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ERP correlates of cognitive control and food-related processing in normal weight and severely obese candidates for bariatric surgery: Data gathered using a newly designed Simon task

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ABSTRACT

Although there have been suggestions that altered cognitive control and food reward sensitivity contribute to overeating in obese individuals, neurophysiological correlates of these mechanisms have been poorly investigated. The current study investigated event-related potentials (ERP) in 24 severely obese and 26 normal weight individuals in fasting condition, using a novel Simon task with food and object distractors.

The study showed that conflict in the Simon task for the food distractor increased with hunger in both groups but was larger with respect to a neutral condition only in the obese individuals. ERP showed higher N1amplitudes in both groups for food distractor, reflecting early food processing. The P2 latency was delayed and the effect of distractors on N2 amplitude was smaller in the obese subjects, reflecting altered neural mechanisms associated with selective attention and cognitive control, all contributing hypothetically to delay response selection of these individuals faced with food distractor.

1. Introduction

The spreading obesity pandemic represents a challenge to health care systems and means that there is a rapidly growing population of severely obese individuals (i.e. body mass index BMI $>$ 40 kg/m^2) who are at higher risk of mortality and medical co-morbidities such as arterial hypertension, type 2 diabetes, obstructive sleep apnea syndrome, non-alcoholic fatty liver, and steatohepatitis (Ogden, Yanovski, Carroll, & Flegal, 2007). It has recently been suggested that an obesogenic environment (i.e., a place where highly caloric foods are readily available) drives the so-called "hedonic" feeding (i.e., feeding in response to pleasure rather than to nutritional needs; (Appelhans, 2009) increasing the risk of overeating and weight gain. Repeated pairing of palatable foods with rewarding outcomes seems to contribute to the development of maladaptive stimulus-response (S-R) associations between those types of foods and eating behaviors, promoting a hypersensitivity of the striatal dopaminergic reward system to food stimuli (for a review see Kenny, 2011). Following the sensitization of S-R associations, food stimuli become particularly salient, automatically capturing attention. Different experimental paradigms and methodologies have uncovered enhanced attentional salience toward food (i.e. food-related attentional bias) in overweight and obese individuals (for a review see Hendrikse et al., 2015).

Growing evidence suggests that adiposity also has an adverse effect on the brain in terms of functional and structural alterations (García-García et al., 2015; Gustafson, Lissner, Bengtsson, Björkelund, & Skoog, 2004; Kullmann, Schweizer, Veit, Fritsche, & Preissl, 2015; Kurth et al., 2013; Marqués-Iturria et al., 2013; Stanek et al., 2011). Cognitive alterations, such as executive dysfunction (e.g. poor decision-making and poor cognitive control, for reviews, Fagundo et al., 2012; Fitzpatrick, Gilbert, & Serpell, 2013; Prickett, Brennan, & Stolwyk, 2015; Spitznagel et al., 2015) have been reported in severely obese individuals. Deficits in cognitive control processes (e.g. response inhibition, interference control; for a review see Braver, 2012) may contribute to dysregulating food consumption and reducing an obese individual's ability to achieve long-term goals such as successfully losing weight and maintaining weight loss.

Some studies in the literature have described findings showing that there is an imbalance between food-reward sensitivity and cognitive control abilities in obese individuals (Ziauddeen, Alonso-Alonso, Hill,

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Kelley, & Khan, 2015). Consistent with this view, behavioral studies have reported finding a correlation between BMI values and response inhibition ability during go/no-go tasks designed to study food-related stimuli in normal weight and obese individuals (Houben, Nederkoorn, & Jansen, 2014) after a 2–3 h fast (Price, Lee, & Higgs, 2005). Neuroimaging studies have also been uncovering higher reactivity to food-related stimuli in reward-related brain regions (e.g. the striatum, amygdala and the orbitofrontal cortex) and lower activity in brain regions linked to cognitive control (e.g. the lateral prefrontal cortex) in obese individuals (Brooks, Cedernaes, & Schiöth, 2013; Nummenmaa et al., 2012). An inverse correlation between BMI values and the activity in frontal brain regions was, moreover, described in connection to a go/no-go task with food-related stimuli (Batterink, Yokum, & Stice, 2010; He et al., 2014). Unfortunately, studies comparing behavioral measures of cognitive control in obese and normal weight individuals have produced conflicting results. While some studies have reported reduced inhibitory control (Calvo, Galioto, Gunstad, & Spitznagel, 2014; Chamberlain, Derbyshire, Leppink, & Grant, 2015; Grant, Derbyshire, Leppink, & Chamberlain, 2015; Mole et al., 2015) and interference control in obese individuals, others have not (Bongers et al., 2015; Hendrick, Luo, Zhang, & Li, 2012).

In view of the excellent temporal resolution provided by event-related potentials (ERP), the technique seems particularly useful to investigate selective attention toward food and cognitive control and to expand our knowledge on the neurocognitive correlates of obesity. To date, only a few studies have investigated both cognitive control and reward sensitivity to food stimuli in simply overweight individuals (i.e., with a BMI between 25 and 30 Kg/m^2) and in patients with class I obesity (i.e., with a BMI between 30 and 35 Kg/m^2) (Carbine et al., 2018; Hume, Howells, Rauch, Kroff, & Lambert, 2015; Nijs, Franken, & Muris, 2010). An elevated BMI has nevertheless been associated with reduced cognitive control and neurocognitive alterations that are particularly relevant in severely obese patients who are candidates for bariatric surgery.

A novel "affective" Simon task utilizing task-irrelevant images of food or objects was designed for the current investigation. The Simon task, a behavioral measure of interference/conflict resolution, was used here to study spatial S-R interference control (i.e., the ability to select the proper response even when other competing responses are present). Despite the fact that the stimuli's spatial position is irrelevant to accurate performance of the task, the reaction times (RTs) are faster when the stimuli and response positions spatially correspond or match (i.e., corresponding trial) than when they do not (i.e. non-corresponding trial) – this is so-called "Simon effect". Conflict between a fast direct automatic pathway and a slow, indirect controlled pathway seems to affect response selection during Simon tasks (Lu & Proctor, 1995; Ridderinkhof, 2002; Simon & Rudell, 1967; Umiltá & Nicoletti, 1990). The stimuli's location seems to automatically activate the spatially corresponding response arising from long-term S-R associations between perceptual and motor processes and linked to genetic factors or to the synaptic consolidation of over-learned S-R associations (Cohen, Dunbar, & McClelland, 1990; Tagliabue, Zorzi, Umiltà, & Bassignani, 2000). A slower indirect (controlled) route, instead, controls goal-directed behavior activating the appropriate response depending on task demands.

It is thought that a dual-route model could explain "addictive-like" behaviors (Bechara, 2005; Evans & Coventry, 2006; Wiers & Stacy, 2006; Wiers, Gladwin, Hofmann, Salemink, & Ridderinkhof, 2013), which depend on an interaction between two pathways of information processing, known as the reflective and impulsive systems (Hofmann, Friese, & Wiers, 2008). In the former, decisions are made in connection to subjective goals and are elicited as a consequence of voluntary decision processes, including executive functions. In the latter, over-learned behavioral repertoires originating from S-R associations stored in long-term memory in close interaction with perceptual stimulus input are activated. As the two pathways interact during response selection and decision-making processes, it can be assumed that enhanced attention toward food-related stimuli is driven by the impulsive system leading to impulsive eating behaviors in obese individuals in whom inhibitory and cognitive control processes in the reflective pathway are weakened.

