Inhibitory control pathway to disinhibited eating: A matter of perspective?

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ABSTRACT

Recent studies highlight the importance of disinhibited eating and underlying inhibitory control deficits in the maintenance of obesity. So far, inhibition facets have been examined in isolation and findings are inconsistent due to different measures. This study illustrates the multifaceted nature of inhibitory control by comparing different inhibition stages in outpatients with chronic overweight (with binge eating disorder, BED, n = 24; Non-BED, n = 47) and healthy controls (HC, n = 30). Besides reporting impulsive patterns (UPPS), participants performed the Food Stroop (FST), Door Opening (DOT) and Stop Signal (SST) task with food and generic stimuli. The results showed a significant influence of self-reported inhibition deficits on body weight in outpatients irrespective of binge eating. On a behavioral level, BED exhibited deficits in focusing on a task (FST) but not to Non-BED but performed better in inhibiting an already initiated response (SST) compared to Non-BED and HC regardless of stimulus category. In sum, first-stage deficits in interference inhibition might be attributable to the initiation of eating episodes, while deficits in the late-stage interruptive inhibition might result in loss of control over an eating episode especially in BED if executive resources are depleted. Under executive control, BED might perform better, given their daily practice. The inclusion of a holistic inhibitory control pathway offers a further step in obesity research.

1. Introduction

In the context of our obesogenic environment, most treatment-seeking patients with overweight find it challenging to maintain their reduced weight after a moderate weight loss of between 5% and 10% of their initial weight (Bischoff et al., 2012). This unsuccessful weight loss maintenance is often accompanied by feelings of guilt and shame, which further reinforce patients' low expectations of self-efficacy and ultimately lead to chronicity. Such findings are alarming given the steady rise in rates of overweight and obesity (BMI ≥ 25 m/kg²) in adults, currently lying at 39.2% of women and 38.5% of men worldwide (World Health Organization [WHO], 2017). Since 1975, especially obesity rates (BMI ≥ 30 m/kg²) have nearly tripled both in the United States (female +19.5%, male +18.5%) and in Europe (female +11.6%, male +15.5%) with no current prospect of reaching a plateau (NCD Risk Factor Collaboration [NCD-RisC], 2017; OECD Health Statistics, 2018; World Health Organization, 2017).

Previous studies conducted to improve the understanding of maintaining factors of obesity highlighted the importance of disinhibited eating patterns in a specific subgroup of individuals with overweight and obesity (Bryant, King, & Blundell, 2008; O’Neill et al., 2012) such as “overeating”, “stress-induced/emotional eating”, “craving for sweets”, “grazing” (uncontrolled, repetitive eating of small amounts of food all day long) and “binge eating” (Burgmer et al., 2005; Carter & Jansen, 2012; Chacko, Chioldi, & Wee, 2015). At the same time, individuals with disinhibited eating might exert more cognitive restraint at times in order to compensate for their greater disinhibition, offering a good explanation for yo-yo effects of dieting (Spinella & Lyke, 2004). In some cases, these disinhibited eating patterns develop into full-blown eating disorders (EDs). According to the DSM-5, 9% of adults seeking weight-loss treatment meet criteria for binge-eating disorder (BED) and 21% for other specified feeding or eating disorders (OSFED), including...
Deficits in interference inhibition

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Table 1

Definition and widely used measurement instruments for distinct inhibitory control processes.

<table>
<thead>
<tr>
<th>Deficit in interference inhibition</th>
<th>Measurement instruments</th>
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<tbody>
<tr>
<td>Inhibition of an already initiated motor response</td>
<td>Go/No-Go task (action restraint), Stop Signal task (action cancellation)</td>
</tr>
<tr>
<td>Inhibition of a predominant or already initiated motor response</td>
<td>Door Opening task, Iowa gambling task, Game of Dice task</td>
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<tr>
<td>Inhibition of a pre-potent response for which immediate reinforcement is available, emphasizing relatively better processing mode</td>
<td>Delay/Temporal discounting task, Door Opening task, Iowa gambling task</td>
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<tr>
<td>Inhibition of task-irrelevant competing information</td>
<td>Visual-probe task, (Food) Stroop task</td>
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<tr>
<td>Inhibition of an urge to grab food that is inappropriate, in conflict with current goals</td>
<td>Antecede task, inhibition of return in Attentional/Spatial working memory</td>
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Moreover, the distinction between a proactive (preparation for action) and reactive (response inhibition, action withholding [inhibition of motor action impulses] and action cancelation [inhibition of already initiated actions]) inhibition processes can be divided into cognitive (including mental and attentional processes that suppress task-irrelevant information in the sense of a interference control) and behavioral mechanisms. In turn, behavioral inhibition includes reward-based or motivational inhibition, such as delayed gratification, and response inhibition, defined as the ability to withhold a motor response (see classification by Bartholdy et al., 2016 and Wu et al., 2016, based on Bari & Robbins, 2013). Various authors suggest that these inhibition processes are necessary at different points between the period when an internal stimulus (e.g. feeling sad and thinking about eating as a method to console oneself) or external stimulus (e.g. the smell of tasty cake) emerges and a response - to eat or not to eat - is generated, carried out or inhibited (Barkley, 1997, 2010; Bartholdy et al., 2016; Bjorklund & Harnishfeger, 1995). Dawe and Loxton (2004) postulated for example, that experiencing certain food stimuli as highly rewarding is associated with the initiation of eating, whereas only rash-spontaneous impulsiveness leads to objective binge eating episodes in BED. Neurocognitive findings confirm different successive, but interfering inhibition processes along a temporal dimension through overlapping brain activation patterns (see e.g. Chambers, Garavan, & Bellgrove, 2009; Sebastian et al., 2013): interference inhibition, action withholding [inhibition of motor action impulses] and action cancelation [inhibition of already initiated actions].

In the current field of research on impulsivity and obesity, the Dual Process Model of Strack and Deutsch (2004) is often used to explain impulsive actions as a conflict between an impulsive and reflective processing mode (see e.g. Jones, Hardman, Lawrence, & Field, 2018). Automated “bottom-up” processes seem to arise from stimulus-response pairs of food and reward experience, and lead to impulsive, resource-saving responses to food-related stimuli. An excessively activated automated system may result in disinhibited eating if it encounters a weakened reflective system (Hofmann, Friese, & Roefs, 2009). Reflective “top-down” processes are closely related to the executive functions, which include abilities of working memory, set-shifting as well as inhibitory control and emotion regulation (Hofmann, Friese, & Strack, 2009).

