

A Behavioral and Event-Related Potentials Study of Food-Related Inhibitory Control in Probable Binge Eating Disorder

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Background: Similar to addictive disorders, deficits on cognitive control might be involved in the onset and development of Binge Eating Disorder (BED). However, it remains unclear whether general or food-related inhibitory control impairments would be basically linked to overeating and binge eating behaviors. This study thus aimed to investigate behavioral performance and electro-physiological correlates of food-related inhibitory control among individuals with binge eating behavior.

Methods: Sixty individuals with probable BED (pBED) and 60 well-matched healthy controls (HCs) were assessed using the typical Stop-Signal Task, a revised Go/No Go Task, and a food-related Go/No Go Task. Besides, another separate sample, including 35 individuals with pBED and 35 HCs, completed the food-related Go/No Go Task when EEG signals were recorded with the event-related potentials (ERPs).

Results: The data revealed that the pBED group performed worse with a longer SSRT on the Stop-Signal Task compared with HCs (Cohen's $d = 0.58$, $p = 0.002$). Moreover, on the food-related Go/No Go Task, the pBED group had a lower success rate of inhibition in no-go trials (Cohen's $d = 0.47$, $p = 0.012$). The ERPs data showed that in comparison with HCs, the pBED group exhibited increased P300 latency (FC1, FC2, F3, F4, FZ) in the no-go trials of the food-related Go/No Go Task (Cohen's $d = 0.56-0.73$, all $p < 0.05$).

Conclusion: These findings suggested that individuals with binge eating could be impaired in both non-specific and food-related inhibitory control aspects, and the impairments in food-related inhibitory control might be linked to P300 abnormalities, implying a behavioral-neurobiological dysfunction mechanism implicated in BED.

Keywords: binge eating disorder, inhibitory control, stop-signal, Go-No Go, ERPs

Introduction

Binge eating disorder (BED) has been recognized as a fully diagnostic category since the introduction of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). As a public health issue, BED is one of the most prevalent eating disorders, with a lifetime prevalence rate of 2.8% for women and 1.0% for men.^{1,2} The 12-month prevalence estimates of BED are 1.4% for women and 0.6% for men.^{1,3} Notably, youths and adolescents are at a high risk for BED, possibly due to their immature cognitive control abilities,^{4,5} with past-year prevalence estimates of 1.8–3.6% in girls, 0.2–1.2% in boys, and 1.5% in gender-diverse youths.^{6,7} BED is associated with significant psychiatric and physical complications, as well as an increased risk of obesity. It can also lead to impairments in quality of life, a substantial burden of disease, and excess mortality.^{8,9} Neurobiological dysfunctions in reward processing, emotion regulation, and inhibitory control are highlighted in individuals with BED, and these domains have been considered as potential targets for emerging treatment approaches.^{10–12} However, the pathogenesis of BED remains unclear, and ongoing efforts are necessary to gain a better understanding of its underlying causes and to improve prevention and treatment outcomes.¹³

Similar to addictive disorders, diminished control over behavior is a hallmark feature of BED, with the experience of losing control over eating behavior being an integral part of its core psychopathology.¹⁴ Impaired self-control, including

impulsive and compulsive transdiagnostic features, may thus represent a key alteration characterizing BED.¹⁵ As an operational paradigm of self-control, inhibitory control or cognitive control reflects the ability of top-down control and recruits prefrontal networks to regulate behavior,^{16–18} and it has been widely studied in substance use disorders, behavioral addictions,^{19–21} as well as in BED.^{15,22–24}

Based on cognitive tasks of inhibitory control such as the Stop-Signal Task and Go/No-Go Task, previous studies have provided substantial evidence linking poor inhibitory control to BED.^{22,25–28} However, recent systematic reviews have argued that the current literature on general inhibitory control in BED has yielded mixed findings and equivocal data (or a lack of available evidence), which seemingly does not support the notion that individuals with BED definitively and consistently exhibit impairments in inhibitory control compared to normal-weight healthy control groups.^{15,24,29,30} Heterogeneity in sample characteristics, task methodologies, and comorbidities may be important confounding factors that contribute to the limited power of conclusions.^{13,15,24} Nonetheless, inhibitory control abnormalities associated with BED continue to be a matter of concern, given that failed inhibitory control is indicated as a potential contributor to unhealthy food consumption and an early marker of disordered eating behaviors in adolescents,^{31,32} and inhibitory control may serve as a target for reducing overeating.¹⁰