The current study was designed to utilize ERP to distinguish between the fast automatic/impulsive processes, possibly occurring early after stimulus onset, and the slow/reflective processes, occurring at later stages of processing and linked to deliberate behaviors. Its aim was to compare the neurophysiological correlates of food-related processing and cognitive control as well as eating attitudes and trait impulsivity in severely obese candidates for bariatric surgery (body mass index BMI $> 40\ kg/m^2$ or a BMI $> 35\ kg/m^2$ with comorbid conditions) and in normal weight individuals. We expected the images of the distracting food stimuli to interfere with the obese participants' selective attention and cognitive control processes.

As some studies have suggested that food-related modulations might occur at later stages of information processing (e.g. P2 and P3) in obese/overweight individuals (Hume et al., 2015; Nijs et al., 2010), we expected to find enhanced amplitudes of these components in the presence of food stimuli with a more pronounced effect in obese individuals. We also chose to analyze the N2 and P3 components because of their connection with cognitive control (Folstein & Van Petten, 2008; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004; Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003; Roche, Garavan, Foxe, & O'Mara, 2005), selective attention and working-memory updating (Polich, 2007). In addition, according to some studies, the P3 is modulated by the S-R interference effect in the Simon task (Leuthold, 2011; Schiff et al., 2014). Independently of food-related processing, we expected obesity-related cognitive dysfunction (Spitznagel et al., 2015) to manifest itself through smaller amplitudes and/or delayed latencies of late ERP components. In other words, we expected to see that differences between normal weight and obese individuals are reflected in modulations in neurophysiological indexes of selective attention, working memory updating (e.g. P2, P3), and executive cognitive control (i.e. N2). In addition, as a recent study reported finding larger N1 amplitude to food cues in hungry non-obese and persons with a history of dieting (Feig et al., 2017), we expected to see a larger N1 in our obese and normal weight participants that should be larger for food distractors with respect to other distractors.

2. Methods

2.1. Participants

Twenty-four severely obese and 26 normal weight age and education level matched individuals were enrolled in the study (See Table 1). An *a priori* power analysis was not performed because this was the first time that a modified affective Simon task was adopted to investigate cognitive control in normal weight and obese individuals. During a pilot study utilizing the same task to investigate normal weight individuals with different subjective hunger levels (high-hunger vs. low-hunger), we found a significant group x distractor x correspondence interaction in the groups made up of 18 individuals. The sample size that we decided upon for the current study was also based on data gathered from previous studies (Hume et al., 2015; Nijs et al., 2010) investigating cognitive control using no more than 20 subjects per group of normal weight and obese individuals. We decided to use slightly larger groups to further increase the statistical power of our analyses.

The obese individuals, all candidates for laparoscopic sleeve gastrectomy, were recruited from the Bariatric Surgery Unit of Padua University Hospital. The study's exclusion criteria were: neurological diseases, psychiatric disorders, and age < 18 or > 60 years. Obese participants presented some relevant medical conditions: type 2 Diabetes (8.3%); obstructive sleep apnea syndrome (OSAS) (33.3%); hypertension (37%); dyslipidemia (33.3%). Prior to enrollment the

Table 1
Mean (SD) demographics variables and self-report measures.

		Normal weight	Obese
Gender (F/M)		22/4	19/5
Age (years)		32.8 (9.79)	37.6 (10.1)
Education (years)		11.9 (3.45)	13.6 (3.69)
Weight (kg)		60.4 (6.37)	123 (19.1)
Height (m)		1.66 (.70)	1.66 (.89)
BMI (kg/m^2)		21.7 (1.75)	44.71 (6.44)
Self-report measures	Chronbach α		
BES	.918	4.58 (4.31)	14.3 (9.26)
YFAS	.898	1.04 (.916)	3.26 (1.98)
EAT-26	.923	4.73 (5.43)	10 (6.78)
DEBQ-restrained	.895	2.44 (.78)	2.68 (.71)
DEBQ-emotional	.975	1.82 (.70)	3.16 (1.28)
DEBQ-external	.841	2.66 (.45)	3.01 (8.14)
PFS-available	.882	1.44 (0.55)	2.06 (.81)
PFS-present	.876	1.69 (0.63)	2.55 (1.27)
PFS-tasted	.802	2.06(0.51)	2.38(1.05)
PFS-total	.950	1.68 (.471)	2.31 (1.06)
BIS-11 attentional	.706	14.11 (2.12)	15.54 (3.47)
BIS-11 motor	.755	18.7(4.75)	20.45(5.70)
BIS-11 non-planning	.787	24.3 (4.71)	26.9 (5.22)
BIS-11-total	.875	57.2 (9.77)	60.4 (15.1)

Notes: SD = standard deviation; F = female; M = male; m = meter; kg = kilogram; BMI = Body Mass Index; BES = Binge Eating Scale; YFAS = Yale Food Addiction Scale (total score); EAT-26 = Eating Attitude Test; DEBQ = Dutch Eating Behavior Questionnaire (subscales: restrain, emotional, external); PFS = Power of Food Scale; BIS-11 = Behavioral Inhibition Scale (total score). * $p < .05$; ** $p < .001$.

participants were provided information about the experimental procedure and were asked to sign consent statements. The study was performed in accordance with the Helsinki Declaration (Editors, International Committee of Medical Journal Editors, 2004) and approved by the local Ethical Committee.

2.2. Materials

2.2.1. Affective Simon task (see Fig. 1)

The trials were carried out in a dimly lit room with the participants seated in front of a 15-in. cathode ray tube (CRT) computer screen at a distance of 58 cm. The session consisted of 600 experimental trials presented in five blocks of 120 trials each. Each trial started with a central black fixation cross subtending 0.5° of visual angle, displayed on a light gray background. The fixation cross was surrounded by a black square perimeter with the side subtending 3° of the visual angle. After intervals ranging between 2000 and 3500 ms, the target stimuli were presented at an eccentricity of 4.5° of the visual angle on the left or right of the fixation cross for 147 ms. The stimuli were 4 × 4 red-and-black or green-and-black checkerboards subtending 1.48° of the visual angle. A 4 × 4 black-and-white checkerboard was presented together with the target as the contralateral filler. At the same time, a central distractor was shown together with the target stimulus inside the square surrounding the fixation cross for 2000 ms. The distractors consisted of food and object images projected on a white background or an empty square with a white background. Inter-trial intervals ranged from 1000 to 2000 ms. Ten food items and ten objects were selected from a validated dataset (Blechert, Meule, Busch, & Ohla, 2014[1]).

The participants were instructed to maintain central fixation and to respond to the lateral stimuli as quickly and accurately as possible. Half of the participants were instructed to press the left button (the letter 'Z' of the keyboard) with their left index finger if the target was the red-and-black checkerboard and the right button (the letter 'M') with their right index finger if it was the green-and-black one, independently of its spatial position. The instructions were inverted for the other half of the participants. In half of the trials, the responding hand was on the same side as the target (corresponding condition – C); in the other half, it was on the other side (non-corresponding condition – NC). There were corresponding and non-corresponding conditions for all three types of distractors: a food item, an object, and a neutral condition (i.e., a white square). The mean RT and response accuracy were calculated separately for the two types of correspondence and for three distractors. The differences in the RTs between the non-corresponding and corresponding trials were analyzed for each type of distractor (i.e., food, object, neutral) as a measure of interference control over task-irrelevant spatial information (i.e., the Simon effect or the S-R interference effect).