Inhibitory control, as one important reflective executive process, is defined as the overall ability to inhibit a planned, predominant or already initiated response (Bartholdy, Dalton, O’Daly, Campbell, & Schmidt, 2016; Logan, 1994; Verbruggen & Logan, 2009), such as an urge to grab food that is inappropriate, in conflict with current goals (e.g. goal to lose 5 kg) or no longer required, implying a certain overlap with impulsivity. Following Bari and Robbins (2013) and Nigg (2000), inhibition processes can be divided into cognitive (including mental and attentional processes that suppress task-irrelevant information in the sense of a interference control) and behavioral mechanisms. In turn, behavioral inhibition includes reward-based or motivational inhibition, such as delayed gratification, and response inhibition, defined as the ability to withhold a motor response (see classification by Bartholdy et al., 2016 and Wu et al., 2016, based on Bari & Robbins, 2013). Various authors suggest that these inhibition processes are necessary at different points between the period when an internal stimulus (e.g. feeling sad and thinking about eating as a method to console oneself) or external stimulus (e.g. the smell of tasty cake) emerges and a response - to eat or not to eat - is generated, carried out or inhibited (Barkley, 1997, 2010; Bartholdy et al., 2016; Bjorklund & Harnishfeger, 1995). Dawe and Loxton (2004) postulated for example, that experiencing certain food stimuli as highly rewarding is associated with the initiation of eating, whereas only rash-spontaneous impulsiveness leads to objective binge eating episodes in BED. Neurocognitive findings confirm different successive, but interfering inhibition processes along a temporal dimension through overlapping brain activation patterns (see e.g. Chambers, Garavan, & Bellgrove, 2009; Sebastian et al., 2013): interference inhibition, action withholding [inhibition of motor action impulses] and action cancelation [inhibition of already initiated actions].

Besides this theoretical discussion, factor-analytic conceptions suggest two main dimensions, termed “reward sensitivity/drive” and “rash-spontaneous impulsiveness” (Dawe, Gullo, & Loxton, 2004) or a four-factor model based on previous measurement instruments for impulsivity, abbreviated UPPS (Whiteside, Lynam, Miller, & Reynolds, 2005). In the last few years, the assumption of a superior trait “impulsivity” with underlying subcomponents has been increasingly viewed critically (Allom, Panetta, Mullan, & Hagger, 2016).

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inhibition in anticipation of a stimulus, like attentional processes) and a reactive (inhibition of a response) inhibition has become more established (Aron, 2011; Bartholdy et al., 2017).

Research on the maintenance of obesity conducted over the past two decades suggests a strong role of impairments in 1. interference inhibition (or cognitive inhibition), 2. decision-making (or reward-based or motivational inhibition) and 3. response inhibition (Bartholdy et al., 2017; Giel, Teufel, Junne, Zipfel, & Schag, 2017; Wu et al., 2016). However, studies using performance-based measures have reported conflicting findings due to different methods and sample compositions (Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016). To date, inhibition processes have only been examined in isolation and only one study has integrated them into an explanatory model of BED (see food addiction model by Treasure, Leslie, Chami, & Fernández-Aranda, 2018). Table 1 displays an inhibitory control pathway suggesting how impulses to grab food might successively pass through different inhibition processes and lead to disinhibited eating, thus maintaining obesity.

Although no studies have evaluated this model of inhibitory control pathway in individuals with obesity per se, we can highlight recent studies that support the key inhibition processes of the model.

Previous research concerning interference inhibition found no differences in RT for high-caloric palatable food words between adults with obesity and normal-weight controls; the same applied for unspecified color-word versions of the Stroop paradigm (Kittel, Brauhardt, & Hilbert, 2015; Nijs, Franken, & Muris, 2010). In an Attentional cueing task, females without BED experienced more difficulties in disengaging their attention from food than did normal-weight females (Carters, & Hilbert, 2015; Nijs, Franken, & Muris, 2010). In an Attentional cueing task, females without BED experienced more difficulties in disengaging their attention from food than did normal-weight females (Carters, Rieger, & Bell, 2015). Likewise, individuals with BED made significantly more incorrect initial saccades in an Antisaccade task compared to weight-matched controls without BED (Zeehr et al., 2016).

With regard to impulsive decision-making, adults with obesity performed worse than normal-weight controls in reward-related decision making, irrespective of whether or not they had been diagnosed with BED (Davis, Patte, Curtis, & Reid, 2010; Fitzpatrick, Gilbert, & Serpell, 2013; Wu et al., 2016). Nevertheless, the severity of binge eating was strongly positively associated with the performance on the Iowa gambling task (Danner, Onwehoud, van Haastert, Hornsveld, & de Ridder, 2012). Moreover, successful weight loss maintainers made fewer impulsive reward-related decisions in a Game of Dice task compared to their unsuccessful counterparts (Brockmeyer, Simon, Becker, & Friederich, 2017).

Relating to response inhibition, inefficient inhibitors in a Stop Signal task (SST) consumed more in a subsequent snack test if they were also high-restrained eaters (Jansen et al., 2009). Furthermore, participants with normal weight and inhibitory deficits in food-specific SSTs were found to have a higher BMI (Houben, Nederkoorn, & Jansen, 2014) and experienced stronger food craving (Meule, Lutz, Vögele, & Kübler, 2014). Individuals with obesity and normal weight (Hendrick, Luo, Zhang, & Li, 2012; Mühlberg, Mathar, Villringer, Horst, & de Ridder, 2014) and those with obesity and normal weight (Hendrick, Luo, Zhang, & Li, 2012; Mühlberg, Mathar, Villringer, Horst, & de Ridder, 2014) and those with BED and an age- and BMI-matched healthy control group (Wu et al., 2013) showed similar performance in a generic SST. By contrast, Mole et al. (2015) found greater impairments in generic SST performance only in individuals with obesity and no BED. However, individuals with obesity and BED demonstrated more disruptive inhibition deficits compared to a weight-matched group without BED in a food-specific SST (Svaldi, Naumann, Trentowska, & Schmitz, 2014) and irrespective of stimulus category (Manasse et al., 2016).

To summarize, compared to normal-weight controls, individuals with obesity generally show impulsive decision-making specifically towards food. Findings are inconsistent with regard to interference and response inhibition, showing a tendency towards greater food-specific impairments in those with BED (Giel et al., 2017). The past decade has seen rapid advances with respect to the understanding of the complex nature of impulsivity and its role in obesity. However, although experimental studies considering different inhibition stages as interfering processes would provide the opportunity to explain the previous discrepant findings, such studies are lacking. Therefore, it is unsurprising that it currently appears to be a matter of perspective whether inhibitory control deficits underlie the inability to inhibit eating impulses.

Consequently, the aim of the current study was to generate an integrated approach hypothesizing that compared to healthy controls, individuals with overweight and disinhibited eating should show impairments in (i) sustaining their attention on the ongoing task (poor interference inhibition), (ii) delaying gratification (impulsive decision-making) and (iii) inhibiting an already initiated response (poor response inhibition). Moreover, we expected that food-specific stimuli would provoke larger inhibitory control deficits than generic stimuli. Individuals with BED should perform even worse concerning food-specific interference and response inhibition, whereas those without BED should perform better in response inhibition compared to interference inhibition and decision-making.