Compared to general inhibitory control dysfunctions, impairments in food-related inhibitory control appear to be more likely involved in BED.^{24,29} Indeed, it has been suggested that a food-specific focus on inhibitory control and impulsivity might allude to processes closer to the core pathology of BED.³³ One prior study compared behavioral performance on the Stop-Signal Task with both neutral and food stimuli between individuals with BED and overweight/obese individuals without BED. The findings indicated that individuals with BED exhibited greater difficulty in inhibiting responses triggered by food trials.³⁴ Furthermore, food-related rash-spontaneous behaviors towards food were found to be increased in individuals with BED, as assessed by adapted inhibitory control tasks involving food pictures.^{35–37} In addition, incorporating food-specific inhibition training protocols has been suggested as a beneficial component in the treatment of BED.^{38,39} However, controversial evidence claimed that the inhibitory deficits associated with binge eating were not specific to food.⁴⁰ By comparing overweight/obese subjects with and without BED on a Stop-Signal Task that contained food-specific stimuli, positive non-food stimuli, and neutral stimuli, this study exhibited poorer inhibitory control across different stimuli types in the BED group, but the deficits appeared not to be specific to food stimuli.⁴⁰ Therefore, further replication research is still needed to clarify the inhibitory control related to food in BED.

In the current study, we aimed to further investigate the cognitive characteristics of general and food-related inhibitory control in a non-clinical sample of Chinese young adults at risk for BED, considering that the majority of studies in the literature have focused on clinical samples of BED and were primarily conducted in Western cultures.¹³ A large sample of 120 young adults, consisting of individuals with probable BED (pBED) and well-matched healthy controls, was assessed using the typical Stop-Signal Task, a revised Go/No Go Task, and a food-related Go/No Go Task. Furthermore, the electrophysiological correlates of food-related inhibitory control have been of great interest in the search for possible biological markers in BED.^{41–43} Therefore, we included an additional separate sample of 35 individuals with pBED and 35 healthy controls, who completed the food-related Go/No Go Task when electroencephalogram (EEG) signals were recorded with the event-related potentials (ERPs). It was hypothesized that the individuals with pBED would prominently exhibit impairments in food-related inhibitory control, which might be closely linked to abnormalities in certain neurobiological indicators (eg, N200 and P300).

Materials and Methods

Participants

The participants were recruited from a local university in Guiyang, China between September and November 2021. All of them were young adult students who attended three-year public psychology courses. Firstly, the students were invited to complete a short self-report screening questionnaire during a 45-min psychology class. This questionnaire consisted of demographic information (ie, age, gender, years of education, ethnicity, home locality, smoking and drinking behaviors) and a Chinese version of the Binge Eating Scale (BES),^{44,45} which has been properly used among Chinese college

students in previous studies.^{46,47} A total of 862 students voluntarily responded to the screening questionnaire, 63 of whom were identified as individuals with pBED, with a total score ≥ 18 on the BES.^{45,48} All 63 students voluntarily participated in our study and were subsequently scheduled to complete a person-to-person interview in the laboratory. This interview included a brief checklist regarding physical diseases/conditions, current or past brain trauma, and history of psychoactive substance use/abuse, major psychiatric disorders, neurological diseases, and other mental disorders (see [Supplementary Tables S1](#) for details). Afterwards, these students completed several cognitive tasks. Inclusion criteria included: (1) 18–25 years of age, and (2) willingness to participate in this study. Exclusion criteria included: (1) current or past severe psychiatric disorders (eg, schizophrenia, psychosis, bipolar disorder, major depressive disorder), neurological diseases, or other mental disorders; (2) a history of psychoactive substance use/abuse (eg, cocaine, heroin, ketamine, amphetamine); (3) current or past brain trauma; and (4) severe physical diseases or conditions that were inappropriate to complete the tasks, all of which were evaluated by self-reports. In total, three students were excluded based on one or more of these exclusion criteria. Finally, 60 individuals with pBED (aged 19.13 ± 0.89 years, from 18 to 23 years; 46 females, 76.7%) were included.

Sixty healthy control students (HCs) were recruited through advertisements and posters in the same university classes, matched with the pBED group on body mass index (BMI), age, gender, and educational level. The HCs were voluntarily enrolled in this study. They completed the self-report screening questionnaire, underwent the person-to-person interview, and performed the cognitive tasks in the laboratory. Inclusion criteria included: (1) 18–25 years of age, (2) willingness to participate in this study, and (3) a total score of 17 or less on the BES. Exclusion criteria included: (1) current or past severe psychiatric disorders, neurological diseases, or mental disorders; (2) current or past psychoactive substance abuse; (3) a history of brain trauma; and (4) severe physical diseases. The HCs were aged 19.37 ± 1.18 years (18–23 years; 47 females, 78.3%).

In addition to the 60 individuals with pBED and 60 HCs who were only tested on cognitive tasks (ie, the Stop-Signal Task, Go/No Go Task, and food-related Go/No Go Task), another separate sample was recruited at the same university using a similar procedure, with the same inclusion and exclusion criteria. This sample included 35 students with pBED and 35 HCs, who were matched on BMI, age, gender, and educational level. They completed a food-related Go/No Go Task when EEG signals were recorded in the ERPs study. The two samples were comparable on demographics and task scores ([Tables 1–3](#)). All subjects were compensated with RMB ¥ 50.