2.2.2. Self-report measures

Questionnaires were used to assess dysfunctional eating behaviors and attitudes toward food: The Binge Eating Scale (BES, Gormally, Black, Daston, & Rardin, 1982) was used to assess the presence of binge eating behavior; the Yale Food Addiction Scale (YFAS, Innamorati et al., 2015) was used to assess the presence of food addiction; the Power of Food Scale and the Dutch Eating Behavior Questionnaire were used respectively to assess emotional eating behavior (PFS, Lowe et al., 2009; DEBQ, Van Strien, Frijters, Bergers, & Defares, 1986). Finally, the Barratt Impulsiveness Scale (BIS- 11, Fossati, Di Ceglie, Acquarini, & Barratt, 2001) was used to assess impulsivity traits.

2.3. Procedures

The participants were instructed to fast for 6 h prior to the experimental session, which took place in all cases at the same time of the day (12–2 p.m.). The questionnaires were administered, completed, and collected at the beginning of the experimental session (T0). The participants were also asked about subjective levels of hunger/satiety/desire-to-eat, which were rated on a Likert scales ranging from −5 to +5. They participated in the experimental session as explained above while simultaneously undergoing EEG recording. At the end of the session (T1), they once again rated their levels of hunger/satiety/desire-to-eat.

2.4. EEG recording/preprocessing

Electroencephalographic signal (EEG) was continuously acquired with Micromed BQ3200S equipment (Mogliano, Veneto, Italy) from 29 Ag/AgCl electrodes pre-cabled on an elastic cap according to the 10–20 international EEG system. The left mastoid and Fpz were used as the online and ground reference, respectively, and all the EEG data were re-referenced offline to the right mastoid. The signals from all the channels were digitized with a sampling rate of 512 Hz and 8 bit/channel resolution. The signals were filtered online in the 0.03–30 Hz range. The impedance was kept below 5 KΩ. Offline EEG analyses were performed with EEGlab (Delorme & Makeig, 2004) applying the following: band-pass filter (0.1–30 Hz); segmentation in 3000 ms epochs (1500 ms pre-stimulus, 2000 ms post-stimulus); eye-blink and artifact correction using independent component analysis (ICA); rejection of trials with an amplitude exceeding ± 100 μV; and baseline correction was applied adopting a pre-stimulus interval between −200 and 0 ms. Segments between −200 and 1000 ms were averaged separately depending on the distractors (i.e., food, object, neutral) and correspondence (i.e., C, NC). The number of valid segments for each condition was as follows: neutral C (90 ± 9; mean ± SD); neutral NC (90 ± 13); food C (93 ± 10); food NC (89 ± 11); object C (90 ± 9); object NC (87 ± 11); there were no significant differences across conditions or groups (all *ps* > .05).

[1] Image numbers were: 18, 32, 45, 46, 54, 107, 110, 145, 167, 176, 1008, 1025 1033, 1036, 1044, 1060, 1096, 1081, 1117, 1137.

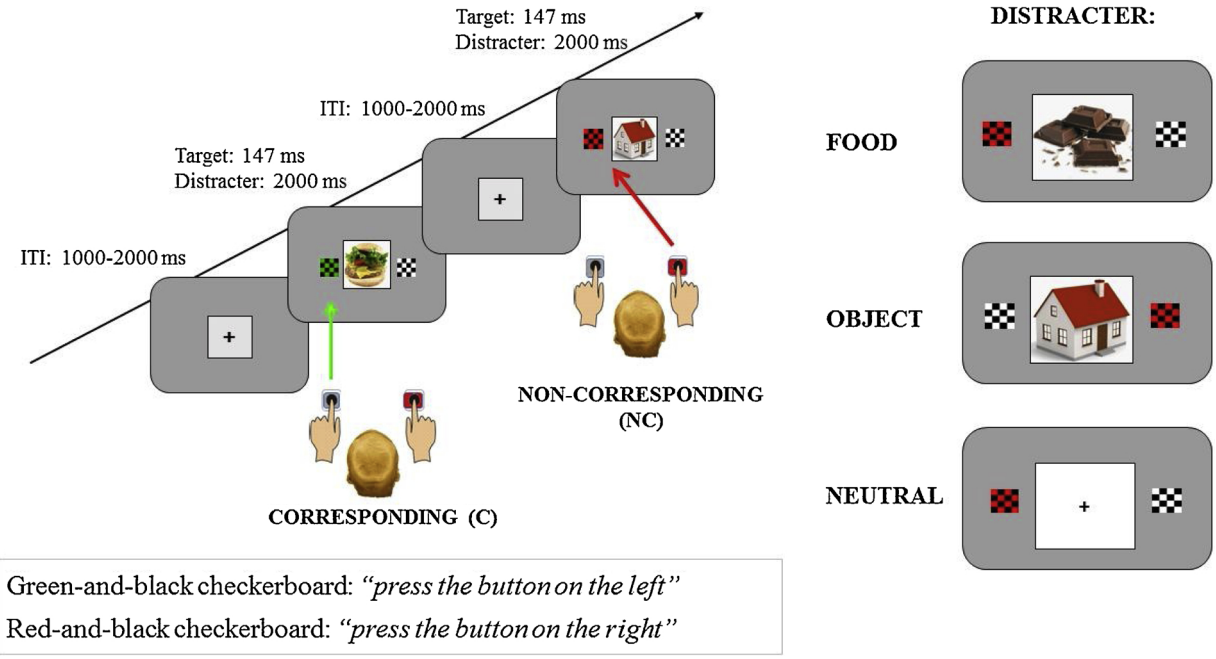

Fig. 1. A schematic drawing of the trials using a Simon task and examples of both corresponding (C) and non-corresponding (NC) conditions (on the left) and the three types of distractors presented during the task: a food item, an object, and a neutral distractor (on the right).

2.5. Data analysis

2.5.1. Self-report measures

Between-group differences in hunger/satiety/desire-to-eat scales were measured with mixed analysis of variance (ANOVA) using the group (obese, normal weight) as the between-subject factor and time (T0, T1) as the within-subject factor. The scores of the two groups of participants were compared using independent samples *t*-tests.

2.5.2. Affective Simon task

The accuracy and the RTs were analyzed with separate 2 × 3 × 2 mixed ANOVA with the group (obese, normal weight) as the between-subject factor, and the distractor (food, object, neutral) and correspondence (C, NC) as the within-subject factors. The Simon effect (i.e., the differences in the RTs between the non-corresponding and corresponding trials) was analyzed by a 2 × 3 mixed ANOVA with the group (obese, normal weight) as the between-subject factor, and the distractor (food, object, neutral) as the within-subject factors. Bonferroni post-hoc correction for multiple comparisons was calculated for the significant effects.