2. Material and methods

2.1. Participants

Treatment-seeking outpatients with disinhibited eating behaviors and overweight (BMI 25–29.9 kg/m², n = 20) or mild to moderate obesity (BMI 30–39.9 kg/m², n = 51) were recruited through notices in medical centers and from the waiting list of the affiliated outpatient clinic for psychotherapy. To be included in the study, they had to report the therapeutic objective of weight reduction and at least two long-term unsuccessful weight maintenance efforts (i.e. not maintaining a weight loss of over 5% (BMI 25–35 kg/m²) or 10% (BMI > 35 kg/m²) of initial body weight for at least one year; Montesi et al., 2016). To reduce variance in the level of cognitive abilities, only participants aged between 18 and 50 years were included. Exclusion criteria were bulimia nervosa, current dieting, medication-induced or organically caused weight change, pregnancy, presence of substance use disorder, severe self-injurious behavior, acute suicidal ideation or current or past psychotic disorders. The clinical sample was split into a BED group (n = 24), meeting the diagnostic criteria for BED according to DSM-5 and a Non-BED group (n = 47). Furthermore, a healthy control group (HC; n = 30), which did not show disinhibited eating or current or past EDs, was stratified by age and highest level of education. We also included overweight controls in the healthy control group in order to model the average BMI 25.5 kg/m² of 35-40-year-olds in Germany (Federal Statistical Office, 2017). The final sample consisted of N = 101 participants.

2.2. Materials

2.2.1. Measures of inhibitory control

To measure interference inhibition, the Food Stroop task (FST; Pinnow & Kirkcaldy, 2012) was administered. Participants were instructed to tap the corresponding key for the color of the presented word as fast and accurately as possible, whilst inhibiting reading the content of the word. Words of three stimulus categories were used (36 food-related words, e.g. “chocolate”, “bread”; 36 body-related words, e.g. “stomach”, “figure” and 36 semantically unrelated words, e.g. “computer”, “alarm clock”). The words were confirmed as most frequently labeled in the corresponding category and controlled for length (Pinnow & Kirkcaldy, 2012). After a practice block (24 number words), two blocks were presented. Each block included 108 words that were presented in a randomized order. The intertrial interval was 3 s (see Fig. 1). Subsequently, we calculated the speed of correct responses (RTs in ms) for each of the three word categories and food- and body-interference indices (RT differences of the food- and body-condition: foodRT – controlRT and bodyRT – controlRT, respectively). No subjects in the current study fulfilled the outlier criteria defined by Schulz and Liebing (1991), i.e. all RTs were between 150 ms and 1500 ms. To
control for the influence of stimuli characteristics, the emotional valence of all words was rated on a scale from 1 (unpleasant) to 5 (pleasant). The test-retest-reliability was found to be lower for interference indices than for response latencies alone, but nevertheless still adequate (Strauss, Allen, Jorgensen, & Cramer, 2005). Regarding predictive validity, food-interference indices were related to weight gain at 1-year follow-up (Calitri, Pothos, Tapper, Brunstrom, & Rogers, 2010).

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To assess impulsive decision-making, we used an adjusted version of the Door Opening task (DOT. Nederkoorn, Braet, van Eijs, Tanghe, & Jansen, 2006), a gambling task evaluating the individual decision making or response perseverance in pursuit of risky rewards. Participants were able to open a virtual closed door by pressing a key. Behind the door, they found either a) a happy face combined with a low-caloric food stimulus (e.g. tomatoes) or a natural stimulus (e.g. flower), winning a point, or b) a sad face combined with a high-caloric food stimulus (e.g. pizza) or a neutral stimulus (e.g. kitchen utensil), losing a point. The probability of finding a happy face behind a closed door decreased from 100% to 0% over every 10-door trial. Participants were instructed that after each opened door, they could decide whether to open another door or to finish the game and cash the points they had earned, with the objective of earning as many points as possible (see Fig. 1). They were told that they were taking part in a competition with the other participants, starting with a credit of ten points, and that at the end, the “winner” with the most points would receive a voucher for 50 Euros. Participants who open more doors seem to be more reward-sensitive in terms of showing a greater tendency to continue to pursue a response set for reward despite “punishment”. After a small practice block, the food-specific block (food stimuli) and then the generic block (natural and inorganic neutral stimuli) were performed. Each block comprised a maximum of 110 doors. All stimuli generated by Blechert, Meule, Busch, and Ohla (2014) were standardized in terms of background color, distance and figure-ground composition. In previous studies, the DOT was found to differentiate between children with and without impulsive behavior patterns (such as obesity/BED and ADHD) and was thus interpreted as a valid task to measure inhibition of impulsive decision-making (Nederkoorn, Braet et al., 2006; Scholten, Schrijvers, Nederkoorn, Kremers, & Rodenburg, 2014; Verbeken, Braet, Claus, Nederkoorn, & Oosterlaan, 2009).