Measures

Demographics

A brief self-report questionnaire was used to collect demographic data (eg, age, gender, years of education, ethnicity, and home locality). Standard procedures were used to measure weight and height, and then BMI was calculated by weight divided by the square of height (ie, kg/m^2). Smoking and drinking behaviors were assessed using one single question

Table 1 Demographic Characteristics of the Groups in the Behavioral Study

Variables	pBED Group (n=60)	HCs (n=60)	χ^2/t	p
Age, years (M \pm SD)	19.13 \pm 0.89	19.37 \pm 1.18	-1.223	0.224
Gender, Female n (%)	46 (76.7)	47 (78.3)	0.048	0.827
Years of education (M \pm SD)	13.08 \pm 0.28	13.18 \pm 0.47	-1.420	0.158
BMI, kg/m^2 (M \pm SD)	21.95 \pm 3.66	21.04 \pm 1.69	1.760	0.082
Ethnicity, Hans n (%)	45 (75.0)	46 (76.7)	0.045	0.831
Home locality, Urban n (%)	45 (75.0)	44 (73.3)	0.043	0.835
Smoking status, yes n (%)	2 (3.3)	0 (0.0)	2.034	0.154
Drinking status, yes n (%)	3 (5.0)	2 (3.3)	0.209	0.648
BES scores (M \pm SD)	21.50 \pm 3.28	5.57 \pm 2.21	31.191***	<0.001

Note: *** $p < 0.001$.

Abbreviations: pBED, probable Binge Eating Disorder; HCs, Healthy Controls; BES, Binge Eating Scale.

Table 2 Inhibitory Control Performance of the Groups on the Cognitive Tasks (M±SD)

Variables	pBED Group (n=60)	HCS (n=60)	t	p
Stop-Signal Task				
Correct accuracy in Go-trials (%)	95.22±4.26	94.63±6.73	0.577	0.565
Correct accuracy in Stop-trials (%)	52.89±4.71	53.09±4.67	−0.244	0.808
Response time in Go-trials (ms)	619.94±184.50	631.45±187.37	−0.399	0.735
Stop-Signal Reaction Time (ms)	269.83±44.60	247.89±29.81	3.168**	0.002
Go/No Go Task				
Correct accuracy in frequent-go trials (%)	94.26±6.12	94.50±4.41	−0.256	0.799
Correct accuracy in rare-go trials (%)	85.11±12.28	84.78±9.81	0.161	0.873
Correct accuracy in no-go trials (%)	65.21±13.60	64.45±12.86	0.314	0.754
Response time in frequent-go trials (ms)	240.15±33.68	241.38±28.98	−0.214	0.831
Response time in rare-go trials (ms)	243.89±33.95	243.36±34.02	0.085	0.932
Food-related Go/No Go Task				
Correct accuracy in frequent-go trials (%)	95.09±5.32	95.19±5.26	−0.103	0.918
Correct accuracy in rare-go trials (%)	84.56±11.23	85.56±11.49	−0.485	0.629
Correct accuracy in no-go trials (%)	58.88±15.43	65.80±14.22	−2.555*	0.012
Response time in frequent-go trials (ms)	244.43±37.45	239.17±32.11	0.825	0.411
Response time in rare-go trials (ms)	249.47±41.42	244.48±36.83	0.696	0.488

Notes: *p<0.05, **p<0.01.

Abbreviations: pBED, probable Binge Eating Disorder; HCs, Healthy Controls.

Table 3 Demographics and Task Scores of the Sample in the Event-Related Potentials (ERPs) Study

Variables	pBED Group (n=35)	HCS (n=35)	χ^2/t	p
Age, years (M±SD)	19.31±0.90	19.57±1.04	−1.108	0.272
Gender, Female n (%)	26 (74.3)	25 (71.4)	0.072	0.788
Years of education (M±SD)	13.14±0.36	13.26±0.51	−1.095	0.278
BMI, kg/m ² (M±SD)	21.66±3.54	20.53±1.37	1.763	0.085
Ethnicity, Hans n (%)	23 (65.7)	24 (68.6)	0.065	0.799
Home locality, Urban n (%)	25 (71.4)	23 (65.7)	0.265	0.607
Smoking status, yes n (%)	1 (2.9)	0 (0.0)	1.014	0.314
Drinking status, yes n (%)	2 (5.7)	1 (2.9)	0.348	0.555
BES scores (M±SD)	20.97±2.93	5.26±1.88	26.718***	<0.001
Food-related Go/No Go Task (M±SD)				
Correct accuracy in frequent-go trials (%)	94.91±5.95	95.18±5.68	−0.197	0.844
Correct accuracy in rare-go trials (%)	85.27±10.70	85.13±11.81	0.052	0.959
Correct accuracy in no-go trials (%)	58.98±14.53	67.52±13.14	−2.581*	0.012
Response time in frequent-go trials (ms)	244.55±35.90	244.82±30.51	−0.033	0.974
Response time in rare-go trials (ms)	246.40±39.14	251.80±34.61	−0.612	0.542

Notes: *p<0.05, ***p<0.001.