2.5.3. ERP analysis

Given the novelty of the task, exploratory analysis using a multivariate partial least square (PLS) technique, a data-driven approach making it possible to explore differences between conditions without *a priori* assumptions of the results (Lobaugh, West, & McIntosh, 2001), was performed (see Supplementary material for more details). This was followed by traditional ERP analyses based on both visual inspection of the ERP and PLS results. In agreement with previous studies (Bar-Haim, Lamy, & Glickman, 2005; Folstein & Van Petten, 2008; Mapelli, Di Rosa, Cavalletti, Schiff, & Tamburin, 2014; Polich & Kok, 1995; Thai, Taber-Thomas, & Pérez-Edgar, 2016), adaptive mean amplitudes (μV) and peak latencies (ms) were extracted for P2 (120–250 ms) and N2 (150–300 ms) in the fronto-central midline electrodes (Cz, Fz), and for P3 (250–500 ms) in the centro-parietal midline electrodes (Cz, Pz), based on their usual topographical distribution. In addition, PLS analysis made it possible to detect an early prefrontal N1 component (here called PF-N1) sensitive to the type of distractor (see PLS LV3 results in the supplementary materials for details), that we further explored using traditional ERP analyses in the 50–150 ms time window in the prefrontal electrode sites (Fp1, Fp2, F3, F4, Fz, Cz).

The amplitudes and latencies of the ERP differences in the obese and normal weight individuals were investigated applying a 2 × 3 × 2 mixed ANOVA with the group (obese, normal weight) as the between-subject factor and the distractor (food, object, neutral) and correspondence (C, NC) as the within-subject factors. Bonferroni post-hoc correction for multiple comparisons was calculated for the significant effects.

2.5.4. Correlations

Pearson partial correlations adjusted for group were calculated. The hunger/satiety/desire–to-eat ratings at T0 and T1 were correlated with RTs for food distractor in the C and NC conditions and with the magnitude of the Simon effect for the food distractor. Additional correlations were calculated between the hunger/satiety/desire-to-eat ratings and those ERP components modulated by food-related stimuli.

3. Results

3.1. Self-report measures

An analysis of the questionnaires showed that the obese individuals had higher scores on the BES $t(47) = 4.71$, $p = .0001$, Y-FAS $t(47) = 3.67$, $p = .0001$, EAT-26 $t(47) = 3.04$, $p = .01$, DEBQ-emotional $t(47) = 4.61$, $p = .0001$, and PFS $t(47) = 2.72$, $p = .01$ with respect to the normal weight individuals. No significant differences were observed in the DEBQ-external, DEBQ-restrained or self-reported impulsivity (all p's > .05; see Table 1 for details).

An analysis of the subjective hunger/satiety/desire-to-eat ratings showed a significant effect of group for the hunger $F(1, 46) = 7.33$, $p = .009$, $\eta_p^2 = .14$, satiety $F(1, 46) = 10.7$, $p = .002$, $\eta_p^2 = .19$ and the desire-to-eat $F(1, 46) = 6.57$, $p = .001$, $\eta_p^2 = .13$ (see Table 2). The obese individuals reported lower levels of hunger and desire-to-eat, and a higher level of satiety with respect to the normal weight group (Table 2).

Table 2
Mean (SD) Likert scales hunger/satiety/desire to eat.

	Normal weight	Obese
Hunger	1.87 (.392)	.375 (−392)
Satiety	3.08 (.441)	1.04 (.441)
Desire to eat	2.29 (.497)	.488 (.497)

Lower values indicate lower hunger, satiety, and desire to eat; *Notes*: SD = standard deviation. * p < .05; ** p < .001.

3.2. Modified affective Simon task

The ANOVA on the RTs showed a main effect of distractor: $F(2, 96) = 27.7$, $p = .00001$, $\eta_p^2 = .37$, with faster RTs for the neutral distractor with respect to the other two distractors (neutral: 577 ms ± 85; food: 591 ms ± 87; object: 589 ms ± 88; mean ± SD; Bonferroni correction: neutral vs. food, $p = .0001$; neutral vs. object, $p = .0001$), and a main effect of correspondence: $F_{1,\ 48} = 107$, $p = .000001$, $\eta_p^2 = .69$, with faster RTs in the C (565 ms ± 85) with respect to the NC (606 ms ± 90). A significant distractor x correspondence interaction was found: $F(2, 96) = 8.52$, $p = .0003$, $\eta_p^2 = .15$, with faster RTs in the NC trials with a neutral distractor with respect to the other two distractors (neutral NC = 593 ms ± 88; food NC = 615 ms ± 90; object NC = 610 ms ± 94; Bonferroni correction: neutral NC vs. food NC, $p = .0001$; neutral NC vs. object NC, $p = .0001$). Interestingly, a significant distractor x correspondence x group interaction was found: $F(2, 96) = 3.73$, $p = .027$, $\eta_p^2 = .07$, although the post-hoc tests did not reveal any significant differences between the group and correspondence. To further explore the triple interaction, a second ANOVA was performed on the magnitude of the Simon effect (i.e., the difference in the RTs between the NC and C trials) separated for the three distracting conditions. The results showed a main effect of distractor: $F(2, 96) = 8.52$, $p = .0003$, $\eta_p^2 = .151$, highlighting a larger interference effect for both the food and object distractors with respect to the neutral distractor (neutral = 32 ms ± 32; food = 47 ms ± 30; object = 42 ms ± 33; Bonferroni correction: neutral vs. food, $p = .0001$; neutral vs. object, $p = .022$). A significant distractor x group interaction was found: $F(2, 96) = 3.73$, $p = .027$, $\eta_p^2 = .07$, showing that in the obese group the interference effect was larger in the presence of food distractors with respect to the neutral one (see Fig. 2, Table 3); there were no differences between the other two distractors (Bonferroni correction: food vs. neutral, $p = .0001$; neutral vs. object, $p = .12$; food vs. object, $p = .72$). In the normal weight group there were no differences across distractors (all p's > .1).

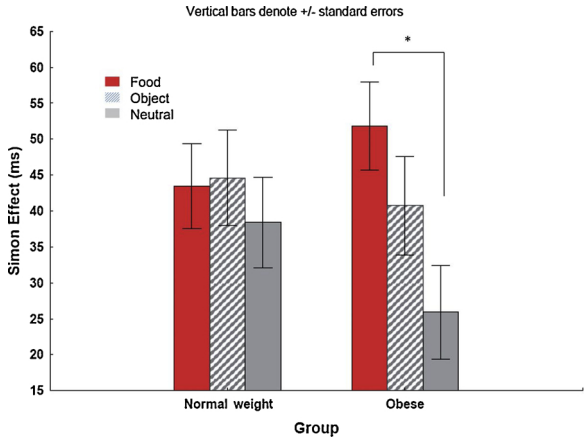

Fig. 2. The Simon effect (ms) in the obese and normal weight subjects during the trials with different distractors: food (red), object (grey stripes), neutral (grey). The vertical bars denote standard errors. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 3
Mean (SD) in the Simon task.

	Normal weight	Obese
RTs	ms	ms
Food C	567 (96)	567 (79)
Food NC	611 (96)	619 (84)
Object C	566 (93)	569 (78)
Object NC	611 (106)	610 (68)
Neutral C	558 (90)	563 (83)
Neutral NC	596 (99)	589 (75)
Accuracy (%)	%	%
Food C	98 (2)	97 (2)
Food NC	93 (5)	92 (5)
Object C	98 (2)	97 (2)
Object NC	93 (5)	92 (5)
Neutral C	97 (3)	97 (2)
Neutral NC	95 (3)	94 (4)
Simon Effect (NC-C)	ms	ms
Food	43 (33)	52 (25)
Object	45 (43)	41 (16)
Neutral	38 (39)	26 (21)

Notes: SD = standard deviation; C = corresponding trials; NC = non-corresponding trials.