A food-specific Stop Signal task (SST; Logan & Cowan, 1984) was used to assess response inhibition, i.e. inhibiting a response to a choice reaction time task once an auditory stop signal is presented. On Go trials (75% of trials), participants were instructed to indicate the direction of the presented arrow within appetitive food stimuli (first block) or neutral stimuli (second block). We used 64 high-caloric food stimuli and 64 different neutral stimuli from the previously mentioned food-pics database (Blechert et al., 2014) due to the greater difficulty of inhibiting eating impulses in the context of appetitive food. The response keys were “D” (for left arrow) and “K” (for right arrow). In Stop signal trials (25% of trials), a 100 Hz tone of 100 ms was produced, signaling that the participants should inhibit their responses. The participants were instructed to press the corresponding keys as quickly as possible and not to wait for the Stop signal (see Fig. 1). Each block includes a practice phase of 16 trials and a randomly presented experimental phase of 64 trials. When the delay between Go stimulus and Stop signal (SSD), initially set at 250 ms, increases, the probability of responding correctly on Stop signal trials (p(respond | signal)) increases. The Stop signal reaction time (SSRT) represents the estimated covert latency of stopping, computed by subtracting the average SSD from the median RT in Go trials, with the assumption of an underlying “horse race” between inhibitory and excitatory processes (Logan & Cowan, 1984). Prolonged SSRTs can be interpreted as poorer inhibitory control (e.g. food-specific SSRT = 236 ms (SD = 36 ms) for a BED group vs. 208 ms (SD = 36 ms) for a weight-matched control group, see Svaldi et al., 2014). Reliable estimations of SSRT can be generated if the mean p(respond | signal) is 0.5. Therefore, SSD was increased by 50 ms in the next trial if inhibition was successful and decreased by 50 ms if participants failed to withhold response. In the current study, 17 (food-specific block) and 11 (generic block) subjects, respectively, were identified as outliers following the criteria defined by Congdon et al. (2012), i.e. their percent inhibition on Stop trials was less than 25% or greater than 75%. To ensure sufficient power, these outliers were replaced by mean values. The SSRT showed acceptable test-retest-reliability, with r = .65, p < .001 (Weafer, Baggott, & Witt, 2013) and low convergent validity with other self-control measures (Duckworth &
current study, SSRT was positively correlated with Go RT (r = .467, p < .001), suggesting that our participants did not develop waiting strategies for the Stop signal (Sylwan, 2004). The UPPS Impulsive Behavior Scale (UPPS; Kämpfe & Mitte, 2009; Whiteside et al., 2005) was implemented as a self-report measure to assess distinct impulsive components leading to disinhibited behavior. The 45-item scale yields scores for the following four subscales: “Lack of perseverance” illustrates deficits in the ability to resist intrusive thoughts and remain focused until the end of the task (e.g. “My thinking is usually careful and purpose.”), inverted. “Lack of premeditation” denotes a tendency to make risky decisions without considering the consequences (e.g. “There are so many little jobs to be done that sometimes I just ignore them all.”). “Sensation seeking” describes the willingness to take risks for novel experiences (e.g. “I’ll try anything once.”). “Urgency” describes a tendency to engage in rash action in response to extreme affect (e.g. “When I feel upset, I will often say things that I later regret.”). Items are rated on a 5-point scale (disagree strongly to agree strongly). Acceptable reliability indices were shown in the current study, ranging from Cronbach’s α = .63 (lack of perseverance) to α = .75 (lack of premeditation). The subscales correlated moderately (r = .43 for urgency, lack of premeditation and perseverance, p < .01) to strongly (r = .65 for sensation seeking, p < .01) between self- and peer-assessment and with the BIS-15 (0.37 ≤ r ≤ .67, p < .01), suggesting good convergent validity (Kämpfe & Mitte, 2009; Meule, Vogele, & Kübler, 2011).

2.2.2. Measures of disinhibited eating behavior and ED pathology

The subscale “Disinhibition of control” of the Three-Factor Eating Questionnaire (TFEQ; Pudel & Westenhofer, 1989) was administered to assess subjective loss of control when eating, also referred to as disinhibited eating triggered by emotional or situational cues. The scale consists of 16 items, e.g. “When I feel anxious, I find myself eating.” Some items have a true-false response format, while others are rated on Likert scales of frequency, varying from “never/no” to “always/a lot.” “Disinhibition” was positively correlated with increased food intake and predicted weight loss outcome (Legenbauer et al., 2018). The subscale “Cognitive restraint of eating” (rigid control scale with 16 items) describes a cognitive override of physiological signs of hunger to reduce or maintain weight. The scale was used as a control variable. In the current study, low internal consistencies were found with Cronbach’s α ranging from .65 to .74.

The Eating Disorder Examination-Questionnaire (EDE-Q; Hilbert & Tuschen-Caffier, 2016a) was used to assess the frequency of disordered eating symptoms over the past 28 days. The EDE-Q consists of 20 items, e.g. “Has thinking about food, eating or calories made it very difficult to concentrate on things you are interested in?”, which are summed up to form a global score and the four subscales “Restraint eating”, “Eating concern”, “Shape concern” and “Weight concern”. Two additional items assess the frequency of episodes of overeating and days with objective binge eating episodes in the last 28 days. Cronbach’s α varied from α = .77 (weight concern) to α = .92 (total score), indicating high internal consistency in the current study. Reference values are available for individuals with atypical EDs including BED (M = 3.39, SD = 1.38) and healthy controls (M = 1.44, SD = 1.22) (Hilbert & Tuschen-Caffier, 2016a).

2.3. Procedure

The study was approved by the local ethics committee and registered in the German Clinical Trials Register (DRKS-ID: DRKS00005250). The clinical samples and the HC group were screened for their corresponding inclusion and exclusion criteria by telephone. Afterwards, the diagnostic and experimental sessions took place on separate days in a controlled laboratory environment at two different psychotherapeutic outpatient clinics. Trained graduate and doctoral students in clinical psychology and psychotherapy conducted the diagnostic interviews and measurements. All participants signed a written statement of informed consent and were debriefed in depth at the end of the study.

In the diagnostic session, lifetime EDs of treatment-seeking participants according to DSM-5 criteria were investigated with the Eating Disorder Examination (EDE; Hilbert & Tuschen-Caffier, 2016b). All other diagnoses for Axis I and borderline personality disorder (BPD) for Axis II were checked using the Structured Clinical Interview for DSM-IV (SCID-I/I; First, Spitzer, Gibbon, & Williams, 1997; Wittchen, Zaudig, & Fydrich, 1997). Furthermore, the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 2006) was used to evaluate the severity of comorbid depressive symptoms. The healthy control group was screened for EDs and depressive syndrome, completing corresponding items of the Patient Health Questionnaire (PHQ; Spitzer, Kroenke, & Williams, 1999). Additionally, two items of the SCOFF questionnaire (Morgan, Reid, & Lacey, 1999) related to anorexia nervosa pathology were added. If a current or past ED was indicated, the ED section of the SCID-I was administered.

For the experimental session, the participants were instructed to arrive at the session without feeling hungry. First, all participants completed the UPPS and their height and weight were measured. Furthermore, only the clinical samples filled out the EDE-Q and the disinhibition scale of the TFEQ to ensure test economy. Next, all participants were instructed to evaluate their current level of hunger, satiation, tiredness and mood on a 5-point scale, word-anchored at its endpoints (“not at all/positive” to “extremely/negative”). Following this, they performed the PST, DOT and SST on Inquisit 4 Web (Millisecond Software, Seattle, US), with the order of the experimental tasks being counterbalanced across participants.

2.4. Statistical analyses

Assuming at least moderate differences between the BED and Non-BED group (d = .778 with regard to SSRT, see Svaldi et al., 2014), a priori calculations suggested a total sample size of 69 to provide 80% power at two-sided α = .05 in order to detect main effects. Applying the same conditions, a sample size of 101 was required to detect moderate interaction effects (according to n² = .09, Svaldi et al., 2014).