Abbreviations: pBED, probable Binge Eating Disorder; HCs, Healthy Controls; BES, Binge Eating Scale.

each (ie, “Have you smoked at least one cigarette in the past 14 days” and “Did you take at least one drink in the past 14 days”, with 0 = No and 1 = Yes, respectively).

Binge Eating

The BES^{44,45} was designed to screen binge eating behavior. It is a 16-item self-report scale assessing behavioral, emotional, and cognitive symptoms of binge eating. Each item consists of four or three statements reflecting a range of severity (0 = no problem, 3 = severe problem), with a possible total score ranging from 0 to 46. A Chinese version of the BES^{46,47} was adopted. Higher total scores of the BES indicate more severe binge eating problems, with a total score ≥ 18 indicating pBED.^{45,48} The *Cronbach's* α was 0.860 in the present study.

Stop-Signal Task (SST)

The SST⁴⁹ was a typical measure of inhibitory control, which requires individuals to make rapid responses on Go-trials (ie, left button-press for a left-pointing arrow, right button-press for a right-pointing arrow), but to inhibit their responding on Stop-trials when an auditory stop signal (a 300-Hz tone) was presented. There were 5 blocks of 64 trials each. In the Stop-trials (25%), stop signals occurred after the Go stimulus with a variable delay (stop-signal delay, SSD). The SSD was initially 200 ms, and adjusted using a staircase procedure to identify a point at which the participants inhibited successfully on about 50% of the Stop-trials, thus allowing the estimation of the Stop-Signal Reaction Time (SSRT). After a successfully inhibited Stop-trial, the SSD increased by 50 ms (making the next Stop-trial more difficult), whereas the SSD decreased by 50 ms if the subject failed to stop (making the next Stop-trial easier). The SSRT estimates the time required for one subject to withhold responses, with a longer SSRT indicating poorer inhibitory control.

Go/No Go Task

A previously modified and validated Go/No Go Task^{50,51} was employed to investigate response inhibition. This task was essentially designed to separate cognitive processing of infrequent stimuli (stimulus-driven attention) from inhibitory processes by including three different types of colored circles: (a) frequent-go trials (frequent gray, $n = 388$, about 75%), (b) infrequent-go trials (rare yellow, $n = 65$, about 12.5%), and (c) no-go trials (rare blue, $n = 65$, about 12.5%). Contrast of infrequent-go trials versus no-go trials thus was expected to detect response inhibition (excluding stimulus-driven attention). In this task, a colored circle was presented on the black screen for 400 ms, with a 400-ms inter-stimulus interval, lasting for about 7 minutes. Participants were told to press a button as soon as possible in response to gray and yellow circles, but to refrain from responding to blue circles. The frequent-go, infrequent-go, and no-go trials were intermixed in a pseudo-random order. Prior to the formal experiments, participants practiced 30 filler trials (10 gray, 10 yellow, and 10 blue circles). This task was programmed using the E-prime Version 2.0. Response accuracy in three types of trials and reaction time (RT) in frequent-go and infrequent-go trials were recorded and analyzed.

Food-Related Go/No Go Task

A food version of the original Go/No Go Task^{50,51} was also adopted in this study. It was an analogue of the original task, but consisted of three different types of object squares/pictures instead of colored circles: (a) frequent-go trials (natural flowers, $n = 388$, about 75%), (b) infrequent-go trials (daily-use articles, such as teacups, $n = 65$, about 12.5%), and (c) no-go trials (delicious high-calorie foods, such as pizza, $n = 65$, about 12.5%). In this task, the object square was similarly presented on the black screen for 400 ms, with a 400-ms inter-stimulus interval. Participants have to quickly press a button in response to flowers and daily-use articles, but to withhold a response to the foods. These three types of trials were also intermixed in a pseudo-random order. This task was programmed using the E-prime Version 2.0. Response accuracy in all trials and RT in frequent-go and infrequent-go trials were analyzed.

EEG Recording and Preprocessing

ERPs Experimental Design

In the food-related Go/No Go Task, an object square/picture (ie, either natural flowers, daily-use articles, or high-calorie foods) was randomly presented on the central visual area of a black computer screen for 400 ms, followed by an inter-trial interval of 400 ms. Participants were instructed to press a button in response to flowers (left key) and daily-use articles (right key) as rapidly as possible, but to withhold a response to foods (no responses). This task consisted of 388 frequent-go trials (natural flowers, 75%), 65 infrequent-go trials (daily-use articles, 12.5%) and 65 no-go trials (high-calorie foods, 12.5%), lasting for about 7 minutes. All stimulus pictures were collected from the image database on the Internet and edited to be homogeneous with respect to color, brightness, contrast, viewing distance, and visual complexity. The task was programmed by the E-prime 2.0. Accuracy and RT were recorded and analyzed.