The ANOVA on the accuracy showed a main effect of distractor: $F(2,96) = 3.97$, $p = .022$, $\eta_p^2 = .08$, with a higher accuracy for the neutral distractor with respect to the object but not to the food distractor (neutral = 96 % ± 3; food = 95 % ± 3; object = 95 % ± 3; Bonferroni correction: neutral vs. object $p = .03$). The analysis also indicated a main effect of correspondence: $F(1, 48) = 53.2$, $p = .000001$, $\eta_p^2 = .53$, with a higher accuracy in the C (98 % ± 2) with respect to the NC (93 % ± 5) trials as well as a significant distractor x correspondence interaction: $F(2, 96) = 12.8$, $p = .000001$, $\eta_p^2 = .21$, showing a higher accuracy for the neutral distractor with respect to the object and food distractors, but only during the NC trials (neutral NC = 95 % ± 41; food NC = 93 % ± 6; object NC = 93 ± 5; Bonferroni correction: neutral vs food, $p = .00001$; neutral vs. object, $p = .0003$). The means and standard deviations of the RTs and the accuracy in the different task conditions in each group are outlined in Table 3.

The Pearson correlation between the cognitive control measures for food distractors and subjective hunger/satiety/ and desire-to-eat perceptions uncovered that he desire to eat at T0 correlated with both RTs in the NC trials with food distractors ($r = -.326$, $p = .022$; Fig. 3A) and with the magnitude of the Simon Effect in the food condition ($r = -.346$, $p = .015$, Fig. 3B), showing slower RTs and a higher Simon Effect in those individuals who expressed a greater desire to eat at baseline.

3.3. ERP results

The PLS results highlighted the principal effect of the type of distractor, the correspondence and the group in the different time-windows and topographies (see Supplementary material). Only the traditional ERP analysis of the amplitudes and latencies are outlined here (see Fig. 4 for an overview of the ERP components and topographies).

The ANOVA for the PF-N1 amplitude showed a main effect of distractor: $F(2, 96) = 10.7$, $p = .00006$, $\eta_p^2 = .18$, showing a higher PF-N1 for food with respect to the other two distractors (neutral = −2.22 μV ± 1.37; food = −3.19 μV ± 2.11; object = −2.59 μV ± 2.10; Bonferroni correction: food vs. neutral, $p = .00001$; food vs. object, p = .01).

The ANOVA for the PF-N1 latency showed a significant effect of distractor: $F(2, 96) = 29.5$, $p = .0000001$, $\eta_p^2 = .38$, with a longer latency for the neutral distractor with respect to the other two (neutral = 91 ms ± 10; food = 81 ms ± 9; object = 80 ms ± 9; Bonferroni correction: neutral vs. food, $p = .0001$; neutral vs. object

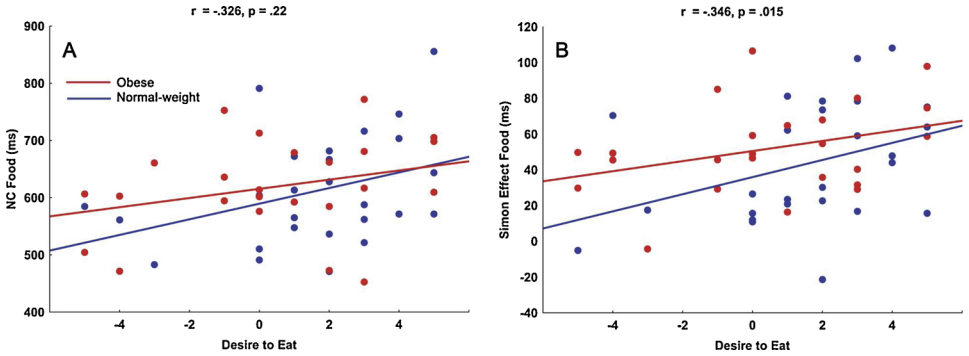

Fig. 3. Pearson partial correlations adjusted for the two groups (obese, normal weight): A) The RTs during the NC trials with food distractors vs the desire-to-eat; B) The Simon Effect for the food distractors vs the desire-to-eat.

p = .0001). The mean and standard deviations of the PF-N1 amplitude and latency are depicted in Table 4.

The ANOVA for the P2 amplitude showed a main effect of distractor: $F(2, 96) = 4.64$, $p = .01$, $\eta_p^2 = .09$, showing a greater P2 amplitude for the object distractor compared to food distractor (neutral = 3.55 μV ± 2.78; food = 3.13 μV ± 3.05, object = 3.99 μV ± 2.99; Bonferroni correction: food vs. object, $p = .0001$). No other main effects or interaction, including the groups, were found for the P2 amplitude.

The ANOVA for the P2 latency highlighted a significant effect of distractor: $F(2, 96) = 40.3$, $p = .00001$, $\eta_p^2 = .46$, showing a longer P2 latency for the neutral distractor with respect to the food and object distractors (neutral = 157 ms ± 25; food = 134 ms ± 18, object = 135 ms ± 14; Bonferroni correction: neutral vs food $p = .0001$; neutral vs object, $p = .0001$). A significant main effect of group was also found: $F(1, 48) = 4.64$, $p = .036$, $\eta_p^2 = .46$, with the obese showing longer latencies (146 ms) with respect to their counterparts (137 ms ± 14) (see Fig. 5). A significant interaction between group and distractor was found: $F(2, 96) = 3.07$, $p = .05$, $\eta_p^2 = .06$. The post-hoc analysis showed a shorter latency for food and object distractors with respect to the neutral distractor in both the obese (neutral = 157 ms ± 25; food = 141 ms ± 18; object = 141 ms ± 12; Bonferroni correction: neutral vs food, $p = .006$; neutral vs object, $p = .002$) and normal weight individuals (neutral = 156 ms ± 25;