First, to take into account the multifaceted nature of the inhibitory control pathway, task performances were z-transformed and analyzed using a mixed-design MANOVA with the between-subjects factor “group” (HC, Non-BED, BED) and within-subjects factors “stimulus category” (generic, food-specific) and “inhibition process” (interference inhibition, decision-making, response inhibition). Next, we entered BMI as a continuous covariate in order to test whether weight influenced the effect of group on inhibition process. Additional exploratory analyses with both clinical samples were calculated, including cognitive restraint of eating as a covariate (see Spinella & Lyke, 2004). To break down the interaction results, group differences between outpatients with disinhibited eating and healthy controls were analyzed using one- or two-way independent ANOVAs for each performance-based inhibition process. Furthermore, a one-way MANOVA was performed to investigate group differences in self-reported inhibitory control. If Mauchly’s test indicated violation of the sphericity assumption, degrees of freedom were corrected using Greenhouse-Geisser estimates. Dummy-coded contrasts for planned comparisons were applied using Holm-Bonferroni corrections for multiple testing (cf. aBH). To exclude potential confounding effects of baseline states (tiredness, hunger, satiation and mood) and emotional valence of the stimuli characteristics concerning the PST, separate ANCOVAs were tested if the independence from the experimental effect and the assumption of homogeneity of regression slopes were confirmed. Again, planned contrasts with Holm-Bonferroni corrections were calculated using the regression procedure for
Finally, Pearson’s correlation coefficients were calculated in order to analyze associations between inhibitory control, disinhibited eating, ED psychopathology and BMI. Moreover, we analyzed commission and omission errors of the FST (RT for incorrect trials per condition, % incorrect trials per condition) and the SST (% missing in Go trials, % incorrect Go trials) for associations. Additionally, convergent validities between self-reports and performance-based measures of inhibitory control were calculated. Effect sizes were computed as $r$ (small $\geq .10$, medium $\geq .30$, large $\geq .50$) or $\eta^2_p$ (small $\geq .01$, medium $\geq .06$, large $\geq .14$). All statistical analyses were performed using SPSS 23.

3. Results

3.1. Preliminary analyses

Sample characteristics. There was a significant main effect of group on BMI, Welch’s $F(2, 52.94) = 102.430, p < .001, \eta^2_p = .588$, indicating that in line with expectation, the clinical samples including outpatients with and without BED had a significantly higher BMI than the HC group (both $p < .001$). The groups did not differ significantly with respect to age, $F(2, 98) = .225, p = .799$, or level of education, Fisher’s Exact Test $= 8.078, p = .147$. However, there was a significant association between sex and group, Fisher’s Exact Test $= 14.351, p < .001$. Separate $\chi^2$-tests revealed that there were significantly more men in the HC group compared to the Non-BED group ($p < .001$) but not compared to the BED group ($p = .070$). Moreover, as expected, the BED group reported higher levels of depression, $t(69) = 3.083, p = .003, r = .348$, disinhibited eating, $t(61.73) = 3.877, p < .001, r = .433$, and ED psychopathology than the Non-BED group, e.g. regarding objective binge eating episodes, $t(26.72) = 5.094, p < .001, r = .701$. The HC group scored below the cut-off of 5 on the PHQ-D depression scale, demonstrating an absence of increased depressiveness. Further group-related details are summarized in Table 2.

Baseline states and emotional valence of the stimuli characteristics. Analyses of treatment-independent variables indicated no group differences regarding baseline levels of tiredness, hunger and satiety, all $F_s \geq 2.612, \text{all } p_s \geq .078$. However, there was a significant interaction between baseline mood and group, $F_{(2, 98)} = 8.084, p < .001, \eta^2_p = .142$, with the HC group reporting significantly more positive mood at baseline than the two clinical groups (both $p \leq .005$). Moreover, analyses revealed a significant interaction between the emotional valences of the food- and body-related FST stimuli and group, $F_{(2, 98)} = 10.672, p < .001, \eta^2_p = .179$. The BED group estimated food- and body-related words to be more unpleasant than did the Non-BED group ($p \leq .015$) and to the HC group (both $p < .001$). With respect to body-related words, there was also a significant difference between the Non-BED group and the HC group ($p < .001$).

Self-reported inhibitory control. Using Pillai’s trace, there was a significant main effect of group on self-reported inhibitory control, $V = 0.437, F(8, 192) = 6.710, p < .001, \eta^2_p = .219$. Separate univariate ANOVAs revealed significant group effects on three UPPS subscales, $F_{(2, 98)} \geq 6.756, p \leq .002, .121 \geq \eta^2_p \geq .305$. Planned contrasts showed that compared to the HC group, both clinical groups had significantly higher scores on lack of perseverance, $t(98) = 3.586, p < .001, r = .341$ (Non-BED), $t(98) = 2.559, p = .012, r = .250$ (BED) irrespective of the diagnosis of BED, $t(98) = 0.547, p = .586$ (see Table 3). Moreover, both clinical groups reported a significantly greater lack of premeditation, $t(98) = 3.891, p < .001, r = .366$ (Non-BED), $t(98) = 2.896, p = .002, r = .281$ (BED), and more urgency, $t(98) = 5.243, p < .001, r = .468$ (Non-BED), $t(98) = 6.085, p < .001, r = .524$ (BED). Again, no differences were found between the BED and the Non-BED group, $t(98) = 0.463, p = .644$ (lack of premeditation), $t(98) = 1.759, p = .082$ (urgency). There were no significant group differences with respect to sensation seeking, $F_{(2, 98)} = 1.461, p = .237$.

3.2. Group differences in inhibition processes

Differences between inhibition processes. Using Pillai’s trace, there was a significant interaction effect between group and inhibition process, $V = .134, F(4, 196) = 3.514, p = .009, \eta^2_p = .068$. However, the findings did not provide sufficient evidence of any other main or interaction effect, all $V_s \leq .053, F_s \geq 1.339, p_s \geq .257$. Separate univariate ANOVAs did not reveal significant main effects of group, $F_{(2,}$

