on impulsive behavior.

Authors contributions

RO, CB and UC designed the research; RO recruited participants; RO, CB collected the data; RO, CB, FM, AH collaborated on the data analysis; RO, CB, FM, AH and UC collaborated on the writing of the manuscript.

Conflict of interest

All Authors declare to have no financial, biomedical or any sort of conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.appet.2018.12.043.

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