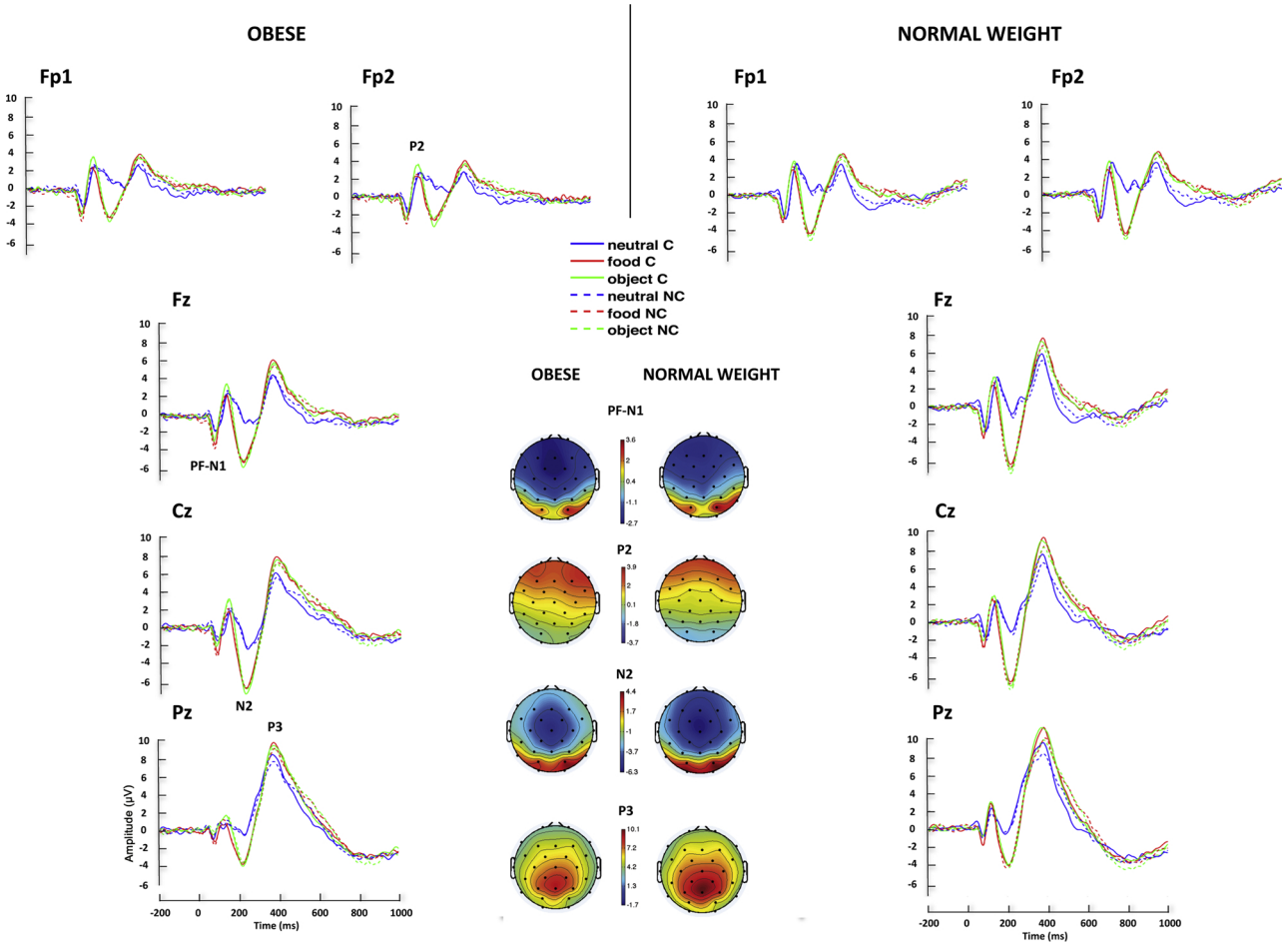

Fig. 4. The ERP waveforms and amplitude distribution of the PF-N1, P2, N2, and P3 components for the obese (on the left) and normal weight (on the right) participants.

Table 4
Mean (SD) PF-N1 amplitude (μV) and latency (ms).

	Normal weight	Obese
PF-N1 Amplitude	μV	μV
Food C	−3.11 (2.40)	−3.01 (2.15)
Food NC	−3.22 (2.10)	−3.42 (2.23)
Object C	−2.62 (2.19)	−2.68 (1.67)
Object NC	−2.73 (2.29)	−2.31 (2.51)
Neutral C	−2.42 (1.67)	−1.98 (1.21)
Neutral NC	−2.70 (1.57)	−1.76 (1.14)
PF-N1 Latency	ms	ms
Food C	81 (10)	84 (14)
Food NC	79 (9)	81(8)
Object C	78 (10)	81 (10)
Object NC	80 (9)	81 (14)
Neutral C	93 (10)	90 (12)
Neutral NC	91 (12)	88 (10)

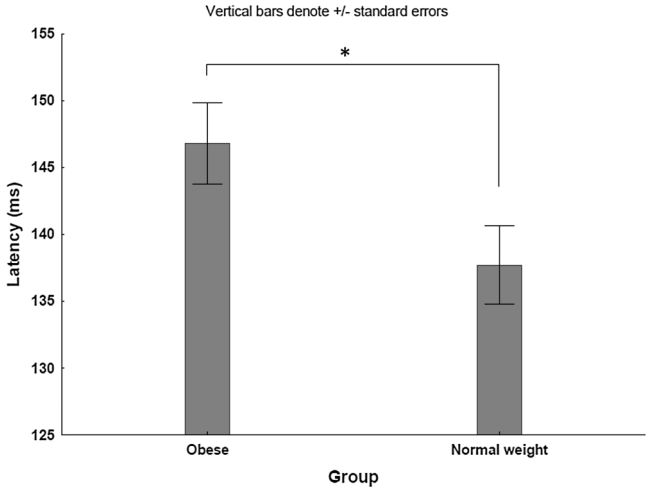

Fig. 5. P2 latency (ms) in the obese (on the left) and normal weight (on the right) subjects. The vertical bars denote standard errors.

Table 5
Mean (SD) P2 amplitude (μV) and latency (ms).

	Normal weight	Obese
P2 Amplitude	μV	μV
Food C	3.26(2.99)	2.94 (3.39)
Food NC	3.25 (3.29)	3.06 (2.95)
Object C	3.87 (3.09)	4.02 (2.93)
Object NC	3.97 (3.33)	4.12 (2.98)
Neutral C	3.91 (3.15)	3.17 (2.63)
Neutral NC	3.64 (3.06)	3.47 (2.51)
P2 Latency	ms	ms
Food C	127 (19)	144 (27)
Food NC	127 (13)	138(12)
Object C	127 (13)	143 (14)
Object NC	130 (13)	139 (13)
Neutral C	158 (26)	159 (26)
Neutral NC	154 (25)	155 (28)

food = 127 ms ± 15; object = 129 ms ± 12 ms; neutral vs food, $p = .0001$; neutral vs object, $p = .0001$). To further explore this interaction, the effect of the food and object distractors were isolated by separately subtracting their latency from the latency of the neutral distractor (i.e. Food-Neutral; Object-Neutral). The ANOVA analysis did not however uncover a significant main effect of the group: $F(1, 48) = 3.85$, $p = .055$, $\eta_p^2 = .07$. The mean and standard deviations of the P2 amplitude and latency are depicted in Table 5.

The ANOVA for the N2 amplitude showed a main effect of distractor: $F(2, 96) = 124$, $p = .00001$, $\eta_p^2 = .72$, with a smaller amplitude for the neutral distractor with respect to the other two (neutral = −2.69 μV ± 3.53; food = −6.84 μV ± 4.17, object = −7.01 μV ± 3.84; Bonferroni correction: neutral vs food, p = .0001; neutral vs object, p = .0001). A significant distractor x group interaction was also found: $F(2, 96) = 3.77$, $p = .026$, $\eta_p^2 = .073$, and post-hoc analysis revealed a larger N2 amplitude for food and object distractors with respect to the neutral distractor in both the obese (neutral = −3.2 μV ± 4.08; food = −6.64 μV ± 4.27: object = −6.73 μV ± 4.09; Bonferroni correction: neutral vs food, p = .0001; neutral vs object, p = .0001;) and normal weight individuals (neutral = −2.22 μV ± 2.94; food = −7.02 μV ± 4.16; object = −7.28 μV ± 3.64; Bonferroni correction: neutral vs food, $p = .0001$; neutral vs object, $p = .0001$). Just as for the P2 latency, the effect of food and object distractors were isolated by separately subtracting the ERP amplitudes of the two conditions from the one of the neutral (i.e. Food-Neutral; Object-Neutral). The ANOVA on these differential scores showed a main effect of group: $F(1, 48) = 4.06$, $p = .049$, $\eta_p^2 = .08$, with a smaller amplitude of the N2 for the obese (−3.47 μV ± 2.41) with respect to their counterparts (−4.93 μV ± 2.68); see Fig. 6. No significant interaction between the group and distractor was found.