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Sociodemographic characteristics and psychopathology presented separately for healthy controls (HC) and outpatients with overweight and disinhibited eating (BED, Non-BED).</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HC (n = 30)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.96 (2.46)</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>36.30 (12.13)</td>
</tr>
<tr>
<td>Highest level of general education</td>
<td></td>
</tr>
<tr>
<td>- still student</td>
<td>0 (0)</td>
</tr>
<tr>
<td>- secondary education</td>
<td>3 (33.3)</td>
</tr>
<tr>
<td>- university entrance (13 yrs)</td>
<td>20 (66.7)</td>
</tr>
<tr>
<td>Sex, female (n)</td>
<td>20 (66.7)</td>
</tr>
<tr>
<td>Disinhibited eating (TFEQ)</td>
<td>-</td>
</tr>
<tr>
<td>ED pathology (EDE-Q Global)</td>
<td>-</td>
</tr>
<tr>
<td>Objective overeating (EDE)</td>
<td>-</td>
</tr>
<tr>
<td>Objective binge eating (EDE)</td>
<td>-</td>
</tr>
<tr>
<td>Depression (PHQ/BDI-II)</td>
<td>4.27 (2.80)</td>
</tr>
<tr>
<td>Current comorbidities (SCID)</td>
<td></td>
</tr>
<tr>
<td>- At least one comorbidity</td>
<td>-</td>
</tr>
<tr>
<td>- Any depressive disorder</td>
<td>-</td>
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<tr>
<td>- Any anxiety disorder</td>
<td>-</td>
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<tr>
<td>Disinhibitory psychopathology</td>
<td></td>
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<tr>
<td>- Borderline personality disorder</td>
<td>-</td>
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<tr>
<td>- ADHD</td>
<td>-</td>
</tr>
<tr>
<td>- Skin picking disorder</td>
<td>-</td>
</tr>
<tr>
<td>- Substance abuse</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. Means (SD) and numbers (%). BED/Non-BED = Individuals with overweight and/without binge eating disorder. TFEQ = Three-Factor Eating Questionnaire; EDE-Q = Eating Disorder Examination-Questionnaire; EDE = Eating Disorder Examination, episodes of overeating and binge eating are recorded for the last 28 days; PHQ = Patient Health Questionnaire; BDI-II = Beck Depression Inventory-II, total scores ≥14.29 indicate clinical severity (14–19 mild, 20–28 moderate, 19–63 severe); SCID = Structured Clinical Interview for DSM-IV, ADHD = attention deficit hyperactivity disorder. Test statistic = $\chi^2$-or Fisher’s Exact Test for categorical variables and $t$-or $F$-test for metric variables. Group differences are analyzed with Bonferroni post-hoc tests at *$p < .05$, **$p < .01$. |
98) = 0.929, \( p = .399 \), stimulus category, \( F_{(1,98)} = 0.002, p = .967 \), and inhibition process, \( F_{(1,196)} = 0.090, p = .914 \), but a significant interaction effect emerged between group and inhibition process, \( F_{(4,196)} = 3.559, p = .008, \eta^2_p = .068 \). This indicates that performance on the different measures of inhibitory control differed between the HC, Non-BED and BED groups irrespective of the stimulus category.

BMI was not significantly related to inhibition performances, \( F_{(1,97)} = 0.787, p = .379 \). The results were similar when BMI was taken into account: The main effect of group remained non-significant, \( F_{(2,97)} = 1.317, p = .273 \), and the group x inhibition process interaction remained significant, \( F_{(4,194)} = 2.781, p = .028, \eta^2_p = .054 \).

Viewing at the interaction graph (see Fig. 2 for overall inhibition performance) suggests an interaction when comparing interference inhibition performance (RT of FST) to response inhibition performance (SSRT of SST), as confirmed by further analyses, \( F_{(2,98)} = 7.240, p < .001, \eta^2_p = .129 \). Planned contrasts revealed that the BED group showed increased deficits regarding interference inhibition compared to the HC group, \( t_{(98)} = 2.055, p = .043, r = .203 \), after protecting against Type 1 error (cf. \( \alpha_{HB} = .017 \)), but did not differ from the Non-BED group, \( t_{(98)} = 0.616, p = .539 \). Surprisingly, the BED group performed better on response inhibition compared to the Non-BED group, \( t_{(98)} = 3.012, p = .003, r = .291 \). To further clarify these unexpected effects of response inhibition, cognitive restraint of eating was entered as a covariate. Cognitive restraint was not significantly related to inhibition performance, \( F_{(1,68)} = 1.065, p = .306 \). After controlling for the effect of cognitive restraint, the results were similar; however, the group x inhibition process x stimulus category reached a trend for significance.

### Table 3
Means (SD/SE) of measures of inhibitory control presented separately for healthy controls (HC) and outpatients with overweight and disinhibited eating (BED, Non-BED).

<table>
<thead>
<tr>
<th></th>
<th>HC (( n = 30 ))</th>
<th>Non-BED (( n = 47 ))</th>
<th>BED (( n = 24 ))</th>
<th>Group Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Self-reported inhibitory control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>UPPS</td>
<td></td>
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<tr>
<td>Lack of perseverance</td>
<td>2.26 (0.44)</td>
<td>2.69 (0.50)</td>
<td>2.61 (0.59)</td>
<td>HC &lt; **Non-BED, HC &lt; *BED, Non-BED = BED</td>
</tr>
<tr>
<td>Lack of premeditation</td>
<td>2.26 (0.48)</td>
<td>2.74 (0.53)</td>
<td>2.68 (0.56)</td>
<td>HC &lt; **Non-BED, HC &lt; **BED, Non-BED = BED</td>
</tr>
<tr>
<td>Sensation seeking</td>
<td>2.73 (0.57)</td>
<td>2.95 (0.53)</td>
<td>2.92 (0.65)</td>
<td>HC = Non-BED = BED</td>
</tr>
<tr>
<td>Urgency</td>
<td>2.39 (0.57)</td>
<td>3.01 (0.47)</td>
<td>3.23 (0.49)</td>
<td>HC &lt; **BED, HC &lt; *Non-BED, Non-BED = BED</td>
</tr>
<tr>
<td><strong>Performance-based inhibition processes</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Food Stroop task (FST)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RT (ms)</td>
<td>615.55 (18.57)</td>
<td>660.87 (14.30)</td>
<td>685.52 (21.32)</td>
<td>HC &lt; BED, HC = Non-BED, Non-BED = BED</td>
</tr>
<tr>
<td>Interference index (ms)</td>
<td>3.61 (23.55)</td>
<td>4.53 (23.27)</td>
<td>2.80 (22.44)</td>
<td>HC = Non-BED = BED</td>
</tr>
<tr>
<td>Door Opening task (DOT)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Opened doors (n)</td>
<td>61.40 (24.30)</td>
<td>62.43 (18.55)</td>
<td>60.81 (21.42)</td>
<td>HC = Non-BED = BED</td>
</tr>
<tr>
<td>Stop Signal task (SST)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SSRT (ms)</td>
<td>329.50 (79.46)</td>
<td>333.50 (65.75)</td>
<td>283.09 (43.68)</td>
<td>HC &gt; **BED, HC = Non-BED, Non-BED &gt; **BED</td>
</tr>
<tr>
<td>Go RT (ms)</td>
<td>642.63 (169.68)</td>
<td>565.83 (117.33)</td>
<td>558.08 (147.03)</td>
<td>HC &lt; **Non-BED, HC &lt; *BED, Non-BED = BED</td>
</tr>
</tbody>
</table>

**Note.** Means (SD/SE). BED/Non-BED = Individuals with overweight and/or binge eating disorder. UPPS = UPPS Impulsive Behavior Scale. Higher scores on all measures indicate more inhibitory control deficits (except for Go RTs). Group differences are analyzed with planned contrasts at \( p < .05, **p < .01 \) with Holm-Bonferroni corrections.