EEG Recording

EEG signals were recorded, using the EEG system (Brain Amp MR plus, Gilching, Germany), from a 32 electrode cap (Easy-Cap, Herrsching-Breitbrunn, Germany) positioned according to electrode montage of the international 10/20 system. All the leads were referenced to the linked earlobes. A ground electrode was placed on the forehead. The

vertical electro-oculogram (VEOG) was recorded using an electrode placed approximately 1 cm below the left eye. The horizontal electro-oculogram (HEOG) was recorded using an electro-oculogram signal from one electrode placed approximately 1 cm from outer canthi of the left eye. The impedance was kept below 5 k Ω during EEG recording. Signals were recorded using a sampling rate of 500 Hz, with a band-pass from 0.1 to 70 Hz.

EEG Data Preprocessing

The EEG data were processed offline using the Brain Vision Analyzer software (Brain Products, Munich, Germany). Firstly, the VEOG and HEOG were deleted, and the data were then re-referenced to TP9 and TP10 (bilateral mastoid electrodes). The remaining EEG data were filtered with a band-pass of 0.01–40 Hz and were divided into epochs of 700 ms duration (–800 to 2000 ms). Baseline corrections were made based on the time interval of –800 to 0 ms. Bad-segment interpolation and rejection were conducted for each channel and each subject. The eye artefacts, head movement, muscle artefacts, frequency interference, and electrocardiographic activity were then removed from the data using an independent component analysis (ICA). All channels were subjected to additional correction, and trials with drifts larger than ± 100 μ V in any scalp electrode were rejected. The data were averaged for each trial condition (ie, frequent-go trials, infrequent-go trials, and no-go trials). Two subjects (one from pBED group, another from HCs) were excluded from analysis due to the number of usable no-go trials less than 32 (ie, 50% of no-go trials), resulting in a final sample of 68 subjects ($n = 34$ in each group). The peaks of N200 (150–300 ms) and P300 (250–450 ms) were automatically marked using the “peak finder” function of the Brain Analyzer. Since N200 and P300 were both located mainly at the fronto-central electrode sites, amplitude and latency of them were analyzed from these electrodes (ie, FC1, FC2, F3, F4, CZ, PZ, FZ, FCz), as suggested before.⁵²

Statistical Analyses

Data analysis was conducted with the Statistical Package for the Social Sciences for Windows, Version 22.0 (SPSS Inc., Chicago, IL, USA). Chi-square tests were performed to test between-group differences on categorical variables (ie, gender, ethnicity, home locality, smoking and drinking status). T-tests were used to analyze group differences on age, years of education, BMI, and BES scores. Multivariate analysis of variance (mANOVA) models were used to compare task scores between the two groups. If mANOVA models indicated significant effects, additional pairwise comparisons on the task scores were performed using *t*-tests. Differences on the amplitude and latency of N200 and P300 were tested by a 2 (group: pBED, HCs) \times 3 (trial type: frequent-go trials, infrequent-go trials, no-go trials) \times 8 (electrode: FC1, FC2, F3, F4, CZ, PZ, FZ, FCz) design, with trial type and electrode as the within-subject factors and group as the between-subject factor. Simple contrasts were then conducted using *t*-tests on each electrode if group effects were significant. Statistical significance was set as $p < 0.05$, two-tailed.

Results

Demographic Characteristics

As seen in Table 1, no between-group differences were found on BMI, age, years of education, gender, ethnicity, home locality, smoking and drinking status (all $p > 0.05$).

Inhibitory Control Task Performance

On the Stop-Signal Task, the mANOVA models revealed significant group differences on the SSRT ($F_{(1, 117)} = 11.296$, $p = 0.001$, $\eta_p^2 = 0.088$), but not on the correct accuracy in Go-trials ($F_{(1, 117)} = 0.111$, $p = 0.740$), correct accuracy in Stop-trials ($F_{(1, 117)} = 0.022$, $p = 0.883$), or RT in Go-trials ($F_{(1, 117)} = 0.085$, $p = 0.772$). Pairwise comparisons (Table 2) indicated that the pBED group performed worse with a longer SSRT than HCs ($t = 3.168$, $p = 0.002$, Cohen's $d = 0.58$).