The ANOVA for the N2 latency showed a significant distractor effect: $F(1, 48) = 5.37$, $p = .006$, $\eta_p^2 = .10$, suggesting a longer latency for the neutral distractor with respect to the other two (neutral = 239 ms ± 39; food = 228 ms ± 27; object = 230 ms ± 24; Bonferroni correction: food vs. neutral, $p = .009$; neutral vs object, $p = .04$). The mean and standard deviations of the N2 amplitude and latency are depicted in Table 6.

The ANOVA for the P3 amplitude highlighted the main effects of distractor: $F(2, 96) = 52.9$, $p = .000001$, $\eta_p^2 = .52$, showing greater amplitudes for both the food and object distractors with respect to the neutral one (neutral = 8.48 μV ± 3.68 μV; food = 10.3 μV ± 4.31; object = 10.01 μV ± 4.15; Bonferroni correction: neutral vs food, $p = .0001$; neutral vs object, $p = .0001$), as well as a main effect of correspondence: $F(1, 48) = 28.2$, $p = .000001$, $\eta_p^2 = .37$, showing a greater P3 amplitude for the C (10.3 μV ± 4.21) with respect to the NC trials (9.18 μV ± 3.83). No other main effects or interactions were found.

The ANOVA for the P3 latency highlighted a main effect of distractor: $F(2, 96) = 6.94$, $p = .002$, $\eta_p^2 = .13$, with a shorter latency for the neutral distractor with respect to the other two (neutral = 387 ms ± 60; food = 401 ms ± 54: object = 403 ms ± 62; Bonferroni correction: neutral vs food, $p = .022$; neutral vs object, $p = .005$); a main effect of correspondence: $F(1, 48) = 8.61$, $p = .005$, $\eta_p^2 = .15$, with a shorter latency in the C (392 ms ± 54). With respect to the NC trials (401 ms ± 59). The main effect of group did not reach statistical significance: $F(1, 48) = 3.86$, $p = .055$, $\eta_p^2 = .08$, although the obese showed a numerical delay in the P3 latency (413 ms ± 72) with respect to their counterparts (382 ms ± 31). The mean and standard deviations of the P3 amplitude and latency are depicted in Table 7.

Finally, despite the fact that the food distractor had an effect on the PF-N1 amplitude, no correlations were found between the ERP and the hunger/satiety/desire-to-eat perception.

4. Discussion

The current study used behavioral and ERP measures to investigate cognitive control in the presence of food and object distracting images in severely obese and normal weight individuals. Interference control was assessed using a novel affective Simon task utilizing corresponding and non-corresponding target stimuli and three distractors (i.e. food, object and neutral). After a 6 h fast, the participants completed questionnaires investigating eating-related attitudes and trait impulsivity, answered questions about hunger and desire-to-eat, and participated in the experimental session. At the end of the session, the participants

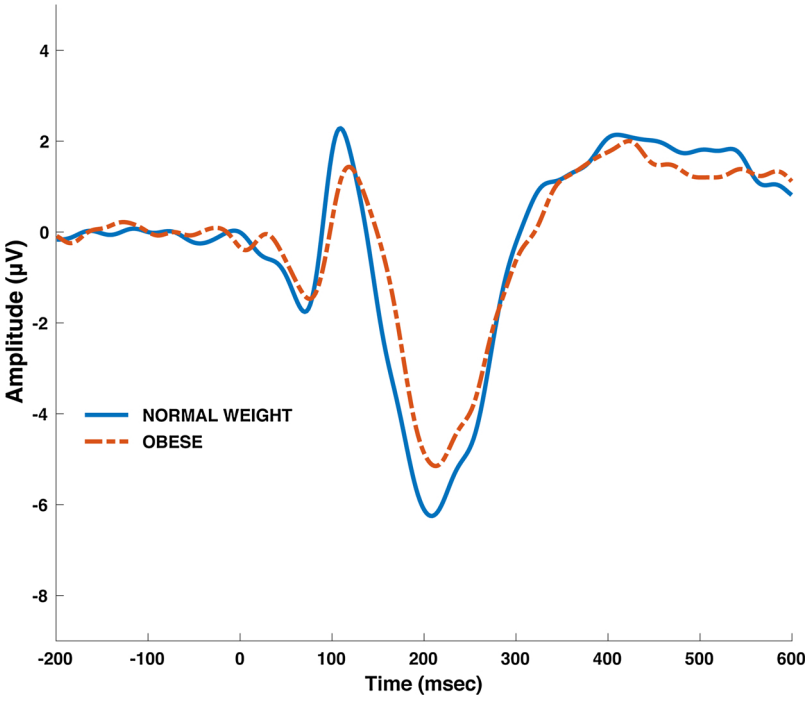

Fig. 6. ERP difference between Food/Object distractors and Neutral, which depicts the N2 amplitude's interaction between group and distractor. Obese (red line), normal weight (blue line). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 6
Mean (SD) N2 amplitude (μV) and latency (ms).

	Normal weight	Obese
N2 Amplitude	μV	μV
Food C	−6.85 (3.92)	−6.67 (4.27)
Food NC	−7.18 (4.47)	−6.61 (4.33)
Object C	−7.11 (3.66)	−6.98 (4.39)
Object NC	−7.45 (3.72)	−6.47 (3.89)
Neutral C	−2.20 (3.01)	−3.03 (4.27)
Neutral NC	−2.24 (3.06)	−3.39 (4.04)
N2 Latency	ms	ms
Food C	232 (43)	239 (37)
Food NC	227 (38)	234 (43)
Object C	221 (20)	237 (34)
Object NC	221 (29)	234 (28)
Neutral C	223 (43)	304 (25)
Neutral NC	226 (38)	238 (31)

Table 7
Mean (SD) P3 amplitude (μV) and latency (ms).

	Normal weight	Obese
P3 Amplitude	μV	μV
Food C	11.4 (4.45)	10.1 (4.58)
Food NC	10.3 (4.41)	9.18 (4.03)
Object C	11.1 (4.67)	9.96 (4.20)
Object NC	9.93 (4.08)	9.19 (3.89)
Neutral C	9.49 (4.09)	8.63 (3.72)
Neutral NC	8.50 (3.61)	8.22 (3.61)
P3 Latency	ms	ms
Food C	383 (28)	410 (70)
Food NC	387 (24)	421 (74)
Object C	388 (44)	410 (72)
Object NC	392 (41)	422 (87)
Neutral C	360 (38)	407 (72)
Neutral NC	379 (55)	406 (78)

were once again questioned about their hunger and desire-to-eat.

Slower RTs were observed for the non-corresponding conditions and for the trials with distracting images. When the interaction between the correspondence, distractor, and group was analyzed, we found that the correspondence effect (i.e., the Simon effect) was larger for the food images with respect to the neutral distractor *only* in the obese individuals. These results indicate that distractors interfered with cognitive control mechanisms during response selection in both groups, although the effect was greater for the food images in the obese population. This is consistent with the theory that obese individuals are able to exercise cognitive control over some stimuli but with lesser extends over food (Houben et al., 2014; Price et al., 2005), probably because of a hypersensitivity of the food reward system.