Fig. 2. Z-scores. Performance on interference inhibition (Food Stroop task, FST; RT), decision-making (Door Opening task, DOT; opened doors) and response inhibition (Stop Signal task, SST; SSRT), presented separately for individuals with overweight and disinhibited eating (BED, Non-BED) and healthy controls (HC). High performance represents good inhibitory control abilities which means less impulsive reactions. Error bars represent values for ± 1 SE.
Examination-Questionnaire, episodes of overeating and days with binge eating were assessed for the last 28 days. Results are reported in the case of significance, Note.

Table 4

<table>
<thead>
<tr>
<th align="left">Material (Fig. S1, Table S2).</th>
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</table>
| Table 3. Differences concerning interference inhibition. No significant differences in opened doors (DOT) were found between the three groups, F(2, 98) = 0.052, p = .949. Moreover, baseline states did not affect the three groups differently in the gambling period in pursuit of risky rewards, all F(23, 98) ≤ 5.219, all ps ≥ .049. Considering the door selection strategies recommended by Verbeek et al. (2009), an additional Chi-square analysis was performed comparing between conservative (1–41 doors, 10–39 points), optimal (42–74 doors, 40–45 points) and perseverative selections (75–110 doors, 39–10 points). Likewise, the groups did not differ with respect to these gambling strategies, χ²(4) = 7.341, p = .119. (ii) Differences concerning response inhibition. The main effects of group on SSRT and on Go RTs (SST) reached statistical significance, with F(2, 98) = 5.459, p = .006, n² = .100 and F(2, 98) = 4.285, p = .016, n² = .080. Planned contrasts revealed that the BED group showed better inhibition performance (shorter SSRT and longer Go RTs) compared to the HC group, F(2, 98) = 2.889, p = .005, r = -.280 and to HC, t(98) = 3.027, p = .003, r = .292. No differences in inhibition performance were found between the Non-BED and the HC group, t(98) = .445, p = .657. Moreover, the BED group was faster in responding correctly to the choice reaction time task (shorter RTs in Go trials) compared to the HC group, t(98) = 2.295, p = .024, r = .226, but not compared to the Non-BED group, t(98) = .958, p = .343. However, the comparison between the HC group and the Non-BED group narrowly missed the alpha level, at t(97) = 1.944, p = .055. (iii) Differences concerning response inhibition. The main effects of group on SSRT and on Go RTs (SST) reached statistical significance, with F(2, 98) = 5.459, p = .006, n² = .100 and F(2, 98) = 4.285, p = .016, n² = .080. 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3.3. Inhibition processes and their associations with disinhibited eating and BMI

Correlation analyses between measures of inhibitory control and measures of disinhibited eating behavior, ED psychopathology and BMI are listed in Table 4. Self-reported urgency was moderately associated with disinhibited eating, shape and weight concern and BMI. Moreover, there were significant correlations between BMI and lack of perseverance and premeditation. Contrary to expectation, food-specific SSRT was negatively correlated with eating concern, shape and weight concern and global ED psychopathology. All error rates did not correlate significantly with global eating pathology or BMI (.004 ≤ r ≤ −.171, all p ≥ .154), with the exception that BMI was significantly correlated with omission errors in Go trials for the food-specific SST, r = −.217, p = .029. Concerning convergent validity between self-report and performance-based measures of inhibitory control, only food-specific SSRT was negatively, albeit weakly, correlated with UPPS subscales, −.216 (urgency) ≤ r ≤ −.211 (lack of perseverance), all ps < .05. All inter-correlations across measures of impulsivity are represented in the Supplementary Material (Table S1).

4. Discussion

4.1. General discussion

The purpose of the current study was to illustrate the multifaceted nature of inhibitory control by focusing on different inhibition processes in outpatients with overweight and disinhibited eating behaviors in comparison to healthy controls. As assumed in our first hypothesis (i), outpatients with BED demonstrated impaired interference inhibition in the FST compared to healthy controls, requiring significantly more time to inhibit interference words. Contrary to our second hypothesis (ii), the study did not provide sufficient evidence of impulsive decision-making in the DOT in our clinical samples. Moreover, in contrast to our third assumption (iii), outpatients with BED performed better in the inhibition of already initiated responses in the SST compared to those without BED and healthy controls. Furthermore, both clinical groups were faster in Go trials of the SST, suggesting above-average abilities with regard to response selection, initiation and switching. The inclusion of baseline states as covariates did not alter the results. Against our expectations, no specific inhibition deficits for high-calorie cues emerged. Outpatients with BED showed more deficits in interference inhibition and by trend also in decision-making, while this pattern was reversed in healthy controls, and those without BED achieved comparable performance across all inhibitory processes. When the questionnaire results are incorporated, the picture contradicts the behavioral data: Both clinical groups reported increased deficits in resisting intrusive thoughts, in considering the consequences while making decisions, and in inhibiting rash action in response to extreme affects. Several aspects need to be discussed with respect to these heterogeneous results.

(i) We found a poorer performance on interference inhibition (prolonged RTs in the FST) in outpatients with BED compared to healthy controls but not compared to those without BED. This finding is in line with Lavagnino et al. (2016), who assumed impaired conflict monitoring processes in obese individuals regardless of binge eating patterns, while other researchers reported conflicting findings (Carters et al., 2015; Kittel et al., 2015; Nijs et al., 2010). One possible explanation for this discrepancy might lie in the comparison between different paradigms (e.g. Stroop, Attentional bias) for the same construct. Dohle, Diel, and Hofmann (2018) distinguished between updating/monitoring working memory and task-switching/shifting functions in interference inhibition, clarifying that it remains difficult to the classify of subtle distinctions in executive functioning into separate neurocognitive domains (Fitzpatrick et al., 2013). Hence, comparisons between different behavioral task measures or even between different variants of the same measures should be interpreted with caution. Interestingly, some researchers view performance-based findings in the FST from a different angle, claiming that a cognitive processing bias toward food-related stimuli may contribute to successful weight loss maintenance and rather depicts a functional coping strategy (Demos, McCaffery, Cournoyer, Wunsch, & Wing, 2013; Phelan et al., 2011). Accordingly, treatment-seeking samples in general should differ from samples who are not aiming to lose weight (like in Nijs et al., 2010), which was confirmed by the current findings.

(ii) Our findings did not provide sufficient evidence of impulsive decision-making (comparable number of opened doors in the DOT) in individuals with overweight, contradicting previous results (Giel et al., 2017; Nederkoorn, Braet et al., 2006; Scholten et al., 2014). Moreover, we found no significant relationship between the DOT and self-reported inhibitory control, thus calling into question the appropriateness of the DOT for assessing impulsive decision-making in adults. To our knowledge, four previous studies have examined the relationship between weight and the DOT, and only included children and adolescents aged 8–15 years (Guerrieri, Nederkoorn, & Jansen, 2008; Nederkoorn et al., 2006; Scholten et al., 2014; Verbeken et al., 2009). It is possible that adults are quicker to see through the rationale of decreasing probability of winning a point due to the monotonous task characteristics.