On the Go/No Go Task, the mANOVA models revealed no significant group differences on the correct accuracy in frequent-go trials ($F_{(1, 117)} = 0.037$, $p = 0.847$), correct accuracy in rare-go trials ($F_{(1, 117)} = 0.627$, $p = 0.430$), correct accuracy in no-go trials ($F_{(1, 117)} = 0.187$, $p = 0.666$), RT in frequent-go trials ($F_{(1, 117)} = 0.156$, $p = 0.693$), or RT in rare-go trials ($F_{(1, 117)} = 0.013$, $p = 0.910$).

On the food-related Go/No Go Task, the mANOVA models revealed significant group differences on the correct accuracy in no-go trials ($F_{(1, 117)} = 6.947, p = 0.010, \eta_p^2 = 0.056$), but not on the correct accuracy in frequent-go trials ($F_{(1, 117)} = 0.120, p = 0.730$), correct accuracy in rare-go trials ($F_{(1, 117)} = 0.071, p = 0.790$), RT in frequent-go trials ($F_{(1, 117)} = 0.283, p = 0.595$), or RT in rare-go trials ($F_{(1, 117)} = 0.234, p = 0.629$). Pairwise comparisons (Table 2) indicated that pBED group had a lower correct accuracy in no-go trials than HCs ($t = -2.555, p = 0.012$, Cohen's $d = 0.47$).

Event-Related Potentials (ERPs) Study Outcomes

As seen in Table 3, there were no between-group differences on BMI, age, gender, years of education, ethnicity, home locality, smoking and drinking status (all $p > 0.05$).

Behavioral Data

The mANOVA models revealed significant group differences on the correct accuracy in no-go trials ($F_{(1, 67)} = 5.895, p = 0.018, \eta_p^2 = 0.081$), but not on the correct accuracy in frequent-go trials ($F_{(1, 67)} = 0.106, p = 0.745$), correct accuracy in rare-go trials ($F_{(1, 67)} = 0.002, p = 0.961$), RT in frequent-go trials ($F_{(1, 67)} = 0.096, p = 0.758$), or RT in rare-go trials ($F_{(1, 67)} = 0.768, p = 0.384$). Pairwise comparisons (Table 3) showed that the pBED group had a lower correct accuracy in no-go trials than HCs ($t = -2.581, p = 0.012$, Cohen's $d = 0.62$).

EEG Data

Regarding N200, the 2 (group: pBED, HCs) \times 3 (trial type: frequent-go trials, infrequent-go trials, no-go trials) \times 8 (electrode: FC1, FC2, F3, F4, CZ, PZ, FZ, FCz) between-within models revealed no significant main effects of the group, trial, and electrode on N200 amplitude or N200 latency (all $p > 0.05$). The interaction effects of trial \times group, electrode \times group, trial \times electrode, and group \times trial \times electrode were also not significant (all $p > 0.05$). See more details of N200 amplitude and latency in [Supplementary Tables S2](#) and [S3](#), respectively. Regarding P300, the 2 \times 3 \times 8 between-within models revealed no significant main effects of group, trial, and electrode on P300 amplitude (all $p > 0.05$), without significant interaction effects of trial \times group, electrode \times group, trial \times electrode, and group \times trial \times electrode (all $p > 0.05$). See more details of P300 amplitude in [Supplementary Tables S4](#). Instead, the 2 \times 3 \times 8 between-within models revealed significant main effects of group on P300 latency ($F_{(1, 65)} = 15.048, p < 0.001, \eta_p^2 = 0.188$), although without main effects of trial and electrode or interaction effects of trial \times group, electrode \times group, trial \times electrode, and group \times trial \times electrode (all $p > 0.05$). Simple contrasts at each electrode point (Table 4) revealed significant increases in P300 latency (FC1, FC2, F3, F4, FZ) during the no-go trials for the pBED group compared to HCs (Cohen's $d = 0.56$ – $0.73, ps < 0.05$). This suggests that impairments in food-related inhibitory control may be closely linked to increased P300 latency. We further tested the correlations between correct accuracy in no-go trials (ie, food-related inhibitory control) and P300 latency (FC1, FC2, F3, F4, FZ) using Pearson correlation. Data showed that there were significant negative

Table 4 Comparisons of P300 Latency (Ms) on the Food-Related Go/No Go Task ($M \pm SD$)

Electrode Sites	pBED Group (n=34)	HCs (n=34)	t	p
Frequent-go trials				
FC1	367.82 \pm 33.47	350.77 \pm 37.33	1.984	0.051
FC2	371.03 \pm 42.48	349.85 \pm 40.18	2.112*	0.038
F3	366.32 \pm 42.98	351.06 \pm 37.22	1.565	0.122
F4	371.06 \pm 35.60	352.68 \pm 41.77	1.930	0.058
CZ	369.27 \pm 42.75	346.38 \pm 47.37	2.091*	0.040
PZ	334.15 \pm 43.93	318.18 \pm 45.25	1.477	0.145
FZ	366.94 \pm 42.63	350.85 \pm 39.15	1.621	0.110
FCz	370.62 \pm 34.41	354.71 \pm 48.45	1.561	0.123

(Continued)

Table 4 (Continued).