Independently of BMI values, the desire-to-eat correlated positively with the magnitude of the Simon effect for the food images and with the RTs in the NC trials for food distractor. This result seems to suggest that food images delayed the response selection in the more demanding cognitive control conditions (e.g. NC trials), especially in those individuals experiencing a strong interest in/desire for food intensified in this case by fasting which could have modulated the ability to exert top-down cognitive control enhancing attentional resources toward food stimuli.

ERP analysis revealed an early PF-N1 response with a higher amplitude for the food distractors. Just as in Feig et al.'s study (2017), the PF-N1 evoked by the food cues seemed to reflect an early facilitation in object recognition of behavioral-relevant environmental stimuli. Indeed, a magnetoencephalography (MEG) study showed that a cortical network including regions of the orbitofrontal cortex (OFC) mediated object recognition at early stages of information processing at ≈50 ms after stimulus onset (Bar et al., 2001). The OFC, which is part of the dopaminergic reward system, is known to be involved in hedonic food processing in reinforcement learning and in the formation and long-term consolidation of S-R associative clusters. In view of these considerations, the PF-N1 could be considered an early neural marker of evaluative processes of sensory information, probably associated with

the fast/indirect impulsive pathway.

Although we expected to find some correlations between the PF-N1 for the food distractors and the subjective hunger/desire-to-eat, the results did not prove to be statistically significant. Nevertheless, although all the participants were tested while they were in the same metabolic state (i.e., fasting), the two groups showed differences in their hunger perception and desire-to-eat. If a relationship does indeed exist between the two variables, it could have been masked by the differences in the two groups. Other ERP studies are needed to better control these variables.

The analysis of the P2 component showed shorter latencies for the distractors (i.e. food and object) at the middle/later stages of information processing (150–400 ms). Although specific food-related modulations of the P2 or other later ERP components (e.g. P3) were not present, as has been reported by other studies (Hume et al., 2015; Nijs et al., 2010), the considerable differences in the task adopted (Stroop task) and the participants' metabolic state of fasting could explain the discordant results. Nevertheless, the P2 is thought to reflect mechanisms of selective attention and feature detection (Hillyard, Teder-Sälejärvi, & Münte, 1998; Luck & Hillyard, 1994), in which case shorter latencies for food and object images could suggest that distracting stimuli engage attentional resources required to process information needed for task accomplishment. Interestingly, longer overall P2 latencies were detected in the obese individuals regardless of the type of distractors or correspondence, reflecting a delayed engagement of attentional processing, which could be explained by an altered control of selective attention and/or suppression of distracting information (Prickett et al., 2015).

The analysis of the N2 component, which is considered a neural marker of the engagement of selective attention toward relevant and irrelevant information, revealed longer latencies and higher amplitudes for both the food and object distractors. The N2 is usually enhanced when different sources of perceptual information compete to recruit attentional resources depending on task demands (Folstein & Van Petten, 2008). Interestingly, when the effect of distractors was isolated from the neutral condition (i.e., the differential scores: food-neutral; object-neutral), a smaller N2 amplitude was detected in the obese individuals. This is coherent with the key role played by the N2, whose neural generator is probably localized in the anterior cingulate cortex, in conflict detection and cognitive control (Folstein & Van Petten, 2008). The reduced effect of distractors on the N2 amplitude that was observed during the Simon task in the obese individuals showed that at least in this case it reflected reduced cognitive control ability.

The P3 analysis uncovered longer latencies and higher amplitudes for both distractors. The P3, which is thought to be associated with working memory updating and speed of information processing (Polich, 2007), is a positive deflection, reaching its maximum amplitude over the parietal sites of the scalp, with peak latency occurring at roughly 300–400 ms after stimulus onset. The effect of distracting images on this component may be regarded as a neurophysiological correlate of the conscious categorization of stimuli and of directing attention toward task-related information. A longer P3 latency and lower amplitude in the non-corresponding condition were detected here, confirming evidence in the literature on the effects of spatial S-R correspondence on the P3 parameters (Donchin & Coles, 1988; Leuthold & Sommer, 1999; Leuthold, 2011; Ragot & Renault, 1981; Ragot, 1984; Smulders, 1993).

Study findings on the whole suggest that the P2, N2 and P3 ERP components reflect the activity of a frontal-parietal network known to be involved in deliberate attention and cognitive control processes (Polich, 2007; Sur & Sinha, 2009) that should be considered neural markers of processing within the reflective indirect pathway during deliberate response selection (Polich, 2007). Given the link between these components and the reflective indirect pathway, the fact that the obese participants showed overall delayed P2 latency and smaller N2 amplitude to distractors would support the hypothesis of weakened deliberative cognitive control ability.

All things considered, the present findings seem to point in the direction of the dual-route model of healthy/unhealthy behaviors (i.e., overeating; Wiers et al., 2013). Since obese individuals seem to be unable to inhibit prepotent responses in the presence of food distractors when cognitive control is required, the attentional bias toward food found at a neurophysiological level (i.e. larger PF-N1) may contribute to reducing cognitive control towards food stimuli in this population (i.e., larger Simon effect for food distractors). This is probably linked to an alteration in deliberative processes associated with the reflective system, evidenced by the longer P2 latency for distractors and smaller N2 amplitude, and reflecting an imbalance between the impulsive and reflective systems leading to impulsive responses toward food.

Our data also suggest that reduced control for food distractor could have been linked to the participants' metabolic state and subjective hunger as a correlation between the magnitude of the Simon effect and the desire-to-eat value was found in both the groups at a behavioral level. Unfortunately, similar results were not observed for neurophysiological data.

The study has some limitations. First, many of the obese patients seeking bariatric treatment presented serious medical co-morbidities such as diabetes, cardiovascular diseases and OSAS. Given the potential influence of these comorbidities over cognitive functions, it will need to be controlled during future studies. Second, only severely obese individuals and normal weight subjects were studied. Future studies will need to examine subjects with various grades of obesity and different dieting histories. Third, as mentioned above, the participants were tested only in a fasting state; the results may have been different if the participants had recently eaten as other factors may have been involved in the participants' cognitive control. Finally, the sample size of the study was relatively small. Studies examining larger groups of individuals will be able to produce more generalizable findings.

In conclusion, the study showed evidence of an early neural marker of sensory evaluation of behavioral relevant food-related information in the prefrontal areas (i.e., PF-N1) probably linked to the impulsive information processing system. The fact that the obese participants showed delayed P2 latency and smaller N2 amplitude in response to distracting stimuli probably reflects weakened processes in the reflective system. Study findings suggest that the interaction between the two information processing systems may affect cognitive control and response selection toward food stimuli in connection to the individual's metabolic state and, in obese individuals, to a dysfunctional neural mechanism of cognitive control.

Looking ahead, it will be interesting to evaluate the obese participants' cognitive and behavioral control after surgery and weight loss. Identifying the mechanisms that can modulate healthy/unhealthy eating behaviors will help investigators develop measures to enhance cognitive control abilities and to modify non-conscious processes associated to unhealthy behaviors (Wiers et al., 2013).

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.biopsycho.2019.107804.

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