(iii) The most surprising finding is the absence of significant impairments in response inhibition (comparable SSRTs, more specifically shorter SSRTs in the BED group, in the SST) in outpatients with overweight and disinhibited eating compared to healthy individuals. This is in line with some previous studies (Hendrick et al., 2012; Mühlberg et al., 2016; Wu et al., 2013) but rather contradicts other individual findings (Manasse et al., 2016; Mole et al., 2015; Svaldi et al., 2014) and a recent meta-analytic finding of group differences (SMD = 0.30, p = .007; Lavagnino et al., 2016). From a clinical perspective, the lack of impairments is easy to explain: Most individuals with obesity have good inhibitory control abilities at times and do not show overeating or binge episodes in the presence of other persons (Fairburn & Wilson, 1993). While they have learned to exercise inhibitory control in social contexts, in specific contexts and with specific moods (e.g. watching television, loneliness), they are unable to inhibit eating impulses which are triggered directly by food-specific (e.g. leftover food in the kitchen) or generic stimuli (e.g. partnership conflicts). Interestingly, a better food-specific response performance tended to merge in the BED group if cognitive restraint of eating was taken into account, strengthening our interpretation that especially individuals with BED may perform better due to their ability to control food intake at times. However, contrary to this potential explanation, the treatment-seeking participants did not show similar performance to the healthy control group in the FST. Therefore, it might be argued that response inhibition might be particularly affected by confounding factors. First, Mühlberg et al. (2016) observed that higher BMI was predictive of longer SSRT, but only for individuals with overweight and low to normal self-reported impulsivity indicating a complex interplay between different inhibitory control components. Interestingly, urgency in response to extreme affect, theoretically linked to response inhibition (r = −.216 in the current study), was positively correlated with disinhibited eating, ED psychopathology and BMI (Manjrekar, Berenbaum, & Bhayani, 2015; Mobbs, Crepin, Thiery, Golay, & van der Linden, 2010; Racine et al., 2015; see also Table 4). These results further support the idea of an emotion-based inhibitory control pathway raising the question of how impulsivity interacts with emotional awareness and emotion regulation in obesity and BED (Kittel et al., 2015). Recent reviews
reported that increases in negative affect have been found to be antecedents of binge eating in BED, whereas the evidence for the relief component is very limited (Dingemans & van Furth, 2012; Leehr et al., 2015). Notably, Loeb et al. (2018) found that only those women with obesity and BED, which were restrained eaters and in a positive mood while testing, showed impairments in response inhibition while other studies stressed the importance of negative mood (Leehr et al., 2018; Nicholls, Devonport, & Blake, 2016). In the current study, mood was not induced experimentally; therefore, we cannot rule out that individuals with disinhibited eating and overweight merely remained inhibited in this response inhibition process, due to the lack of emotional triggers. Second, Nederkoorn, Braet, et al. (2006) and Nederkoorn, Smulders, et al. (2006) found higher SRRTs for women with obesity only in the final block of four blocks of 128 trials emphasizing a normal inhibition performance in the initial phase of a generic SST. Moreover, Mühlig et al. (2016) allowed participants a 30 sec break after every 200 trials, and failed to find sufficient evidence of group differences in SRRT between obese and lean participants. Therefore, it seems possible that impairments might have been masked in the current SST in light of the small number of trials (64 for each block), including only 32 Stop trials in total. These two explanations are strengthened by the conclusions of the review by Bartholy et al. (2016), which suggested that impaired inhibitory control as measured by the SST only emerges only if executive resources are depleted and food-specific stimuli are involved.

However, the latter expectation could not be confirmed either for the SST or for the FST and DOT, supporting the idea of a general impairment of inhibition performance irrespective of stimulus category in individuals with overweight and disinhibited eating. This rather contradictory result might be attributed to the composition of the sample, which compromised a chronic subgroup of outpatients with unsuccessful weight loss maintenance, who probably have impulsivity problems in several spheres of life.

4.2. Strengths and limitations

A strength of the current study was that it focused on the homogeneous subgroup of outpatients with overweight and disinhibited eating, and excluded subgroups with other key issues in the obesity spectrum (e.g. social eating, heavy drinking, Caroleo et al., 2018; Jimenez, Green, Subramanian, & Razak, 2018). The application of structured clinical interviews ensured a valid diagnosis. Moreover, we used different methods to measure inhibitory control, using a more holistic interpretation of an inhibitory control pathway. However, several limitations need to be considered when interpreting the current findings. First, inhibitory control is a broad construct that encompasses, but is not limited to, the inhibition processes assessed. The inclusion of reward-related processes in the inhibition construct can be seen as questionable (see e.g. Dawe et al., 2004; Dawe & Loxton, 2004). Moreover, the fixed order of the generic and food-specific blocks in the FST and DOT, which did not exclude transfer effects (e.g. training effect), should be viewed critically. As only stimuli characteristics in the FST were controlled for emotional valence, we cannot rule out that the results in the SST and DOT may have been affected by the hedonic value of the presented food-related stimuli. Given that not every individual with disinhibited eating has strong implicit approach associations with the same food, stimuli should have been individualized regarding palatability ratings (Jones et al., 2018). At the very least, the individual preference for the food stimuli might be examined in order to increase task relevance and ecological validity. Moreover, impulsive eating patterns might also be present to a lesser degree in individuals with overweight of the healthy control group with poor self-awareness. Unfortunately, due to our study design, we were only able to control for cognitive restraint of eating in the two clinical groups. Finally, due to our unbalanced allocation of men and women, we cannot rule out gender-specific differences, as found in previous studies (Gerlach, Loeber, & Herpertz, 2016; Koritzky, Yechiam, Bukay, & Milman, 2012).

4.3. Conclusions and future directions

Taken together, the current findings suggest that first-stage impairments in overcoming interference from distracting information might be attributable to the initiation of eating episodes, which are performed and reported by outpatients with overweight and obesity regardless of binge eating patterns (see also Manasse et al., 2016). By contrast, impairments in the late-stage response inhibition might result in a loss of control over an eating episode especially in outpatients with BED if executive resources are depleted (e.g. negative mood, overexertion, starvation or restricted eating). Under executive control, those exhibiting binge eating might show better performance in terms of inhibiting an already initiated response, given their daily practice of inhibitory control in social situations. These initial findings can be used to develop inhibitory control interventions aimed at enhancing weight loss maintenance. However, further investigations employing mood, overexertion or starvation induction are needed to examine these preliminary data. Moreover, future studies need to consider crucial methodological aspects such as a greater number of trials, individualized food-specific stimuli, and the examination of different successive, but interfering inhibition processes in one sample. The consideration of such an inhibitory control pathway, which depicts the complex interplay between impulsivity, inhibitory control and disinhibited eating, might deepen the understanding and constitute a further step in obesity research.

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Appendix A. Supplementary data

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References