Electrode Sites	pBED Group (n=34)	HCs (n=34)	t	p
Rare-go trials				
FC1	393.94±33.62	376.03±41.84	1.946	0.056
FC2	388.97±47.95	371.74±39.39	1.620	0.110
F3	387.91±43.39	376.47±44.40	1.075	0.286
F4	391.97±41.95	376.85±42.78	1.471	0.146
CZ	391.74±39.23	377.82±41.81	1.415	0.162
PZ	380.97±48.68	367.00±60.20	1.052	0.297
FZ	389.32±44.05	378.44±39.32	1.075	0.286
FCz	382.82±43.29	357.09±39.33	2.566*	0.013
No-go trials				
FC1	379.06±39.19	350.79±45.49	2.745**	0.008
FC2	389.53±38.38	368.15±34.56	2.414*	0.019
F3	388.77±40.23	360.24±38.47	2.989**	0.004
F4	388.06±37.08	368.24±33.70	2.307*	0.024
CZ	385.53±41.09	370.09±46.27	1.455	0.150
PZ	386.77±40.96	376.71±44.21	0.973	0.334
FZ	390.94±33.21	366.21±34.53	3.011**	0.004
FCz	388.09±36.86	371.97±44.57	1.625	0.109

Notes: * $p < 0.05$, ** $p < 0.01$.

Abbreviations: pBED, probable Binge Eating Disorder; HCs, Healthy Controls.

correlations between P300 latency (FC1, FC2, F3, F4, FZ) and correct accuracy in no-go trials ($r_{(68)} = -0.257$ to -0.309 , $ps < 0.05$).

Discussion

In this study, we investigated aspects of general and food-related inhibitory control in a non-clinical sample of young adult students with pBED, and linked food-related inhibitory control to potential electrophysiological correlates. Our data revealed that the individuals with pBED were compromised in both non-specific and food-related inhibitory control aspects, with a longer SSRT on the Stop-Signal Task and a lower successful inhibition rate on a food-related Go/No Go Task, compared with well-matched healthy controls. Additionally, impaired food-related inhibitory control in the pBED individuals was associated with biological abnormalities (increased P300 latency) in fronto-central sites (FC1, FC2, F3, F4, FZ). These findings implied a behavioral-neurobiological dysfunction mechanism of inhibitory control implicated in BED.

Inhibitory control is a crucial neurocognitive construct, underlying both impulsivity and compulsivity mechanisms in various psychiatric disorders such as addictive behaviors^{53,54} and eating disorders.^{55,56} With reference to BED, a loss of control over eating behavior has been included in the core psychopathology,¹³ and accordingly, deficient inhibitory control is believed to stand at the core of the pathogenesis of BED.¹⁵ However, despite a large number of related studies, the literature on inhibitory control in BED remains inconclusive. A meta-analysis (up to December 2019) identified global cognitive impairments among individuals with BED, including inhibitory control, cognitive flexibility, attention and planning.²² Conversely, other systematic reviews did not draw definitive impairments of inhibitory control in BED populations compared to normal-weight control groups.^{15,24,30} Insufficient data from small studies and heterogeneity in methodologies might account for the mixed conclusions.^{13,30} In our study, non-clinical samples of BED exhibited worse inhibitory control performance on the Stop-Signal Task compared to well-matched healthy controls, potentially indicating a significant deficit in non-specific inhibitory control for BED.^{22,23,40} In addition, dysfunctional inhibitory control is considered an early marker of eating problems in adolescents³¹ and one treatment target for BED.¹⁰ Therefore, our findings of impairments in inhibitory control in a non-clinical sample of individuals with BED should provide further

firsthand evidence for the literature, and support the pivotal role of general aspects of inhibitory control implicated in the development of BED.¹³ Interestingly, patients with BED exhibited an impaired ability to estimate time, which positively correlated with compulsive self-monitoring and impulsivity,⁵⁷ thus suggesting a possible link between impulsivity, compulsivity, and self-awareness in BED. Impaired inhibitory control has been considered a common neurocognitive mechanism underlying compulsivity and impulsivity,¹⁶ hence it might also play a role in self-awareness in BED, which should be studied in future.

Comparatively speaking, food-specific inhibitory control domains have been supposed to be consistently compromised in BED,^{24,34–37} in spite of the limited research. However, previous studies also indicated that overweight/obese individuals with BED had poorer inhibitory control on tasks across food-specific and non-food stimuli, suggesting that inhibitory deficits associated with binge eating were not food-specific.⁴⁰ In our study, we utilized both a typical Go/No Go Task and an analogue with food stimuli (ie, the food-related Go/No Go Task), affording a direct comparison between food-related and non-food-related inhibitory control with the same task paradigm among the pBED group and HCs. Interestingly, our data revealed that individuals with pBED had a lower successful inhibition rate (ie, a lower correct accuracy in no-go trials) on the food-related Go/No Go Task with a moderate effect size (Cohen's $d = 0.47$), whereas they had a normal performance equivalent to the HCs on the typical Go/No Go Task (Table 2). Similar findings were detected in the smaller separate sample (Table 3), indicating that individuals with pBED exhibited poorer inhibitory control performance on the food-related Go/No Go Task compared to HCs, with a large effect size (Cohen's $d = 0.62$). These convergent data pointed to an explicit food-related inhibitory control deficit in BED (assessed with the Go/No Go paradigm), in addition to the impairments of general/non-specific inhibitory control (evaluated by the Stop-Signal Task). Our results thus provided more evidence for the proposals that emerging treatments targeting food-specific inhibition training might serve as effective novel approaches to treating BED.^{13,38,39} Specifically, in a recent randomized controlled trial, food-related inhibitory control assessed using the antisaccade paradigm was increased in BED patients who attended an impulsivity-focused group intervention, indicating that food-related inhibitory control/impulsivity could be effectively modified due to the training effects of certain precise treatments such as the IMPULS.⁵⁸ In this respect, our findings may suggest a potential target for impulsivity-focused neurocognitive training programs in the treatment of BED, such as SSRT or correct accuracy in no-go trials. This is because both the Go/No Go and Stop-Signal tasks are typical paradigms of impulsivity (inhibitory control). Nevertheless, future randomized controlled trials are warranted.

In the present study, to link food-related inhibitory control with potential neurobiological markers in BED, we further investigated the electrophysiological correlates of dysfunctions in food-related inhibitory control in a separate sample of individuals with pBED and HCs. Previous neuroimaging studies suggested a generalized dysfunction in the fronto-striatal areas (eg, orbitofrontal cortex) during inhibitory control processes in BED.^{59,60} Besides, significant electrophysiological differences in the N200, P200, P300, and LPP components were found in participants with binge-purge eating disorders compared to healthy controls.^{43,61} Importantly, inhibitory control-related ERP components (ie, N200 and P300) increased during food-specific no-go trials in eating/weight disorders.⁴² In our study, the ERPs data showed that impaired food-related inhibitory control in the pBED group could be associated with a longer P300 latency at the fronto-central sites (FC1, FC2, F3, F4, FZ), suggesting increased recruitment (or a blocked functioning) of inhibitory control in response to food-specific stimuli in the BED subjects.⁶² Moreover, previous studies found that overweight individuals with BED did not show an increased N200 latency related to conflict processing on a food-related antisaccade task.³⁶ Similarly, our pBED group did not display abnormalities in N200 components (ie, latency and amplitude) on the food-related No-Go trials or non-food Go trials compared to HCs, although we used a revised food-related Go/No Go Task. Nonetheless, future similar studies should overcome methodological shortcomings with consistent tasks. Our ERPs results, together with previous findings, indicated that P300, rather than N200 (at least in the current samples), might represent a potential neurophysiological marker for food-related inhibitory control in binge eating.^{41,63}

There were several limitations that should be noted in the present study. First, this study was essentially a cross-sectional design, and thus could not draw a causal conclusion between the inhibitory control processes and BED. Future longitudinal or follow-up studies are needed. Second, our samples mainly consisted of non-clinical young adult college students who were not diagnosed with clinical criteria for BED. In particular, the students were those who have attended three-year public psychology courses. Such a population might be likely to possess an advanced awareness of their

behaviors, emotional states, and cognitive symptoms, which could introduce certain subjective biases into the findings. As a result, our findings could not be generalized to clinical samples with serious binge-eating problems, and the results should be explained carefully. In addition, although we included two separate samples in this study, the sample size in the ERPs design was not extremely large and the electrophysiological findings might be limited partially by the sample characteristics. Finally, despite its temporal precision, the ERPs technology could not offer an exact spatial orientation related to the inhibitory control areas. Therefore, more intensive studies using neuroimaging methods are warranted to uncover underlying neural bases of general and food-related inhibitory control in BED, for a better understanding of its pathogenesis.

Conclusions

Despite these limitations, our study suggested that individuals with pBED might be compromised in both non-specific and food-related inhibitory control aspects. Furthermore, their impaired food-related inhibitory control was associated with an increased fronto-central P300 latency. Our study might contribute to a better understanding of the pathology of binge eating.

Data Sharing Statement

Data could be obtained by contacting the corresponding author.

Ethics Approval and Informed Consent

The study was conducted according to the guidelines of the Declaration of Helsinki, and approved by the Human Research Ethics Committee of the Guizhou Medical University (2020LS05). Informed consent was obtained from all subjects involved in the study.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflict of interest.

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